

*Addis Ababa*  
*University*  
*(Since 1950)*



**A D D I S   A B A B A   U N I V E R S I T Y**

**COLLEGE OF HEALTH SCIENCES**

**SCHOOL OF PUBLIC HEALTH**

**ASSESEMENT OF THE EFFECTS OF ETHIOPIAN ORTHODOX  
CHRISTIANS FASTING ON BIOCHEMICAL RISK FACTORS AND  
ANTHROPOMETRIC MEASUREMENTS**

**By: Habtamu Guja**

**Advisors: Dr. Jemal Haidar (Associate Professor)**

**Dr. Kaleab Baye (Assistant Professor)**

**April, 2016**

**Addis Ababa, Ethiopia**

**A D D I S   A B A B A   U N I V E R S I T Y**

**COLLEGE OF HEALTH SCIENCES**

**SCHOOL OF PUBLIC HEALTH**

**ASSESEMENT OF THE EFFECTS OF ETHIOPIAN ORTHODOX  
CHRISTIANS FASTING ON BIOCHEMICAL RISK FACTORS AND  
ANTHROPOMETRIC MEASUREMENTS**

**By: Habtamu Guja**

**Advisors: Dr. Jemal Haidar (Associate Professor)**

**Dr. Kaleab Baye (Assistant Professor)**

**A Thesis submitted to the school of graduate studies Addis Ababa University in  
partial fulfillment of the requirements for the degree of master in public health.**

**April, 2016**

**Addis Ababa, Ethiopia**

**A D D I S   A B A B A   U N I V E R S I T Y**

**COLLEGE OF HEALTH SCIENCES**

**SCHOOL OF PUBLIC HEALTH**

**APPROVED BY THE EXAMINING BOARD**

**Muluken Gizaw (BSc, MPH)**

**Chairperson**

\_\_\_\_\_  
**Signature**

**Jemal Haidar (MD, MSc, CRM, CME)**

**Primary Advisor**

\_\_\_\_\_  
**Signature**

**Kaleab Baye (PhD)**

**Secondary Advisor**

\_\_\_\_\_  
**Signature**

**Robel Yirgu (BSc, MPH)**

**Internal Examiner**

\_\_\_\_\_  
**Signature**

**Aweke Kebede (PhD)**

**External Examiner**

\_\_\_\_\_  
**Signature**

**April, 2016**

**Addis Ababa, Ethiopia**

## **Acknowledgments**

I would like to thank Almighty God for His Peace and filling me in Wisdom, Understanding and Knowledge as in Exodus 31 for executing all my daily activities including this work.

I would like to express my appreciation and thanks to my advisors: Dr. Jemal Haidar and Dr. Kaleab Baye, for their unreserved support, encouragement and supervision throughout the work.

My appreciation also goes to all staffs of MOFAM Pharmaceuticals for providing me the moral support, for their tolerance and gap filling at times when I was not at work. Special thanks to Mr. Tolha Bahru, Mrs. Tsehaynesh Abat, Mrs. Eleni Kassa, Mr. Tigistu Woldie & Mr. Abdulfetah Baharu.

Their Excellencies: Mrs. Tadelech Woldie (Eteyae's Prayer), Mrs. Alemnesh Assefa (Ma), Mr. Reshad Seman, Mrs. Marta Ageba, Mr. Gizaw Getenet, Dr. Woyita Woza, Mrs. Selamawit Guja (Emush), Mr. Binyam Meseret, Mr. Kefyalew Jote, Mr. Eyuel, S/r Haregewoiyn Tilahun are strongly appreciated for their routine support and involvement in my research work in different ways.

I would like to thank Ethiopian Public Health Institute staffs especially at TB/HIV research and development department for carrying out the biochemical analysis. I also thank nutrition department workers for allowing me to store the serum under deep freezer. Addis Ababa University is also strongly acknowledged for continuing education opportunity and internet access.

Finally, I would like to say thank you to my study subjects for their willingness and tolerance during the repeated measurements; thank all my family members and friends who played a significant role in the accomplishment of this work and May God Bless them abundantly!

## **Abstract**

**Background:** As in many developing countries, the prevalence of cardiovascular diseases (CVDs) in Ethiopia has been increasing dramatically. Unhealthy changes in the dietary pattern are among the responsible factors for the rise. Religious beliefs play a significant part in shaping those behaviors; however, there is no study conducted on the role of Ethiopian Orthodox Christians' fasting practice in relation to biochemical indicators for CVD risk factors and anthropometric indices.

**Objective:** To assess the effects of Ethiopian Orthodox Christians' Easter fasting on the anthropometric indices and biochemical risk factors for cardiovascular diseases.

**Methodology:** Before and after study design having controls was used among 88 voluntarily selected participants from eligible population and followed over Easter fasting period in two groups; those who fast (fasting group) and do not fast (control group). Participants were measured for serum lipid profiles and anthropometric indices before the beginning and at the end of Easter fasting. Paired t-test was used for comparison of baseline and endline measurements in each group. Besides, independent sample t-test was used for comparison of differences between groups.

**Results:** In comparison to controls, fasters showed significant mean reduction at the end of fasting; 11% in total cholesterol, 20% in LDL-C, 11% in TG, 21% in LDL/HDL ratio, 12% in TC/HDL ratio, and 7% in blood glucose concentrations. Similarly, significant reductions in the anthropometric measurements were observed ( $p < 0.001$ ); 2% in body weight, BMI and waist to hip ratio whereas, waist circumference was reduced by 3%. However, there was no statistically significant change observed in lipid profiles among control group.

**Conclusion:** Periodic abstinence from animal source foods during the Ethiopian Orthodox Christians' Easter fasting period and shifts to calorie restricted vegetarians' diet style by fasters in this study attributed to the observed biochemical and body measurement changes highlighting that religious fasting has a substantial impact on health-related dietary behaviors.

## Table of Contents

<b>Contents</b>	<b>Page</b>
Acknowledgments	i
Abstract	ii
List of Abbreviations and acronyms	vi
List of Figures	vii
List of Tables	viii
1. INTRODUCTION	1
1.1 Background	1
1.2. Statement of the Problem	3
1.3. Significance of the Study	3
2. LITERATURE REVIEW	4
2.1. Religion in Ethiopia	4
2.2. Fasting in Ethiopian Orthodox Christianity	4
2.3. Religious Fasting and Anthropometric Measurements	5
2.4. Lipids, Lipoproteins and Serum Lipid Levels	6
2.5. Cardiovascular Disease Risk Factors	10
2.5.1. Blood sugar and blood pressure levels as risk factors for CVD	10
2.5.2. Obesity, socioeconomic status, and dietary risk factors for CVDs	11
2.5.3. Behavioral Risk Factors	13
2.5.4. Biological, Genetic and Environmental Risk factors for CVDs	15
3. OBJECTIVE	19
3.1. General Objective	19
3.2. Specific Objectives	19
4. METHODOLOGY	20
4.1. Study area and period	20
4.2. Study design	20
4.3. Study Population, Study Subjects and Sampling Procedure	20

4.4.	Eligibility Criteria	21
4.4.1.	Inclusion Criteria	21
4.4.2.	Exclusion Criteria	21
4.5.	Sample Size Determination	21
4.6.	Data Collection and Instruments for Data Collection	24
4.6.1.	Sampling and Data Collection Framework	25
4.6.2.	Questionnaires	26
4.6.3.	Anthropometric variables	27
4.6.4.	Biochemical assays	28
4.7.	Data Quality Control Methods	29
4.8.	Data Management	29
4.9.	Data Analysis	30
4.10.	Operational Definitions	31
4.11.	Ethical Considerations	32
4.12.	Study Variables	32
4.12.1.	Dependent Variables	32
4.12.2.	Independent Variables	32
4.13.	Dissemination of Results	32
5.	RESULTS	33
5.1.	Scio-demographic, life style characteristics and practice of fasting	33
5.2.	Baseline characteristics of study groups	35
5.3.	Dietary data	36
5.4.	Comparisons of pre and endline-fasting values in the fasting and controls groups	38
5.5.	Comparisons of changes in the fasters and controls groups	40
5.6.	Effect of fasting and other predictors on endline-fasting values	41
6.	DISCUSSIONS	44
7.	STRENGTH AND LIMITATIONS OF THE STUDY	50
8.	CONCLUSIONS AND RECOMMENDATIONS	51
	REFERENCES	52

## ANNEXES

Annex 1:	Approval Letter of Ethical Clearance from school of public health, Addis Ababa University	70
Annex 2:	Participant's Information Sheet and Consent Form	71
Annex 3:	English versions Questionnaire	74
Annex 4:	Amharic versions Questionnaire	79
Annex 5:	Declaration	84

## **Lists of Abbreviations and Acronyms**

ANOVA	Analysis of variance
ApoE	Apolipoprotein E
ASF	Animal Source Foods
BMI	Body Mass Index
CVDs	Cardiovascular diseases
EOC	Ethiopian Orthodox Christian
EPHI	Ethiopian Public Health Institute
EU	European Union
FAO	Food and Agriculture Organization
FBS	Fasting Blood Sugar
GOC	Greek Orthodox Christians
HDL	High-Density Lipoprotein
HK	Hexokinase
HMG-CoA	3-Hydroxy-3-Methyl Glutaryl-Coenzyme A
LDL	Low-Density Lipoprotein
LDL/HDL	Low-density lipoprotein to High-density lipoprotein ratio
LPL	Lipoprotein Lipase
mg/dL	Milligrams per deciliter
mmHg	Millimeters of Mercury
NCDs	Non-Communicable Diseases
NCEP-ATP	National Cholesterol Education Program, Adult Treatment Panel
SPSS	Statistical Package for Social Sciences
STEPs	Stepwise approach for non-communicable disease surveillance
TC	Total Cholesterol
TC/HDL	Cholesterol to High-Density Lipoprotein ratio
TG	Triglycerides
VLDL	Very Low-Density Lipoprotein
WC	Waist Circumference
WHO	World Health Organization

## List of Figures

Figure 1:	Compositions of lipoproteins	7
Figure 2:	Summary of the fates of lipoproteins produced by the liver	9
Figure 3:	Conceptual frame work of risk factors for cardiovascular diseases	18

## List of Tables

Table 1:	Paired samples T-test with mean pre-fasting values compared to mean end-fasting results from Sarie <i>et al</i> [11].	23
Table 2:	Scio-demographic and life style characteristics of the study population, April 2015, Addis Ababa.	34
Table 3:	Baseline characteristics of serum lipids, blood glucose level, blood pressure and anthropometric measurements between fasters and control group, April 2015, Addis Ababa.	35
Table 4:	Result of seven days frequency of food intake between fasters and controls at end fasting, April 2015, Addis Ababa.	37
Table 5:	Serum lipids level, blood pressure and anthropometric measurements between baseline and endline among fasting and control group.	39
Table 6:	Mean difference comparison of serum lipids, blood glucose, blood pressure and anthropometric measurements between fasting and control group, April 2015, Addis Ababa.	40
Table 7:	Effects of different predictor variables on serum lipids, Anthropometric, blood pressure and glucose level among study participants (n = 88), April 2015, Addis Ababa.	42

# 1. INTRODUCTION

## 1.1. Background

The global prevalence of chronic non-communicable diseases (NCDs) is on the rise, majority of the growth occurring in developing countries. In sub Saharan Africa, NCDs are projected to surpass infectious diseases by 2030 (1). Cardiovascular diseases (CVDs) are the leading causes of morbidity and mortality worldwide, causing nearly one-third of all deaths worldwide (2).

Many sub-Saharan African countries are already experiencing lifestyle and behavioral changes which are responsible for substantial increases in the prevalence of intermediate CVDs risk factors including hypertension and obesity (3). The prevalence of CVDs in Ethiopia has been increasing dramatically (4-5). According to a cross-sectional survey conducted in the mid 1990s in Addis Ababa, only 0.7% of men and 6% of women were obese, and 7.1% of the study population had elevated blood pressure (4). However, in 2008 a community-based study in Addis Ababa showed that 20% of men and 38% of women were overweight and 10.8% of these women were obese. In addition, 31.5% of men and 28.9% of women had high blood pressure (5).

Epidemiological transition attributed to increased urbanization, westernization, and economic development has led to a nutritional transition characterized by a shift to consumption of higher caloric diet and experiencing behavioral changes that are risk to health; such as physical inactivity, and increased tobacco use. Such changes are more likely to have abnormal serum lipids or cholesterol levels (3, 6), hyperglycemia, excess abdominal or body fat, and elevated blood pressure which are contributory risk factors for cardiovascular diseases (1).

Religious beliefs play a significant part in shaping social behavior and they tend to influence the way people live, the dietary choices they make (7). Study done by Belwal and Tafesse indicated that religious factors affects the demand for food consumed during fasting by the Ethiopian Orthodox Christians (8). Periodic abstention from animal source foods is associated with a better insulin sensitivity, lower levels of serum lipids and blood pressure in subjects at risk indicating that the beneficial effect of such diet for the cardiovascular system (9). Orthodox Christian diet is periodically interchanging to a low fat diet (10).

Fasting or the voluntary abstention from all restricted foods is a feature of many religions and the putative health benefits have attracted both scientific and popular interest. Commonly, religious doctrines proscribe foods from animal sources permanently or for particular periods (11). Different cultural and religious groups and the extent to which they adhere to culturally-based dietary instructions, has influences on food habits, nutritional adequacy, and overall health. However, a relatively small proportion of research studies conducted to date have explored the effect of religious fasting (12). Among religions those have been studied regarding their relation to health, Judaism (13-14), Islam (15-18), Seventh-Day-Adventists (19-20), and Greek Orthodox Christians (11). Unlike the Greek, Ethiopian Orthodox Christians (EOC) in addition to animal source foods proscription, calorie restriction has also been practiced by most of the fasters; however, to the best of the researcher's knowledge, there is no study conducted on the role Ethiopian Orthodox Christians' fasting practice in relation to biochemical indicators for CVD risk factors and anthropometric indices.

This study assesses the effects of Ethiopian Orthodox Christians Easter fasting on serum lipids profile and on the anthropometric indices of obesity.

## **1.2. Statement of the Problem**

Cardiovascular diseases are the leading causes of morbidity and mortality worldwide (2). Within two decades the prevalence of CVDs in Ethiopia has increased dramatically (3). Unhealthy dietary behaviors like consumption of calorie-dense foods are among the responsible risk factors for the increased prevalence of cardiovascular diseases (1). Unlike the Greek, Ethiopian Orthodox Christians (EOC) in addition to animal source foods proscription, calorie restriction has also been practiced by most of the fasters and the duration of Easter fasting period is prolonged by one week; however, to the best of the researcher's knowledge, there is no study conducted on the role Ethiopian Orthodox Christians' fasting practice in relation to biochemical indicators for CVD risk factors and anthropometric indices (21-22).

To date, only very few investigations have examined the health-related effects of these fasting periods. Those few studies in Greek again presented conflicting findings on blood pressure (23-24), similar inconsistent findings on lipid profile, blood glucose levels and anthropometric measurements (23-25) that additionally highlights more work remains to be performed.

## **1.3. Significance of the Study**

Most religions have long established fasting periods that convey religious identity; however, the importance of dietary rules and the degree to which they are observed by followers vary considerably over time, often in response to a changing environment and hence, it is important to investigate and document the role of religious fasting with respect to health. Moreover, the study being the first in the country, findings will provide baseline data for elucidating the effect of Ethiopian Orthodox Christians fasting on cardiovascular disease risk factors that will serve as background information for further future studies.

## **2. LITERATURE REVIEW**

### **2.1. Religion in Ethiopia**

The 2007 National Population and Housing Census of Ethiopia (26) indicated that Orthodox Christians form the largest religious group (43.5 %) followed by the Muslims (33.95 %), Protestants (18.52 %), Catholics, traditional religion and others (4.03 %). Diet plays an integral role in the religious customs of a variety of faiths. For many religions, this role is manifested in the form of specialized fasting periods (22).

### **2.2. Fasting in Ethiopian Orthodox Christianity**

Fasting is defined as a partial or total abstention from all foods, or a select abstention from prohibited foods. Fasting has been the subject of numerous scientific investigations, the three most commonly studied fasts are caloric restriction, alternate-day fasting, and dietary restriction (22).

Ethiopian Orthodox Church has seven fasting periods to be observed by all believers and during those periods, the believers are not allowed to eat any animal or dairy source foods unless exempted from these fasts because of serious sickness, breastfeeding mothers, and children less than seven years of age (27). Greek Orthodox Christians fast for a total of 180 - 200 days each year (22), while Ethiopian Orthodox Christians fast for 250 days each year, of which about 180 are obligatory for all, and the rest are only for special groups in the church (priests, monks, and nuns) and their main fasting periods are the Fast of Prophets (*Tsome Nebiyat* or *Gena* 40 days prior to Christmas), the Great Lent (55 days prior to Easter), the fasting of Salvation (all Wednesdays and Fridays, except for the fifty days after Easter) and the Assumption of the Virgin Mary (*Tsome Filseta*, 15 days in August) (21).

The Great Lent or *Abye Tsome*, also called *Hudade* is the longest fasting period and the major fast of the church. This Lent is observed as a remembrance of the 40 days and nights fasting of Lord Jesus Christ after His baptism. Later the Ethiopian church added 15 more days to it and made 55 days (21).

### **2.3. Religious Fasting and Anthropometric Measurements**

Religious fasts are partaken primarily for spiritual purposes; they also have the potential to greatly affect one's physical health. Regarding anthropometric outcomes, Greek Orthodox Christian monks were observed to decrease significantly during fasting periods (22).

Anthropometric measures of adiposity such as body mass index (BMI), waist circumference (WC), and waist-to-hip ratio (WHR) have been shown to correlate differently with CVD risk. The risk of death from cardiovascular disease increases with excessive fat (28-32) and obesity is shown to adversely affect cardiac function, increases the risk factors for coronary heart disease, and is an independent risk factor for cardiovascular disease (33). Dyslipidemia, hypertension, and other CVD risk factors are highly correlated with increasing BMI (34-35) and excessive abdominal adiposity is also a strong independent predictor (36).

BMI is the most frequently used measure of adiposity in epidemiologic studies; however, some investigators have reported that using BMI alone is not the most accurate measure of increased CVD risk; instead, other studies argued that WC as a better predictors of future CVD risk (37-38). Since BMI does not accurately reflect the degree of body fat and body fat distribution (39-40). Abdominal fat measurement is advocated as a better indicator of (40). There is a large body of evidence that suggests abdominal fat distribution measured by waist circumference (WC) may be more closely tied to metabolic risks than BMI (41-41). To this effect, the US National Institutes of Health has recommended combined measurements of WC and BMI as an assessment tool for CVD risk (43). Thresholds of BMI in accordance with WHO protocol (Undeweight:  $<18.5 \text{ kg/m}^2$ ; Normal:  $18.5\text{--}24.9 \text{ kg/m}^2$ ; Overweight:  $25.0\text{--}29.9 \text{ kg/m}^2$ ; Obese  $\geq 30 \text{ kg/m}^2$ ). Abdominal obesity is explained by having a waist circumference of  $\geq 94 \text{ cm}$  for men and  $\geq 80 \text{ cm}$  for women (3).

Waist to hip ratio (i.e. the waist circumference divided by hip circumference) suggested as an additional measure of body distribution. This ratio can be measured more precisely than skin fold and it provides an index for both subcutaneous and intra abdominal adipose tissue. The cutoff point for risk of metabolic complication, WHR  $>0.90$  for males and  $>0.85$  for females (44).

## 2.4. Lipids, Lipoproteins and Serum Lipid Levels

Lipids are characterized by their insolubility in water and include a family of compounds; triglycerides, phospholipids, and sterols. Sterols are compounds with a multiple ring structure; the most common sterol is cholesterol (45).

Cholesterol is an essential substance involved in many functions, such as maintaining cell membranes where it is mainly incorporated in to the plasma membranes of cells and regulates membrane fluidity conferring a higher degree of rigidity. Furthermore, it is the precursor for the synthesis of fat soluble vitamins, all steroid hormones, bile acid and help cell connections in the brain [46]. While in circulation, cholesterol, being a lipid, requires a transport vesicle to shield it from the aqueous nature of plasma. Complex, micelle-like join up of various proteins and lipids achieve cholesterol transport through the vascular system. These particles are known as lipoproteins (46).

Lipoproteins are heterogeneous in size, shape, composition, function, and perhaps most importantly, their contribution to vascular disease. All types of lipoproteins carry all classes of lipids: triacylglycerides, cholesterol, phospholipids and amphipathic proteins called apolipoproteins (46). Lipoproteins can be differentiated on the basis of their density, as depicted in Figure 1, the degree of lipid in a lipoprotein affects its density—the lower the density of a lipoprotein, the more lipid it contains relative to protein. The four major types of lipoproteins are chylomicrons, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL) (45-46).

**Chylomicrons and VLDL:** These two lipoproteins are rich in triglyceride. Chylomicrons are synthesized by enterocytes from lipids absorbed in the small intestine and VLDL is synthesized in the liver. Their function is to deliver energy-rich triacylglycerol (TAG) to cells in the body. TAG is stripped from chylomicrons and VLDL through the action of lipoprotein lipase, an enzyme that is found on the surface of endothelial cells. This enzyme digests the TAG to fatty acids and monoglycerides, which can then diffuse into the cell to be oxidized, or in the case of an adipose cell, to be re-synthesized into TAG and stored in the cell (45-46).

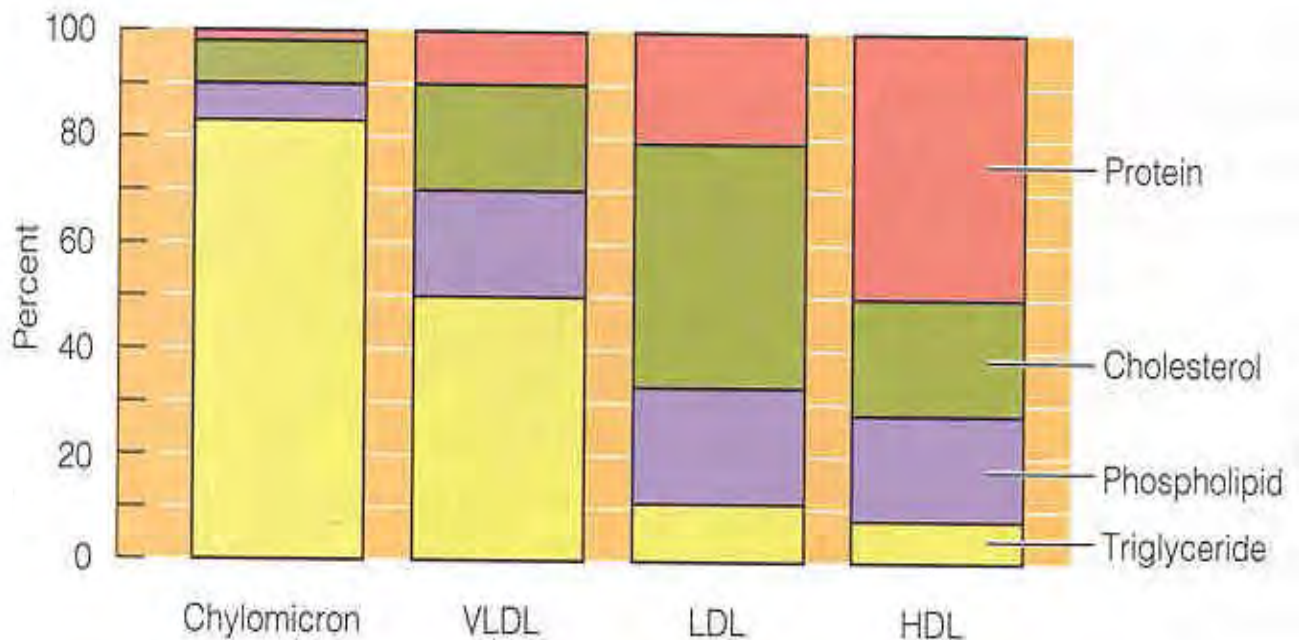


Figure 1: Compositions of lipoproteins. Source: Whitney and Rolfes, 2008 (45).

**Low Density Lipoproteins (LDL):** As VLDL particles are stripped of triacylglycerol, they become denser and these particles are remodeled at the liver and transformed into LDL. The function of LDL is to deliver cholesterol to cells, where it is used in membranes, or for the synthesis of steroid hormones. Cells take up cholesterol by receptor-mediated endocytosis when LDL binds to its receptor and internalized in an endocytic vesicle. Receptors are recycled to the cell surface, while hydrolysis in an endolysosome releases cholesterol for use in the cell (6, 45-46). High levels of LDL cholesterol (the so-called “bad cholesterol”) greatly increase the risk for atherosclerosis because LDL particles contribute to the formation of atherosclerotic plaques (46).

**High Density Lipoprotein (HDL):** Excess cholesterol is eliminated from the body via the liver, as excess cholesterol from cells is brought back to the liver by HDL in a process known as reverse cholesterol transport. HDL is synthesized and secreted by the liver and it travels in the circulation where it gathers cholesterol to form mature HDL, which then returns the cholesterol to the liver via various pathways (6, 45). Cholesterol delivered to the liver via HDL enters the bile acid synthesis pathway also known as the cholesterol catabolic pathway (46). Low HDL levels (“good cholesterol”) are an independent risk factor, because reverse cholesterol transport

works to prevent plaque formation, or even cause regression of plaques once they have formed. HDL may also have anti-inflammatory properties that help reduce the risk of atherosclerosis and thereby promote vascular health (46).

Liver is central to the regulation of cholesterol levels in the body; the liver is not only synthesizing cholesterol for export to other cells, but it also removes cholesterol from the body by converting it to bile salts and putting it into the bile where it can be eliminated in the feces. Furthermore, the liver synthesizes the various lipoproteins involved in transporting cholesterol and lipids throughout the body. Cholesterol synthesis in hepatocytes is under negative feedback regulation; increased cholesterol in the cell decreases the activity of 3-hydroxy-3-methyl glutaryl-coenzyme A (HMG-CoA reductase), the rate-limiting enzyme in cholesterol synthesis (45). When cholesterol levels rise in the blood, they can, however, have dangerous consequences. Homeostasis of cholesterol is centered on the metabolism of lipoproteins, which mediate transport of the lipid to and from tissues (46). Figure 2 below summarizes the fates of lipoproteins produced by the liver.

Abnormal levels of these lipoproteins in blood are linked to increase risk of atherosclerosis. Atherosclerosis is a cardiovascular disease in which lipids and inflammatory cells accumulate in plaques within the walls of blood vessels. As a result, vessel walls are narrowed and clots may form, impeding blood flow and oxygen delivery and causing tissue injury. Heart disease occurs because the coronary arteries supplying the heart are a major site where atherosclerotic plaques form. Although atherogenesis is a multifactorial process, abnormalities in lipoprotein metabolism are one of the key factors, representing around 50% of the population-attributable risk of developing cardiovascular disease (47).

Serum lipid profile is measured for cardiovascular risk prediction and has now become almost a routine test including four basic parameters: total cholesterol (TC), high density lipoproteins (HDL) cholesterol, low density lipoproteins (LDL) cholesterol and triglycerides (48). However, lipoproteins ratios, TC/HDL cholesterol and LDL/HDL cholesterol, are being used as risk indicators with greater predictive value than isolated parameters used independently, particularly the former. These two indices can be regarded as similar; since two thirds of plasma cholesterol is found in LDL, total and LDL cholesterol are closely correlated. Moreover, an increased in the

level of the denominator, HDL cholesterol is more prevalently associated with plaque regression, while a decrease in LDL cholesterol would slow down progression. Both predict greater cardiovascular risk for a wide range of cholesterol concentrations. However, when there is no reliable determination of LDL cholesterol, as in cases of hypertriglyceridemia, it is preferable to use the total/HDL cholesterol ratio (47).

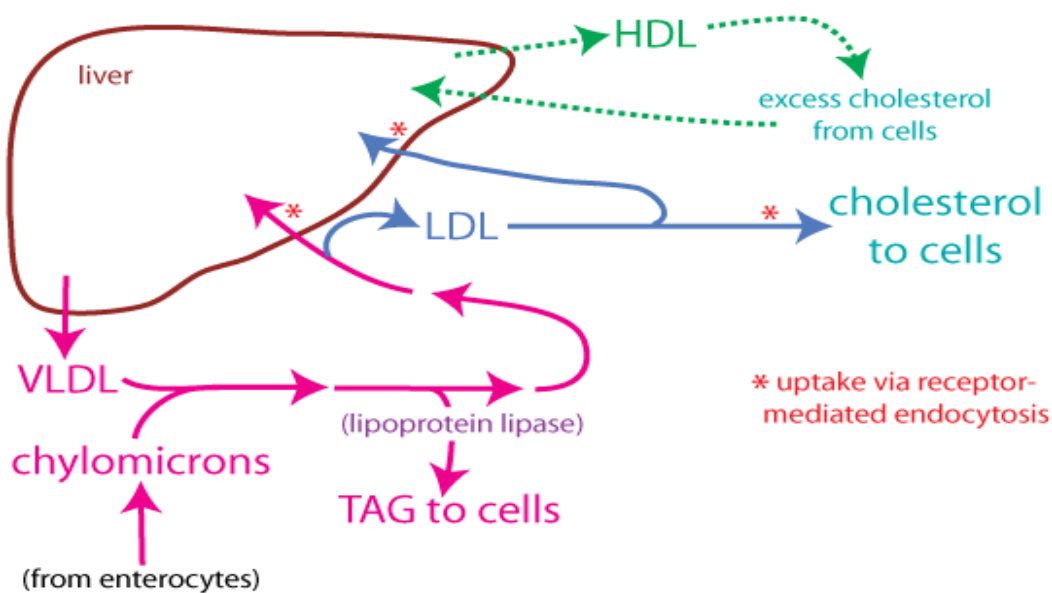


Figure 2: Summary of the fates of lipoproteins produced by the liver: VLDL and chylomicrons (pink pathway); LDL (blue pathway); and HDL (green pathway). Source: <http://courses.washington.edu/conj/bess/cholesterol/liver.html> [Accessed on September 14, 2015]

In the presence of infection or inflammation, the level of total cholesterol and HDL decreases and triglyceride level increases (49) whereas the ratio of total cholesterol to HDL (TC/HDL) remains relatively stable (48). This ratio may therefore serve as a more reliable measure of serum lipid level (50). Individuals with a high TC/HDL or LDL/HDL ratio have greater cardiovascular risk owing to the imbalance between the cholesterol carried by atherogenic and protective lipoproteins; this may be due to an increase in the atherogenic component contained in the numerator, a decrease in the anti-atherosclerotic trait of the denominator, or both. TC/HDL ratio is a more sensitive and specific index of cardiovascular risk than total cholesterol; for this reason, the ratio is also known as atherogenic or Castelli index (47).

Lipid profile measurement is usually done in fasting blood specimen, fasting refers to 12–14 h overnight complete dietary restriction with the exception of water and medication. This may hold true due to two main reasons: the first being postprandial triglycerides remain elevated for several hours (51), and secondly, most reference values for serum lipids are established on fasting blood specimen (48).

Desirable blood lipid profiles are total cholesterol <200mg/dL, LDL cholesterol <100mg/dL, HDL cholesterol  $\geq$ 60mg/dL, and triglycerides <150mg/dL. Low HDL-C was defined to be <40 mg/dL in men and <50 mg/dL in women (3, 45, 52). Lipoprotein ratios therapeutic target cut-off values are < 3.7 for LDL/HDL cholesterol and < 5 for TC/HDL cholesterol ratio (47).

## **2.5. Cardiovascular Disease Risk Factors**

Cardiovascular diseases cause nearly one-third of all deaths worldwide and risk factors such as elevated cholesterol levels, hypertension, elevated glucose level, obesity or being overweight, socioeconomic status and behavioral risk factors like cigarette smoking, physical inactivity, unhealthy diets, alcohol and Khat consumption are among the major risk factors (2, 28-31).

### **2.5.1. Blood sugar and blood pressure levels as risk factors for CVD**

Cardiovascular diseases due to hypertension and diabetes mellitus or elevated glucose level are among the top causes [31]. Hypertension is a growing public health problem, with remarkable contribution to cardiovascular diseases morbidity (53) and it is major risk factor of causing heart attack (54).

Cardiovascular complications are now the leading causes of diabetes-related morbidity and mortality. The public health impact of cardiovascular disease in patients with diabetes is already enormous and is increasing. The adverse influence of diabetes extends to all components of the cardiovascular system: the microvasculature, the larger arteries, and the heart, as well as the kidneys (55). Cut off values with mean systolic blood pressure (SBP)  $\geq$  140 mmHg (millimeters of mercury) or diastolic blood pressure (DBP)  $\geq$  90 mmHg are considered to be hypertensive, while fasting blood sugar level >110mg/dL are considered as hyperglycemic (56).

### **2.5.2. Obesity, socioeconomic status, and dietary risk factors for CVDs**

Rapid lifestyle changes fast food, obesity, and lower socioeconomic status are major factors in the development of CVD (67).

**Obesity:** Obese individuals are more likely to have elevated total cholesterol, triglycerides, low density lipoprotein (LDL) cholesterol and decreased high density lipoprotein (HDL) cholesterol. These metabolic profiles are most often seen in obese people with a high accumulation of intra-abdominal fat and have consistently been related to an increased risk of coronary heart disease (CHD). With weight loss, the levels of triglycerides can be expected to improve. A 10 kg weight loss can produce a 15% decrease in LDL cholesterol levels and an 8% increase in HDL cholesterol (6, 54).

**Dietary Factors:** A healthy diet can contribute to a healthy body weight, a desirable lipid profile and a desirable blood pressure (57). High dietary intakes of saturated fat, trans-fats and low intake of fruits, vegetables and fish are linked to cardiovascular risk. Moreover, frequent consumption of high-energy foods, such as processed foods that are high in fats and sugars, promotes obesity compared to low-energy foods (57). Elimination of trans-fat and replacement of saturated with polyunsaturated vegetable oils lowers the risk of CVD (54, 57).

Adherence to plant based diet is modestly associated with a better insulin sensitivity, lower levels of total cholesterol and lower levels of systolic blood pressure in overweight and obese subjects. This may suggest that compared to general population, the beneficial effect of this diet in cardiovascular system of excess body weight people is limited (9). Greek Orthodox Christian (GOC) diet is periodically interchanging to a vegetarian-type diet and shares all the features of the plant based or Mediterranean diet (12).

**Socioeconomic Status:** Socioeconomic status (SES) indicators are usually measured by determining education, income, wealth index, occupation, marital, social status and neighborhood environment or a composite of these indicators which are an important contributor to one's health (28, 58). In recent years, as cardiovascular disease (CVD) has emerged as a leading cause of death in developing countries, it is important to identify and target people who are at risk given that a third of all deaths are expected to be due to CVD by 2020 (58).

Studies have shown socio-economic status and risk factors for CVD vary with the country's social and economic development. In developed countries, the association between socio-economic status and CVD risk factors is negative, with a higher prevalence of CVD risk factors among people of lower SES (59-61). However, the evidence from developing countries has been inconsistent (62-65).

CVD has been described as a marker of modernization and social affluence and can mirror a society's stage of economic development (28); nutrition transition is predicted to lead to changing relationships between SES and CVD risk. The nutrition transition is defined as changes in dietary intake patterns because of the adoption of 'modern' lifestyles due to social and economic development. As a consequence, disease patterns initially shift towards nutrition-related chronic diseases like CVD. In the early stages of such a transition, risk factors tend to be concentrated among the high SES groups and urban dwellers, which have earlier access to these 'modern' lifestyles (66). Experience in high-income settings has shown that as the transition progresses, people of higher SES start to change their behavior and adopt healthier lifestyles, probably due to multiple factors (greater awareness, greater self-efficacy, better access to healthy diets), leading to a lowering of their risk, while the burden of disease shifts to lower SES groups (58).

Education reflects degree of knowledge and skill, along with the ability to attract material wealth (58). Higher levels of education may positively affect health by allowing the person to gather the necessary skills and assets required to insulate them from adverse factors or to reduce exposure to negative influences on health, adopting positive health behavior and having access to preventive health services (67). Several studies, including Tadesco *et al* (67) have demonstrated that the relationship between education and hypertension, cigarette smoking and high cholesterol have generally found a negative association. However, the study done by Yu *et al* (28) have shown that better educational level did not seem to be able to prevent the worsening of the risk factors (like smoking) in those highly educated people even though educated people still consistently had the lowest levels of risk factors (28, 67).

Income reflects current economic or materialistic welfare; the study by Samuel *et al* (58) used wealth index, which is based on asset ownership, could be considered an indicator of long-term economic status, as household assets are unlikely to change in response to short-term economic shocks. Unlike educational status, income status and risk for developing CVD is less consistent as it vary significantly with the levels of country's social and economic development.

### **2.5.3. Behavioral Risk Factors**

Regular physical activity would help to control the growing danger of obesity and co-morbidities of metabolic syndrome (68). Cigarette smoking influences cardiovascular system because of carbon monoxide and nicotine leading to a reduction in myocardial oxygen intake (69). Cardiovascular diseases typically occur in middle age or later, however lifestyle behaviors are learned in early life and maintained throughout adulthood (69). Among other behavioural risk factors, regular khat consumption (53) and alcohol consumption (70) shown to give rise to CVD conditions.

**Khat consumption:** Among the behavioral risk factors, khat consumption, where Khat or chat is a natural stimulant with amphetamine-like effects which is commonly used for social recreation in Ethiopia. Regular chewing of Khat is associated with elevated mean diastolic blood pressure, which may have sustained effects on the cardiovascular system that can contribute to elevated blood pressure at the population level (53). Regular and repeated intake of Khat has recently been reported to be associated with increased risk of acute myocardial infarction (71) and high blood pressure (72-73).

**Cigarette smoking:** Smoking is a major risk factor for atherosclerotic cardiovascular diseases through leading to abnormal levels of lipids in the blood (74); in comparison to non-smokers, cigarettes smokers had significantly higher TC (3%), TG (9.1%), VLDL (10.4%), LDL (1.7%), and lower concentrations of HDL (-5.7%) (75). Moreover, a dose-response relationship was found between the number of cigarettes smoked and the change in lipid or lipoprotein variable (75). In addition, smoking causes an acute increase in arterial stiffness and associated with greater endothelial dysfunction which results in high blood pressure (76) which is resulted because of smoking, through its basic ingredients nicotine and carbon monoxide, increases

circulating free radicals that pose oxidative stress and the development of arteriosclerosis (74). In persons with CHD, smoking cessation reduces coronary event rate by about 50% within one to two years of stopping; in addition, cessation has a benefit of 5-10% increase in HDL-C (30).

**Alcohol intake:** Studies have indicated that moderate alcohol drinkers have a lower risk of heart disease than abstainers (77). Light-to-moderate alcohol consumption in healthy men and women is associated with enhanced insulin-mediated glucose uptake, lower plasma glucose and higher HDL-cholesterol concentration with a modest 5–15%. These changes contribute to coronary protective effect of moderate alcohol intake (30, 78).

Moderate consumption of alcohol defined by U.S. Dietary Guidelines as up to two standard drinks a day for men and one for women has been associated with a reduced risk of coronary heart disease (77). Women appear to incur greater risks for overall mortality at greater than 1 standard drink per day while men appear to have the greatest benefit at 2 standard drinks per day (79). Despite countries have different national guidelines in the amount of standard drink, most countries national guidelines set around 20g pure alcohol which comply with the WHO low risk responsible drinking guideline (80-81).

The WHO uses four risk drinking groups founded on epidemiological evidence (82-83). The first group refers to abstainers; the second are grouped under category I, which indicates drinkers at low risk (below 20g of pure alcohol daily for women and 40g for men). The third, category II consists of alcohol use at risk for health ( $\geq 20$ -40g for women and  $\geq 40$ -60g for men). Category III consists of alcohol consumption that is already causing harm to the drinker, who may also have symptoms of dependence ( $\geq 40$ g for women and  $\geq 60$ g for men).

**Physical activity:** it can help people achieve a variety of goals, including increased cardio-respiratory fitness, increased strength, improved glycemic control, decreased insulin resistance, improved lipid profile, blood pressure reduction and maintenance of weight loss [84]. Physical activity or exercise can be broadly classified in to three types: aerobic exercise is physical activity, such as walking, bicycling or jogging that involves continuous, rhythmic movements of large muscle groups; resistance exercise is physical activity involving brief repetitive exercises with weights, weight machines, resistance bands or one's own body weight (e.g. pushups) to

increase muscle strength and/or endurance; flexibility exercise is a form of activity, such as lower back or hamstring stretching that enhances the ability of joints to move through their full range of motion; and some types of exercise, such as yoga, can incorporate elements of both resistance and flexibility exercise (84).

There is strong and consistent evidence that regular aerobic exercise alone or combined with resistance exercise reduce triglycerides, increase high-density lipoprotein cholesterol (HDL-C), reduce blood pressure, waist circumference and improve insulin sensitivity and glucose homeostasis (30, 85-86) and thereby contributes to the prevention of several chronic diseases and reduced risk of premature death (87); however, exercise alone has little effect on LDL-C level (30).

Moderate intensity exercise, including walking at a moderately brisk pace, done regularly (30 minutes 3-5 times a week) shown to raise HDL-C (30). However, for patients with cardiac ischemia and angina, exercise must be tailored to the degree of disease and should be done at levels that do not aggravate the condition (30).

#### **2.5.4. Biological, Genetic and Environmental Risk factors for CVDs**

These risk factors include age, sex, family history/heredity or defect in gene and environmental factors. Unlike the modifiable risk factors, these risk factors cannot be changed; however, people in these risk categories should receive early screening and regular check-ups (88).

**Age:** Cardiovascular disease becomes increasingly common with advancing age; as a person gets older, the heart undergoes subtle physiologic changes, even in the absence of disease. The heart muscle of the aged heart may relax less completely between beats, and as a result, the pumping chambers become stiffer and may work less efficiently. About 80% of heart disease deaths occur in people aged 65 or older (54).

**Sex:** Men of younger age are more vulnerable to attacks than pre-menopausal women of the same age and the risk of cardiovascular disease begins to rise in postmenopausal women with more than 55 years of age are more susceptible than men (88). Women before menopause are protected against CAD, because of the impact of sex hormones function (88).

**Family history:** Premature coronary vascular disease occurs in families with the history of CAD. The disease can be prevented by identifying the families with positive history of CAD, especially in people who have parents or siblings with artery disease or death in the family because of a history of CAD at an early age (55 years for a male and 65 for female relative). The risk of getting coronary heart disease among first-degree relatives was 2.5 to 7 times higher than the relatives of the controls (88).

**Genetic factor:** Cardiovascular risk factors, such as myocardial infarction locus (chromosomal loci) have strong genetic components (54), subjects with apolipoprotein E (apoE) polymorphism become less responsive to serum lipids in response to the dietary intervention (11). Familial hypercholesterolaemia is also another genetic condition explained by mutations in the low density lipoprotein (LDL) receptor gene and characterized by markedly elevated LDL cholesterol levels and increased risk of premature coronary heart disease (89). Similarly, the deletion or insertion polymorphism of the angiotensinogen gene M235T and angiotensin-converting enzyme gene A1166C are associated with hypertension and coronary heart disease, respectively (90).

**Environmental factors:** Seasonal variability in humans can partly be attributed to endogenous biological rhythms which exert an independent effect on variations in lipid and lipoprotein concentration. Environmental factors such as photoperiod or endocrine factors such as the concentration of steroid hormones can be correlated and/or involved in the regulation of these quantitative variations (90). Seasonal variations were shown statistically significant changes in lipid levels irrespective of the geographical area, and irrespective of the age, sex, ethnicity, and baseline lipid levels of the individuals (92). Cholesterol levels tend to be higher in the winter months and lower in the summer months (93). According to the study carried out by Robinson *et al* (94), the mean cholesterol levels were three to five percent higher in winter than in summer and the mean monthly cholesterol levels were negatively correlated with mean

monthly air temperatures ( $r=-0.60$  to  $-0.71$ ). Another study by Fuller *et al* (95) have demonstrated a significant fall in plasma triglyceride level from summer to winter and a smaller fall from spring to autumn. Seasonal changes in plasma volume explained a substantial proportion of the observed variation; a relative plasma hypervolemia during the summer seems to be linked to increases in temperature and physical activity (93, 96). Lipoprotein lipase (LPL) enzyme activity is also regulated seasonally; where adipose tissue LPL provides free fatty acids for storage in adipocytes, whereas in skeletal muscle LPL provides free fatty acids for oxidation. The activity of this enzyme is also higher in winter than in summer (96).

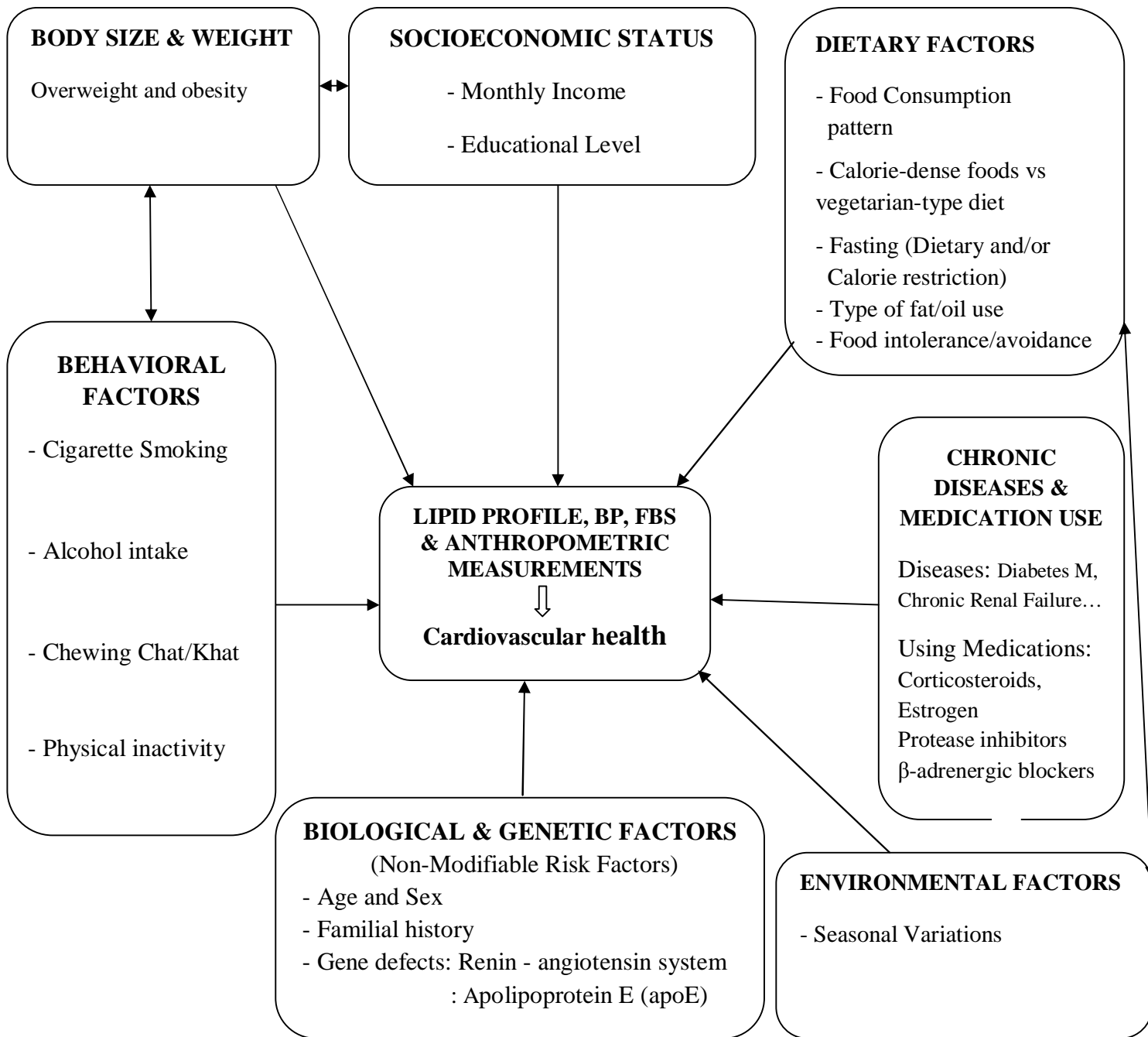


Figure 3: Conceptual frame work of risk factors for cardiovascular diseases [Developed from the reviewed literatures]

### **3. OBJECTIVE**

#### **3.1. General Objective:**

- The objective of this study is to assess the effects of Ethiopian Orthodox Christians' Easter fasting on the anthropometric indices and biochemical risk factors for cardiovascular diseases.

#### **3.2. Specific Objectives:**

- To measure the effect of fasting on biochemical changes;
- To assess the effect of fasting on anthropometric measurement.

## **4. METHODOLOGY**

### **4.1. Study area and period**

The study was conducted in Addis Ababa, the capital city of Ethiopia, from February 10 to April 10, 2015. According to the Ethiopian National Population and Housing Census of 2007 (26), Addis Ababa has a total population of 2,737,551. About 23% of the total urban population of Ethiopia lives in Addis Ababa and with respect to religion 74.7% are Orthodox Christians, 16.2% Islam and 7.8% are Protestants (26).

### **4.2. Study design**

Community based before and after study design having controls was used among voluntarily selected participants from eligible population and followed over Easter fasting period in two groups; those who fast (exposed) and non-fast (control) group. Within the two conditions, participants were measured on the dependent variables before and at the end. A major advantage of this, within subject, study design is that participants serve as their own controls, thus reducing the error variance and increasing the statistical power of the test with considerably fewer participants (97-99).

A pair of measurement was made for two groups before the beginning (pre or baseline) and before the completion (endline) of the Easter fasting period. Measurements include fasting blood collection for biochemical tests, anthropometric and blood pressure measurements, and the completion of questionnaires.

### **4.3. Study Population, Study Subjects and Sampling Procedure**

Study populations were Orthodox Christians living in Addis Ababa. Study population were chosen using convenience sampling technique from four sub-cities (Addis Ketema, Gulelie, Lideta and Bole) from faith based groups within the community like *Yetsewa Mahiber* and other informal groups (groups facilitating Ethiopian Epiphany called *yetemeket beal astebabariwoch*) on the basis of their willingness to participate and satisfying the inclusion criteria of the study.

Among Ethiopian Orthodox Christian adults of both sexes, faithful fasters who have been strictly adhering to Orthodox Christian fasting practices of the major recommended fasting periods regularly for  $\geq 5$  years and those who are planning to fast the upcoming Easter fasting were participated in this study as fasters. Control group included subjects that do not fast or do not avoid animal source foods consumption during the upcoming season of Easter fasting.

#### **4.4. Eligibility Criteria**

##### **4.4.1. Inclusion Criteria**

Volunteers, aged 18 years and older giving consent for the study were recruited from both sexes in the group of fasters and controls. Subjects who were permanent residents or temporarily residing in Addis Ababa during the study period were included. For the fasters group, subjects that have been fasting the major fasting periods of Ethiopian Orthodox Church were included. For the control group, those subjects that do not fast during the great lent fasting were included. Those who were free from chronic illnesses related to CVD and not taking long term medication that are known to alter the BP, blood glucose and lipid profiles were included.

##### **4.4.2. Exclusion Criteria**

Subjects who were not permanent residents or who do not stay in Addis Ababa during the study period; those who were with chronic illnesses and taking long term medication that are known to alter the blood glucose and lipid profiles were excluded. Subjects aged  $< 18$  years old; those who were unwilling to participate in the study; lactating and pregnant women were excluded.

#### **4.5. Sample Size Determination**

To determine sample size, the study used GPower Version 3.1.9.2 as a tool, this software provides sample size and power analyses for tests that use F, t, chi-square, or z distributions and various distributions for nonparametric applications. GPower is the one of the software packages that performs sample size calculations like Minitab and Epi-info covering a wider range of study designs (100-101). As an input GPower requires selecting appropriate test family (t-test in our case), type of statistical test within test family (dependant sample t-test), specifying  $\alpha$  error probability, power ( $1-\beta$  error probability), and determining effect size (102).

An effect size is the difference between two means (e.g., treatment minus control) divided by the standard deviation of the two conditions. It is the division by the standard deviation that enables us to compare effect sizes across experiments (103). Effect size can be used at planning stage to find the sample size required for sufficient power for study and for the purpose of calculating a reasonable sample size and effect size can be estimated by pilot study results, similar work published by others, or the minimum difference that would be considered important by experts (104). To calculate effect sizes from similar published research articles, a simplified methodology by Thalheimer and Cook (103) was used, employing equations 1 and 2;

$$d = \frac{\bar{x}_t - \bar{x}_c}{S_{pooled}} \quad \text{Eq. 1}$$

$d$  = Cohen's  $d$  effect size

$X$  = mean (average of treatment or comparison conditions)

$S$  = standard deviation

Subscripts:  $t$  refers to the treatment condition and  $c$  refers to the comparison condition (or control condition).

$$S_{pooled} = \sqrt{\frac{(n_t - 1)s_t^2 + (n_c - 1)s_c^2}{n_t + n_c}} \quad \text{Eq. 2}$$

Where:  $S$  = standard deviation

$n$  = number of subjects

Subscripts:  $t$  refers to the treatment condition and  $c$  refers to the comparison condition (or control condition).

From the study done by Sarie *et al* (11), fasters, as compared to their pre-fasting status, have showed a decreased levels of end total cholesterol, LDL-c and BMI as presented in Table 1. From that study, calculating for effect size among the three variables using equations 1 and 2 indicated that BMI (with mean difference of 0.4 and pooled standard deviation of 0.7) had relatively smaller or least detected difference with Cohen’s **d** effect size of 0.56.

Table 1: Paired samples T-test with mean pre-fasting values compared to mean end-fasting results from Sarie *et al* (11).

Variables	Pre-Fasting (n=43)		End-Fasting (n=43)		P-value	Effect Size ( <i>d</i> )
	Mean	Stan Dev	Mean	Stan Dev		
Total cholesterol (mmol/L)	5.6	0.15	5.1	0.14	<0.001	3.40
LDL cholesterol (mmol/L)	3.3	0.1	3.2	0.1	<0.001	0.61
BMI (kg/m <sup>2</sup> )	28	0.7	27.6	0.7	<0.001	0.56

Generally, effect sizes of 0.20 are considered small, 0.50 are medium, 0.80 are large and 1.3 are very large (104-105), these known benchmarks enable us to compare the above calculated effect size (0.56) to be categorized around medium effect size. Therefore, the present study considers medium effect size (0.5); power (1-β) of the study (0.85) and α-error probability (0.05) to have a sufficient sample size so as to detect differences that might present in the study variables; since sample size increases with increase in power, with a decrease in effect size and with decreasing level of significance.

Incorporating the above assumptions for sample size calculation using GPower software, sample size was found to be 38 for each group.

$$38 \times 2 \text{ (Control and Fasting group)} = 76$$

Considering 20% non-response, 16 subjects were added and the total number of study subjects were totally 92 (46 fasters and 46 controls).

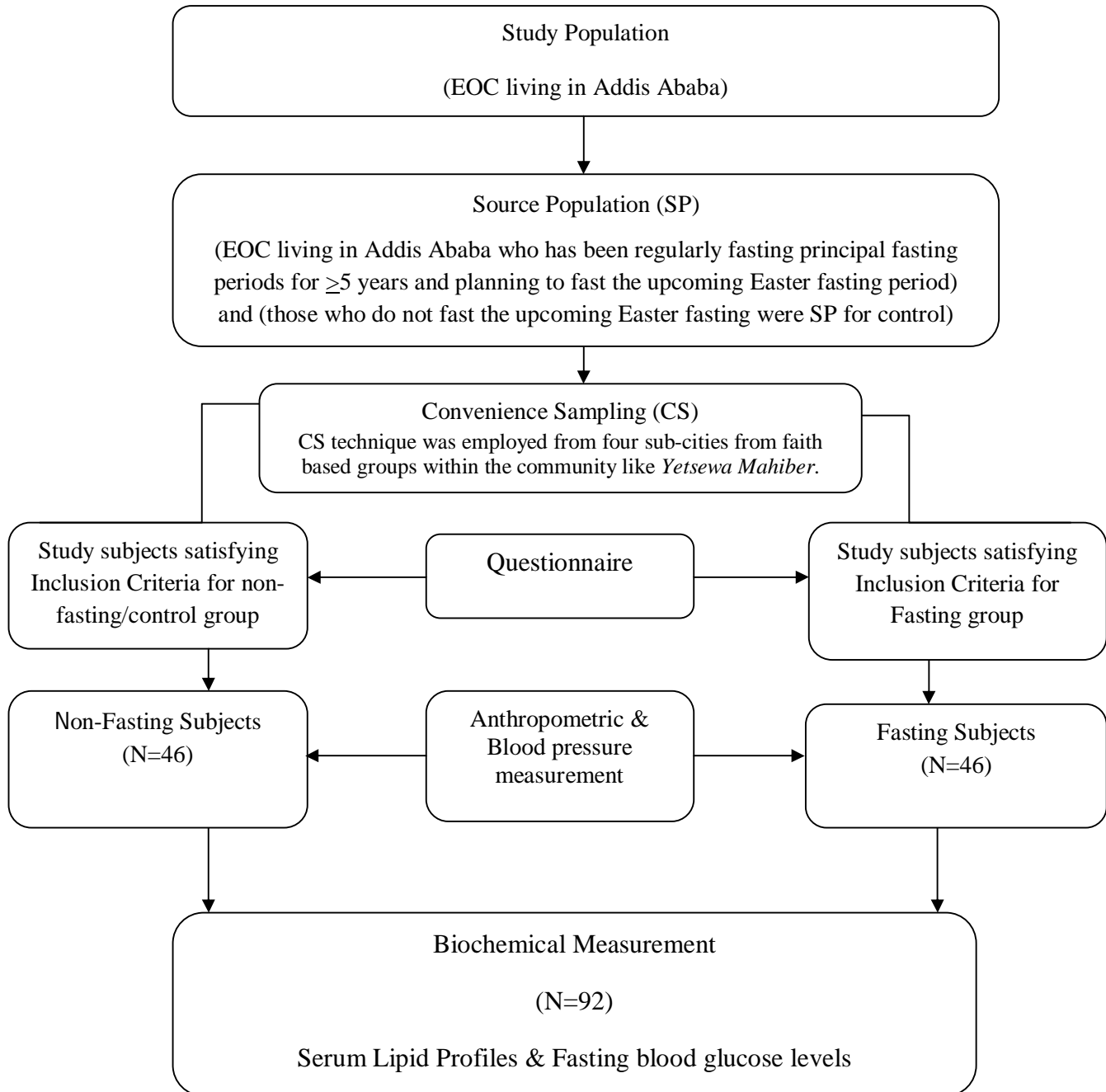
Similar previous studies conducted to evaluate the effect of religious fasting on biochemical and anthropometric variables used comparable sample sizes, to mention some: study by Mansi (17), used a total of 70 study subjects; Maislos *et al* (15), used 24 subjects; Thannoun & Mahmoud (18), used 31 subjects; Sarri *et al* (11), used 40 fasters and 31 controls; Sarraf-Zadegan *et al* (106), used a total of 50 study subjects and again Sarri *et al* (23), used 38 fasters and 29 matched controls. In repeated measures design fewer participants are often involved because of subject differences are minimized and hence reduces the error variability (98). Ha and Ha (107) suggests that if dependent variables of the study are either an interval or ratio scale, the sample size per group should be greater or equal to 30 ( $n \geq 30$ ).

#### **4.6. Data Collection and Instruments for Data Collection**

The instruments used for data collection were adapted mainly from the WHO's stepwise (STEPs) approach for non-communicable disease surveillance (108) and partly from Sarri *et al* (11), and Amole *et al* (6). STEP is the WHO-recommended surveillance tool for chronic disease risk factors and chronic disease-specific morbidity and mortality which is intended to serve as an entry point for low and middle-income countries into surveillance of chronic diseases and their risk factors (108). This approach is characterized by the use of questionnaires to gain information on risk factors, simple physical measurements (anthropometric and blood pressure measurements) and biochemical measurements (lipid profile and glucose level).

Subjects were examined and measured 1 week before the beginning (pre) and the last 5 days before the completion (end) of the Easter fasting period. After overnight fasting, measurements were made in the morning between 8 AM and 10 AM which include fasting blood collection (for biochemical analysis), anthropometric measurements (height, weight, waist and hip circumference), and blood pressure, the completion of questionnaires regarding their fasting habits, their health status, medication use, and their behavioral risk factors such as cigarette smoking, alcohol intake, physical inactivity, khat chewing, and the dietary habit (11).

#### 4.6.1. Sampling and Data Collection Framework



#### 4.6.2. Questionnaires

All volunteers participating in the study were interviewed face-to-face by the principal investigator or assistants using a modified standard questionnaire (6, 11, 108). The questionnaire has four components: questions on socio-demographic characteristics; behavioral risk factors and fasting experience; health status and medication use; and dietary assessment.

Socio-demographic characteristics included were age, sex, marital status, educational level, and economic status. Behavioral risk factors for cardiovascular health: physical inactivity, khat chewing, smoking and alcohol consumption. Questions on previous fasting experience that help to identify devoted fasters those who have been fasting regularly during the principal fasting periods. Regarding health status and medication use, subjects with chronic diseases related to CVD and medications use that have a potential to adversely affect the biochemical tests were screened and filtered at this stage.

Level of alcohol intake in this study considered the WHO risk of drinking (82-83), low risk subjects are those who consume below 20g of pure alcohol daily for women and 40g for men; and subjects at risk for health are those who consume  $\geq 20$ -40g for women and  $\geq 40$ -60g for men. Twenty grams pure alcohol is equivalent to 400ml of 5% alcohol beer, 182ml of 11% wine, 50ml of 40% alcohol spirits, 182ml of 11% *Tej*, 388ml of 5.16% alcohol *Tella*, and 54ml of 37% alcohol *Areki/Katikala* (109). The study considered the following volumes per container as indicated by Tekle-Haimanot and Haile (110) for locally produced alcoholic drinks in Ethiopia: *Tella* sold in tin containers called *Tass* of 750 ml volume; *Tej* which is drunk in funnel necked glass bottle known as *Birelle* with a volume of 300 ml; *Katikala* is a locally distilled spirit consumed in 15 ml small glass called *melekia*.

There are a wide range of methods that could be used for the dietary assessment of individuals and groups; but food frequency questionnaire (FFQ) is the most commonly used for investigating the relationship between dietary factors and chronic diseases (111). It is used to evaluate population intakes relative to reference values quantitatively or rank individuals as high and low consumers, qualitatively (112-113). FFQs is used to capture habitual/usual dietary intakes (114) and this study used predefined food lists within 13 food groups (115) and portion size was not

estimated. The food groups were adapted from FAO/EU guideline for measuring individual dietary diversity (116) the reference period of 7 days was used since short reference periods like the 24 hours recall period does not provide an indication of individual's habitual diet [116]. The 7 days reference period was converted in to different 'times' of frequencies of intake; never,  $\leq 2$  times per week, 3-6 times per week, once daily, and  $\geq 2$  times per day (117). In addition to FFQ, subjects' consumption of fast foods and practice of snacking were asked as indicated by Amole *et al* (6). Moreover, subjects were asked for dietary restrictions because of food intolerance or food allergies if any (118).

#### **4.6.3. Anthropometric variables**

Body weight was measured two times at pre-stage and end of fasting by a digital scale (Seca, Hamburg, Germany) to the nearest 100g, placed in flat surface. Subjects were weighed barefoot in very light clothing. Standing height was measured with an adjustable wooden measuring board once, without shoes to the nearest 0.1 cm with the shoulders in relaxed position, arms hanging freely, feet together, heels against the back board and knees straight. Body Mass Index (BMI) was calculated by dividing weight (kg) by height squared ( $m^2$ ) (3, 11).

Waist circumference was measured at the midpoint between the lower margin of the least palpable rib and the top of the hip or minimal waist using stretch-resistant tape. Hip circumference was measured around the widest portion of the buttocks, with the tape parallel to the floor. For both measurements, the subject stand with feet close together thereby body weight evenly distributed, arms at the side and wearing light clothing. When the subject become at relaxed state measurement was taken at the end of normal expiration and these measurements were done in a private place (108). The cut-off points for waist to hip ratio above 0.90 for males and above 0.85 for females used to indicate CVD risk (44, 108, 119).

Blood Pressure (BP) was measured with an automatic blood pressure monitor (OMRON, Model: HEM-711ACN, Illinois 60015) after individuals had been resting for 5 min (108). Subjects place their right arm on the table with the palm facing upward, three BP measurements were taken with 3 min elapsing between successive measurements and according to WHO recommendation,

the mean systolic blood pressure (SBP) and diastolic BP (DBP) from the second and the third measurements were considered for the analysis (3, 11, 108). Digital BP measuring machines (automated devices) are recommended by WHO steps for CVD risk assessment. Even though it is less accurate than manual devices, calibration to the standard method enhances the accuracy (120). Additionally, automated devices reduce higher degree of deviation in measuring diastolic blood pressure using manual devices (121).

#### **4.6.4. Biochemical assays**

Subjects were told to fast overnight, blood samples were obtained in the morning after 12 hours over night fast from an antecubital vein. Blood samples of 6-10 ml were collected in to plain vacuum tube (122), using proper sanitation and infection prevention techniques (3) and after 30 minutes, the collected blood was centrifuged at 3000 revolution per minute for 10 minutes (6); the separated serum was placed in cold box carrier containing ice packs that maintains the temperature around  $-4^{\circ}\text{C}$  and transported to the Ethiopian Public Health Institute (EPHI) to be kept in deep freezer at a temperature of  $-40^{\circ}\text{C}$  until the samples analyzed. The serum was later used to determine participants' fasting triglycerides, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol and glucose concentrations.

The serum levels of glucose, TC, HDL-c, LDL-c and TG were measured using COBAS INTEGRA 400 (Roche Diagnostics GmbH, Sandhofer Strasse 116, D-68305 Mannheim Germany) random access full automated auto analyzer at Ethiopian Public Health Institute, TB/HIV research and development laboratory. Glucose was determined by Hexokinase (HK) method, TG and total TC were evaluated with enzymatic colorimetric method and HDL-c and LDL-c were analyzed by homogenous enzymatic colorimetric method (123-124) and the results were reported as mg/dL.

The cutoff points to categorize dyslipidemia defined as TC  $\geq 200$  mg/dL, HDL-c  $< 40$  mg/dL, LDL-c  $\geq 130$  mg/dL, TG  $\geq 150$  mg/dL and hyperglycemia as glucose  $\geq 110$  mg/dL based on the classification of the United States National Cholesterol Education Program, Adult Treatment Panel (NCEP-ATP) III guideline for CVD risk factor (52). Lipoprotein ratios for therapeutic target cut-off values are  $< 3.7$  for LDL/HDL-c and  $< 5$  for TC/HDL-c ratio (47).

#### **4.7. Data Quality Control Methods**

The questionnaires were originally written in English, then translated in to Amharic and back translated to English by experts to ensure the quality of translation and consistency. Anthropometric measuring instruments and biochemical analyzers were calibrated by their respective reference materials. Digital blood pressure measuring machine was calibrated to the gold standard method for measuring blood pressure, mercury sphygmomanometer (125), using method agreement for clinical measurements as indicated by Bland and Altman (126).

Four assistant data collectors were involved one nurse for BP measurement, two laboratory professionals (1 technician and 1 technologist) for drawing blood specimen and one nutritionist (MSc holder) for the dietary assessment and anthropometric measurement. Two days training on the contents of the questionnaire, data collection techniques, and ethical conduct of human research was given for those assistants and any doubts/question in the method they were going to undertake was clarified.

Pretest of the questionnaire was conducted in 10 volunteer subjects (~10% study sample size) for validation of questionnaire two weeks prior to actual data collection and some adjustment on additional preparations was made. During the actual data collection period, the questionnaire was checked for completeness every night after data collection. Feedback on previous day activities was given for the assistant data collectors before the next day data collection and the overall coordination was made by the principal investigator.

#### **4.8. Data Management**

Data was checked for completeness and entered into SPSS version 20 computer soft ware package. After complete entry of all the questionnaires, soft copy was checked with its hard copy to see the consistency. After cross checking, cleaning was made to avoid missing values, outliers and inconsistencies before analysis. Study participant who could not complete their end status measurement because of various reasons were also excluded from analysis.

#### 4.9. Data Analysis

A statistical analysis program SPSS (version 20 for Windows) was used to analyze the data. Frequency distribution of socio-demographic characteristics of the study population was determined. Chi-square test was used to evaluate the difference in the distribution of categorical variables of the study groups. The Shapiro-Wilk test was performed to assess the normality of the distribution. For normally distributed variables, within group comparisons of pre and end fasting test results were analyzed using paired t-test and to detect differences between the fasting and the control groups, independent samples t-test was used after obtaining differences in result values before and at the end for test variables.

One way ANOVA and Kruskal Wallis tests were used to compare variables having more than two groups for parametric and nonparametric tests, respectively and Duncan *post hoc* test was used for mean separation. For the data that were not-normally distributed (pre- and end- TG values), the appropriate non-parametric tests Mann-Whitney and Wilcoxon's tests were employed to compare between group and within group comparisons, respectively. Regression analysis was used to compare variables' end-fasting concentrations and anthropometric measurements with their respective pre-fasting values, age, sex, socioeconomic and behavioral risk factors. Continuous variables were presented as mean values  $\pm$  standard deviation, while categorical variables were presented as absolute numbers and relative frequencies. All differences were considered statistically significant at  $p < 0.05$ .

#### 4.10. Operational Definitions

Physical activity: subjects who are engaged in leisure time physical activity (walking, fitness training and sports) for greater than or equal to three times per week of thirty minutes per occasion will be classified as physically active (127).

Body mass index (BMI): defined as the weight in kilograms divided by the square of height in meters ( $\text{kg}/\text{m}^2$ ) is used to measure the “degree of fatness”. Normal weight is characterized by a BMI of between 18.5 and 24.9  $\text{kg}/\text{m}^2$  (6). Overweight is defined as BMI values between 25 and 29.9  $\text{kg}/\text{m}^2$  (6).

Obesity is defined as a condition of abnormal or excessive fat accumulation in the adipose tissue of the body which is explained by BMI value  $\geq 30\text{kg}/\text{m}^2$  (6).

Fasters are defined as those who have been fasting regularly and decided to fast the Easter fasting period through avoiding consumption of all animal source foods during the period (11).

Current smokers are defined as those who smoked at least one cigarette per day (11).

Low risk alcohol consumption defined as drinkers who consume below 20g of pure alcohol daily for women and 40g for men; the levels above which are categorized in to risk to health (82-83).

Elevated blood pressure (BP) defined as a systolic blood pressure (SBP) of  $\geq 140$  mm Hg or a diastolic blood pressure (DBP)  $\geq 90$  mm Hg (52).

Elevated fasting blood glucose (FBG) to be  $\geq 110$  mg/dL or with a previous history of diabetes (52).

Dyslipidemia is the presence of abnormal levels of lipids in the blood, characterized by an elevation of the concentration of total cholesterol (TC), low-density lipoprotein (LDL), and triglycerides (TG), and a decrease in high-density lipoprotein cholesterol (HDL) (75).

#### **4.11. Ethical Considerations**

The study was conducted after obtaining ethical clearance from the Ethical Committee of Addis Ababa University, College of Health Science, School of Public Health with Reference number SPH/596/07; Dated February 10, 2015 (See Annex 1). Informed written consent was obtained from study participants after subjects get informed about the purpose and the benefit of the study along with their full right to refuse or completely reject from participation. Finally, interview record, anthropometric measurements, and biochemical analysis result were strictly kept confidential.

#### **4.12. Study Variables**

##### **4.12.1. Dependent Variable**

Anthropometric measurements (body mass index, waist circumference and waist to hip ratio); mean systolic and diastolic blood pressure; biochemical tests (fasting blood glucose, total cholesterol, low density lipoprotein, high density lipoprotein, triglyceride levels and lipoprotein ratios).

##### **4.12.2. Independent Variable**

The independent variables were individual's position based on fasting status; either fasting or non-fasting. In addition, socio-demographic characteristics such as age, sex, educational and income status; behavioral risk factors such as cigarette smoking, alcohol drinking, khat chewing and dietary practices.

#### **4.13. Dissemination of Results**

The result of this study will be submitted and presented to the School of Public Health, College of Health Sciences, Addis Ababa University as partial fulfillment of a master's degree in public health. The findings will also be disseminated through publishing on national or international journal and presentations on scientific conferences.

## 5. RESULTS

### 5.1. Scio-demographic, life style characteristics and practice of fasting

Socio-demographic and life style characteristics of the study population ( $n = 88$ ) are presented in Table 2. The study participants were volunteers that constituted two groups (each  $n = 44$ ) of fasters and non-fasters (controls) and with male to female ratio of nearly 1. The mean age of fasters ( $29 \pm 7$  years) and controls ( $31.4 \pm 7$  years); there were no statistically significant differences between fasters and controls group with respect to age, sex, educational level, and monthly income, with  $p$ -values 0.17, 0.67, 0.40 and 0.10, respectively.

Regarding the behavioral risk factors among fasters, there were no smokers, 36.4% reported to drink an alcohol, 9% of them do regular physical activity, 50% had a practice of eating fast foods and snacking was practiced by 28%. Between groups comparison of behavioral risk factors, showed no statistically significant differences in cigarette smoking ( $p = 0.21$ ), alcohol intake ( $p = 0.69$ ), physical activity ( $p = 0.06$ ), practice of eating fast foods ( $p = 0.21$ ) and snacking practice ( $p = 1.0$ ).

The subjects in the fasters group had been practicing the fasting rituals for a mean of  $10.7 \pm 4.3$  years; among the fasters, 56.8% practiced all the seven fasting periods of the Ethiopian Orthodox Church and the rest 43.2% practiced at least three of the principal fasting periods of the church (fasting of salivation, the great lent fasting preceding Easter and fasting preceding Christmas). During the fasting period, all the fasters had a practice of avoiding animal source foods; in addition, 61.4% skip breakfasts and 22.7% fasted until 3PM. The fasters' compliance with the fasting rules was 100%.

Table 2: Scio-demographic and life style characteristics of the study subjects, April 2015, Addis Ababa.

<b>Variables</b>	<b>Fasters N (%)</b>	<b>Controls N (%)</b>	<b>p-value</b>
<b>Sex</b>			0.67
Males	22 (50)	24 (54.55)	
Females	22 (50)	20 (45.45)	
<b>Age†</b>	29.34 ± 7.14	31.43 ± 6.99	0.17
<b>Educational Status</b>			0.40
Elementary School/ No education	8 (18.1)	9 (20.5)	
Secondary school	14 (31.8)	16 (36.4)	
> Secondary school	22 (50)	19 (43.2)	
<b>Monthly income</b>			0.10
<900 Eth Birr	12 (27.3)	10 (22.7)	
1000-1999 Eth Birr	10 (22.7)	7 (15.9)	
2000-2999 Eth Birr	7 (15.9)	13 (29.5)	
≥ 3000	15 (34.1)	14 (31.8)	
<b>Cigarette Smoking</b>			0.21
Current smokers	0 (0)	3 (6.8)	
Current Non-smokers	44(100)	41 (93.2)	
<b>Alcohol intake</b>			0.69
Non-consumers	28 (66.6)	35 (79.5)	
Moderate level consumers	7 (15.9)	1 (2.3)	
Risk to health level consumers	9(20.5)	8(18.2)	
<b>Physical Activity</b>			0.06
Yes	9 (20.5)	3 (6.8)	
No	35 (79.5)	41 (93.2)	
<b>Practice of Eating Fast Foods</b>			0.13
Yes	22 (50)	29 (65.9)	
No	22 (50)	15 (34.1)	
<b>Snacking Practice</b>			1.00
Yes	28 (63.6)	28 (63.6)	
No	16 (36.4)	16 (36.4)	

*P* - values are from comparison between fasters and controls using Chi-square. † Independent sample t - test was used

All study subjects in both groups reported that they did not suffer from any chronic diseases like thyroid related, diabetes, cardiovascular diseases and did not take any medication or food supplements that affect the dependent variables of the study. In addition, there was no reported food avoidance because of food allergy or intolerance in both groups.

## 5.2. Baseline characteristics of study groups

Baseline characteristics of serum lipids, blood glucose level, blood pressure and anthropometric measurements between fasters and control group are shown in Table 3 and showed no statistically significant differences among all variables across the two groups.

Table 3: Baseline characteristics of serum lipids, blood glucose level, blood pressure and anthropometric measurements between fasters and control group, April 2015, Addis Ababa

Variables	Fasters (N = 44)		Controls (N = 44)		p-value
	Mean	St. Dev	Mean	St. Dev	
TC (mg/dL)	172.27	40.63	164.73	32.89	0.34
HDL-c (mg/dL)	43.84	10.85	42.64	8.39	0.56
LDL-c (mg/dL)	109.71	37.48	98.14	28.89	0.11
TG (mg/dL) †	112.58	48.44	117.8	44.21	0.27
TC/HDL	4.18	1.48	4.08	1.33	0.73
LDL/HDL	2.69	1.23	2.45	1.05	0.34
FBS (mg/dL)	86.84	8.92	89.09	9.07	0.24
SYST BP (mm Hg)	118.1	12.32	123.59	14.38	0.06
DIST BP (mm Hg)	75.71	8.20	78.02	9.42	0.22
BMI (Kg/m <sup>2</sup> )	22.15	3.81	23.00	4.18	0.32
WC (cm)	81.64	11.77	84.45	11.07	0.25
WHR	0.83	0.068	0.862	0.086	0.07

FBS, fasting blood glucose; TC, total cholesterol; HDL-c, high density lipoprotein; LDL-c, low density lipoprotein; TG, triglyceride; TC/HDL, ratio of total cholesterol to high density lipoprotein; LDL/HDL, ratio of low density lipoprotein to high density lipoprotein; BMI, body mass index; WC, waist circumference; WHR, waist to hip ratio; SYST BP, systolic blood pressure; DIST BP, diastolic blood pressure. † Non-parametric Mann-Whitney test was used.

### **5.3. Dietary data**

Food frequency result are shown in Table 4, the data show statistically non-significant difference between groups with respect to consumption of grains/cereals, fruits, fats/oils, tea/coffee, house hold sugar/honey, confectionary foods. However, animal source food groups (milk and milk products, fish, egg, and meat) were not consumed by fasters during the fasting period and frequency of intake by control groups were significantly higher. Fasters have shown significantly higher frequency intake of vegetables, pulses/legumes, and roots/tubers than controls ( $p < 0.001$ ,  $p = 0.002$ , and  $p < 0.001$ , respectively).

Table 4: Result of seven days frequency of food intake between fasters and controls at end fasting, April 2015, Addis Ababa.

Food Groups	Category	Frequency of intake in previous week					p-value
		Never N (%)	≤2X/Week N (%)	3-6X/Week N (%)	Once per day N (%)	≥2x/day N (%)	
<b>Fruits</b>	Fasters (N=44)	0 (0)	21 (47.7)	16 (36.4)	6 (13.6)	1 (2.3)	0.118
	Controls (N=44)	4 (9.1)	16 (34.4)	21 (47.7)	3 (6.8)	0 (0)	
<b>Vegetables</b>	Fasters (N=44)	0 (0)	0 (0)	23 (52.3)	19 (43.2)	2 (4.5)	<0.001
	Controls (N=44)	1 (2.3)	8 (18.2)	35 (79.5)	0 (0)	0 (0)	
<b>Grains/Cereals</b>	Fasters (N=44)	0 (0)	0 (0)	0 (0)	0 (0)	44 (100)	0.315
	Controls (N=44)	0 (0)	0 (0)	0 (0)	1 (2.3)	43 (97.7)	
<b>Pulses/Legumes/Nuts</b>	Fasters (N=44)	0 (0)	0 (0)	0 (0)	35 (79.5)	9 (20.5)	0.002
	Controls (N=44)	0 (0)	0 (0)	0 (0)	44 (100)	0 (0)	
<b>Roots &amp; Tubers</b>	Fasters (N=44)	1 (2.3)	8 (18.2)	32 (72.7)	3 (6.8)	0 (0)	<0.001
	Controls (N=44)	2 (4.5)	30 (68.2)	11 (25.0)	0 (0)	1 (2.3)	
<b>Milk &amp; milk products</b>	Fasters (N=44)	44 (100)	0 (0)	0 (0)	0 (0)	0 (0)	
	Controls (N=44)	10 (22.7)	14 (31.8)	15 (34.1)	3 (6.8)	0 (0)	
<b>Fish</b>	Fasters (N=44)	44 (100)	0 (0)	0 (0)	0 (0)	0 (0)	
	Controls (N=44)	41 (93.2)	2 (4.5)	1 (2.3)	0 (0)	0 (0)	
<b>Egg</b>	Fasters (N=44)	44 (100)	0 (0)	0 (0)	0 (0)	0 (0)	
	Controls (N=44)	8 (18.2)	15 (34.1)	21 (47.7)	0 (0)	0 (0)	
<b>Meat</b>	Fasters (N=44)	44 (100)	0 (0)	0 (0)	0 (0)	0 (0)	
	Controls (N=44)	2 (4.5)	4 (9.1)	28 (63.6)	10 (22.7)	0 (0)	
<b>Fats/Oils</b>	Fasters (N=44)	0 (0)	0 (0)	0 (0)	6 (13.6)	38 (86.4)	0.502
	Controls (N=44)	0 (0)	0 (0)	0 (0)	4 (9.1)	40 (90.9)	
<b>Tea/Coffee</b>	Fasters (N=44)	3 (6.8)	1 (2.3)	4 (9.1)	9 (20.5)	27 (61.4)	0.747
	Controls (N=44)	1 (2.3)	1 (2.3)	2 (4.5)	11 (25.0)	29 (65.9)	
<b>Household sugar/Honey</b>	Fasters (N=44)	0 (0)	1 (2.3)	2 (4.5)	13 (29.5)	28 (63.6)	0.943
	Controls (N=44)	0 (0)	1 (2.3)	3 (6.8)	11 (25.0)	29 (65.9)	
<b>Confectionary foods</b>	Fasters (N=44)	6 (13.6)	16 (36.4)	15 (34.1)	6 (13.6)	1 (2.3)	0.075
	Controls (N=44)	1 (2.3)	10 (22.7)	18 (40.9)	11 (25.0)	4 (9.1)	

P - values are from comparison between fasters and controls using Chi-square ( $X^2$  under linear by linear association).

#### **5.4. Comparisons of pre and endline-fasting values in the fasting and control groups**

Changes in serum lipids level, blood pressure and anthropometric measurements between baseline and endline among fasting and control groups are shown in Table 5. In the fasting group, there was statistically significant decline in the levels of fasting blood sugar ( $p = 0.001$ ), total cholesterol ( $p < 0.001$ ), LDL-c ( $p < 0.001$ ), TG ( $p = 0.031$ ), lipoprotein ratios ( $p < 0.001$ ), weight ( $p < 0.001$ ), BMI ( $p < 0.001$ ), waist to hip ratio ( $p < 0.001$ ), systolic and diastolic blood pressure ( $p < 0.001$ ). However, there was no statistically significant change in the HDL cholesterol concentrations.

In control group, there were no statistically significant changes observed in the level of total cholesterol ( $p = 0.44$ ), LDL-c ( $p < 0.31$ ), HDL-c ( $p = 0.14$ ), TG ( $p = 0.46$ ), lipoprotein ratios ( $p > 0.05$ ), fasting blood glucose concentration ( $p = 0.061$ ), systolic and diastolic blood pressure  $p < 0.80$  and  $p = 0.44$ , respectively. However, statistically significant increases were seen in the levels of all anthropometric variables: weight ( $p = 0.001$ ), BMI ( $p = 0.001$ ), waist circumference ( $p < 0.001$ ) and waist to hip ratio ( $p < 0.001$ ).

Table 5: Serum lipids level, blood pressure and anthropometric measurements between baseline and endline among fasting and control group, April 2015, Addis Ababa.

Variables	Fasters (N = 44)			Controls (N = 44)		
	Pre	Endline	<i>p</i> -value	Pre	Endline	<i>p</i> -value
FBS (mg/dL)	86.84 ± 8.92	80.86 ± 9.58	0.001	89.10 ± 9.07	93.83 ± 8.86	0.061
TC (mg/dL)	172.27 ± 40.63	151.89 ± 33.14	<0.001	164.73 ± 32.89	166.98 ± 32.93	0.441
HDL-c (mg/dL)	43.84 ± 10.86	43.52 ± 10.02	0.778	42.41 ± 8.39	44.55 ± 9.63	0.144
LDL-c (mg/dL)	109.71 ± 37.48	87.44 ± 29.96	<0.001	98.14 ± 28.89	95.65 ± 26.51	0.306
TG (mg/dL) †	112.57 ± 48.44	100.70 ± 44.41	0.037	117.80 ± 44.21	122.66 ± 48.48	0.052
TC/HDL	4.18 ± 1.48	3.67 ± 1.20	<0.001	4.08 ± 1.33	3.90 ± 1.00	0.365
LDL/HDL	2.69 ± 1.23	2.13 ± 0.94	<0.001	2.45 ± 1.05	2.23 ± 0.68	0.062
Weight (Kg)	59.71 ± 12.74	58.43 ± 12.47	<0.001	65.72 ± 13.29	66.30 ± 13.54	0.001
BMI (Kg/m <sup>2</sup> )	22.15 ± 3.81	21.69 ± 3.76	<0.001	23.01 ± 4.18	23.21 ± 4.26	0.001
WC (cm)	81.64 ± 11.77	79.56 ± 11.44	<0.001	84.45 ± 11.06	85.75 ± 11.00	<0.001
WHR	0.83 ± 0.068	0.815 ± 0.068	<0.001	0.862 ± 0.086	0.873 ± 0.083	<0.001
SYST BP (mmHg)	118.1 ± 12.32	113.63 ± 10.72	<0.001	123.59 ± 14.38	123.41 ± 14.75	0.801
DIST BP (mmHg)	75.71 ± 8.20	72.30 ± 7.15	<0.001	78.02 ± 9.41	78.52 ± 10.20	0.436

Values are presented as mean ± standard deviation; FBS, fasting blood glucose; TC, total cholesterol; HDL-c, high density lipoprotein; LDL-c, low density lipoprotein; TG, triglyceride; TC/HDL, ratio of total cholesterol to high density lipoprotein; LDL/HDL, ratio of low density lipoprotein to high density lipoprotein; BMI, body mass index; WC, waist circumference; WHR, waist to hip ratio; SYST BP, systolic blood pressure; DIST BP, diastolic blood pressure. † Non-parametric Wilcoxon Signed Ranks test was used.

## 5.5. Comparisons of changes in the fasters and controls groups

Changes between groups are presented in Table 6 showing statistically significant differences in most lipid profiles: TC ( $p < 0.001$ ), LDL-c ( $p < 0.001$ ), TG ( $p = 0.008$ ) and LDL/HDL ( $p = 0.036$ ); however, the values for HDL-c and TC/HDL-c ratio were shown insignificant between group differences. In addition, all anthropometric, blood pressure and blood glucose measurements indicated that fasters had significantly lower values than controls.

Table 6: Mean difference comparison of serum lipids, blood glucose, blood pressure and anthropometric measurements between fasting and control groups, April 2015, Addis Ababa

Variables	Fasters (n = 44)		Controls (n = 44)		p-value
	Mean Diff	SEMD	Mean Diff	SEMD	
FBS (mg/dL)	- 5.98	1.60	4.73	1.20	<0.001
TC (mg/dL)	- 20.37	2.93	2.25	2.89	<0.001
HDL-c (mg/dL)	- 0.32	1.12	1.91	1.29	0.194
LDL-c (mg/dL)	- 22.27	3.55	- 2.50	2.41	<0.001
TG (mg/dL) †	- 11.87	5.33	4.86	6.47	0.008
TC/HDL	- 0.51	0.12	- 0.17	0.19	0.137
LDL/HDL	- 0.56	0.11	- 0.22	0.12	0.036
Weight (Kg)	- 1.28	0.19	0.58	0.16	<0.001
BMI (Kg/m <sup>2</sup> )	- 0.46	0.07	0.20	0.06	<0.001
WC (cm)	- 2.08	0.25	1.30	0.20	<0.001
WHR	- 0.02	0.002	0.11	0.002	<0.001
SYST BP (mm Hg)	- 4.47	0.87	- 0.18	0.71	<0.001
DIST BP (mm Hg)	- 3.41	0.66	0.50	0.64	<0.001

Mean difference (endline - baseline) comparisons between groups using independent samples t-test; SEMD, Standard error of the mean difference; FBS, fasting blood glucose; TC, total cholesterol; HDL-c, high density lipoprotein; LDL-c, low density lipoprotein; TG, triglyceride; TC/HDL, ratio of total cholesterol to high density lipoprotein; LDL/HDL, ratio of low density lipoprotein to high density lipoprotein; BMI, body mass index; WC, waist circumference; WHR, waist to hip ratio; SYST BP, systolic blood pressure; DIST BP, diastolic blood pressure. † Non-parametric Mann-Whitney test was used.

## 5.6. Effect of fasting and other predictors on endline-fasting values

Multiple linear stepwise regression analysis identified fasting to be a strong predictor for most of the dependent variables that have shown an inverse relation to endline values of total cholesterol ( $\beta = - 20.8, p < 0.001$ ), LDL cholesterol ( $\beta = - 16.0, p < 0.001$ ), LDL/HDL ratio ( $\beta = - 0.25, p < 0.03$ ), blood sugar ( $\beta = - 11.45, p < 0.001$ ), systolic ( $\beta = - 4.92, p < 0.001$ ), diastolic blood pressure ( $\beta = - 4.20, p < 0.001$ ), BMI ( $\beta = - 0.69, p < 0.001$ ) and endline waist circumference ( $\beta = - 3.31, p < 0.001$ ), showing that fasters have lower levels of these variables (Table 7).

Age of participants was positively related to endline TC/HDL ratio ( $\beta = 0.034, p=0.01$ ) and endline blood glucose levels ( $\beta = 0.27, p = 0.037$ ). Similarly, smoking status was positively related to endline-LDL/HDL ratio ( $\beta = 0.80, p < 0.011$ ). Whereas, educational status was inversely related to endline-TG level ( $\beta = -17.32, p = 0.02$ ); and body mass index was also negatively related to participants' monthly income ( $\beta = - 0.22, p < 0.031$ ). The practice of eating fast foods was positively related to both endline-HDL levels and endline-waist circumference with  $\beta = 3.32, p < 0.001$  and  $\beta = 7.35, p < 0.032$ , respectively. However, other predictors like alcohol intake, khat chewing, sex, physical activity and snaking practice were not show a significant relationship to any of the dependent variables of this study.

Table 7: Effects of different predictor variables on serum lipids, Anthropometric, blood pressure and glucose level among study participants (n = 88) in Addis Ababa, April 2015.

Dependant variables	Predictors	Beta Coefficient ( $\beta$ )	Standard Error	p-value	95% Confidence interval for $\beta$
Endline Total Cholesterol	Pre-fasting TC	0.763	0.051	<0.001	[0.66, 0.86]
	Fasting	- 20.84	3.711	<0.001	[-28.22, -13.46]
Endline LDL Cholesterol	Pre-fasting LDL	0.678	0.055	<0.001	[0.57, 0.79]
	Fasting	- 16.03	3.70	<0.001	[- 23.37, - 8.68]
Endline HDL Cholesterol	Pre-fasting HDL	0.66	0.081	<0.001	[0.50, 0.82]
	Eating fast food	3.32	1.57	<0.001	[0.204, 6.44]
Endline TC/HDL Cholesterol	Pre-fasting TC/HDL	0.48	0.067	<0.001	[0.34, 0.61]
	Age	0.034	0.013	0.010	[0.004, 0.057]
	Smoking	1.035	0.471	0.031	[0.098, 1.97]
Endline LDL/HDL Cholesterol	Pre-fasting LDL/HDL	0.529	0.052	<0.001	[0.426, 0.60]
	Smoking	0.795	0.306	0.011	[0.186, 1.41]
	Age	0.015	0.008	0.083	[-0.002, 0.035]
	Fasting	- 0.249	0.113	0.03	[-0.475, -0.024]
Endline Fasting Blood Sugar	Pre-fasting FBS	0.42	0.101	<0.001	[0.22, 0.62]
	Fasting	- 11.45	1.733	<0.001	[- 14.90, - 8.0]
	Age	0.27	0.128	0.037	[0.17, 0.53]
Endline Mean Systolic BP	Pre-fasting Systolic BP	0.89	0.041	<0.001	[0.805, 0.97]
	Fasting	- 4.92	1.11	<0.001	[- 7.12, - 2.72]
Endline Mean Diastolic BP	Pre-fasting Diastolic BP	0.878	0.051	<0.001	[0.78, 0.98]
	Fasting	- 4.20	0.902	<0.001	[-5.99, - 2.40]
Endline fasting BMI	Pre-fasting BMI	1.01	0.012	<0.001	[0.983, 1.03]
	Fasting	- 0.685	0.093	<0.001	[- 0.87, - 0.50]
	Monthly Income	- 0.218	0.100	0.031	[- 0.416, - 0.02]
Endline fasting WC	Pre-fasting WC	0.982	0.014	<0.001	[0.954, 1.01]
	Fasting	- 3.31	0.318	<0.001	[- 0.87, - 0.5]
	Eating fast food	7.35	0.330	0.29	[0.079, 1.39]
Endline fasting Triglyceride	Pre-fasting Triglyceride	0.636	0.081	<0.001	[0.47, 0.80]
	Fasting	- 17.46	7.49	0.022	[-32.35, -2.56]
	Educational status	- 17.29	7.50	0.024	[-32.21, -2.37]

Multiple Linear Stepwise Regression Analysis with mean end-TC, end-LDL cholesterol, end-HDL cholesterol, end-BMI, end-WC and the end-ratios of TC/HDL-C and LDL-C/HDL-C as the dependent variables. Independent variables were age, mean pre-BMI, fasting status, smoking, sex, educational level, WHR, pre-ratios of TC/HDL-C and LDL-C/HDL-C and the pre-values of the dependent variables. Sex, fasting, smoking, physical activity, monthly income, eating fast foods and snacks are dummies (sex: 0 = females and 1 = males, fasting status: 0 = control subjects and 1 = fasters, smoking: 0 = non-smokers and 1 = smokers, Education status: < Secondary = 0 and  $\geq$  Secondary = 1, Eating fast food: not = 0 and yes = 1, Monthly income: < 2000 birr = 0 and  $\geq$  2000 = 1, Physical activity: yes = 0 and no = 1, Snacking: not = 0 and yes = 1).

Analysis of variance test across the three levels of alcohol consumption (non-consumers, moderate level consumers and risk to health level consumers) have showed statistically insignificant differences across groups. However, the types of oils used have shown statistically significant difference in endline fasting blood glucose and triglyceride concentrations with p-values 0.001 and  $< 0.001$ , respectively. Fasting blood glucose concentration is significantly higher in palm oil users ( $91.85 \pm 9.66$ ) than Niger seed oil ( $83.26 \pm 11.18$ ) and sunflower oil ( $81.67 \pm 11.27$ ) users. Similarly, endline triglyceride concentration was significantly higher among palm oil users ( $129.93 \pm 48.33$ ) than niger seed oil ( $97.17 \pm 41.39$ ) and sunflower oil users groups ( $75.25 \pm 23.56$ ).

## 6. DISCUSSION

The most important finding of this study is that most serum lipids and anthropometric variables significantly decreased among the fasting group over the fasting period. Despite similar biochemical, anthropometric, and socio-demographic characteristics among fasters and controls at baseline, fasters had decreased levels of total cholesterol, LDL-c, TG, LDL/HDL-c ratio, blood pressure, fasting blood sugar, WHR and BMI at the end of fasting period.

Between the baseline and end line assessments, among fasters total cholesterol decreased by 11%, LDL-C by 20%, TG by 11%, LDL/HDL ratio by 21%, TC/HDL ratio by 12%, and blood glucose 7%. In contrast, HDL-c only decreased by 0.7%. Most of these findings are in agreement with the results reported by previous studies (11, 128-134).

A longitudinal study by Sarri *et al* 2003 (11) compared serum lipids and obesity indices between fasters and non-fasters over the three principal fasting periods of Greek Orthodox Church and have demonstrated that fasters had 10% reduction in TC, 12% reduction in the LDL-c levels, body weight and BMI were decreased by 2% during the Easter fasting period. Similarly, Barnard *et al* (128) who conducted a strict vegetarian-diet intervention study for 5 weeks on 35 healthy women with comparable age (22 to 48 years) to the present study placed study subjects on the intervention diet consisted grains, legumes, vegetables and fruit. After the intervention diet phase total cholesterol and LDL were decreased by 13.2% and 16.9% respectively (128). Goff *et al* (129) reported a significant decrease in triglyceride level in vegans compared to omnivores. Likewise, Nieman *et al* (130) and Toohey *et al* (131) investigated Seventh-Day Adventists with comparable dietary habits to the fasting group in our study. They found that lifetime strict vegetarians had lower concentrations of total-, and LDL cholesterol, as well as blood glucose concentrations when compared with non-vegetarians and lacto-ovo-vegetarians ( $p < 0.05$ ) (130). Toohey *et al* (131) also found lower levels of BMI, triacylglycerols and TC/HDL ratio.

In the present study, the level of HDL-c was not changed significantly between baseline and end line as well as across groups. This may be partly explained by the different dynamics of changes in HDL cholesterol (132); in locations where a low-fat diet is a norm, HDL-c levels are low and upon dietary fat restriction, further decrease in HDL-c become insignificant. In contrast, increasing the amount of fat and cholesterol in the diets of such people with a habitually low fat diet may increase their HDL-c, because the body will respond in a way to get eliminate the extra fat and cholesterol by increasing the number of available cholesterol reverse transporters (HDL-c); and hence, reducing dietary fat and cholesterol may cause a decrease in HDL-c because there is less need for it (132).

In support of the above explanation, previous study by Kahleova *et al* (133) that followed subjects with mean baseline HDL-c levels of experimental group ( $41.38 \pm 11.6$ ) and control group ( $42.15 \pm 7.7$ ) provided with calorie restricted vegetarian diets over 24 weeks have shown no significant change in HDL cholesterol within the intervention group and between comparison groups ( $p=0.07$ ). Similarly, Astazole *et al* (134), conducted a crossover study in individuals with a low levels of HDL-c at baseline, providing them with a low-fat diet over six weeks resulted in insignificant decrease in their HDL-C, but substantial decreases in their LDL-c concentrations resulted in a significant change in the LDL-c/HDL-c ratio. In the present study, the baseline mean HDL-c concentration of the subjects were not far from the borderline ( $43.84 \pm 10.85$ ) for the fasting and  $42.64 \pm 8.39$  for the control groups; further restriction in dietary fat because of fasting have resulted in statistically insignificant changes ( $p=0.78$ ) which is in agreement with the above studies (133-134).

Lipoproteins ratios TC-c/HDL-c and LDL-c/HDL-c were reported to remain unchanged after a Greek orthodox Christians fasting ritual (11), and low-fat vegetarian diet (128), possibly because of simultaneous decreases in the concentrations of TC, LDL-c and HDL-c. However, in the present study, both lipoproteins ratios were decreased significantly among fasters as a result of decrease in the levels of TC and LDL-c with insignificant change in the level of the denominator (i.e., HDL-c). This finding is supported by Toohey *et al* (131) that compared strict vegetarians (vegans) to lacto-ovo vegetarian (LOV) in African-Americans. They reported that serum total cholesterol, LDL-c, and triglycerides were significantly lower in vegans compared to LOV, but

there were no differences in HDL-c; and the ratio of total to HDL-c was significantly lower in vegans because of the isolated decreases in the numerator variable (i.e., TC). In the present study, for the given significantly unchanged HDL-c levels, the 20% reduction in the LDL-c brought a significant intergroup difference in LDL-c/HDL-c ratio ( $p=0.036$ ); in contrast, the 10% reduction in TC concentrations led to insignificant TC/HDL-c ratio intergroup variability ( $p=0.137$ ).

Fasting blood glucose concentrations were reduced by a mean difference of  $-6\text{mg/dL}$ , which is 7% in the present study. This finding is in line with Nieman *et al* (130) who showed that in comparison to non-vegetarians, vegetarians had significantly lower serum glucose levels. Correspondingly, Goff *et al* (129) have reported  $7\text{mg/dL}$  mean reduction in fasting blood glucose in vegans than omnivores. Calorie-restricted vegetarian diets have been shown to increase insulin sensitivity through reduced volume of visceral fat and body weight (133).

The anthropometric measurements have shown a significant change during the fasting period among fasters' group ( $p < 0.001$ ); a reduction of 2% in body weight, BMI and waist to hip ratio whereas, waist circumference was reduced by 3%. At baseline there were no significant difference between groups as presented in Table 3; however, these anthropometric variables have shown statistically significant increases in the control group ( $p = 0.001$ ) (Table 5).

Fasting and control groups had mean BMI in the normal category at both pre and endline fasting states. Fasting had a statistically significant impact on fasters' BMI at the end of the fasting period. In accordance to the results in this study, Sarrie *et al* (11) reported 2% reduction in the level of both body weight and BMI in the Greek Orthodox Christians Easter fasters. Haddad *et al* (137), studying a group of vegans and non-vegetarians has found a significantly lower BMI levels in the vegan group (137). Similar findings were reported for BMI reduction by Toohey *et al* (131) and a significant reduction in waist circumference by Kahleova *et al* (133) after calorie-restricted vegetarian diet. Greater improvement in risk factors occurs with greater body weight losses (138), even a modest weight loss of 5-10% of initial body weight was shown to substantially improve insulin sensitivity, glycemic control, hypertension and dyslipidemia in people at risk (139).

Seasonal changes were shown to affect anthropometric measurements (135-136) and lipid profiles (93-96). In the present study, endline measurements were taken around mid-April, which is two weeks before May, the hottest month of the year in Ethiopia. In warmer weather body water increases because of fluid conserving hormone which allow the kidney to retain more fluid and reduce the amount of salt in sweat leading to more water retention that account for weight gain. In addition, unlike winter, in summer there is relatively lower metabolic rate and minimum calorie burning to maintain core body temperature (135). The observed increase in the anthropometric variables among controls could be attributed to seasonal change.

However, there were no simultaneous rises in lipid profiles along with the increase in anthropometric variables in control group. Similarly, seasonal variations were shown statistically significant changes in lipid levels irrespective of the geographical area, and irrespective of the age, sex, ethnicity, and baseline lipid levels of the individuals (92). According to the study carried out by Robinson *et al* (94), the mean monthly cholesterol levels were negatively correlated with mean monthly air temperatures. Seasonal changes in plasma volume explain a substantial proportion of the observed variation; a relative plasma hypervolemia during hot season is linked to increase in temperature (93, 96); in addition, the activity of lipoprotein lipase enzyme is also lower in summer (96).

Fasting led to significant reductions in systolic (5%) and the diastolic (4%) blood pressure ( $p < 0.001$ ). The mean difference was -4.5 and -3.4 mmHg for systolic and diastolic blood pressures, respectively. Comparable finding was reported by Appleby *et al* (140) by comparing vegans and meat eaters. Vegans were reported to have a lower prevalence of hypertension and lower systolic and diastolic blood pressure than meat eaters. The mean differences in blood pressure reduction between meat eaters and vegans among participants with no self-reported hypertension were -4.2 mmHg in systolic and -2.8 mmHg in diastolic blood pressure (140). Similar finding was also reported by Goff *et al* (129) that vegans had a significantly lower mean systolic blood pressure when compared to omnivores.

Reduced intakes of dietary saturated fatty acids and cholesterol are known to lower total and LDL cholesterol concentrations and are associated with low risk of cardiovascular diseases (141-142). Reducing dietary saturated fat to 7% of energy was shown to reduce in serum cholesterol

concentration by 10% and mortality from ischemic heart disease by 25-30% (141). Food composition analysis of Greek orthodox Christian nuns' showed that their diet was very low in cholesterol and in saturated fat intake (6% of total energy intake), and high in fiber and antioxidant vitamins that could be attributed to the nuns' high consumption of fruit, vegetables, cereals and legumes (143).

Vegetarians consume more grains, vegetables, fruit, and legumes; as a result their diet consists of more dietary fiber and less dietary cholesterol (137). Calorie restricted vegetarian diet was shown to improve body mass index and LDL-c level more than a conventional diet (139). Meta-analysis of randomized controlled trials found that diets high in dietary pulses/legumes (beans, peas, chickpeas, lentils) reduced TC and LDL-c (144), lowered fasting blood glucose levels (145). Evaluations of nutrient intake levels done by Haddad *et al* (137) have shown that vegan diets provides significantly higher amounts of ascorbate than non-vegeterians and the higher antioxidants level in diet shown to maintain very low levels of LDL cholesterol (143). A vegetarian diet was also reported to reduce intramyocellular lipid concentrations (129).

Frequent consumption of foods high in dietary fibers (vegetables, pulses/legumes, cereals) is associated with a decreased risk of cardiovascular disease (139). In addition, high-fiber foods are low in energy density and high in volume and thus favor satiety and discourage over consumption (138-139); slow gastric emptying and delay absorption of glucose in small intestine, thereby improve postprandial glucose level (139).

Plant sterols are cholesterol-like molecules found in all plant foods, with the highest concentrations occurring in vegetable oils. They are absorbed only in trace amounts, but inhibit the absorption of intestinal cholesterol (146) and hence cholesterol absorption significantly reduced. Total bile acid excretion was also significantly increased with the consumption of plants sterols (147). As LDL cholesterol is a marker for cholesterol synthesis and absorption, a daily two milligrams of plant sterol supplementation for 4 weeks have shown to reduce LDL-c by ~10% (148).

The beneficial changes seen in the biochemical and anthropometric indices among fasters could be from a combination of restriction from all animal source foods/saturated fatty acids to a more plant based diet rich in fibers, antioxidants, and plant sterols; calorie restriction with the majority (84%) skipping breakfasts.

Regression analysis indicated that fasting is a significant determinant for most of the dependent variables. In addition, age of participants was positively related to end TC/HDL ratio and end blood glucose level, as indicated by Huma *et al* (54) that cardiovascular disease risks becomes increasingly common with advancing age and the risk is similar to impaired glucose metabolism (52). Smoking was positively related to end-LDL/HDL ratio which is similar to the finding by Craig *et al* (75) that in comparison to non-smokers, cigarettes smokers had significantly higher LDL and lower HDL cholesterol levels. In this study, the practice of eating fast foods was positively related to both end-HDL levels and end-waist circumference. Previous study (6) indicated that individuals with preference for fast foods were shown to consume fatty foods providing additional calories that are more likely to bring positive energy balance. In the contrary, calorie restricted diet shown a significant reduction in waist circumference (133).

Among the socio-economic factors, educational status was shown to relate negatively to end-TG level; and monthly income was also negatively related to body mass index. A higher level of education positively affects health, and negative relationship between education and lipid profiles were previously reported by Tadesco *et al* (67). Higher income level was similarly shown to provide a better access to healthy diets, leading to a lowering of health risks (58).

Types of oils used have shown statistically significant difference in end-triglyceride and end-blood glucose levels. The levels are significantly higher in palm users than niger seed oil and sunflower oil. The study by Mohammadifard *et al* (149) evaluating different oil types consumption in subjects over 40 days have shown a significant rise in triglyceride levels in hydrogenated vegetable oils (ghee) consumers; and such lipid level changes are associated with reduced insulin sensitivity (52). Hydrogenation produces undesirable *trans* fatty acids that have a negative impact on plasma lipoprotein profiles. Aini *et al* (150) indicated that Palm oil has similar physical characteristics to vegetable ghee. Unlike other vegetable oils, palm oil is rich in saturated fat (52).

## **7. STRENGTH AND LIMITATIONS OF THE STUDY**

The study assessed the effect of religious fasting to cardiovascular disease risk factors for the first time in Ethiopia, and therefore will serve as a baseline for future studies.

The study used participants selected through non-probability sampling method, and thus may affect the external validity of the findings. However, the study used a within-subject design that helped see changes before and after fasting. Besides a control group was also involved to control for environmental variations.

## **8. CONCLUSIONS AND RECOMMENDATIONS**

Periodic abstinence from animal source foods during the Ethiopian Orthodox Christians' Easter fasting period and shifts to strict vegetarians' diet like style by fasters in this study attributed to the observed biochemical and body measurement changes. Compared to controls, fasters presented decreased serum lipoproteins level and anthropometric measurements. Religious fasting has a substantial impact on health-related behaviors and shown to reduce cardiovascular diseases risk factors among fasters. However, whether these effects are sustained after the fasting and how multiple fasting throughout the year affect cardiovascular disease risk factors remains unknown.

Religious fasting practice has a positive effect on the reduction of cardiovascular disease risks; however, periodic abstinences from animal source foods may have an effect on micronutrients status and thus warrant further studies.

This study was not included other predictors like genetic (ApoE polymorphism) that affect serum lipids response to the dietary factors. Future studies need to consider genetic factors, inflammatory markers and plasma insulin levels. In addition, long term effects of oil types that are consumed by the population need to be studied with respect to lipid profiles, blood glucose, plasma insulin and inflammatory markers.

## REFERENCES

1. Tran A, Gelaye B, Girma B, Lemma S, Berhane Y, Bekele T, *et al.* Prevalence of Metabolic Syndrome among Working Adults in Ethiopia. *International Journal of Hypertension* 2011; 2011(Article ID 193719): 1-8. doi:10.4061/2011/193719
2. WHO. The global burden of disease: 2004 update. World Health Organization Press, 20 Avenue Appia, 1211 Geneva 27, Switzerland 2008; 1-160.
3. Wai WS, Dhami RS, Gelaye B, Girma B, Lemma S, Berhane Y, *et al.* Comparison of Measures of Adiposity in Identifying Cardiovascular Disease Risk Among Ethiopian Adults. *Obesity* 2011; 1-9. doi:10.1038/oby.2011.103. Accessed on November 5, 2014 from URL: [http://www.addiscontinental.edu.et/files/aciph\\_STEPS\\_Obesity\\_Paper.pdf](http://www.addiscontinental.edu.et/files/aciph_STEPS_Obesity_Paper.pdf)
4. Betre M, Kebede D, Kassaye M. Modifiable risk factors for coronary heart disease among young people in Addis Ababa. *East Afr Med J* 1997; 74(6):376-81. [Abstract]
5. Tesfaye F, Byass P, Wall S. Population based prevalence of high blood pressure among adults in Addis Ababa: uncovering a silent epidemic. *BMC Cardiovascular Disorders* 2009; 9:39. doi:10.1186/1471-2261-9-39
6. Amole OI, OlaOlorun DA, Odeigah OL. Body size and abnormal lipids among adult patients at the Baptist Medical centre, Ogbomoso, Nigeria. *Afr Health Sci* 2013; 13(1): 32–37. doi: 10.4314/ahs.v13i1.5
7. Fam KS, Waller DS, Zafer BE. The influence of religion on attitudes towards the advertising of controversial product. *Eur J Mark* 2004; 38(5/6): 537-555.
8. Belwal R, Tafesse Y. A study of the impact of orthodox Christians' fasting on demand for biscuits in Ethiopia. *African Journal of Marketing Management* 2010; 2(1): 10-17.
9. Tzima N, Pitsavos C, Panagiotakos DP, Skoumas J, Zampelas A, Chrysohoou C, *et al.* Mediterranean diet and insulin sensitivity, lipid profile and blood pressure levels, in

- overweight and obese people; The Attica study. *Lipids in Health and Disease* 2007; 6:22  
doi:10.1186/1476-511X-6-22
10. Delimaris A. Potential health of the periodic vegetarianism in Greek Orthodox Christian diet: a brief overview scientific chronicles. *Scientific Chronicles* 2012; 17(2): 79-82.
  11. Sarri KO, Tzanakis NE, Linardakis MK, Mamalakis GD, Kafatos AG. Effects of Greek Orthodox Christian Church fasting on serum lipids and obesity. *BMC Public Health* 2003; 3:16.
  12. Shatenstein B, Ghadirian P. Influences on diet, health behaviors and their outcome in select ethno cultural and religious groups. *Nutrition* 1998; 14(2):223-30 [Abstract]
  13. Friedlander Y, Kark JD, Kaufmann NA, Stein Y. Coronary heart disease risk factors among religious groupings in a Jewish population sample in Jerusalem. *Am J Clin Nutr* 1985; 42(3):511-21.
  14. Friedlander Y, Kark JD, Stein Y. Religious observance and plasma lipids and lipoproteins among 17-year-old Jewish residents of Jerusalem. *Prev Med* 1987; 16(1): 70-79.
  15. Maislos M, Khamavsi N, Assali A, Abou-Rabiah Y, Zvili I, Shany S. Marked increase in plasma high-density-lipoprotein cholesterol after prolonged fasting during Ramadan. *Am J Clin Nutr* 1993; 57:640-2
  16. Temizhan A, Donderici O, Ouz D, Demirbas B. Is there any effect of Ramadan fasting on acute coronary heart disease events? *Int J Cardiol* 1999; 70(2):149-53.
  17. Mansi KMS. Study the Effects of Ramadan Fasting on the Serum Glucose and Lipid Profile among Healthy. Jordanian Students. *American Journal of Applied Sciences* 2007; 4 (8): 565-569.
  18. Thannoun AM, Mahmoud ES. Effect of Fasting in Ramadan on Blood Glucose and Lipid Profile. *Mesopotamia J of Agric* 2010; 38 (2): 1-7.

19. Fraser GE. Diet as Primordial Prevention in Seventh-Day Adventists. *Prev Med* 1999; 29(6 Pt 2):S18-23.
20. Fraser GE. Associations between diet and cancer, ischemic heart disease, and all-cause mortality in non-Hispanic white California Seventh-day Adventists. *Am J Clin Nutr* 1999; 70(3 Suppl):532S-538S.
21. Arega R. (2009). Fasting in the Ethiopian Orthodox Church. Ethiopian Orthodox Tewahdo Church Sunday School Department – Mahibere Kidusan. Accessed on November 1, 2014 from URL: [http://www.eotcmk.org/site-en/index.php?option=com\\_content&task=view&id=56&Itemid=1](http://www.eotcmk.org/site-en/index.php?option=com_content&task=view&id=56&Itemid=1)
22. Trepanowski JF, Bloomer RJ. The impact of religious fasting on human health. *Nutrition Journal* 2010; 9:57: 1-9. doi:10.1186/1475-2891-9-57
23. Sarri K, Linardakis M, Codrington C, Kafatos A. Does the periodic vegetarianism of Greek Orthodox Christians benefit blood pressure? *Prev Med* 2007; 44(4):341-8. [Abstract]
24. Papadaki A, Vardavas C, Hatzis C, Kafatos A. Calcium, nutrient and food intake of Greek Orthodox Christian monks during a fasting and non-fasting week. *Public Health Nutr* 2008; 11:1022-9.
25. Sarri K, Bertias G, Linardakis M, Tsibinos G, Tzanakis N, Kafatos A. The effect of periodic vegetarianism on serum retinol and alpha-tocopherol levels. *Int J Vitam Nutr Res* 2009; 79:271-280. [Abstract]
26. Ethiopian Census Report 2007. Accessed on November 2, 2014 from URL: <http://www.csa.gov.et/>
27. Ethiopian Orthodox Church Information. Seven Official Liturgical Fasting Days, Ethiopian Orthodox Liturgical Calendar Holy Days. Accessed on November 2, 2014 from URL: [http://www.ethiopianorthodoxchurch.info/CalanderFastsFeastsDays.html# anchor\\_89](http://www.ethiopianorthodoxchurch.info/CalanderFastsFeastsDays.html# anchor_89)

28. Yu Z, Nissinen A, Vartiainen E, Song G, Guo Z, Zheng G, *et al* Associations between socioeconomic status and cardiovascular risk factors in an urban population in China. *Bulletin of the World Health Organization* 2000; 78(11):1296–1305.
29. Getahun W, Gedif T, Tesfaye F. Regular Khat (*Catha edulis*) chewing is associated with elevated diastolic blood pressure among adults in Butajira, Ethiopia: a comparative study. *BMC Public Health* 2010; 10:390.
30. Fenske JN, Van Harrison R, Jackson EJ, Marcelino MA. UMHS Lipid Therapy Guideline Update, May 2014. Accessed July 19, 2015 from, <http://www.med.umich.edu/1info/FHP/practiceguides/lipids/lipidsupdate.pdf>
31. Wong ND. Epidemiological studies of CHD and the evolution of preventive cardiology. *Nature Reviews Cardiology* 2014; 11: 276–289. doi:10.1038/nrcardio.2014.26
32. Calle EE, Rodriguez C, Walker-Thurmond K. Over weight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *N Engl J Med* 2003; 348: 1625-1638.
33. Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, *et al*. *Circulation* 2004; 110: 2952-2967. doi: 10.1161/01.CIR.0000145546.97738.1E
34. Rabkin SW, Chen Y, Leiter L. Risk factor correlates of body mass index. Canadian Heart Health Surveys Research Group. *CMAJ* 1997; 157(suppl1): S26-31.
35. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. *World Health Organ Tech Rep Ser* 2000; 894:1-253. i-xii.
36. Despres JP, Lemieux I, Prudhomme D. Treatment of obesity: need to focus on high risk abdominally obese patients. *BMJ* 2001; 322: 716-20.
37. Menke A, Muntner P, Wildman RP, Reynolds K, He J. Measures of adiposity and cardiovascular disease risk factors. *Obesity* 2007; 15:785–795.

38. Lee CM, Huxley RR, Wildman RP, Woodward M. Indices of abdominal obesity are better discriminators of cardiovascular risk factors than BMI: a meta-analysis. *J Clin Epidemiol* 2008; 61:646–653. [Abstract]
39. Song-Ming DU, Guan-Sheng MA, Yan-Ping LI, Fang H, Xiao-Qi HU, Yang X, *et al.* Relationship of Body Mass Index, Waist Circumference and Cardiovascular Risk Factors in Chinese Adult. *Biomedical and Environmental Sciences* 2010; 23: 92-101.
40. Hsieh SD, Yoshinaga H, Muto T. Waist-to-height ratio, a simple and practical index for assessing central fat distribution and metabolic risk in Japanese men and women. *Int J Obes Relat Metab Disord* 2003; 27(5):610-6.
41. Ardern CI, Katzmarzyk PT, Janssen I, Ross R. Discrimination of health risk by combined body mass index and waist circumference. *Obes Res* 2003; 11(1):135-42.
42. Janssen I, Katzmarzyk PT, Ross R. Body mass index, waist circumference, and health risk: evidence in support of current National Institutes of Health guidelines. *Arch Intern Med* 2002; 162(18):2074-9.
43. National Institute of Health. The Practical Guide. Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. NHLBI Obesity Education Initiative. NIH Publication Number 00-4084. October 2000; 1-97. Accessed on November 5, 2014 from URL: [http://www.nhlbi.nih.gov/files/docs/guidelines/prctgd\\_c.pdf](http://www.nhlbi.nih.gov/files/docs/guidelines/prctgd_c.pdf)
44. WHO. Waist Circumference and Waist-Hip Ratio Report of a WHO Expert Consultation Geneva, December 2008b. Accessed on November 5, 2014 from URL: [http://whqlibdoc.who.int/publications/2011/9789241501491\\_eng.pdf](http://whqlibdoc.who.int/publications/2011/9789241501491_eng.pdf)
45. Whitney E., Rolfes SR. *Understanding nutrition*. 11<sup>th</sup> Edition. 2008, Thomson Learning, Inc., ISBN-13: 978-0-495-11669-1, 10 Davis Drive, Belmont, CA 94002-3098, USA. Page 139-169.
46. Daniels TF, Killinger KM, Michal JJ, Wright Jr. RW, Jiang Z. Lipoproteins, cholesterol homeostasis and cardiac health. *Int J Biol Sci* 2009; 5(5):474-488. doi:10.7150/ijbs.5.474.

47. Millan J, Pinto X, Munoz A, Zuniga M, Rubies-Prat J, Pallardo LF, *et al.* Lipoprotein ratios: Physiological significance and clinical usefulness in cardiovascular prevention. *Vasc Health Risk Manag* 2009; 5: 757–765.
48. Nigam PK. Serum Lipid Profile: Fasting or Non-fasting? *Ind J Clin Biochem* 2011; 26(1): 96–97. DOI10.1007/s12291-010-0095-x
49. Alvarez C, Ramos A. Lipids, lipoproteins and apolipoproteins in serum during infection. *Clin Chem* 1986; 32: 142–145.
50. Nawaz H, Comerford BP, Njike VY, Dhond AJ, Plavec M, Katz, DL. Repeated serum lipid measurements during the peri-hospitalization period. *Am J Cardiol* 2006; 98(10):1379-82.
51. Campose H, Khoo C, Sacks FM. Diurnal and acute pattern of postprandial apolipoprotein B-48 in VLDL, IDL and LDL from normolipidemic human. *Atherosclerosis* 2005; 181: 345–51.
52. NCEP: Third report of the national cholesterol education program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III) final report. *Circulation* 2002; 106(25):3143-3421.
53. Getahun W, Gedif T, Tesfaye F. Regular Khat (*Catha edulis*) chewing is associated with elevated diastolic blood pressure among adults in Butajira, Ethiopia: a comparative study. *BMC Public Health* 2010; 10:390.
54. Huma S, Tariq R, Amin F, Mahmood KT. Modifiable and Non-modifiable predisposing Risk Factors of Myocardial Infarction - A Review. *J Pharm Sci & Res* 2012; 4(1): 1649-1653.
55. Joint Editorial Statement Diabetes Mellitus: A Major Risk Factor for Cardiovascular Disease. A Joint Editorial Statement by the American Diabetes Association; the National Heart, Lung, and Blood Institute; the Juvenile Diabetes Foundation International; the

- National Institute of Diabetes and Digestive and Kidney Diseases; and the American Heart Association. *Circulation* 1999; 100: 1132-1133. doi: 10.1161/01.CIR.100.10.1132.
56. Alberti KG, Zimmet P, Shaw J. Metabolic syndrome – a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabet Med* 2006; 23:469–480.
  57. World Health Organization. Global Atlas on Cardiovascular Disease Prevention and Control. Mendis S, Puska P, Norrving B. Editors. World Health Organization (in collaboration with the World Heart Federation and World Stroke Organization), Geneva 2011.
  58. Samuel P, Antonisamy B, Raghupathy P, Richard J, and Fall CHD. Socio-economic status and cardiovascular risk factors in rural and urban areas of Vellore, Tamilnadu, South India. *International Journal of Epidemiology* 2012; 41: 1315–1327. doi:10.1093/ije/dys001
  59. Wamala SP, Wolk A, Schenck-Gustafsson K, Orth-Gomer K. Lipid profile and socioeconomic status in healthy middle aged women in Sweden. *Journal of Epidemiology and Community Health* 1997; 51: 400-407.
  60. Lawlor DA, Batty GD, Morton SMB, Clark H, MSc, Macintyre S, and Leon DV. Childhood Socioeconomic Position, Educational Attainment, and Adult Cardiovascular Risk Factors: The Aberdeen Children of the 1950s Cohort Study. *Am J Public Health* 2005; 95(7): 1245–1251. doi: 10.2105/AJPH.2004.041129
  61. Batty GD, Leon DA. Socio-economic position and coronary heart disease risk factors in children and young people: Evidence from UK epidemiological studies. *European Journal of Public Health* 2002; 12: 263–272.
  62. Gupta R, Gupta VP, Ahluwalia NS. Educational status, coronary heart disease, and coronary risk factor prevalence in a rural population of India. *BMJ* 1994; 309:1332. doi: <http://dx.doi.org/10.1136/bmj.309.6965.1332>

63. Reddy KS, Prabhakaran D, Jeemon P, Thankappan KR, Joshi P, Chaturved V, *et al.* Educational status and cardiovascular risk profile in Indians. *Proceedings of the National Academy of Sciences* 2007; 104(41):16263-16268.
64. Ebrahim S, and Smeeth L. Non-communicable diseases in low and middle-income countries: a priority or a distraction? *International Journal of Epidemiology* 2005; 34:961–966. doi:10.1093/ije/dyi188
65. Kinra S, Bowen LJ, Lyngdoh T, Prabhakaran D, Reddy KS, Ramakrishnan L, *et al.* Sociodemographic patterning of non-communicable disease risk factors in rural India: a cross sectional study. *BMJ* 2010; 341:c4974. doi:10.1136/bmj.c4974
66. Popkin BM. The Shift in Stages of The Nutrition Transition in the Developing World Differs from Past Experiences? *Public Health Nutrition* 2002; 5(1A):205–214. DOI:10.1079/PHN2001295
67. Tedesco MA, Di-Salvo G, Caputo S, Natale F, Ratti G, Iarussi D, *et al.* Educational level and hypertension: how socioeconomic differences condition Health care. *J Hum Hypertens* 2001; 15:727–731.
68. Sharma M, Mahna R. Obesity, Metabolic Syndrome and Physical Activity in Indian Adults. *J Metabolic Synd* 2012; 1:114.
69. Cuhadar S, Atay A, Saglam G, Koseoglu M, Cuhadar L. Cardiovascular risk factors in young male adults: impact of physical activity and parental education. *Central Asian Journal of Global Health* 2013; 2(1). DOI 10.5195/cajgh.2013.44. Accessed on Nov 30, 2014 from URL: <http://cajgh.pitt.edu/ojs/index.php/cajgh/article/download/44/63>
70. Suhrcke M., Nugent, R.A., Stuckler, D. Rocco, L. *Chronic Disease: An Economic Perspective*. London: Oxford Health Alliance 2006. Retrieved May 20, 2015 from: <http://www.sehn.org/tccpdf/Chronic%20disease%20economic%20perspective.pdf>

71. Al-Motarreb A, Briancon S, Al-Jaber N, Al-Adhi B, Al-Jailani F, Salek MS, *et al.* Khat chewing is a risk factor for acute myocardial infarction: a case-control study. *British Journal of clinical pharmacology* 2005; 59(5): 574-581.
72. Tesfaye F, Byass P, Berhane Y, Bonita R, Wall S. Association of smoking and khat (*Catha edulis* Forsk) use with high blood pressure among adults in Addis Ababa, Ethiopia, 2006. *Prev Chronic Dis* 2008; 5(3): 1-10. Accessed on May 12, 2015 from: [http://www.cdc.gov/pcd/issues/2008/jul/07\\_0137.htm](http://www.cdc.gov/pcd/issues/2008/jul/07_0137.htm).
73. Tesfaye F, Byass P, Wall S. Population based prevalence of high blood pressure among adults in Addis Ababa: uncovering a silent epidemic. *BMC Cardiovascular Disorders* 2009; 9:39. doi:10.1186/1471-2261-9-39
74. Papathanasiou G, Mamali A, Papafloratos S, Zerva E. Effects of Smoking on Cardiovascular Function: The Role of Nicotine and Carbon Monoxide. *Health Science Journal* 2014; 8(2): 274-290.
75. Craig WY, Palomaki GE, Haddow JE: Cigarette smoking and serum lipid and lipoprotein concentrations: an analysis of published data. *BMJ* 1989; 298:784-788.
76. Karatzi K, Papamichael C, Karatzis E. Acute smoke-induced endothelial dysfunction is more prolonged in smokers than in non-smokers. *Intern J Cardio* 2007; 120: 404–406.
77. Mukamal KJ, Rimm EB. Alcohol's effects on the risk for coronary heart disease. *Alcohol Res Health* 2001; 25(4):255-261.
78. Facchini F, Chen YD, Reaven GM. Light-to-moderate alcohol intake is associated with enhanced insulin sensitivity. *Diabetes Care* 1994; 17(2):115-9.
79. Kim SH, Abbasi F, Lamendola C, Reaven GM. Effect of Moderate Alcoholic Beverage Consumption on Insulin Sensitivity in Insulin Resistant, Non-diabetic individuals. *Metabolism* 2009; 58(3): 387–392. doi: 10.1016/j.metabol.2008.10.013

80. AIM Feature. Social aspects and communication regarding alcohol and health via national drinking guidelines. Retrieved on May 17, 2015 from, <http://www.aim-digest.com/digest/members%20over%20yr/guidelinesHC.pdf>
81. Devaux M, Sassi F. Alcohol consumption and harmful drinking: Trends and social disparities across OECD countries, OECD Health Working Papers, No. 79, OECD Publishing, Paris. 2015; Page 15. Accessed on May 13, 2015, from: <http://dx.doi.org/10.1787/5js1qwkwz2p9s-en>
82. Rehm J, Eschmann S. Global monitoring of average volume of alcohol consumption. *Sozial-und Praventivmedizin* 2002; 47(1):48-58.
83. Rehm J, Room R, Monteiro M, Gmel G, Graham K, Rehn N, *et al.* Alcohol use. In Ezzati M, Lopez AD, Rodgers A, Murray CJL, editors. *Comparative Quantification of Health Risks: Global and Regional Burden of Diseases Attributable to Selected Major Risk Factors*, World Health Organization, Geneva; 2004.
84. Sigal RJ, Armstrong MJ, Colby P, Kenny GP, Plotnikoff RC, Reichert SM, *et al.* Physical Activity and Diabetes. *Can J Diabetes* 2013; 37: S40-S44.
85. Thompson PD, Crouse SF, Goodpaster B, Kelley D, Monya, N, Pescatello L. The acute versus the chronic response to exercise. *Med Sci Sports Exerc* 2001; 33(6 Suppl): S438-45; discussion S452-3.
86. Chudyk A, Petrella RJ. Effects of Exercise on Cardiovascular Risk Factors in Type 2 Diabetes: A meta-analysis. *Diabetes Care* 2011; 34(5): 1228–1237.
87. Warburton DER, Nicol CW, Bredin SSD. Health benefits of physical activity: the evidence. *CMAJ* 2006; 174 (6): 801-809. doi: 10.1503/cmaj.051351
88. Tabei SMB, Senemar S, Safari B, Ahmadi Z, Haqparast S. Non-modifiable Factors of Coronary Artery Stenosis in Late Onset Patients with Coronary Artery Disease in Southern Iranian Population. *J Cardiovasc Thorac Res* 2014; 6(1): 51-55. doi: 10.5681/jcvtr.2014.010

89. Real JT, Chaves FJ, Martinez-Usó I, Garcia-Garcia AB, Ascaso JF, Carmena R. Importance of HDL cholesterol levels and the total/HDL cholesterol ratio as a risk factor for coronary heart disease in molecularly defined heterozygous familial hypercholesterolaemia. *European Heart Journal* 2001; 22: 465–471. doi:10.1053/euhj.2000.2408
90. Wang J, Staessen JA. Genetic polymorphisms in the renin–angiotensin system: relevance for susceptibility to cardiovascular disease. *Eur J Pharmacol* 2000; 410 (2-3): 289-302.
91. Wallaert C, Babin PJ. Age-related, sex-related, and seasonal changes of plasma lipoprotein concentrations in trout. *J Lipid Res* 1994; 35: 1619-1633.
92. Kelly GS. Seasonal Variations of Selected Cardiovascular Risk Factors. *Alternative Medicine Review* 2005;10(4):307-320.
93. Ockene IS, Chiriboga DE, Stanek EJ, Harmatz MG, Nicolosi R, Saperia G, *et al.* Seasonal Variation in Serum Cholesterol Levels Treatment Implications and Possible Mechanisms. *Arch Intern Med* 2004; 164:863-870.
94. Robinson D, Hinohara S, Bevan EA, Takahashi T. Seasonal variation in serum cholesterol levels in health screening populations from the U.K. and Japan. *Journal of Medical Systems* 1993; 17(3): 207-211.
95. Fuller JH, Grainger SL, Jarrett RJ, Keen H. Possible seasonal variation of plasma lipids in a healthy population. *Clinica Chimica Acta* 1974; 52(3): 305–310.
96. Donahoo WT, Jensen DR, Shepard TY, Eckel RH. Seasonal Variation in Lipoprotein Lipase and Plasma Lipids in Physically Active, Normal Weight Humans. *The Journal of Clinical Endocrinology & Metabolism* 2000; 85(9): 3065-3068.
97. National Institute for Health and Clinical Excellence (NICE). 3rd Ed. NICE, Mid City Place, 71 High Holborn, London WC1V 6NA. 2012; 185-195. Retrieved November 22, 2014, from: <http://www.nice.org.uk/article/pmg4/resources/non-guidance-methods-for-the-development-of-nice-public-health-guidance-third-edition-pdf>

98. Brown KW, Cozby CP, Kee DW, Worden PE. *Research Methods in Human Development*. 2<sup>nd</sup> ed, Mayfield Publishing Company, 1280 Villa Street, Mountain View, California 94041. 1999; 216-227. Accessed on November 10, 2014, from URL: <https://www.csusm.edu/psychology/docs/ResearchMethodsInHumanDevelopment.pdf>
99. Ellis MV. Repeated Measures Designs. *The Counseling Psychologist* 1999; 27(4): 552-578.
100. Cunningham JB, McCrum-Gardner E (2007) Power, effect and sample size using GPower: practical issues for researchers and members of research ethics committees. *Evidence Based Midwifery* 2007; 5(4): 132–6.
101. McCrum-Gardner, E. Sample size and power calculations made simple. *International Journal of Therapy and Rehabilitation*, January 2010; 17(1): 10-14.
102. GPower Version 3.1.9.2. Program written by Franz Faul, Universitak Kiel, Germany. Copyright (C) 1992-2014. Accessed on November 12, 2014 from URL: [http://www.gpower.hhu.de/fileadmin/redaktion/Fakultaeten/Mathematisch-Naturwissenschaftliche\\_Fakultaet/Psychologie/AAP/gpower/GPowerWin\\_3.1.9.2.zip](http://www.gpower.hhu.de/fileadmin/redaktion/Fakultaeten/Mathematisch-Naturwissenschaftliche_Fakultaet/Psychologie/AAP/gpower/GPowerWin_3.1.9.2.zip)
103. Thalheimer W, Cook S. How to calculate effect sizes from published research articles: A simplified methodology. *A Work-Learning Research Publication*. 2002;1-9. Retrieved November 13, 2014 from [http://www.bwgriffin.com/gsu/courses/edur9131/content/Effect\\_Sizes\\_pdf5.pdf](http://www.bwgriffin.com/gsu/courses/edur9131/content/Effect_Sizes_pdf5.pdf)
104. Sullivan GM, Feinn R. Using Effect Size or Why the P Value Is Not Enough. *Journal of Graduate Medical Education*. 2012 September; 279 – 282. Doi:<http://dx.doi.org/10.4300/JGME-D-12-00156.1> Accessed on November 12, 2014 from: <http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3444174/pdf/i1949-8357-4-3-279.pdf>
105. Cohen J. A power primer. *Psychological Bulletin*. 1992; 112(1): 155-159. Retrieved November 13, 2014 from URL: <http://drsmorey.org/bibtex/upload/Cohen:1992.pdf>

106. Sarraf-Zadegan N, Atashi M, Naderi GA, Baghai AM, Asgary S, Fatehifar MR, *et al.* The Effect of Fasting in Ramadan on the Values and Interrelations between Biochemical, Coagulation and Hematological Factors. *Ann Saudi Med* 2000; 20(5-6):377-381.
107. Ha RR, Ha JC. *Integrative Statistics for the Social and Behavioral Sciences, Introduction to Hypothesis Testing, Two-Sample Tests*, SAGE Publications, Inc, ISBN: 141298744X. 2012; 145-182. Accessed on November 10, 2014, from: [http://www.sagepub.com/upm-data/40287\\_Chapter9.pdf](http://www.sagepub.com/upm-data/40287_Chapter9.pdf)
108. World Health Organization. *Chronic diseases and health promotion. STEPwise approach to surveillance (STEPS). STEPS Manual*. Geneva, World Health Organization; 2008. Accessed on November 3, 2014, from URL: <http://new.paho.org/hq/dmdocuments/2009/STEPSmanual.pdf>
109. Yohannes T, Melak F, Siraj K. Preparation and physicochemical analysis of some Ethiopian traditional alcoholic beverages. *African Journal of Food Science* 2013; 7(11): 399-403.
110. Tekle-Haimanot R, Haile G. Chronic Alcohol Consumption and the Development of Skeletal Fluorosis in a Fluoride Endemic Area of the Ethiopian Rift Valley. *Journal of Water Resource and Protection* 2014; 6: 149-155.
111. Satalic Z, Baric IC, Cecic I, Keser I. Short food frequency questionnaire can discriminate inadequate and adequate calcium intake in Croatian postmenopausal women. *Nutrition Research* 2007; 27:542–547. doi: 10.1016/j.nutres.2007.07.001
112. Johansson G. *Dietary assessments Use, design concepts, biological markers, pitfalls and validation*. Institutionen for kostvetenskap, Umea universitet, 2006; page 15-16. Retrieved on May 12, 2015 from: <http://www.diva-portal.org/smash/get/diva2:464612/FULLTEXT02>
113. Brantsaeter AL. *Design, Use and Interpretation of Food Frequency Questionnaires*. Norwegian Institute of Public Health 2011. Retrieved on July 16, 2015, from

<http://www.birmingham.ac.uk/Documents/college-les/gees/inflame/consortium/atcs/food-frequency-question-brantsaeter.pdf>

114. Wrieden W, Peace H, Armstrong J, Barton K. A short review of dietary assessment methods used in National and Scottish Research Studies. Briefing Paper Prepared for: Working Group on Monitoring Scottish Dietary Targets Workshop, September 2003. Retrieved on June 13, 2015 from: <http://www.food.gov.uk/sites/default/files/multimedia/pdfs/scotdietassessmethods.pdf>
115. Goulet J, Nadeau G, Lapointe A, Lamarche B, Lemieux S. Validity and reproducibility of an interviewer-administered food frequency questionnaire for healthy French-Canadian men and women. *Nutrition Journal* 2004; 3:13. doi:10.1186/1475-2891-3-13
116. FAO/EU. Guidelines for measuring household and individual dietary diversity. Kennedy G, Ballard T and Dop M. Editors. Office of Knowledge Exchange, Research and Extension, FAO, Viale delle Terme di Caracalla, 00153 Rome, Italy. 2013; Page 5-10. Retrieved on June 13, 2015 from: <http://www.fao.org/3/a-i1983e.pdf>
117. Charlton KE, Steyn K, Levit N, Jonathan D, Zulu J, Nel J. Development and validation of a short questionnaire to assess sodium intake. *Public Health Nutrition* 2008; 11 (1): 83-94.
118. Hyson DA, Schneeman BO, Davis PA. Almonds and Almond Oil Have Similar Effects on Plasma Lipids and LDL Oxidation in Healthy Men and Women. *J Nutr* 2002; 132: 703–707.
119. Ross R, Berentzen T, Bradshaw AJ, Janssen I, Kahn SK, Katzmarzyk PT, *et al.* Does the relationship between waist circumference, morbidity and mortality depend on measurement protocol for waist circumference? *Official Journal on the International Chair on Cardiometabolic Risk* 2008; 1(2): 20-22.
120. Appel LJ, Miller ER, Charleston J. Improving the measurement of blood pressure: is it time for regulated standards? *Ann Intern Med* 2011; 154(12):838-40.

121. Karnath B. Sources of Error in Blood Pressure Measurement. Review of Clinical Signs. Turner White Communications Inc., Wayne, PA. 2002; 33-37. Retrieved on May 23, 2015 from: [http://www.turner-white.com/pdf/hp\\_mar02\\_error.pdf](http://www.turner-white.com/pdf/hp_mar02_error.pdf)
122. Deeg M. Variations in Lipid Values. Lipid Topics 2006; 1(3). Retrieved on May 23, 2015 from: [http://www.ptsdiagnostics.com/uploads/2/6/2/8/26289179/tb003\\_variations\\_in\\_lipid\\_values\\_0606\\_4.pdf](http://www.ptsdiagnostics.com/uploads/2/6/2/8/26289179/tb003_variations_in_lipid_values_0606_4.pdf)
123. Owiredu WKBA, Amegatcher G, Amidu N. Precision and Accuracy of Three Blood Glucose Meters: Accu-Chek Advantage, One Touch Horizon and Sensocard. Journal of Medical Sciences, 2009; 9:185-193. Accessed on November 5, 2014 from URL: [http://docsdrive.com/pdfs/ans\\_inet/jms/2009/185-193.pdf](http://docsdrive.com/pdfs/ans_inet/jms/2009/185-193.pdf)
124. Abebe M, Kinde S, Belay G, Gebreegziabxier A, Challa F, Gebeyehu T, *et al.* Antiretroviral treatment associated hyperglycemia and dyslipidemia among HIV infected patients at Burayu Health Center, Addis Ababa, Ethiopia: a cross-sectional comparative study. BMC Research Notes 2014; 7:380. doi:10.1186/1756-0500-7-380
125. Ogedegbe G, Pickering T. Principles and techniques of blood pressure measurement. *Cardiol Clin* 2010; 28(4): 571–586. doi: 10.1016/j.ccl.2010.07.006
126. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurements. *Lancet* 1986; 1: 307-310.
127. Physical Activity and Health: A report of the Surgeon General. Washington DC: US department of health and human services. Accessed on June 12, 2015, from: URL <http://www.surgeongeneral.gov/library/disabilities/calltoaction/whatitmeanstoyou.html#choose>
128. Barnard ND, Scialli AR, Bertron P, Hurlock D, Edmonds K, Talev L. Effectiveness of a low-fat vegetarian diet in altering serum lipids in healthy premenopausal women. *Am J Cardiol* 2000; 85(8):969-72.

129. Goff LM, Bell JD, So P, Dornhorst A, Frost GS. Veganism and its relationship with insulin resistance and intramyocellular lipid. *Eur J Clin Nutr* 2005; 59:291–298.
130. Nieman DC. Dietary status of Seventh-Day Adventist vegetarian and non-vegetarian elderly women *J Am Diet Assoc* 1989; 89(12):1763-9.
131. Toohey ML. Cardiovascular disease risk factors are lower in African-American vegans compared to lacto-ovo-vegetarians *J Am Coll Nutr* 1998; 17(5):425-34.
132. Ornish D. Was Dr Atkins right? *J Am Diet Assoc* 2004; 104:537–542.
133. Kahleova H, Matoulek M, Malinska H, Oliyarnik O, Kazdova L, Neskudla T, *et al.* Vegetarian diet improves insulin resistance and oxidative stress markers more than conventional diet in subjects with Type 2 diabetes. *Diabet Med* 2011; 28(5): 549–559. doi: 10.1111/j.1464-5491.2010.03209.x
134. Asztalos B, Lefevre M, Wong L, Foster TA, Tulley R, Windhauser M, *et al.* Differential response to low-fat diet between low and normal HDL-cholesterol subjects. *J Lipid Res* 2000; 41: 321–328.
135. Van-Ooijena AM, Lichtenbelt WDM, van Steenhovenb AA, Westerterp KR. Seasonal changes in metabolic and temperature responses to cold air in humans. *Physiology & Behavior* 2004; 82: 545–553
136. Marti-Soler H, Gubelmann C, Aeschbacher S, Alves L, Bobak M, Bongard V, *et al.* Seasonality of cardiovascular risk factors: an analysis including over 230,000 participants in 15 countries. *Heart* 2014; 0:1–7. doi:10.1136/heartjnl-2014-305623
137. Haddad EH, Berk LS, Kettinger JD, Hubbard RW, Peters WR. Dietary intake and biochemical, hematologic, and immune status of vegans compared with nonvegetarians. *Am J Clin Nutr* 1999; 70(3 Suppl): 586S-593S.
138. Wharton S, Sharma AM, Lau DCW. Weight management in diabetes. *Can J Diabetes* 2013; 37:S88-S86.

139. Dworatzek PD, Arcudi K, Gougeon R, Husein N, Sievenpiper JL, Williams SL. Nutrition Therapy. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. *Can J Diabetes* 2013; 37: S45-S55.
140. Appleby PN, Davey GK, Key TJ. Hypertension and blood pressure among meat eaters, fish eaters, vegetarians and vegans in EPIC–Oxford. *Public Health Nutrition* 2002; 5(5): 645–654.
141. Law M. Dietary fat and adult diseases and the implications for childhood nutrition: an epidemiologic approach. *Am J Clin Nutr* 2000; 72(5 Suppl):1291S-1296S.
142. Shahar DR, Yerushalmi N, Lubin F, Froom P, Shahar A, Kristal-Boneh E. Seasonal variations in dietary intake affect the consistency of dietary assessment. *Eur J Epidemiol* 2001; 17(2):129-33.
143. Kafatos A. Mediterranean diet of Crete: foods and nutrient content. *J Am Diet Assoc* 2000; 100(12):1487-93.
144. Bazzano LA, Thompson AM, Tees MT, Nguyen CH, Winham DM. Non-Soy Legume Consumption Lowers Cholesterol Levels: A Meta-Analysis of Randomized Controlled Trials. *Nutr Metab Cardiovasc Dis* 2011; 21(2): 94–103. doi:10.1016/j.numecd.2009.08.012
145. Sievenpiper JL, Kendall CW, Esfahani A, Wong JM, Carleton AJ, Jiang HY, *et al.* 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* 2009; 52(8):1479-95. doi: 10.1007/s00125-009-1395-7.
146. Harcombe Z, Baker JS. Plant sterols lower cholesterol, but increase risk for coronary heart disease. *Online J Biol Sci* 2014; 14(3): 167-169. DOI: 10.3844/ojbsci.2014.167.169
147. Carr TP, Cornelison RM, Illston BJ, Stuefer-Powell CL, Gallaher DD. Plant sterols alter bile acid metabolism and reduce cholesterol absorption in hamsters fed a beef-based diet.

Nutrition Research 2002; 22(6): 745–754. DOI: [http://dx.doi.org/10.1016/S0271-5317\(02\)00389-5](http://dx.doi.org/10.1016/S0271-5317(02)00389-5)

148. Malina DMT, Fonseca FA, Barbosa SA, Kasmah SH, Machado VA, Franca CN, *et al.* Additive effects of plant sterols supplementation in addition to different lipid-lowering regimens. *Journal of Clinical Lipidology* 2015; 9(4): 542–552. DOI: <http://dx.doi.org/10.1016/j.jacl.2015.04.003>
149. Mohammadifard N, Hosseini M, Sajjadi F, Maghroun M, Boshtam M, Nouri F. Comparison of effects of soft margarine, blended, ghee, and unhydrogenated oil with hydrogenated oil on serum lipids: A randomized clinical trial. *ARYA Atheroscler* 2013; 9(6): 363-371.
150. Aini IN, Hanirah H, Maimon CH, Zawiah S, Man YBC. Physico-Chemical Properties and Quality of Palm-Based Vegetable Ghee. *Sains Malaysiana* 2010; 39(5): 791–794.

ANNEXES

Annex 1: Letter of Ethical Clearance from school of public health, Addis Ababa University



**ADDIS ABABA UNIVERSITY**  
**College of Health Sciences**  
**School of Public Health**  
**Ethical Clearance Form**

Version 10.Feb.2015

Date: / 10 / 02 / 2015 /  
 Ref.No. SPH/596/07

Project number /011/

Date of approval (D/M/Y) / <u>10/2/15</u> /	
Project Title: Assessment of the Effects of Ethiopian Orthodox Christians Fasting on Serum Lipids and Anthropometric Measurements	
Name of PI: Habtamu Guja	Phone Number <u>+251-911 912460</u>
Institution	School of Public Health
Department	
Decision of Research and Ethics Committee:	<input checked="" type="checkbox"/> Approved <input type="checkbox"/> Approved with Recommendation <input type="checkbox"/> Resubmission <input type="checkbox"/> Disapproved
Valid until	Feb. 10, 2015 - May 10, 2015

Dean, School of Public Health

Signature 

Date 10/2/15



## **Annex 2: Participant’s Information Sheet and Consent Form**

English version information sheet and consent form for participating in the study, “Assessment of the Effects of Ethiopian Orthodox Christian fasting on serum lipids and Anthropometric Measurements” Addis Ababa, Ethiopia

### **A. Information Sheet**

Identification Code: \_\_\_\_\_

Hello, how are you?

My name is Habtamu Guja, I am a Masters Student in AAU, college of health sciences, school of public health, as a principal investigator conducting this study.

The objective of this study is to assess the effects of Ethiopian Orthodox Christians Easter fasting on serum lipids profile and on the anthropometric indices of obesity.

Your cooperation and willingness to participate in the interview, body measurement & providing blood sample is very helpful in identifying the effect of fasting on those dependant variables. I assure you that all information that you give will be kept strictly confidential. Your participation is voluntary and you are not obliged to answer any question you do not want to answer. If you are not still comfortable with interview, please be free to stop me any time you like there is no harm if you don’t answer the questions and no special benefit you get except knowing your lipid profile, blood glucose level, blood pressure and BMI status at the end of the study. If you are willing to participate, the interview questions and measurements will take 30- 40 minutes. I would like to interview you few questions regarding socio-demographic characteristics; health behavior and fasting experience; health related and medication use; and dietary assessment. Body measurements (height, weight, waist and hip), blood pressure and blood samples will be collection twice at pre and of end fasting for biochemical measurements.

We would be thankful if you spend some time with us answering questions.

1. If yes, Name of interviewer\_\_\_\_\_ Signature\_\_\_\_\_
2. If not, skip to the other participant

For more information and question here is the contact address of investigator:

Habtamu Guja: Tele: 0911 912460; e-mail: habtamugujab@yahoo.com

## **B. Consent form**

I \_\_\_\_\_ am informed on study to be conducted by Masters Student in AAU, college of health sciences school of public health on Assessment of the Effects of Ethiopian Orthodox Christian fasting on serum lipids and Anthropometric Measurements. The objective of the study is to assess the effects of Ethiopian Orthodox Christians Easter fasting on serum lipids profile and on the anthropometric indices of obesity and participation to this study is voluntary no obligation to answer any questioner there is no harm by not answering the questions and no special benefit by answering the question except knowing my health status at the end of the study and also the interview will take 30- 40 minutes. I heard all the information mentioned above and willing to participate in the interview.

1. Signature of the study subject agreed to participant as per the above stated information

\_\_\_\_\_

2. Name of interviewer \_\_\_\_\_ Signature \_\_\_\_\_

**በጥናትና ምርምር ላይ ለመሳተፍ ፈቃደኝነትን መጠየቃያ የስምምነት ቅጽ፤**

እንደምን ነህ/ሽ?

እኔ ሀብታሙ ጉጃ እባላለሁ፤

በአዲስ አበባ ዩኒቨርሲቲ የጤና ሳይንስ ኮሌጅ በህብረተሰብ ጤና ትምህርት ውስጥ ለሚያደርገው የማስተርስ ዲግሪ የማሟያ የጥናትና የምርመር ስራ እየሰራሁ ነው ። የኢትዮጵያ ኦርቶዶክስ ተዋህዶ ቤተክርስቲያን ዓብይ ፆም በጸሐፊዎች የደም የቅባት መጠን እና የሰውነት ክብደትና ግዝፈት መጠን ልኬት ላይ ያለውን አስተዋፅኦ ለማጥናት በሚደረገው በዚህ ጥናት ተሳታፊ እንዲሆኑ የእርሶን ፈቃደኝነት በአክብሮት እየጠየኩ፤ የእርሶ ፈቃደኝነትና ቀና ትብብር ጥናቱ ለማጎበረሰባችን የአመጋገብ ሥርዓት እና ጤና መማሻሻል እንደ ግብዓት ትልቅ ጠቀሜታ እንደሚያበረክት በመግለጽ ነው።

በመሆኑም እርስዎ በምርምር ስራው ላይ ለመሳተፍ ፈቃደኛ ከሆኑ ፤ የሥነ - ምግብ ባለሙያው ለሚጠይቁት ጥያቄ የበኩልዎን መረጃ በመስጠት፤ የሰውነት ክብደትና ግዝፈት መጠን ልኬት እና የደም ናሙና በመስጠት እገዛ እንዲያደርጉ በአክብሮት እጠይቃለሁ።

የሚሰጡት ምላሽ እና የደም ናሙና ከጥናትና ምርምር ስራው ውጪ ለሌላ አገልግሎት አይውልም። በመጠይቁ ላይ ከተመለከቱት ጥያቄዎች መካከል መመለስ የማይፈልጉት ጥያቄ ካለ አለመመለስ ይችላሉ። በጥናቱ ላይ በመሳተፍዎ የራስዎን የግል የጤና ሁኔታ በጥናቱ መጨረሻ ላይ ከማወቅ ውጪ የተለዩ ጥቅም አያገኙም።

ከላይ በተገለጸውን ቅጽ መስማማትዎን ፊርማዎ ያረጋግጡ \_\_\_\_\_

መረጃው የተሰጠበት ቀን \_\_\_\_\_

የአጥኚው ፊርማ \_\_\_\_\_

### Annex 3: Questionnaire (English Version)

Individual's ID: \_\_\_\_\_

Date of Interview: \_\_\_\_ / \_\_\_\_ / 2015

Time started: \_\_\_\_: \_\_\_\_ AM/ PM Time Ended: \_\_\_\_: \_\_\_\_ AM / PM

Name: \_\_\_\_\_ Telephone (Res) \_\_\_\_\_

Telephone (Office) \_\_\_\_\_ Mob \_\_\_\_\_

Physical Address: Sub city: \_\_\_\_\_, Woreda: \_\_\_\_\_ Kebele: \_\_\_\_\_ H.No: \_\_\_\_\_

Role of participant in the study: Faster  Non-faster/Control

Methods of Assessment is classified in to three: I. Questionnaire,

II. Anthropometric Measurements and

III. Biochemical Analysis

#### I. QUESTIONNAIRE

##### 1. Socio-demographic Characteristics

No	Questions	Responses	Code
1	Age	_____ ( Completed years)	
2	Sex	1. Male <input type="checkbox"/> 2. Female <input type="checkbox"/>	
3	Educational Level	1. No formal Education <input type="checkbox"/> 2. Primary Level <input type="checkbox"/> 3. Secondary Level <input type="checkbox"/> 4. Tertiary Level <input type="checkbox"/>	
4	Marital Status	1. Single <input type="checkbox"/> 2. Married <input type="checkbox"/> 3. Divorced <input type="checkbox"/> 4. Widowed <input type="checkbox"/>	
5	Monthly Income (Eth Birr)	1. No regular income <input type="checkbox"/> 2. Less than 999 <input type="checkbox"/> 3. From 1,000 to 1,999 <input type="checkbox"/> 4. From 2,000 to 2,999 <input type="checkbox"/> 5. $\geq$ 3,000 <input type="checkbox"/>	

## 2. Behavioural Risk Factors & Fasting Experience Assessment

No	Questions	Responses	Code
1	Do you Smoke?	1. Current smoker <input type="checkbox"/> 2. Current non-smoker <input type="checkbox"/>	
2	Do you drink an alcohol?	1. Yes <input type="checkbox"/> 2. No <input type="checkbox"/>	
	If yes, how often & amount?	Frequency: _____ Type: _____ Amount per occasion: _____	
3	Do you chew Chat/Khat?	1. Yes <input type="checkbox"/> 2. No <input type="checkbox"/>	
4	Do you do physical activity ( $\geq$ 30min walk, or fitness training $\geq$ 3 times per wk)?	1. Yes <input type="checkbox"/> 2. No <input type="checkbox"/>	
5	Do you participate in EOC fasting practice?	1. Yes <input type="checkbox"/> 2. No <input type="checkbox"/>	
5.1	If yes, among the 7 fasting periods in which of them do you participate?	1. Salivation fasting (wednesday & friday) <input type="checkbox"/> 2. Fasting preceding Christmas (Gena) <input type="checkbox"/> 3. Fasting preceding Easter (Fasika) <input type="checkbox"/> 4. Sene <input type="checkbox"/> 5. Nenewie <input type="checkbox"/> 6. Assumption of the Virgin Merry (Filseta) <input type="checkbox"/> 7. Tsegie <input type="checkbox"/>	
5.2	For how many years have you participated in the above chosen fasting periods?	_____ (in years)	
5.3	During fasting, to what extent do you limit your intake?	1. Deprivation of all ASF + eat breakfast <input type="checkbox"/> 2. Deprivation of all ASF except fish <input type="checkbox"/> 3. Deprivation of ASF + skip breakfast <input type="checkbox"/> 4. Deprivation of ASF + fasting until 3PM <input type="checkbox"/> 4. Other _____	

ASF Animal Source Foods, EOC Ethiopian Orthodox Church



#### 4.5. Food Frequency Questionnaire;

How often, in the past 7 days, did you eat the following?

List of foods	Range of frequencies				
	Never	Weekly		Daily	
	Never	≤2x/wk	3-6x/week	Once/day	≥2x day
<b>Fruit</b> (fresh & canned; bananas, oranges, papaya, avocado, mango, guava, peach, pineapple, apples, etc. or juices made from them)					
<b>Vegetables</b> (fresh & canned; tomato, cabbage, kale, spinach, broccoli, carrots, lettuce, mushrooms, etc.)					
<b>Pulses/legumes/nuts:</b> (chickpeas, Lentils, peas, beans, broad beans, soya, groundnut & other pulses. Foods made from them; stew, peanut butter, testi soya, dry roasted or boiled pulses etc.					
<b>Grains/Cereals</b> (rice, wheat, sorghum, millet, teff, maize, oats, barley, finger millet etc. or foods made from them; injera, bread, porridge, dry roasted or boiled grain, pasta/macaroni)					
<b>Milk &amp; Milk products</b> (cow milk, powder milk, yogurt, cheese, whey)					
<b>Fish</b> (fresh, dried & canned like roasted, stew, lebeleb, etc.)					
<b>Eggs</b> (fried, boiled, omelet in salad, in baked goods, etc.)					
<b>Meat</b> (chicken, beef, goat, sheep or meat products sausages, stew)					
<b>Cooking Oil/Fat:</b> oils, fats or butter added to food or used for cooking					
<b>Roots and Tubers</b> (cassava, taro, white potato, beetroot, sweet potato, pumpkin, bulla, kocho, manioc etc )					
<b>Coffee and Tea</b>					
<b>Household sugar/honey</b>					
<b>Confectionery</b> (ice-cream, candy, chocolates, biscuits, cakes, cookies, Soft Drinks, energy drinks, sweetened juice drinks)					

## II. ANTHROPOMETRIC AND BLOOD PRESSURE MEASUREMENTS

No	Variables	Pre-Fasting	End of fasting	Difference
1	Weight (Kg)			
2	Height (Cm)			
3	Waist Circumference (Cm)			
4	Hip Circumference (Cm)			
5	BMI (Kg/m <sup>2</sup> )			
6	Waist-Hip Ratio			
7	Systolic BP (mmHg) Measurement 1			
	Systolic BP (mmHg) Measurement 2			
	Systolic BP (mmHg) <b>Average</b>			
8	Diastolic BP (mmHg) Measurement 1			
	Diastolic BP (mmHg) Measurement 2			
	Diastolic BP (mmHg) <b>Average</b>			

## III. BIOCHEMICAL ASSAYS

No	Parameters	Pre-Fasting	End of fasting	Difference
1	FBS (mg/dL)			
2	Triglyceride (mg/dL)			
3	LDL-Cholesterol (mg/dL)			
4	HDL-Cholesterol (mg/dL)			
5	Total-Cholesterol (mg/dL)			

FBS Fasting Blood Sugar; LDL & HDL Low & High Density Lipoprotein

**Annex 4: Questionnaire (Amharic Version) መጠይቅ**

የግለሰብ/ዊ መለያ ኮድ: \_\_\_\_\_ ቃለ-መጠይቁ የተደረገበት ቀን: \_\_\_/\_\_\_/2007 ዓ.ም.

ቃለ-መጠይቁ የተደረገበት ሰዓት: ከ\_\_\_:\_\_\_ ያበቃበት ሰዓት: \_\_\_:\_\_\_

የግለሰብ/ዊ ስም: \_\_\_\_\_ ስልክ (የቤት): \_\_\_\_\_

ስልክ (የሥራ): \_\_\_\_\_ (የሞባይል): \_\_\_\_\_

አድራሻ: ክፍለ ከተማ: \_\_\_\_\_ ወረዳ: \_\_\_\_\_ ቀበሌ: \_\_\_\_\_ የቤት ቁጥር: \_\_\_\_\_

ግለሰብ/ዊ በጥናት ውስጥ የሚኖራቸው ሚና፤ ጿሚ  የማይገኝ/ከንትርል/

ይህ ጥናት ሦስት ዓብይ የዳሰሳ መንገዶች አሉት፡ 1ኛ. ቃለ-መጠይቅ,

2ኛ. የሰውነት ክብደትና ግዝፈት መጠን ልኬት

3ኛ. የደም ናሙና ምርመራ

**I. ቃለ-መጠይቅ**

**1. የግለሰብ/ዊ ህበራዊ፣ ሥነ-ህዝባዊ እና ኢኮኖሚያዊ ሁኔታ**

ተራ ቁ.	ጥያቄዎች	ምላሾች	ኮድ
1	ዕድሜ	_____	
2	ፆታ	1. ወንድ <input type="checkbox"/> 2. ሴት <input type="checkbox"/>	
3	የትምህርት ደረጃ	1. መደበኛ ትምህርት ያልተማሩ <input type="checkbox"/> 2. መጀመሪያ ደረጃ <input type="checkbox"/> 3. ሁለተኛ ደረጃ <input type="checkbox"/> 4. ከሁለተኛ ደረጃ በላይ <input type="checkbox"/>	
4	የጋብቻ ሁኔታ	1. ያላገቡ <input type="checkbox"/> 2. ያገቡ <input type="checkbox"/> 3. የተፋቱ <input type="checkbox"/> 4. የትዳር አጋራቸውን በሞት ያጡ <input type="checkbox"/>	
5	ወርሃዊ ገቢ (ብር)	1. መደበኛ የገቢ ምንጭ የሌላቸው <input type="checkbox"/> 2. ከ 999 ብር ያነሰ <input type="checkbox"/> 3. ከ 1,000 እስከ 1,999 ብር <input type="checkbox"/> 4. ከ 2,000 እስከ 2,999 ብር <input type="checkbox"/> 5. 3,000 ብር እና ከዚያ በላይ <input type="checkbox"/>	

2. የግል ባህሪ እና የጾም ልምድ መጠይቅ

ተራ ቁ.	ጥያቄዎች	ምላሾች	ክድ
1	ሲጋራ ያጨሳሉ?	1. አዎ <input type="checkbox"/> 2. የለም <input type="checkbox"/>	
2	የአልኮል መጠጥ ይጠጣሉ?	1. አዎ <input type="checkbox"/> 2. የለም <input type="checkbox"/>	
	የአልኮል መጠጥ የሚጠጡ ከሆነ፤ በየምንያክል ጊዜ? _____ በየምንያክል ጊዜ እና መጠኑን ቢገልፁ? የመጠጡ ዓይነት? _____ በአንድ ጊዜ የሚወስዱት መጠን? _____		
3	ጫት ይቅማሉ?	1. አዎ <input type="checkbox"/> 2. የለም <input type="checkbox"/>	
4	የአካል ብቃት እንቅስቃሴ ያደርጋሉ (≥ 30ደቂቃ ፈጣን እርምጃ አልያም ስፖርት ≥3 በየሳምንቱ)?	1. አዎ <input type="checkbox"/> 2. የለም <input type="checkbox"/>	
5	በኢትዮጵያ ኦርቶዶክስ ተዋህዶ ቤተክርስቲያን ዋና ዋና የማት ላይ ይሳተፋሉ?	1. አዎ <input type="checkbox"/> 2. የለም <input type="checkbox"/>	
	5.1. ምላሽዎ አዎ ከሆነ፤ ከተዘረዘሩት 7 ዋና ዋና የማት በየትኛው/ኞቹ ይሳተፋሉ?	1. ረቡዕ እና አርብ <input type="checkbox"/> 2. ገና <input type="checkbox"/> 3. ፋሲካ <input type="checkbox"/> 4. ሰኔ <input type="checkbox"/> 5. ነጥረ <input type="checkbox"/> 6. ፍልሰታ <input type="checkbox"/> 7. ጽጌ <input type="checkbox"/>	
	5.2. ከላይ የመረጧቸውን የማት ለስንት ዓመት/ታት ጽመዋል?	_____ (ዓመት/ታት)	
	5.3. በጾም ወቅት የሚከተሉትን የአመጋገብ ሥርዓት ቢገልፁ?	1. ሁሉንም የእንስሳት ተዋህዶ አልመገብም በተጨማሪ ቁርስ እመገባለሁ <input type="checkbox"/> 2. ሁሉንም የእንስሳት ተዋህዶ ከዓሳ ውጭ አልመገብም <input type="checkbox"/> 3. ከሁሉም የእንስሳት ተዋህዶ በተጨማሪም ቁርስ አልመገብም <input type="checkbox"/> 4. ከሁሉም የእንስሳት ተዋህዶ በተጨማሪም ከቀኑ እስከ 9 ሰዓት አልመገብም፤ <input type="checkbox"/> 5. ሌላ: _____	



**4.5. ከዚህ በታች የተዘረዘሩትን ምግቦች ምንያክል ጊዜ እንደተመገቡ የሚዳስስ መጠይቅ፤**

**ባለፉት 7 ቀናት ውስጥ ከዚህ በታች የተዘረዘሩትን ምግቦች ምንያክል ጊዜ ተመግበዋል?**

የምግቦች ዝርዝር (በየፈርጁ የተዋቀረ)	የተመገቡባቸው ጊዜያቶች				
	በፍጹም	በየሳምንቱ		በየቀኑ	
	በፍጹም	≤2x/ሳምንት	3-6x/ሳምንት	በቀን 1 ጊዜ	በቀን ≥2x
<b>ፍራፍሬዎች፤</b> ሙዝ፣ ብርትኳን፣ ፓፓዬ፣ አቮካዶ፣ ማንጎ፣ ዘይቱን፣ ኮከ፣ አናናስ፣ ፖም እና ወዘተ በጁስ መልክ ወይም የታሸጉ ምርቶችን ጨምሮ					
<b>አትክልቶች፤</b> ቲማቲም፣ ጥቅል ጎመን፣ የአበሻ ጎመን፣ ቆስጣ፣ ካሮት፣ ሰላጣ፣ እንጉዳይ እና ወዘተ በተለያዩ አዘገጃጀት					
<b>ጥራጥራዎች፤</b> ሽምብራ፣ ምስር፣ አተር፣ ባቄላ፣ አኩሪ አተር፣ ለውዝ እና ወዘተ በተለያዩ አዘገጃጀት ፡- ወጥ፣ ቆሎ፣ ንፍሮ፣ ስልጅ፣ ፉል፣ የለውዝ ቅቤ፣ ቴስቲ ሶያ...					
<b>ሰብሎች፤</b> ሩዝ፣ ሰንዴ፣ ማሽላ/ዘንጋዳ፣ ጤፍ፣ በቆሎ፣ አጃ፣ ጉብስ፣ ዳጉሳ እና ወዘተ በተለያዩ አዘገጃጀት ፡- ፓስታ፣ መኮሮ፣ እንጀራ፣ ዳቦ፣ ገንፎ፣ ቆሎ፣ ንፍሮ...					
<b>ወተት እና የወተት ውጤቶች፤</b> ትኩስ የላም ወተት፣ የዱቄት ወተት፣ እርጎ፣ አይብ፣ አጓት/አሬራ፣ ታሽገው የሚሸጡ የወተት ምርቶችን ጨምሮ					
<b>ዓሣ፤</b> በተለያዩ አዘገጃጀት ፡- የዓሣ ወጥ፣ ጥብስ፣ ለብለብ እና ታሽገው የሚሸጡ የዓሣ ምርቶችን ጨምሮ					
<b>እንቁላል፤</b> በተለያዩ አዘገጃጀት ፡- እንቁላል ፍርፍር፣ የተቀቀለ እና ሌሎች ከእንቁላል የተዘጋጁ ምርቶች					
<b>ሥጋ ፤</b> የበሬ፣ በግ፣ ፍዩል፣ ዶሮ ወዘተ በተለያዩ አዘገጃጀት					
<b>ቅቤ እና የሚረገጉ/ፈላሽ ዘይቶች፤</b> ምግብ ለማብሰል/ለማዘጋጀት የተጠቀሟቸው					
<b>ሥራሥር ምግቦች፤</b> የእንጨት ቦዬ፣ ጎደሬ፣ ድንች፣ ቀይ ሥር፣ ስኳር ድንች፣ ዱባ፣ ቡላ፣ ቆጮ ወዘተ በተለያዩ አዘገጃጀት					
<b>ቡና ወይም ሻይ፤</b>					
<b>ስኳር ወይም ማር፤</b>					
<b>ጣፋጭ ምግቦች፤</b> ቸኮሌት፣ ብስኩት፣ ጣፋጭ ኬኮች፣ አይስ ከሬም፣ የለሰላሳ መጠጦች፣ ኃይል ሰጪ የታሸጉ መጠጦች፣ ጣፋጭ ጁሶች ወዘተ					

**II. የሰውነት ክብደትና ግዝፈት መጠን እና የደም ግፊት ልኬት**

**(ANTROPOMETRIC AND BLOOD PRESSURE MEASUREMENTS)**

No	Variables	Pre-Fasting	End of fasting	Difference
1	Weight (Kg)			
2	Height (Cm)			
3	Waist Circumference (Cm)			
4	Hip Circumference (Cm)			
5	BMI			
6	Waist-Hip Ratio			
7	Systolic BP (mmHg) Measurement 1			
	Systolic BP (mmHg) Measurement 2			
	Systolic BP (mmHg) <b>Average</b>			
8	Diastolic BP (mmHg) Measurement 1			
	Diastolic BP (mmHg) Measurement 2			
	Diastolic BP (mmHg) <b>Average</b>			

**III. ከደም ናሙና የሚሰራ የቅባት እና የስኳርመጠን ልኬት (BIOCHEMICAL ASSAYS)**

No	Parameters	Pre-Fasting	End of fasting	Difference
1	FBS (mg/dL)			
2	Triglyceride (mg/dL)			
3	LDL-Cholesterol (mg/dL)			
4	HDL-Cholesterol (mg/dL)			
5	Total-Cholesterol (mg/dL)			

FBS Fasting Blood Sugar; LDL & HDL Low & High Density Lipoprotein

**Annex 5. DECLARATION**

I, the undersigned, declare that this thesis is my original work and that all sources of materials used for the thesis have been dully acknowledged.

Name: Habtamu Guja

Signature: \_\_\_\_\_

Date: \_\_\_\_\_

This thesis work has been submitted for examination with our approval as university advisor.

Name of Advisors

Signature

Date

Dr. Jemal Haidar (Primary Advisor): \_\_\_\_\_

Dr. Kaleab Baye (Secondary Advisor): \_\_\_\_\_

**ASSURANCE OF PRINCIPAL INVESTIGATOR**

I undersigned here agrees to accept responsibility for scientific ethical and technical conduct of the research project and for provision of required progress reports as per terms and the condition of the research I will communicate to my advisor and other stakeholders involved in this research publications office in effect at the time of grant is forwarded as the result of this application.

Name of the student: Habtamu Guja

Signature \_\_\_\_\_

Date \_\_\_\_\_

Approval of Advisors:

Name of the first advisor: Dr Jemal Haidar (Associate professor)

Signature \_\_\_\_\_

Date \_\_\_\_\_

Name of the second advisor: Dr Kaleab Baye (Assistant professor)

Signature \_\_\_\_\_

Date \_\_\_\_\_