RISK PREDICTION OF CARDIOVASCULAR DISEASES IN SELECTED RURAL COMMUNITY OF NEPAL

Mahesh Kumar Khanal MPhil in Noncommunicable Diseases (Thesis) Session: 2011/12 Registration Number: 301/2011-12



DEPARTMENT OF COMMUNITY MEDICINE
BANGLADESH INSTITUTE OF HEALTH SCIENCES
FACULTY OF BIOLOGICAL SCIENCES
UNIVERSITY OF DHAKA
DHAKA, BANGLADESH

August 2015

DECLARATION

I hereby declare that all the work presented in the study entitled "Risk Prediction of Cardiovascular Diseases in Selected Rural Community of Nepal" was carried out by me. All the sources used in this study have been cited correctly. No part of it has been presented previously in any academic institute or university for any other purpose.

Mahesh Kumar Khanal

MPhil in Noncommunicable Diseases Department of Community medicine Bangladesh Institute of Health Sciences (BIHS) Dhaka, Bangladesh

CERTIFICATE

This is to certify that Mahesh Kumar Khanal has completed the thesis entitled "Risk Prediction of Cardiovascular Diseases in Selected Rural Community of Nepal" in partial fulfillment for the requirement of degree of Master in Philosophy in Noncommunicable Diseases from Bangladesh Institute of Health Sciences (BIHS), under faculty of Biological Sciences, University of Dhaka under my guidance and supervision.

α	•
Su	pervisor

Prof. (Dr.) M.S.A Mansur Ahmed

Head, Department of Community Medicine & Course Chairman of Noncommunicable Diseases Bangladesh Institute of Health Sciences (BIHS)

CERTIFICATE

This is to certify that Mahesh Kumar Khanal has completed the thesis entitled "Risk Prediction of Cardiovascular Diseases in Selected Rural Community of Nepal" in partial fulfillment for the requirement of degree of Master in Philosophy in Noncommunicable Diseases from Bangladesh Institute of Health Sciences (BIHS), under faculty of Biological Sciences, University of Dhaka under my guidance and supervision.

Joint-Supervisor

Prof. Arun Shayami

Head of Department, Cardiology Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Tribhuvan University Kathmandu, Nepal

FACULTY OF BIOLOGICAL SCIENCES UNIVERSITY OF DHAKA

The undersigned certify that they have carefully read and examined the student on this thesis, and being satisfied, recommended to the faculty of Biomedical Sciences, Dhaka University for acceptance of this thesis titled "Risk Prediction of Cardiovascular Diseases in Selected Rural Community of Nepal" by Mahesh Kumar Khanal in partial fulfillment of the requirements for the degree of Master of Philosophy (MPhil) in Noncommunicable Diseases (NCD).

Board of examiners

1) Convener	Signature:	
2) Member	Signature:	Full name: Designation:
3) Member	Signature:	Full name:
Date of approv	val:	Designation:

BIHS, Dhaka, Bangladesh

CONTENTS

List of contents	Page no
Acknowledgments	I
List of Tables	III
List of Figures	IV
List of Annexure	V
Abbreviations	VI
Abstracts	VII
CHAPTER 1: INTRODUCTION	1
1.1 Background Information	2-4
1.2 Justification of the study	5-6
1.3 Research Questions	7
1.4 Research Objectives	8
1.5 Lists of Key Variables	9
1.6 Conceptual Framework	10
1.7 Operational Definitions	11-12
CHAPTER 2: LITERATURE REVIEW	13
2.1 Introduction	14
2.2 Global Burden of Cardiovascular Diseases	14
2.3 Burden of Cardiovascular Diseases in South East Asia Region	. 15
2.4 Burden of Cardiovascular Diseases in Nepal	16
2.5 Economic Burden of Cardiovascular Diseases	17
2.6 Risk Prediction Tools for Cardiovascular Diseases	17-22
2.7 Comparison among different risk scores	22
2.8 WHO/ISH Risk Prediction Chart	22
2.9 Cardiovascular Risk Factors	23-27
CHAPTER 3: METHODOLOGY	28
3.1 Study Design	29
3.2 Study Site	29
3.3 Study Period	29
3.4 Study Population	29
3.5 Inclusion Criteria	29
3.6 Exclusion Criteria	29

3.7 Sample Size	30
3.8 Sampling Technique	30
3.9 Data Collection Procedure	32
3.10 Data Collection Techniques	33
3.11 Data Collection Tools	34
3.12 Validity and Reliability	35
3.13 Pre-testing	35
3.14 Data Quality Control	35
3.15 Data Processing and Logical Checking	35
3.16 Data Analysis	35
3.17 Ethical Consideration	36
CHAPTER 4: RESULTS	37-52
CHAPTER 5: DISCUSSION	53
CHAPTER 6: CONCLUSIONS, RECOMMENDATIONS,	
AND LIMITATIONS	58
6.1 Conclusions	59
6.2 Recommendations	60
6.3 Limitations of the Study	61
CHAPTER 7: REFERENCES	62
CHAPTER 8: ANNEXES	72-107

Acknowledgements

I would like to express my gratitude to all those personnel of different level who were directly or indirectly involved in its accomplishment.

Atfirst, I want to thank Norad's Program for Master Studies(NOMA) of University of Oslo, Norway for their financial assistance throughout my MPhil program at the Bangladesh Institute of Health Sciences under University of Dhaka. I would like to express my gratitude to Prof Liaquat Ali, Director of Bangladesh Institute of Health Sciences (BIHS) and chairman of thesis selection committee for approval of my thesis topic, valuable support and inspiration during my thesis. I must not forget to acknowledge ethical review board of Nepal Health Research Council for permitting me to conduct the study.

Primarily I would like to express sincere gratitude to my supervisor, Prof.(Dr.)MSA Mansur Ahmad, Head of Department of Community medicine and course chairperson of Noncommunicable Diseases, Bangladesh Institute of Health Sciences (BIHS) for his paramount supports and guidance. His inspirational supervision had been a cornerstone in completion of this thesis. Likewise, I owe a special thanks to Prof Dr. Arun Shayami, Head of Department, Cardiology, Manmohan Cardiothoracic Vascular and Transplant Center, Institute of Medicine, Tribhuvan University for being so kind and supportive to me during thesis work.

I am really grateful to co-supervisor Md. Moniruzzaman, Senior Lecturer, Department of Community Medicine, Banglash Institute of Health Sciences(BIHS) for providing innumerous supports, guidance and feedback on each and every moments of thesis work. I am indebted to my co-supervisor Mr. Palash C Banik, Senior Lecturer, Department of Community Medicine, Bangladesh Institute of Health Sciences for critical suggestion and guidance. It would be almost impossible to complete the thesis without their supports.

Also especial thanks to Dr Surya Devkota, Dr. Raja Ram Dhungana, DrSuira Joshi, Dr. Devdeep Mukharjee and Secretary of Bhotewodar and Sundarbazar VDC and to my wife Mrs Pratiksha Bhandari for providing valuable supports and suggestion.

I would also like to thank Ramchandra Adhikari, Director of Karmada Hospital for providing laboratory and supporting on logistic, transportation and managerial support. I am indebted to lab technician team MrBikashChiluwal and MrSailendraPandit for providing valuable time in blood sample collection and analysis. Special thanks to Karmada Hospital for assisting in analyzing blood samples.

I can't forget to mention field supervisor and data enumerator team Miss Pratiksha Adhikari, Miss Yem Kumari Gurung, Mr Anil Koju, Mr Krishna Malakar, Mr Bhuban Gurung, Mr Kushbilash Bagale, Mr Dwarika Mishra, Mr Janak Lal Shrestha and Miss Muna Gurung.

My very special thanks and appreciations to all the respondents of this study who have not only spared their time but also provided valuable information for this study with their kind participation and cooperation during the data collection procedure.

I am also thankful to all my friends who have helped me with their valuable suggestions, support and helping me with the computer works.

Lastly, I would like to express my sincere gratitude with deep appreciation to all well-wishers who are directly and indirectly involved to help me in so many ways to carry out the research and preparing this report.

List of Tables

Table No	Title	Page No
Table 1	Sociodemographic and economic characteristics of respondents	39
Table 2	Prevalence of behavioral risk factors of cardiovascular diseases by gender	41
Table 3	Distribution of blood pressure and BMI by gender of study subjects	43
Table 4	Distribution of blood sugar by gender of study subjects	44
Table 5	Distribution of cholesterol by gender of study subjects	46
Table 6	Distribution of 10-year CVDs risk by age and sex of study subjects	47
Table 7	Distribution of CVDs risk by gender after including study subjects with high blood pressure	48
Table 8	Distribution of 10 year risk of cardiovascular disease by socioeconomic status	52

List of Figures

Figure	Title	Page
no		no
Figure 1	Percentage Distribution of BMI by international classification	42
Figure 2	Distribution of moderate and high risk by level of education and sex	49
Figure 3	Distribution of moderate and high risk by cast and sex of study subjects	50
Figure 4	Distribution of moderate and high risk by occupation of study subjects	50
Figure 5	Distribution of moderate and high risk by income level of study subjects	51
Figure 6	Distribution of moderate and high risk by economic status gender of the study subjects	51

List of Annexure

Annex no	Title	Page
		no
Annex I	Informed consent form (Nepali)	72
Annex II	Interview questionnaire (English)	73
Annex III	Interview questionnaire (Nepali)	90
Annex IV	WHO/ISH risk prediction chart	103
Annex V	Kish Household Coversheet (English)	104
Annex VI	Respondent Feedback form (Nepali)	105
Annex VII	Study Site/Geographical location	106
Annex VIII	Work Plan	107

ABBREVIATIONS

SCORE	Systematic Coronary Risk Evaluation
ATP	Adult Treatment Panel
BMI	Body Mass Index
BP	Blood Pressure
CABG	Coronary Artery Bypass Graft
CHD	Coronary Heart Disease
CHOD	Cholesteroloxidase
COPD	Chronic Obstructive Pulmonary Disease
CVDs	Cardiovascular Diseases
DALY	Disability Adjusted Life Year
DBP	Diastolic Blood Pressure
GDP	Gross Domestic Product
GLV	Green Leafy Vegetables
GOD	Glucose Oxidase
GPO	Glycerol-3-phosphate Oxidase
HDL	High Density Lipoprotein
HDL-C	High Density Lipoprotein-Cholesterol
LDL	Low Density Lipoprotein
LDL-C	Low Density Lipoprotein-Cholesterol
LMCs	Low and Middle-income Countries
MET	Metabolic Equivalent
NCD	Non-communicable disease
PAL	Physical Activity Level
PROCAM	Prospective Cardiovascular Munster
SIGN	Scottish Intercollegiate Guidelines Network
SBP	Systolic Blood Pressure
TG	Triglycerides
VDC	Village Development Committee
WHO	World Health Organization

ABSTRACT

Background: Cardiovascular Diseases (CVDs) are the number one cause of morbidity and mortality globally and nationally. It is estimated that 17.3 million died globally due to cardiovascular diseases in 2008. In Nepal, estimated proportionate mortality attributable to CVDs is 22% in 2004 and 25% in 2008. Patients suffering from heart diseaseswere40% among all noncommunicable diseases in Nepal.

Objectives: The main objective of the study is to estimate 10-year Cardiovascular Diseases risk among rural community of Nepal who have not yet developed clinically manifest cardiovascular diseases using World Health Organization/International Society of Hypertension (WHO/ISH) risk prediction chart for SEAR D.

Methods: A Cross sectional study conducted among 388 respondents age ranged from 40 to 80 years. The study conducted in Bhotewodar and Sunderbazar VDCs of Lamjung District. From 18 wards, four wards were selected using probability proportion to size method (PPS). Household was selected using systematic random sampling. Study subject from the selected household was decided using kiss table. WHO STEPS questionnaires tool was used for data collection. Data was analyzed by SPSS version 16.0.

Results: The proportions of population who are at moderate risk were 9.3% and high risk were 4.3% for developing cardiovascular disease in next 10 years. More than 10% risk of cardiovascular disease was 13.6% using WHO/ISH chart alone. Risk was increased to 18% after including those having blood pressure of ≥160/100 mmHg. Moderate and high-risk population was significantly associated with age (p=0.001), level of education (p=0.01) and occupation (p=0.001). Prevalence of current smoker was 24.8%, obesity was 13%, hypertension was 40.5%, diabetes was 16.2%, and raised total cholesterol was 11.6%.

Conclusion: One out of seven people of the total population with age 40 to 80 years was affected by >10 %(moderate and high) risk for development of cardiovascular diseases within next 10 years. Risk was higher among low educated, unemployed and homemaker. Immediate planning and implementation is needed to reduce risk factors of cardiovascular diseases.

CHAPTER 1: INTRODUCTION

This Chapter includes:

- 1.1 Background Information
- 1.2 Justification of the Study
- 1.3 Research Questions
- 1.4 Objectives of the Study
- 1.5 List of Key Variables
- 1.6 Conceptual framework
- 1.7 Operational Definitions

1.1 Background Information

Atherosclerotic cardiovascular diseases are ischaemic heart disease or coronary artery disease (heart attack), cerebrovascular disease (stroke) and diseases of aorta and arteries, including hypertension and peripheral vascular disease[1].

Cardiovascular diseases (CVDS) are common in general population and becoming the number one cause of morbidity and mortality. It is estimated that 17.3 million died globally due to cardiovascular diseases in 2008. Ten percent DALY is attributable to CVDs alone[2]. Again, Out of 16 million deaths under the age of 70, 37 % deaths are caused by CVDs. Developing countries are experiencing more inequalities of occurrence and outcome of these diseases. Eighty percent death is in low and middle income countries except Africa[2]. Trends of death are changing among the developing and developed countries[3]. Twenty nine percent of deaths at the age below 60 years are happening in LMIC while it's only 13% in high income countries. The percentage of premature deaths from CVDs ranges from 4% in high-income countries to 42% in low-income countries, leading to growing inequalities in the occurrence and outcome of CVDs between countries and populations. There has been a doubling of CVDs rates in LMICs during recent decades. Almost two thirds of the total CVDS is accounted by ischemic heart disease and cerebrovascular diseases in both male and female[1, 2]. The burden will be severe[4]. It is projected that 23.3 million people will die by 2030 only due to CVDs[5].

In South East Asia region 7.9 million (55%) deaths were caused by Non communicable diseases in 2008. Cardiovascular diseases alone accounted for 25% deaths in the same year. Cardiovascular diseases claimed 3.7 million lives. Trend of disease is changed. Premature death before age of 60 were 34 % in the region, compare to 23% on rest of the world.

In Nepal death due to CVDs also increased from 22% in 2004 to 25% in 2008[3, 6]. According to hospital based survey in Nepal, 36.5% patients were suffering from NCD and among them 40% had heart disease. Estimated proportionate mortality due to CVDS also increased from 22% in 2004 to 25% in 2008[3, 6]. According to the report of Sahid Gangalal National Heart Centre, which is the only specialty hospital in

Nepal, coronary artery disease is the most common cause of admission (42.5% in 2006)[7]

Different risk factors interact together to develope cardiovascular diseases, which has been explained by different studies. Cardiovascular diseases are the most common underlying cause of mortality among diabetes patients. According to multinational study of WHO, fifty two percent of death were attributed to cardiovascular diseases in type II diabetes mellitus and 44% among type I diabetes mellitus[8]. Similarly, among type I diabetes patients 37% mortality was caused by cardiovascular disease mostly of ischemic heart disease [9]. Risk of CVDs increases with raised glucose valuein blood. In a meta-analysis, risk of CVD is 27% greater in mid-point level (150-194 mg/dl) in compared to lowest level (69-107 mg/dl)[10].

Likewise, blood pressure level is positively related to the risk of stroke and coronary heart disease. In age groups 40 to 70 years, the risk of CVDs doubles for each incremental increase of 20/10 mmHg of blood pressure, starting as low as 115/75 mmHg[11]. In addition to coronary heart disease and cerebrovascular disease, uncontrolled blood pressure causes heart failure, renal impairment, peripheral vascular disease and damage to retinal blood vessels and visual impairment.

Smoking is estimated to cause nearly 10% of cardiovascular diseases[12]. While, smoking cessation is beneficial to diminish the mortality of coronary heart disease. A 50-year follow-up of British doctors demonstrated that, among ex-smokers, the age of quitting has a major impact on survival prospects those who quit between 35 and 44 years of age had the same survival rates as those who had never smoked[13].

Raised blood cholesterol increases the risk of heart disease and stroke. Globally, one third of ischemic heart disease is attributable to high cholesterol [12]. Lowering raised serum cholesterol reduces the risk of heart disease. For example, if 10% (0.6 mmol/l) reduction in serum cholesterol in 40-year old men has been reported to result in a 54% reductionin heart disease within five years; the same serumcholesterol reduction for 70-year old men can result in anaverage 20% reduction in heart disease occurrence withinfive years[14].

Many studies reported that physical activities have inverse relationship to CVDs in dose-response fashion [15, 16]. Physical activity is a key determinant of energy expenditure and thus fundamental to energy balance and weight control. Adequate

consumption fruit and vegetables reduces the risk of CVDs. Ahealthy diet can contribute to a healthy body weight, adesirable lipid profile and a desirable blood pressure[17]. Prospective epidemiological studies have shown a relationship between overweight or obesity and cardiovascular morbidity and mortality[18].

These risk factors have been used to develop a number of multivariate risk models by measuring cumulative effect of relative risk of each risk factor to estimate risk of initial cardiovascular events for different population and ethnicity. The original Framingham risk score (1998), was derived from a largely Caucasian population of European descent[19]. SCORE, which was recommended in the 2007 European Society of Cardiology guidelines on cardiovascular disease prevention included data on more than 200,000 patients pooled from cohort studies in 12 European countries[20]. The QRISK and the updated QRISK2 algorithms were developed to predict cardiovascular risk in patients from different ethnic groups living in England and Wales[21].ASSIGN score was derived from Scottish Heart Health Extended Cohort (SHHEC) where participants were ranked according to social deprivation and followed for death due to cardiovascular causes[22]. The World Health Organization/International Society of Hypertension (WHO/ISH) risk prediction charts have been developed for non-western populations of developing countries where cohort data and resources are not readily available for the development of populationspecific risk prediction charts[23].

The choice of a specific risk model for cardiovascular diseases risk assessment should be individualized based on patient-specific characteristics (e.g., age, gender, ethnicity) and risk models which predict hard events (i.e., death, myocardial infarction [MI], stroke)[24]. The WHO/ISH charthas been developed using a modeling approach[23]. In brief, a set of individual-level CVDs risk factor profiles (age, sex, current smoking, systolic blood pressure, total cholesterol and the presence or absence of type 2 diabetes) have been generated using information on the population distribution of these risk factors from the WHO Comparative Risk Assessment study[25]. This chart can stratify the population into different risk groups, which can be utilized to allocate the limited resources in low resource countries like Nepal.

However, inadequate studies performed to estimate the population under risk of developing CVDs in Nepal.

1.2 Justification of the Study

Prevalence of cardiovascular diseases is increasing among developing countries. Eighty percent of death due to noncommunicable diseases occurred in low and middle income countries with 37% death attributed by CVDs[2].

Cardiovascular disease is the most prevalent noncommunicable disease in Nepal. In a hospital-based study, prevalence of cardiovascular disease was40% among all patients of non communicable diseases admitted in non-specialty hospitals in Nepal[26]. Risk factors of cardiovascular diseases are also increasing in Nepal. Prevalence of hypertension was only 21.5% among population of 15 to 64 years of age in 2007[27]. However, the hypertensive population was 25.7% in same age group in 2013. Similarly, prevalence of raised blood sugar was 3.6% and raised total cholesterol was 22.7%. Current smokers were 18.5%[28]. Different risks factors like age, sex, smoking habit, hyperglycemia, and hypercholesterolemia act together in causing the cardiovascular disease.

So, it is reasonable to assume that population under risk for developing cardiovascular diseases are high in Nepal. However, there is still lack of data on proportion of population in different risk levels for development of cardiovascular diseases. The gap of knowledge on level of risk in population of Nepal is essential to know now.

Thus it is urgent to predict the risk for development of cardiovascular diseases in following years and to find out distribution of the level of risk.

Modification of risk factors has been shown to reduce mortality and morbidity in peoplewith diagnosed or undiagnosed cardiovascular diseases. Forexample, during the 10-year period covered by the World Health Organization (WHO) Multinational Monitoring of Trends and Determinants of Cardiovascular Disease initiative(WHO MONICA Project), mortality from coronary heart disease and stroke declined dramatically in many of the 38 MONICA populations[29]. Coronary heart disease mortality rates in England and Wales decreased by 62% in men and 45% in women 25 to 84 years old. Some 42% of this reduction was attributed to treatments and 58% reduction was attributed to population risk factors reduction[30].

Several forms of therapy can prevent cardiovascular diseases. Decisions about whether to initiate specific preventive actions like lifestyle modification or drug intervention along with their degree of intensity should be guided by estimation of

risk of any such vascular events. WHO/ISH risk prediction chart estimates the individual risk of developing fatal and non-fatal cardiovascular diseases to allow treatment to be targeted and degree to be needed based on individual risk factors.

The result of the study will help to find out the proportion of population under mild, moderate, and high risk of CVDs. Thus, the study will help for planning and designing of the necessaryintervention programs to reduce the occurence of cardiovascular diseases. National guidelines for prevention of CVDs can be developed based on the estimation of risk of cardiovascular diseases.

1.3 Research Questions

- What is the proportion of 10-year risk of cardiovascular diseases among rural community in Nepal?
- How 10-year risk of cardiovascular diseases distributed in relation to age, sex, socioeconomic condition of the rural community in Nepal?

1.4 Research Objectives

General objective

The main objective of the study is to estimate 10 year cardiovascular diseases risk among rural community of Nepal who have not yet developed clinically manifest cardiovascular diseases using World Health Organization/International Society of Hypertension(WHO/ISH) risk prediction chart for SEAR D.

Specific objectives

- To estimate proportion of population who are at moderate and high risk category of cardiovascular diseases in following 10 years among rural community of Nepal
- To determine how 10 year risk of cardiovascular disease is distributed in relation to age, gender, socioeconomic condition of rural community of Nepal
- To ascertain the prevalence of risk factors of cardiovascular diseases among rural community of Nepal

1.5 Lists of key variables

Variables required for risk assessment using WHO/ISH chart

- Age
- Sex
- Smoking
- Diabetes
- Systolic Blood Pressure
- Total cholesterol

Demographic variables

- Cast
- Level of Education
- Occupation
- Monthly Household Income

Behavior Related variables

- Current drinking
- Standerd drink per day
- Level of physical activity
- Servings of fruit per day

Servings of vegetable per day

Physical Measurements

- Height
- Weight
- BMI
- Waist circumference
- Diastolic Blood pressure

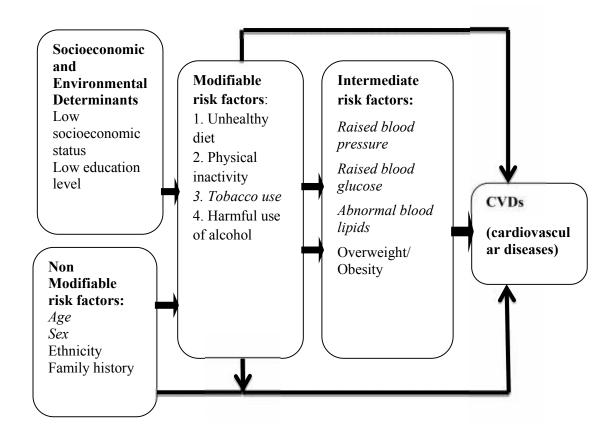
Biochemical Variables

- Fasting Blood sugar
- Post Prandial Blood sugar
- HDL Cholesterol
- Triglycerides
- LDL Cholesterol

Other variables

- Family history of CVDs
- Age of menopause among female

1.6 Conceptual framework



1.7 Operational definitions

- Current smoker: All respondents who are smoking and those who quite smoking less than 30 days before the assessment according to STEPS guidelines.
- **Smoker:** All respondents who are smoking and those who quite smoking less than one year before the assessment[31]. This definition was used for risk prediction chart only.
- Systolic Blood pressure: Mean of last two readings of systolic blood pressure.
- Diabetes: Subject who has venous blood sugar level is 7 mmol/l (126 mg/dl) or who is taking oral hypoglycemic drug or insulin. This definition was used only for risk prediction according to pocket guidelines for prevention of cardiovascular diseases.
- Poor economic status: Economic status is categorized according to family income per person according to Nepal living standard survey 2011. Study subject who had family income per person per month of ≤ 8498 NRswas considered poor economic status. Subjects having 8498 16294 NRs as medium level income and ≥ 16295 NRs as high income status.
- Serving of fruit and vegetable: One serving is equivalent 80 gram of fruit and vegetable measured by showing pictorial show card or measuring cups.
- Sufficient intake of fruits and vegetables: intake for at least five portions (400 gm) of a day = One serving of vegetable = one cup (250 ml) of raw green leafy vegetables and ½ cup (125 ml) of cooked or chopped raw vegetables
- Current drinker: Respondent who is drinking alcohol within last 30 days.
- **Standard drink:** 13.6 gm of alcohol, equivalent to 341 ml of beer, zaand or tongba, 43 ml of local lakshi.

- Metabolic equivalent (MET): MET is defined as the energy cost of sitting quietly and is equivalent to a caloric consumption of 1 kcal/Kg/hour.
- Low level of physical activity: Respondent who is engaging less than 600 MET minutes/week.
- Adequate physical activity: Respondent who spends > 600 MET minutes/week.
- Hypertension: Defined as raised systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg or taking antihypertensive drugs.
- **Body Mass Index (BMI):** International classification of Body Mass Index to categorize underweight (<18.5 kg/m2), normal weight(18.5 to 25.9 Kg/m2), overweight (≥ 25 Kg/m2) and obese(≥ 30 Kg/m2)[32].
- Impaired fasting glucose—According to WHO classification of fasting blood sugar level between 110 and 125 mg/dl[33].
- Impaired glucose tolerance- According to WHO classification of post prandial blood glucose two hours after intake of 75 gm of glucose 140-199 mg/dl[33].
- **Diabetes mellitus** A fasting blood sugar level of 126 mg/dl and higher orpost prandial blood sugar ≥200 mg/dl or taking any anti-diabetic drugs.
- Cholesterol: Cholesterol is categorized based on criteria of third report of National Cholesterol Education Program [34]. Raised total cholesterol, triglyceride and low density lipoprotein was taken for >200 mg/dl, 150 mg/dl and 100 mg/dl respectively. Decreased HDL was taken for <40 mg/dl in male and <50 mg/dl in female.
- Ten year risk category: Risk category for development of fatal and non fatal CVDs are mild risk for <10%, moderate risk for 10-20% and high risk for >20% risk

CHAPTER 2: LITERATURE REVIEW

This chapter includes:

- 2.1 Introduction
- 2.2 Global Burden of Cardiovascular Diseases
- 2.3 Burden of Cardiovascular Diseases in South East Asia Region
- 2.4 Burden of Cardiovascular Diseases in Nepal
- 2.5 Economic burden of cardiovascular Diseases
- 2.6 Risk prediction tools for cardiovascular diseases
- 2.7 Comparison among different risk scores
- 2.8 WHO/ISH risk prediction chart
- 2.9 Cardiovascular risk factors

2.1 Introduction

Cardiovascular diseases are diseases of the heart, vascular diseases of brain and diseases of blood vessels. CVDs due to atherosclerosis are Coronary heart disease or Ischemic heart disease, cerebrovascular disease or stroke, Diseases of aorta and arteries including hypertensive and peripheral vascular disease. Other CVDs are congenital heart disease, rheumatic heart disease, cardiomyopathies, and cardiac arrhythmias[1]. Atherosclerosis is the underlying disease process of major cardiovascular diseases, Ischemic heart disease and Stroke. In 2012, out of the 17.5 million cardiovascular deaths, heart attacks were responsible for 7.3 million deaths and strokes were responsible for 6.6 million deaths[35].

Atherosclerosis derives its name from the Greek words 'sclerosis' meaning hardening and 'athere' meaning gruel (accumulation of lipid). Atherosclerosis pathogenesis is multifactorial process. The phenomenon is characterized by accumulation of cholesterol, infiltration of macrophages, proliferation of smooth muscle cells (SMC), accumulation of connective tissue components and formation of thrombus[36]. This thrombus when mixed with calcium and other substances becomes plaque which causes the inner surface of the blood vessels to become irregular and the lumen to become narrow, making it harder for blood to flow through. Blood vessels also become less pliable as a result. Eventually, the plaque can rupture, triggering the formation of a blood clot. If the blood clot develops in a coronary artery, it can cause a heart attack; if it develops in the brain, it can cause a stroke.

2.2 Global Burden of Cardiovascular Diseases

Cardiovascular Diseases (CVDS) are the number one cause of morbidity and mortality[37]. It was estimated that among 36 million death were due to Non communicable diseases, 17.3 million mortality is attributable to cardiovascular diseases in 2008. Ten percent DALY were lost due to CVDS alone in 2008[2]. In 2012, cardiovascular disease mortality was 17.5 million (31.4%), which is almost half of total noncommunicable disease mortality (67.8%) among 55.8 million global deaths. Almost two thirds of the total CVDS is accounted by Ischemic Heart disease and Cerebrovascular Diseases in both male and female[1, 2]. Among top ten

leading causes of global death Ischemic heart disease is attributable to 13.2 %, Stroke 11.9% and Hypertensive heart disease 2 percent[35].

Developing countries are experiencing more inequalities of occurrence and outcome of these diseases. Over 80 % of CVDS death takes place in low and middle income countries[2]. In all income countries Ischemic heart disease and stroke are the top two killer except low income country where these are number one killer after lower respiratory infection and diarrhea[35]. Trends of death are changing among the developing and developed countries [3]. Twenty nine percent deaths at the age below 60 years are in LMIC while it is only 13% in high income countries. The percentage of premature deaths from CVDs ranges from 4% in high-income countries to 42% in low-income countries, leading to growing inequalities in the occurrence and outcome of CVDs between countries and populations. Among people below the age of 70, CVDs were responsible for the largest proportion (39%) of NCD deaths. There has been a doubling of CVDS rates in LMICs during recent decades.

According to Disease burden estimate for 2012, 1512 million DALY lost due to noncommunicable disease, which is 55.1 % of total 2743 million DALY. Cardiovascular diseases were responsible for 14.4 %(393 million) DALY loss. Of which, 165 million were due to ischemic heart disease and 141 million were due to stroke. Among total DALY ischemic heart disease was responsible for 6% and stroke was responsible for 5.2 percent loss 2012[38]. In 20012, Years of life lost (YLL) due to premature death caused by CVDs was 370 million, 18.5 % of total 2003 years lost.

Cardiovascular disease mortality will rise even in future[4]. It is projected 23.3 million people will die by 2030 only due to CVDS according to projection from 2002[5]. Total proportion of death due to Cardiovascular disease will be 31.7 % even in 2030[39].

2.3 Burden of Cardiovascular Diseases in South East Asia Region

In South East Asia region 7.9 million (55%) deaths were caused by Non communicable diseases in 2008. Cardiovascular diseases alone accounted for 3.7 million (25%) death in the same year. Trend of disease is changed [40]. In 2012

mortality due to cardiovascular diseases was 3.6 million which is one fourth (26.8 %) of total 13.7 million death in SEA region. Among them ischemic heart disease (11.5%) and Stroke (10.5%) were the top two causes of mortality in 2012[35].

Premature death before age of 60 were 34 % in the region, compare to 23% on rest of the world[40]. Age standardized death rate per 100,000 by cardiovascular diseases and diabetes was highest in Bhutan in male, where as it was in Bangladesh in female among SEA countries in 2008. In Bhutan 465 males and 381 females while in Bangladesh 447 males and 388 females per 1,00,000 were died in 2008[6]. It was 203.7 by ischemic heart disease and 108.3 by Cerebrovascular disease in 2008[1].

DALY lost due to cardiovascular diseases in the region is 97.8 million, 13.2 % of total DALY of 739 million in 2012. Among 551 million years of life lost due to premature death globally 93.4 million(17.2%) years were lost by cardiovascular diseases[38].

In 2030, cardiovascular disease will be responsible for 5.8 million deaths. It is projected that ischemic heart disease and stroke will be top two killer even in 2030, causing 13.8 % and 12.7% of regional death[39].

2.4 Burden of Cardiovascular Diseases in Nepal

Cardiovascular diseases are attributable to major mortality and morbidity in Nepal. Death due to CVDS also increased from 22% in 2004 to 25% in 2008[3, 6]. Among all NCD patients, 40% are admitted after diagnosis of Cardiovascular diseases in Non-specialized hospital in Nepal. Highest percentage of patients are Cardiovascular accident (16%) in non-specialized hospital and Myocardial Infarction(18%) in Specialized hospital[26]. Estimated proportionate mortality due to CVDS also increased from 22% in 2004 to 25% in 2008[3, 6]. Among total death due to all cause, 41,400 died by cardiovascular diseases 2012, which was 22.25% of total deaths[35]. According to 2008 estimate NCD accounted 50% of all deaths. Cardiovascular diseases mortality was 25% among all disease. Age standardized death rate per 1,00,000 caused by cardiovascular diseases and diabetes was 400.2 in male more than in female 301.3 in 2008[41]. Ischemic heart diseases and Stroke were accounted for 152.6 and 82.6 deaths per 100000 for both sexes in 2008 [1].

Shahid Gangalal National Heart Centre is the only one specialty hospital in Nepal, where number of patients is doubling yearly. Ischemic Heart Disease is the second most diagnosis (20% in 2005) in emergency. In medical ward Coronary artery disease is the most common cause for admission (42.5 %, n=2017 in 2006), whose number is increasing yearly. Coronary surgery (11%, n=1012) is the commonest after congenital and valvular surgery. Pattern of mean age of coronary artery disease is decreasing(60.6 in 2000, 58.6 in 2006) [7]. In a cardiac camp organized by Shahid Gangalal National Heart Centre, 15 % (324) were Coronary artery disease in Birgunj [42].

2.5 Economic burden of cardiovascular Diseases

Medical expenditure is high for four major chronic cardiovascular diseases: stroke, hypertension, congestive heart failure, and other heart diseases. As a result, the poorest people in low- and middle-income countries are affected most. At the household level, sufficient evidence is emerging to prove that CVDs and other noncommunicable diseases contribute to poverty due to catastrophic health spending and high out of pocket expenditure.

At macro-economic level, CVDs place a heavy burden on the economies of low-and middle-income countries. Noncommunicable disease including cardiovascular disease and diabetes are estimated to reduce GDP by up to 6.77% in low- and middle-income countries experiencing rapid economic growth, as many people die prematurely [43]. It is also the most costly disease in the United States. Using data from multiple sources, the American Heart Association has compiled a detailed table of 2006 estimates of the direct and indirect costs of heart diseases, coronary heart disease, stroke, hypertensive heart disease, heart failure, and total CVDS[44]. The estimated health care spending and lost productivity (direct and indirect costs) of total CVDS exceed \$400 billion.

2.6 Risk prediction tools for cardiovascular diseases

A number of multivariate risk models have developed for estimating the risk of initial cardiovascular events in apparently healthy, asymptomatic individuals based upon assessment of multiple variables. While all of the risk models have advantages

and disadvantages, no single risk model will be appropriate for all patients. The choice a specific risk model for cardiovascular disease (CVDs) risk assessment should be individualized based on patient-specific characteristics (eg, age, gender, ethnicity). Most experts feel that the use of risk models that predict hard CVDs events (ie, death, myocardial infarction [MI], stroke) are preferred over those which include other endpoints (ie, revascularization).

Framingham Heart Study

It is long term ongoing study of cardiovascular diseases in Framingham, Massachusetts conducted by National Heart, Lung and Blood institute in collaboration with Boston University (since 1971). The study was started from 1948 to identify common factors and characteristics that contribute to cardiovascular diseases in participants who had not yet developed overt symptoms of coronary heart disease, stroke, hypertensive heart disease, intermittent claudication.

The researchers recruited 5,209 men and women between the ages of 30 and 62 from the town of Framingham, Massachusetts, and began the first round of extensive physical examinations and lifestyle interviews that they would later analyze for common patterns related to CVDS development. Since 1948, the subjects have continued to return to the study every two years for a detailed medical history, physical examination, and laboratory tests, and in 1971, the Study enrolled a second generation - 5,124 of the original participants' adult children and their spouses - to participate in similar examinations. In 1994, the need to establish a new study reflecting a more diverse community of Framingham was recognized, and the first Omni cohort of the Framingham Heart Study was enrolled. In April 2002 the Study entered a new phase, the enrollment of a third generation of participants, the grandchildren of the Original Cohort. In 2003, a second group of Omni participants was enrolled[45].

In 1960s it was identified that cigarette smoking, increased cholesterol, elevated blood pressure, obesity increase risk of heart disease and physical activity reduce the risk of heart disease. In 1970s it was found that high blood pressure increases the stroke and postmenopausal women are in increased risk for heart disease. In same way, in 1980s it was published that high HDL reduces the risk of heart diseases[46].

The original Framingham risk score (1998), published in 1998, was derived from a largely Caucasian population of European descent [19]. Subsequent studies have suggested that the Framingham risk score performs well for prediction of CHD events in black and white women and men. Framingham Heart Study presented single multivariable function that predicts risk of developing all CVDs and its constituents in 2008[47].

Prediction of Coronary Heart Disease

For thsi study, sample were men (2489) and female (2856) aged 30 to 74 years, followed for 12 years. Blood pressure, cholesterol, LDL-C, HDL-C, Smoking, age and sex were considered for risk prediction. Outcome variable was coronary heart disease only. Family history, physical activity, obesity, postmenopausal, treatment of high blood pressure and high cholesterol were not included in the formulations. Subjects were followed for development of CHD (angina pectoris, myocardial infarction, coronary insufficiency, coronary heart disease death) [19].

ATP III hard CHD risk score

The Framingham risk score was modified (2002) by the third Adult Treatment Panel (ATP III) for screening and treatment of dyslipidemia. The modifications include elimination of diabetes from the algorithm, since it was considered to be a CHD equivalent; broadening of the age range; and inclusion of hypertension treatment and age-specific points for smoking and total cholesterol. Prediction variables used in ATP III hard CHD risk score were age, gender, total cholesterol, HDL cholesterol, systolic blood pressure, blood pressure treatment and current smoking. However, diabetes and family history of CVDSwere not used. CHD death and non fatal MI were end points assessed[34].

Framingham General Cardiovascular Risk Score

The original 1998 and revised 2002 Framingham risk scores do not include all of the potential manifestations and adverse consequences of atherosclerosis, such as stroke, transient ischemic attack, claudication and heart failure (HF) (although manifestations of aortic atherosclerosis were omitted). These patient-important vascular outcomes were included in the development of the 2008 Framingham general cardiovascular risk score, which was shown to have reliable predictive

ability. The estimated risk of developing a cardiovascular event will be higher with this risk score than with those that predict only CHD events. Study participants were 8491 (4522 female, mean age 49) between age of 30 to 74 years who were free of CVDS. Outcomes were CVDS events and death. CVDS includes CHD (coronary death, myocardial infarction, coronary insufficiency and angina) and Cerebrovascular events (including ischemic stroke, hemorrhagic stroke, and transient ischemic attack), peripheral artery disease (intermittent claudication), and heart failure. Age, total cholesterol, HDL cholesterol, systolic blood pressure, antihypertensive medicationuse, current smoking, and diabetes status were used for CVDS risk function[47]. Family history of CVDSwas not used.

SCORE Project

Systematic Coronary Risk Evaluation (SCORE), which was recommended in the 2007 European Society of Cardiology guidelines on cardiovascular disease prevention in clinical practice, included data on more than 200,000 patients pooled from cohort studies in 12 European countries. A unique aspect of SCORE is that separate risk scores were calculated for high- and low-risk regions of Europe. The predictive value of SCORE was high in each component study cohort. SCORE differs from the earlier Framingham risk models (and others) in two important ways: it estimates the 10-year risk of any first fatal atherosclerotic event (eg. stroke or ruptured abdominal aneurysm), not just CHD-related deaths, and it estimates cardiovascular disease mortality. The endpoints assessed wereCardiovascular disease death (including CHD, arrhythmia, HF, stroke, aortic aneurysm, and peripheral vascular disease). Risk factors incorporated into model were age, sex, smoking, systolic blood pressure and region of Europe(high and low risk). There were a pair of chart, one based on cholesterol and another based on cholesterol/HDL cholesterol ratio. Diabetes mellitus, blood pressure treatment, and family history of CVDS were not included[20]

PROCAM study

Prospective Cardiovascular Münster (PROCAM) study recruited 5389 men aged 35 to 65 years of age from 1979 to 1985 and followed up for 10 years for acute coronary events. Cox proportional hazard model was developed using 8 independent risk variables, ranked in order: age, LDL cholesterol, smoking, HDL

cholesterol, systolic blood pressure, family history of premature myocardial infarction, diabetes mellitus, and triglycerides [48].

ASSIGN score

ASSIGN score was derived from 6540 men and 6757 women aged 30–74 years from Scottish Heart Health Extended Cohort (SHHEC), initially free of coronary heart disease, stroke, or transient ischemic attack. Participants were ranked according to social deprivation and followed for death due to cardiovascular causes or any hospital discharge diagnosis post recruitment (potentially several per admission) for coronary heart disease or Cerebrovascular disease or for coronary artery interventions (CABG). Separate model was developed for each sex using age, multiple deprivation index, family history, diabetes mellitus, cigarette smokers, cigarettes per day, systolic blood pressure, total cholesterol, HDL cholesterol. Score was tested against the Framingham score[22]

ORISK

The QRISK and the updated QRISK2 algorithms were developed to predict cardiovascular risk in patients from different ethnic groups living in England and Wales. Main outcome measures are myocardial infarction, coronary heart disease, stroke, and transient ischemic attacks. Risk factors were age, sex, smoking status, systolic blood pressure, ratio of total serum cholesterol to high density lipoprotein, body mass index, family history of coronary heart disease in first degree relative aged less than 60, area measure of deprivation with region of UK, and existing treatment with antihypertensive agent. Finally, this score was validated with Framingham score and ASSIGN score. QRISK2 more accurately identified those at risk than the modified Framingham/ATP III model in this population.[21].

Reynolds Risk Score

The Reynolds risk score for women was developed from a prospective cohort of nearly 25,000 American women without diabetes. The primary differences between the Reynolds risk score and most other risk estimations are the inclusion of family history of MI and high-sensitivity C-reactive protein (hs-CRP) as variables in the risk calculator. Thirty five potential variables were evaluated. Only 9 were included in best fitted model A age, systolic blood pressure, current smoking, apolipoprotein

B-100, high sensitivity CRP, apolipoprotein A-I, parental history of myocardial infarction before age 60 years, and 2 interaction terms, hemoglobin A1c if diabetes was present and lipoprotein(a) level if apolipoprotein B-100 was 100 mg/dl or higher. While clinically simplified model B (Reynolds score) uses age, HbA1C %, current smoking, systolic blood pressure, HDL and total cholesterol, high sensitivity CRP, parental history of MI < 60 years[49].

2.7 Comparison among different risk scores

An important component of multivariate risk models for the estimation of CVDS risk is that many of the risk factors (eg, age, hypertension, serum LDL-cholesterol) are recognized as producing a graded increase in risk[50]. In addition, these models estimate risk of an individual patient over the next 10 years, even though the risk models have been derived from large population-based studies. Several studies have suggested that the Framingham criteria either overestimate or underestimate the risk of initial coronary heart disease (CHD) events in other populations such as Japanese American and Hispanic men, Native American women, European and Asian populations, and African-American men and women, as well as patients older than age 85 years. How a risk score performs is largely dependent on population characteristics along with the presence or absence of primary preventive therapies to address relevant risk factors[51].

2.8 WHO/ISH risk prediction chart

A tool that enables cardiovascular risk assessment and prediction in non-Western populations has been developed. The World Health Organization/International Society of Hypertension (WHO/ISH) risk prediction charts enable the total risk stratification approach for management of CVDS to be introduced in WHO regions where cohort data and resources are not readily available for the development of population-specific risk prediction charts. The charts have been developed using a modeling approach[23]. In brief, a set of individual-level CVDS risk factor profiles (age, sex, current smoking, systolic blood pressure, total cholesterol and the presence or absence of type 2 diabetes) have been generated using information on the population distribution of these risk factors from the WHO Comparative Risk

Assessment study[25]. These risk factor profiles have then been combined with information on the relative risk of each risk factor, along with the population-level estimate of absolute risk. The accuracy and predictive value of current risk predictioncharts could be improved as more epidemiological data become available from individual countries.

For the prevention of CVDs to be cost effective, easy and more accurate prediction of an individual absolute risk of CVDSis essential, and the WHO/ISH risk prediction charts represent an important step forward in this area.

2.9 Cardiovascular risk factors

Most cardiovascular diseases possess constellation of risk factors. These risk factors have conventionally been classified as non-modifiable risk factors like age, gender, ethnicity and family history; modifiable behavioral risk factors like unhealthy diet, physical activity, tobacco use and harmful use of alcohol; and metabolic/physiological risk factors like raised blood pressure, diabetes, dyslipidemia and obesity. However, we have stratified them into primary, intermediate and immediate CVDS risk factors with respect to their nature.

2.9.1 Primary risk factors

Age, gender, family history of CVDs, religion and ethnicity are non-modifiable primary risk factors. With increase in age, there is high likelihood of developing CVDs. After 40 years of age, the lifetime risk of developing CHD is 49% for men and 32% for women. More than four out of five or 81% of the people dying from CHD are 65 years of age or older [52]. Similarly, heredity represents nature's involvement in putting people at risk for CVDS, while other primary risk factors like rapid unplanned urbanization, education, socio-economic status and occupation inversely nurture the dimension for developing the disease [53-56].

2.9.2 Modifiable intermediate risk factors

Tobacco consumption

About 1.3 billion people worldwide smoke any tobacco product today. Among them, about 84% live in developing and transitional economy countries[57]. Worryingly, the number of smokers continues to rise. On the other side, tobacco is

the fourth most common risk factor for disease and the second major cause of death worldwide. It is currently responsible for the death of one in ten adults worldwide (about 4.9 million deaths each year)[58]. If the current smoking pattern continues, it is estimated that deaths from tobacco consumption was about 10 million people per year by 2020[58]. Smoking is markedly attributable for multiple cancers, particularly lung cancer and is at far greater risk of heart disease, stroke, Chronic Obstructive Pulmonary Disease (COPD), diabetes, and other fatal and non-fatal diseases. People who chew tobacco risk cancer of the lip, tongue and mouth[59].

Alcohol consumption

Alcohol consumption is the leading risk factor for disease burden in low mortality developing countries and the third largest risk factor in developed countries[60]. The proportion of disease burden attributable to alcohol use in the developing world is between 2.6% to 9.8% of the total [61]. Besides the direct toxic effects of intoxication and addiction, alcohol use causes about 20% to 30% of each of esophageal cancer, liver disease, homicide, epileptic seizures, and motor vehicle accidents worldwide [60]. It also increases the risk of cardiovascular[62]. Alcohol consumption during pregnancy is related to various risks to the fetus, including Fetal Alcohol Spectrum Disorders, spontaneous abortion, low birth weight and prematurity, and intra-uterine growth retardation[63, 64].

Physical inactivity

Physical inactivity is a major risk factor in promoting obesity, which itself is a risk factor for other chronic diseases[65]. It causes about 1.9 million avoidable deaths per year worldwide[66] which accounts for 21.5% of ischemic heart disease, 11% of ischemic stroke, 14% of diabetes, 16% of colon cancer and 10% of breast cancer[67]. Physically inactive persons have a 20% to 30% increased risk of all-cause mortality as compared to those who adhere to 30 minutes of moderate intensity physical activity on most days of the week[65]. In contrast to this, physical activity may have a protective effect against development of cognitive impairment and dementia, and reduces severity of symptoms among the depressed[68, 69]. Physical activity is associated with the prevention of osteoporosis and related fractures[65].

Fruits, vegetables, fat and salt consumption

Low fruits and vegetable consumption is responsible for loss of 2.7 million lives and 26.7 million (1.8%) DALYs worldwide[58, 70]. Of the burden attributable to low fruit and vegetable intake, about 85% was from cardiovascular diseases and 15% from cancers[58]. Moreover, it is estimated to cause about 19% of gastrointestinal cancer, 31% of ischemic heart disease and 11% of stroke worldwide[58].

The consumption of at least 400g of fruit and vegetables per day is recommended as a population intake goal, to prevent diet-related chronic diseases[71]. Adequate consumption of fruit and vegetables reduces the risk for cardio vascular diseases, stomach cancer and colorectal cancer[71].

There is convincing evidence that high intake of high-energy foods such as processed foods high in fats and sugars promote obesity compared to low-energy foods such as fruits and vegetables[71]. Higher unsaturated fatty acids from vegetable sources and polyunsaturated fatty acids have been associated with a reduced risk of type 2 diabetes[72, 73]. They also lower coronary heart disease risk [74]. In the contrary, partial hydrogenation to increase the shelf life of poly unsaturated fatty acids creates trans fatty acids[71]. Such trans fatty acids increase the risk of coronary heart disease and render the plasma lipid profile even more atherogenic than saturated fatty acids by elevating LDL cholesterol and decreasing HDL cholesterol.

Obesity

Overweight and obesity lead to adverse metabolic effects on blood pressure, cholesterol, triglycerides and insulin resistance[58]. There is slightly increased risk of co-morbidities for BMI 25.0 to 29.9, and moderate to severe risk of co morbidities for BMI greater than 30 [75]. With increasing degree of BMI, risks of coronary heart disease, ischemic stroke and type 2 diabetes mellitus also increase [58] resulting in high Mortality rates [71]. At least 2.6 million people die each year as a result of being overweight or obese [70]. WHO has recommended the median BMI for an adult population should be in the range of 21 to 23 kg/m2, while the goal for individuals should be to maintain BMI in the range 18.5 to 24.9 kg/m2 to achieve optimum health [70]. More than BMI, waist circumference is an approximate index

of intra-abdominal fat mass and total body fat. Changes in waist circumference reflect changes in risk factors for cardiovascular disease and other forms of chronic diseases [71].

High blood pressure

Raised blood pressure is a major risk factor for coronary heart disease and ischemic as well as hemorrhagic stroke[71]. Blood pressure levels have been shown to be positively and continuously related to the risk of stroke and coronary heart disease[76]. The risk of cardiovascular disease doubles for each increment of 20/10 mm Hg of blood pressure, starting as low as 115/75[77]. Complications of raised blood pressure include heart failure, peripheral vascular disease, renal impairment, fundal hemorrhages, and papilledema[78]. Treating systolic blood pressure and diastolic blood pressure to targets that are less than 140/90 is associated with a decrease in cardiovascular complications[79]. Stage 1/Grade 1 hypertension, is defined in a clinical setting when the mean blood pressure is equal to or above 140/90 and less than 160/100 on two or more measurements on each of two or more visits on separate days[71, 77, 80]. Stage 2/Grade 2 hypertension is defined in a clinical setting when the mean blood pressure is equal to or more than 160/100 and less than 180/110 on two or more measurements on each of two or more visits on separate days[71, 77, 80]. Stage 3/Grade 3 hypertension is defined in a clinical setting when the mean blood pressure is equal to or more than 180/110 during two or more measurements on each of two or more visits on separate days[71, 77, 80].

Raised blood glucose

It is predicted that there was at least 366 million people in the world with diabetes by the year 2030 [81]. The excess mortality attributable to diabetes in the year 2000 was estimated to be 2.9 million deaths, equivalent to 5.2% of all deaths. In people 35-64 years old, 6-27% of deaths were attributable to diabetes [82].Impaired glucose tolerance and impaired fasting glycaemia are risk categories for future development of diabetes and cardiovascular disease [83]. The age-adjusted mortality, mostly due to coronary heart disease in many populations, is 2-4 times higher than in the non-diabetic population [84]. People with diabetes have a twofold increase risk of stroke [85]. It is also the leading cause of renal failure in many populations in both developed and developing countries [86]. Lower extremity amputations are at least

10 times more common in people with diabetes than in non-diabetic individuals in developed countries, and more than half of all non-traumatic lower limb amputations are due to diabetes [87]. People with diabetes require at least 2-3 times the health care resources than people who do not have diabetes, and diabetes care accounts for up to 15% of national healthcare budgets [88].

Diabetes mellitus substantially increases the risk of developing coronary, cerebrovascular, and peripheral arterial disease. The pathophysiology of vascular disease in diabetes involves abnormalities in endothelial, vascular smooth muscle cell, and platelet function. The metabolic abnormalities that characterize diabetes, such as hyperglycemia, increased free fatty acids, and insulin resistance, each provoke molecular mechanisms that contribute to vascular dysfunction that increase the propensity to thrombosis, contribute to atherosclerosis and its complications[89].

Raised lipid

Raised total cholesterol is a major cause of disease burden in both the developed and developing world as a risk factor for Ischemic heart disease and Stroke [90]. It was estimated to cause 18% of the global cerebrovascular disease and 56% of global ischemic heart disease. Overall this amounts to about 4.4 million deaths (7.9% of total) and 40.4 million DALYs (2.8% of total) [58]. A 10% reduction in serum cholesterol in men aged 40 can result in a 50% reduction in heart disease within 5 years, while an average of 20% reduction in heart disease occurs within 5 years in men aged 70 years [14]. A 4.6% reduction of population mean of total cholesterol had the greatest impact of all risk factors in decreasing CHD mortality in Ireland; a full 30 % reduction in mortality was attributable to this reduction alone [91]. Plasma HDL cholesterol is inversely related to coronary artery disease incidence, and the relationship is independent of total cholesterol, LDL and triglyceride levels [92]. Increased triglycerides is an independent risk factor for coronary heart disease after controlling for LDL and HDL cholesterol [93].

CHAPTER 3: METHODOLOGY

This chapter contains:

- 3.1 Study design
- 3.2 Study site
- 3.3 Study Period
- 3.4 Study Population
- 3.5 Inclusion Criteria
- 3.6 Exclusion criteria
- 3.7 Sample Size
- 3.8 Sampling Technique
- 3.9 Data Collection Procedure
- 3.10 Data collection techniques
- 3.11 Data collection tools
- 3.12 Validity and Reliability
- 3.13 Pre-testing
- 3.14 Data Quality control
- 3.15 Data Processing and Logical Checking
- 3.16 Data analysis
- 3.17 Ethical consideration

3.1 Study design

This was community based cross-sectional study conducted among population aged 40 to 80 years residing in rural Community of Nepal.

3.2 Study site

Study conducted in two VDCs of Lamjung District of Nepal, 100 kilometer far from Kathmandu. Lamjung is one district among 75 districts of Nepal, covers an area of 1,692 km² and has a population of 140,266 according to census of 2011. There are 61 Village Development Committees (VDCs) in this district. Total population of Two VDCs Bhotewodar (7560) and Sunderbazar (6715) was 14275. Most of the population in that community involved in agritulral works. In Lamjung 31% (52,421) population are Gurung and 7.2 % (12,121) are Tamang 2.2 % (3,757) are Magar[94] who are exposed to risky behavior culturally. (Annex VI)

3.3 Study Period

Study period of this thesis work was July 2014 to August 2015. (Annex VIII)

3.4Study Population

All male and female population of 40 to 80 years of age who do not have clinically manifest cardiovascular diseases.

3.5 Inclusion Criteria

- Age 40 to 80 years
- Without established MI, angina, stroke, Intermittent claudication
- Permanent resident of study area for at least 6 months

3.6 Exclusion criteria

- Pregnant women
- Mentally retarded persons

3.7 Sample Size

Sample size calculated using formula for estimation of proportion for one sample situation. Calculated sample size was 343. Here, P=9.5 % was taken for the proportion of population who were moderate to high risk category in the study done by Koju R et al in suburban population of Nepal[95].

Samplesize (n) =
$$Z^2pq/d^2 = 207$$

where we considered

- 5 % level of significance, z= 1.96
- Allowable error (d) =4%
- Expected prevalence of 9.5 %, p = 0.095, q = 0.905
- Correction for finite population: n/(1+n/N) = 195
- Design effect 1.5 and non-response rate 25%
- Total sample size was 388

3.8 Sampling Technique

Sampling was done in multistageas follows.

- a) Cluster selection: Among 18 wards 4 wards (25%) selected according to probability proportion to size (PPS), where ward was primary sampling unit (PSU). To select the ward, we created a list of wards from both VDCs with their population of age 40 to 80 using national census 2011 data. We put another column containing cumulative size of wards. In this study, sampling interval was 928 obtained after dividing total study population (3712) by required no of sampling units. After taking 928 as a random number, ward number eight of Bhotewodar selected first. Again, other wards selected after adding 928 in that random number. Thus, ward number eight and nine of Bhotewodar and four and nine of Sunderbazar selected for primary sampling units or clusters.
- **b)** Household selection: From four wards required household number decided according to population proportion to size of total population. Thus168 households in Bhotewodar eight, 103 in Bhotewodar nine, 84 Sunderbazar four and 33 in Sunderbazar nine selected. First household of the ward was taken systematically. First one corner was visited randomly. Then nearby road and then right or left side

of the road was chosen randomly. The first household of that road was visited. Then every 6th household was taken as study household with interval of five.

c) Participant selection: From each household one member who met the inclusive criteria selected using KISS table. (Annex V)

d) Flow Chart of sampling technique

- Bhotewodar and Sunderbazar
- total population=14275 with total wards 18
- study pop(40-80 yrs)=3712
- Four ward selected using probability to proportion method
- Required household was decided by population proportion to size.
- Household was visited using systematic random sampling (interval of 5)

Bhotewodar 8
Total HH-960
Selected HH-168
Bhotewodar9
Total HH- 960
Selected HH168
Sunderbazar 4
Total HH- 460
Selected HH84
Sunderbazar 9
Total HH- 177
Selected HH33

- From each household one member who meets the inclusive criteria wasselected using KISS table. Total sample size will be 388.
- Total sample attended for fasting blood sugar and fasting lipid profile was 345.
- Total sample attended for postprandial blood sugar was 327

3.9 Data Collection Procedure

- a) Training conducted for data collector on data collection tools and techniques.
- b) Data was collected in two phases. In first phase interviewer visited the household decided by systematic random selection. They explained the purpose of study, benefit of the study, inclusion and exclusion criteria. To exclude established cardiovascular diseases interviewer asked whether they were said by the physician that they have heart problem or *mutukosamasya*, stroke or *pakhshyaghat*, and blood vessel disease or *raktanalikoroga*. They asked whether they were taking drugs for such diseases. They asked about state of pregnancy and duration of stay in the community. Interviewer then listed all the members who can be included in the study in the KISS table to select one member. Written informed consent was taken using consent form. Information for step one of the questionnaires gathered using face to face interview. Then interviewer finished physical measurements. Interviewer provided information slip of ID no, respondents name with the date, time and place for blood sample collection to the respondents and requested to fast from eight pm to eight am of the blood sample collection date.
- c) In second phase trained technician went to the blood sample collection site. First technician conformed overnight 12 hours fasting. Participant requested to sit on chair and venous blood (volume 4 ml) was taken using flashback needle under aseptic precaution by trained technician and kept in plain and fluoride treated sterile tubes separately. Technician tagged the respondent's ID no on the test tubes and sent the sample to the laboratory of the hospital in Ice pack within two hours.

Then participants requested to drink 75 gm of anhydrous glucose mixed with 250 ml of water within 5 min. They requested to come after 2hrs of taking glucose. Another 1 ml venous blood was taken, put in fluoride treated tube, labelled and sent to the laboratory in ice packing within two hours. Laboratory analysis was done in laboratory of Karmada Hospital Pvt. Lt. Lamjung, using semi-automated machine of Erba Mannheim (Germany), chem5 v3 model. In the laboratory blood sample stored at 0-4 degree centigrade.

d) After finishing analysis, participants wereinformed about their physical measurements and fasting blood sugar, OGTT 2 hour postprandial blood sugar in respondent feedback form. (Annex VI)

3.10 Data collection techniques

Interview

Face to face Interview taken by trained interviewer using Standard STEPs instrument version 2.2.

Physical measurements

All physical measurementswere taken in a separate room. Blood pressure, height, weight, waist circumference, and hip circumference were then measured in sequence.

Blood pressure measurement

For measuring blood pressure doctors, sphygmomanometer and stethoscopewere usedwith appropriate sized cuff. First reading was taken after requesting participants to take 15 min rest crossing leg. Second and third reading was taken at three minutes rest after each reading. Sphygmomanometer was placed on unclothed left arm above the elbow while putting the arm in the table facing palm up.

Height measurement

Height was measured in portable standard height measuring scale. Height was measured without head gear or foot bear. The participants was requested to stand on the flat surface, with straight knee, feet together and heels against the backboard. It was made sure that respondent was looking straight to the interviewer, not tilt their head up and eyes and ears at same level.

Weight

Weight was measured using portable digital weighing scale. The scale put in firm flat surface. The participant requested to step onto the scale only wearing light cloths removing foot wear and socks. Then standing straight, facing forward and placing arms on the side.

Waist and hip circumference measurement

Waist and hip circumference using a constant tension tape. A private area, such as a separate room within the house, was used and the measurement was taken over light clothing. Waist circumference was taken at the end of a normal expiration with the arms relaxed at the sides at the midpoint between the lower margin of the last

palpable rib and the top of the iliac crest (hip bone). Hip circumference was at the maximum circumference over the buttocks. Participants was requested to wrap the tape around themselves. Measurement was taken at the nearest 0.1 cm, making sure to keep the measuring tape snug.

Biochemical measurements

Plasma glucose was estimated by Trinder's Method using reagent GOD-PAP (glucose oxidase/peroxidase – phenol-4-amenoantipyrine). Serum total cholesterol was determined by anCHOD-PAP method using CHOD-PAP (cholesteroloxidase/peroxidase – 4-phenol-aminoantipyrine). Serum triglyceride was estimated using the GPO-PAP (glycerol-3-phosphate oxidase/peroxidase-4-chlorophenol and 4-aminophenazone) method. HDL cholesterol was determined by Phosphotungstic Acid method. For this LDL, VLDL cholesterol the chylomicron fraction from the serum samples was first precipitated out. The clear supernatant was then analyzed using cholesterol reagent.

3.11 Data collection tools

STPEPs Instrument version 2.2: This is the standardized tool focuses on collecting core data about established risk factors of noncommunicable diseases including CVDs. In Step one demographic data, socio-economic data, information on tobacco and alcohol use, some measures of nutritional status, physical activity level and history of blood pressure and diabetes were gathered. In step two height, weight, waist and hip circumference were collected. In step three, biochemical measurements were recorded. (Annex II and III)

Risk Prediction Chart: WHO/ISH risk prediction chart for SEAR D used to stratify individual into different categories for 10 years cardiovascular risk. This chart estimate 10 year risk of a fatal or non-fatal major cardiovascular events according to age, sex, blood pressure, smoking status, total cholesterol and presence or absence of diabetes for SEAR D of WHO epidemiological sub region. The chart approximately estimatescardiovascular risk in people who do not have established coronary heart disease, stroke or other atherosclerotic diseases.(Annex IV)

Measuring tape, Weighing Machine, Sphygmomanometer, Laboratory instrument were other tools used in the study.

3.12 Validity and Reliability

STEPS questionnaires was already tested and translated used in Nepal before the current study.

3.13 Pre-testing

For pre testing of datawas collected in 15 samples from ward number five of Bhotewodar VDC using the tool by trained interviewer and blood sample collected by qualified and trained technician. On completion of pre-testing, feedback was taken from respondents and then instrument was refined necessarily.

3.14 Data Quality control

One team leader was nominated and controlled sheet was prepared for proper monitoring. Periodic field visit was done to the study sites at the time of implementation. Team leader checked the collected data and provided instant feedback if any problem was identified.

3.15 Data Processing and Logical Checking

Data entry, compilation, edition and checking was done in EpiData to maintain consistency. Repetitions and omissions of data were corrected before coding and entering them in SPSS. Recorded data was then exported to SPSS V.16.0 for further analysis.

3.16 Data analysis

Descriptive statistics used to identify the distribution of risk factors among sociodemographic characteristics. Prevalence of individual risk factors was estimated in male, female and in both sex.Individual 10-year risk was computed using the chart separately. Then total risk was coded into mild, moderate, high risk and very high-risk category. Finally, high risk and very high risk was merged into high risk for further analysis. Again, 10-year risk was estimated after adding the population with blood pressure 160/100 mmHg with the chartaccording.Mild risk

was considered as no risk, moderate and high risk was considered as risk population to determine the distribution of 10 year risk in relation to socioeconomic condition. Chi-square and independent t-test wasconducted for comparing proportions of categorical and mean of continuous variables. All tests were two tailed test and p<0.05 was considered statistically significant. Data presented in frequency tables, diagrams and bar.

3.17 Ethical consideration

At first, ethical review committee of Nepal Health Research Council gave approval for ethical clearance of the protocol. Written informed consent was takenusing a consent form in Nepali language before enrolling the respondent. Title, objective, target population, procedure of data collection, benefit of the study, risk of study, confidentiality and anticipated use of the results were explained in the written consent form(Annex I). Everythingwas clarified in brief by the enumerator before consent. All respondents were informed that they were free to leave or refuse to take part in the study at any time. Confidentiality of the respondent was maintained using separate code for each data collection sheet. Data was entered separately for personal information and rest of other information. Data was analysed using the code so that nobody can identify them.

CHAPTER 4: RESULTS

This chapter includes

- 4.1 Sociodemographic characteristics of the study subjects
- 4.2 Behavioral risk factors of the study subjects
- 4.3 Distribution of family history of CVDs and premature menopause among study subjects
- 4.4 Blood pressure and BMI status of the study subjects by gender
- 4.5 BMI status of study of study subjects
- 4.6 Blood sugar of study subjects
- 4.7 Bloodcholesterol of study subjects
- 4.8 Total 10-year CVDS risk using WHO/ISH chart alone
- 4.9 Total CVDs risk distribution after inclusion of other risk factors
- 4.10 Distribution of total ten year CVDS risk by socioeconomic status of study subjects

4.1 Sociodemographic characteristics of the study subjects

Of the 388 participants selected for the study 345 attended for the biochemical measurements and 327 of these attended for post prandial blood sugar level measurement. Dropout rate of participants for biochemical measurement was 11 % and from fasting to postprandial was 4.6 %. Data available for analysis was 388 for sociodemographic, behavioral and physical measurements, and 345 for biochemical analysis and 327 for postprandial blood glucose.

Of the 388 participants, 44.6 % were male and 55.4 % were female. Mean age was 53.4 years with standard deviation of 10.2 years with similar age between male and female. Fourty percent of respondants were in the age group 40 to 49 years.

Participants had different level of education (p<0.01). The highest number of participants (53.9 %) did not have formal education. Different ethnic/ cast people attended the study with highest percentage of participants from gurung community (33.2 %). Most of the participants were working as house maker (57.2 %) and then self employed (22.2 %). Occupations were different among male and female (p<0.001). Almost all participants (91.5 %) had poor economic status. (Table1).

Table 1: Sociodemographic and economic characteristics of respondents

Characteristics	Male(n=173) n(%)	Female(n=215) n(%)	Total (n=388) n(%)	P value			
Age in years							
40-49	74(42.8)	84(39.1)	158(40.7)				
50-59	53(30.6)	65(30.2)	118(30.4)	0.42			
60-69	35 (20.2)	42 (19.5)	77(19.8)	0.42			
70-79	11(6.4)	24(11.2)	35(9)				
$Mean \pm SD^{\#}$	53±9.7	53.6±10.5	53.4±10.2	0.54			
Level of education [¥]							
No formal schooling	34(19.7)	175(81.4)	209(53.9)				
<pre><pre><pre><pre>primary school</pre></pre></pre></pre>	23(13.3)	15(7)	38(9.8)				
Primary school	32(18.5)	7(3.3)	39(10.1)	0.001*			
Secondary school	43(24.9)	12(5.6)	55(14.2)				
< secondary school	41(23.76)	6(2.8)	47(12.1)				
Cast [¥]							
Bhramhan	45(26.0)	41(19.1)	86(22.2)				
Kshetri	32(18.5)	35(16.3)	67(17.3)				
Gurung	45(26.0)	84(39.1)	129(33.2)				
Dalit	14(8.1)	8(3.7)	22(5.7)	0.01*			
Tamang	4(2.3)	15(7.0)	19(4.9)				
Newar	24(13.9)	11(5.1)	45(11.6)				
Other	9(5.2)	21(9.8)	20(5.2)				
$Occupation^{\Psi}$							
Employed	35(20.3)	8(3.8)	43(11)				
Self-employed	61(35.3)	25(11.6)	86(22.2)				
Unemployed	4(2.4)	3(1.4)	7(1.8)	0.001*			
Homemaker	45(26)	177(82.3)	222(57.2)				
Retired	28(16.2)	2(0.9)	30(7.7)				
Economic status ⁴							
Poor economic status	152(39.2)	203(52.3)	355(91.5)				
Medium economic status	18(4.6)	11(2.8)	29 (7.5)	0.06			
High economic status	3(.8)	1(.3)	4(1)				

Chi-square test was done, Fischer-exact test independent t-test for mean difference, p<0.05 considered as level of significance and, *=significant

4.2 Behavioral risk factors of the study subjects

Smoking:

One-fourth (24.8%) participants were current smoker (table 2). Of current smoker, 86.5% were daily smokers. Of the female participants, 19.5% were smoker whereas 31.4% male were smoker. Smoking was 28.6% after considering smoker who

quitted within one year as used in the WHO/ISH chart for risk prediction. Minimum age of initiation smoking was 10 years and mean age of initiation of smoking was 18.9±6.5 years. Out of daily smokers, average consumption of manufactured ciggarets was 10 sticks per day. Of current smoker 42.5% did not try to stop smoking eventhough, 52.3% got advice from the health professionals. Among all the participants, 18.6% also used smokeless tobacco.

Alcohol:

Current alcohol users who drank alcohol within 30 days were 26.2 % where almost half (49.7%) of male but 7.4 % female were alcohol user (table 2).Harmful use of alcohol (male \geq 5, female \geq 4 standard drink) was 53.9 % among current alcohol users. Life time abstainers were 59.8 %. Average standard drink per day was 5.8 \pm 4.3. Males were drinking more drinks (6.3 \pm 4.4, p=0.006) than female. Among the current users, average drinking per month was 15.5 \pm 12.7 days during last month ranging from daily intake to one day per month.

Fruit and vegetable intake:

Inadequate intake of fruit and vegetable (<5 servings per day) was 72.4 % which was 76.3% among male and 69.3% among female (table 2). On average, they consumed 3.7±1.4 servings of fruit and vegetable per day. They consumed vegetable almost daily (6.1±1.8) where as fruit consumption was only 3±1.9 days per week.

Physical inactivity:

Low level of physical activity (<600 metabolic equivalent-minutes per week) was 11.6 % where 14.9% females and 7.5% male had low level of physical activities (table 2). Low level of physical activity was highest among self-employed (14%) and then among the homemakers (12.2%). They spent 375 \pm 169 minutes in sedentary activities on average per day.

4.3 Family history of CVDs and premature menopause among study subjects

Family history of cardiovascular diseases in first degree relatives was 7.5%. Male participants have higher family history (8.1%) than female participants(7%). Family history of CVD at age of <55 years was 0.02% and at age < 65 years was 0.04%. One tenth of female (10.2%) had premature menopause at <40 years of age(table 2)

Table 2: Prevalence of behavioral risk factors of cardiovascular diseases by gender

Behavrioral variables	Male	Female	Total	_
	(n=173)	(n=215)	(n=388)	P
	n (%) 95% C.I	n (%) 95% C.I	n (%) 95 %C.I	value
Current smoking				
No	119(68.6)	173(80.4)	292(75.2)	
Yes	54(31.4) (26.8-36)	42(19.5) (15.6-23.4)	96(24.8) (20.5-29.1)	0.007*
Current alcohol use	(20.0 20)	(10.0 20.1)	(20.0 25.1)	
No	87(50.3)	199(92.6)	286(73.7)	
Yes	86(49.7) (44.7-54.7)	16 (7.4) (4.8-10)	102 (26.2) (21.8-30.6)	0.17
Fruit and vegetable intake	,	, ,	,	
Adequate	41(23.6)	66(30.7)	107(27.6)	
Inadequate	132(76.3) (72.1-80.5)	149(69.3) (64.7-73.9)	281 (72.4) (68-76.8)	0.12
Mean (servings)±SD#	3.6±1.5	3.7±1.4	3.7±1.4	0.001*
Level of physical activity				
Sufficient	160(92.5)	183(85.1)	342(88.4)	
Insufficient(low)	13(7.5) (4.9-10.1)	32 (14.9) (11.4-18.4)	45 (11.6) (8.4-14.8)	0.009
Family history	(12 21)	(' ' ' ' '	(======================================	
No	149(91.9)	200(93)	359(92.5)	
Yes	14 (8.1) (5.4-10.8)	15(7) (4.5-9.5)	29(7.5) (4.9-10.1)	0.7
Premature menopause	·	22 (10.2)	22 (10.2)	
(female only)		6.2-14.2	6.2-14.2)	

Chi-square test was done with p=0.05 as level of significance, # independent t-test for mean difference with significance level of p=0.05, *= significant

4.4 Blood pressure status of the study subjects

Hypertension due to raised systolic blood pressure including those on medication for raised blood pressure was 19.8 %. Hypertension due to raised diastolic blood pressure ≥90 mmHg was 24.5%. Grade one hypertension was 14.6%, second grade hypertension was 3.6% and third grade hypertension was 1.8%. Hypertension due to raised diastolic blood pressure was 38.8% among male and 18.6% among female (p=0.003) (table 3). Hypertension with raised BP (SBP≥140 and/or DBP≥90 mmHg or currently on medication for raised BP) was 40.5%. Mean Systolic BP including currently on medication was 124.2 ± 18.4 mmHg. Mean Diastolic BP including currently on medication was 82.3±33.6 mmHg. Among all, 18% population was taking antihypertensive drug.

4.5 BMI status of study subjects

Of the the total participants, 46.6% were overweight and 12.9% were obese. Overweight among male was 49.7% and among female was 44.2%. Obesity was 15.3% among male and 9.8% among female. As a whole, overweight and obesity were equally distributed among male (59.5%) and female (59.5%). Mean BMI was 25.9±4.1 Kg/m2. (Figure 1 and table 3)

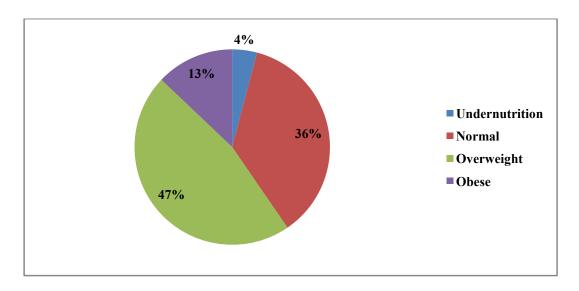


Figure 1: Percentage Distribution of BMIby international classification

Mean waist circumference among male was 92 ± 12 cm where asamong female was 86.5 ± 12.8 cm. Abnormal waist circumference (Male >102cm, Female >88cm) was 32.7 % among male was 16.8% but among female was 45.6 % (table 3).

Table 3: Distribution of blood pressure and BMI by gender of study subjects

Variables	Male(n=173)	Female(n=215)	Total(n=388)	P
	n (%)	n (%)	n (%)	value
Blood pressure				
Normotensive	95(54.9)	136(63.3)	231(59.5)	
95% C.I	49.9-59.9	(58.5-68.1)	(54.6-64.4)	
Hypertension	78(45.1)	79(36.7)	157 (40.5)	0.09
95% C.I	(40.1-50.1)	(31.7-41.5)	35.6-45.4	
Mean SBP±SD(mmHg)	126±18	122±18	124±18	0.03#
Mean DBP±SD (mmHg)	84±33	81±33	82±33	0.43
BMI				
Overweight	102(59.5)	128(59.5)	231 (59.5)	
95% C.I	(54.6-64.4)	(54.6-64.4)	54.6-64.4	0.12
Obesity	17 (9.8)	33 (15.3)	50 (12.9)	
95% C.I	(6.8-12.8)	(11.7-18.9)	(9.6-16.2)	
Mean BMI (Kg/m2)±SD	26±3.5	26±4.5	26±4	0.767
Waist circumference				
Raised	29(16.8)	98(45.6)	127(32.7)	0.001*
95% C.I	(13.1-20.5)	(40.6-50.6)	(28-37.4)	0.001
Mean (cm) ±SD	92±12	86±13	59±12	0.001*

Chi-square test was done with p=0.05 as level of significance, # independent t-test for mean difference with significance level of p=0.05, *= significant

4.6 Blood sugar of study subjects

Raised blood sugar defined by fasting blood glucose \geq 126 mg/dl including those currently on medication was 7.8 %. Raised fasting blood sugar level was higher in male (8.4%) than in female (7.3%). Raisedpostprandial blood sugar measured two hours after oral glucose was 8.3%among those who were non-diabetic before the study. Glucose level after OGTT was same in both male and female.

Prevalence of diabetes mellitus was 16.2% including fasting and postprandial blood suger level along with medication for diabetes. Newly detected diabetes mellitus by OGTTwas 1.7% whose fasting blood glucose was normal. Higher female (2.1%) were detected as diabetic than male (1.3%) after screening by OGTT. Proportion of impaired fasting glucose was 4.3% and impaired glucose tolerance was 8.3%. (table 4)

Table 4: Distribution of blood sugar by gender of study subjects

	Male(n=154)	Female(n=191)	Total(n=345)	P
	n (%)	n (%)	n (%)	value
Fasting Blood sugar [¥]				
Normal	134(87)	169(88.5)	303(87.8)	
IFG C.I DM	7 (4.5) (2.3-6.7) 13(8.4)	8 (4.2) (2.1-6.3) 14 (7.3)	15 (4.3) (2.2-6.4) 27 (7.8)	0.91
C.I	(5.5-11.3)	(10-4.6)	(5-10.6)	
$Mean(md/dl) \pm SD$	92±24	94±40	93±34	0.001*
Post Prandial Blood Sugar [¥] (n=327)				
Normal	115 (79.9)	158(86.3)	273(83.5)	
IGT	17 (11.8) (5.3-11.3)	10 (5.5) (5.3-11.3)	27 (8.3) (5.3-11.3)	0.11
DM	12 (8.3) (5.3-11.3)	15 (8.3) (5.3-11.3)	27 (8.3) (5.3-11.3)	
Mean (mg/dl)± SD (n=327)	128±53	123±56	125±56	0.45
DMwith normal FBS	2(1.3)	4 (2.1)	6 (1.7)	0.84
C.I (n=327)	(0.1-2.5)	(0.6-3.6)	(0.3-3.1)	
DM(FBS+PPBS+medication) (95%C.I	25 (16.2) (12.3-20.1)	31 (16.2) (12.3-20.1)	56 (16.2) (12.3-20.1)	0.99

Chi-square test was done with p=0.05 as level of significance, *fischer exact test, # independent t-test for mean difference with significance level of p=0.05, *= significant

4.7 Blood cholesterol of study subjects

Of the total, 17.1 % had raised total cholesterol. Male (19.5%) had higher prevalence of raised total cholesterol than female (15.2%). Mean total cholesterol was 165±18.8 mg/dl. Raised triglyceride level more than 150 mg/dl 27.2% where higher male (34.4%) than female (21.5%) were affected.

Raised triglyceride level more than 150 mg/dl was 27.2% where higher male (34.4%) than female (21.5%) were affected. Raised triglyceride level more than 200 mg/dl was 11.6%. Male (17.5%) were more than two times more affected than female (6.8%) (p=0.002). Mean triglyceride level was 129±77.9 mg/dl. Similarly, raised low-density lipoprotein (more than 100 mg/dl) was 33%. Prevalence of raised LDL was 39% in male and 28.3% in female with mean level of 86±34.3 mg/dl. Lastly, decreased high-level lipoprotein was 46.7%. Higher prevalence of HDL among male (47.4 %) in comparision of female (46.1%). Mean HDL is 53.2±12.5 mg/dl in both the sexes(Table5).

Table 5: Distribution of blood cholesterol by gender of study subjects

Variable	Male(n=154) n (%)	Female(n=191) n (%)	Total(n=345) n (%)	P value	
Total cholesterol					
Normal	124(80.5)	162(84.8)	286(82.9)		
Raised	30 (19.5)	29(15.2)	59 (17.1)	0.18	
95% C.I	(15.3-23.7)	(11.2-19)	(13.1-21.1)		
Mean [#] (mg/dl)±SD	170 ± 34.4	160±34.8	165 ± 18.8	0.001*	
Triglyceride					
Normal	101(65.6)	150(78.5)	251(72.8)		
Raised	53 (34.4)	41 (21.5)	94 (27.2)	0.08	
95% C.I	(29.4-39.4)	(17.2-25.8)	(22.5-31.9)		
<200 mg/dl	127(82.5)	178(93.2)	305(88.4)		
Raised >200 mg/dl	27(17.5)	13(6.8)	40 (11.6)	0.002^{*}	
95% C.I	(13.5-21.5)	(4.1-9.5)	(8.2-15)		
Mean (mg/dl) #±SD	141±93.3	119±61.4	129±77.9	0.001*	
High Density Lipoprotein					
Normal	81(52.6)	103(53.9)	184(53.3)		
Decreased	73 (47.4)	88 (46.1)	161 (46.7)	0.8	
95% C.I	(42.1-52.7)	(40.8-51.4)	(41.4-52)		
Mean (mg/dl) ±SD [#]	53±12.3	53.3±12.7	53.2±12.5	0.001*	
Low Density Lipoprotein					
Normal	94(61)	137(71.7)	231(67)		
Raised	60 (39)	54 (28.3)	114 (33)	0.036^{*}	
95% C.I	(33.9-44.1)	(23.5-33.1)	(28-38)		
Mean LDL±SD [#]	89.3±35.2	83.4±33.5	86±34.3	0.001*	

Chi-square test was done with p=0.05 as level of significance, # independent t-test for mean difference with significance level of p=0.05, *= significant

4.8 Total 10-year CVD risk using WHO/ISH chart alone

Ten-year risk was calculated using 345 samples. The main outcome of the study is proportion of 10-year risk among participants. Table 6 shows distirbution of total CVDs risk as per WHO/ISH charts. The majority of people had low (<10 %) 10 years risk. Low CVDs risk was 86.4 % (95 % confidence interval was 82.8%-90 %). Moderate risk was 9.3% (95% confidence interval is 6.2-12.4%). Female were

higher (10.5%) than male (7.8%) in moderate risk. High risk was 4.3% (95% confidence interval is 2.2-6.4%) in both sex. High risk was higher in male (4.5%) than in female (4.2%). Among high risk 2.3% were in 20-30% risk, 1.5% were in >30% risk.(table6).

Table6: Distribution of 10-yearCVDs risk by age and gender of study subjects

Variables	Mild risk	Moderate risk	High Risk	P value
	(<10 %)	(10-20 %)	(≥20 %)	
	n (%)	n (%)	n (%)	
Total (n=345)	298(86.4)	32(9.3)	15(4.3)	P value
C.I	(82.8-90)	(6.2-12.4)	(2.2-6.4)	
Sex				
<i>Male (n=154)</i>	135(87.7)	12(7.8)	7(4.5)	0.69
Female	163(85.3)	20(10.5)	8(4.2)	
(n=191)	103(83.3)	20(10.3)	0(4.2)	
Age group [¥]				
40-49 (n=137)	135(98.5)	1(0.7)	1(0.7)	0.001
50-59(n=105)	99(94.3)	4(3.8)	2(1.9)	
60-69(n=71)	51(71.8)	13(18.3)	7(9.9)	
70-79(n=32)	13(40.6)	14(43.8)	5(15.6)	

Chi-square test was done, ^{*}= fischer exact test, *= significant, p=0.05 as level of significance

Among sex 10.5% moderate risk found in female where as 7.8 % moderate risk in male. But high risk was 4.5% in male and 4.2% in female (table 6).

Risk was higher in older age group. Moderate risk proportion was 0.7 % among 40-49 year age, which was 3.8% among 50-59 years of age. Risk was almost five times increased from 50-59 years (3.8%) to 60-69 years (18.3%). Risk was 43.8% among 70-79 years age group, two times higher than 60-69 years age (18.3%). Similarly, high risk proportion was 0.7% among 40-49 years age, 1.9% among 50-59% years of age. Risk was five times higher among 60-70 years than preceding age group. Among 70-79 years age risk was 15.6%, one and half times higher than 60-69 years age group.

4.9 Total CVDs risk distribution after inclusion of other risk factors

Proportion of population under risk for development of CVDs within 10 years were 13.6% (>10% risk) with 95% confidence interval of 10%-17.2 % after simple application of chart only. Higher female (14.7%) were in risk than male (12.3 %). As explained in the WHO pocket guideline for assessment and management of cardiovascular risk, after adding population who had raised blood pressure ≥160/100 mmHg then proportion of population under risk became 15.7% where 16.8% female were in risk and 14.3% male were in risk. Addition of participants with raised DBP ≥100 mmHg, risk was 18% (male 19.5%, female 16.8%). After considering hypertensive population with blood pressure of >160/100 mm Hg as high risk category, total population with high risk will be 8.7 %. There were no participants who had total cholesterol >320 mg/dl, Low-density lipoprotein>240 mg/dl and TC/HDl ratio> 8 (table 7).

Table 7: Distribution of CVDs risk by gender after including study subjects with high blood pressure

Variab	Mild risk			Moderate to high risk		
les	Male	Female	Both sex	Male	Female	Both sex
	n(%)	n(%)	n(%)	n(%)	n(%)	n(%)
	95% C.I	95% C.I	95% C.I	95% C.I	95% C.I	95% C.I
Chart	135(87.7)	163(85.3)	298(86.4)	19(12.3)	28(14.7)	47(13.6)
only	(84.2-91.2)	(81.6-89)	(82.8-90)	(8.8-15.8)	(11-18.4)	10-17.2
Chart+	122(05.7)	150(92.2)	201(04.2)	22(14.2)	22(16.9)	54(15.7)
SBP	132(85.7)	159(83.2)	291(84.3)	22(14.3)	32(16.8)	54(15.7)
>160	(82-89.4)	(79.3-87.1)	80.5-88.1	(10.6-18)	(12.9-20.7)	11.9-19.5
Chart	124(90.5)	150(92.2)	283(82)	30(19.5)	22(16.9)	62(18)
+SBP+	124(80.5)	159(83.2)	(77.9-	(15.3-	32(16.8)	(13.9-
DBP	(76.3-84.7)	(79.3-87.1)	86.1)	23.7)	(12.9-20.7)	22.1)

4.10 Distribution of total ten year CVDs risk by socioeconomic status

Risk of CVDs was different according to level of education (p=0.01). Highest risk was 18.4 % among the participants having no formal education. Among participants having primary school level education, risk was 10% (table 8). Of participants who had, education of secondary and above secondary level, risk was 6.7%. Risk was highest among male (23.3 %) in comparision to female (17.4%) participants without formal education (Figure 2).

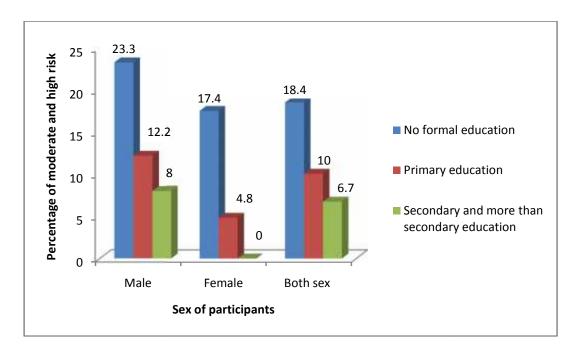


Figure 2: Distribution of moderate and high risk by level of education and gender of study subjects

Moderate to high risk was higher among kshetri, gurung, newar and dalit cast participants. Risk was 17.2% among kshetri, 14.3% among gurung, 15.6% among newar, 14.3% among dalit (table 8). Kshetri female were 19.4% and gurung female were 16.4% in risk. Tamang male were 25 % in risk (chart 3). If ethnicity is divided into two broad category of Tibeto-Burmas(Gurung, Newar, Tamang) and Indo-Aryan (Bramhan, Kshetri and Dalit), then prevalence was 14.7% among tibeto-aryan and 12.3% among Indo-Aryan.

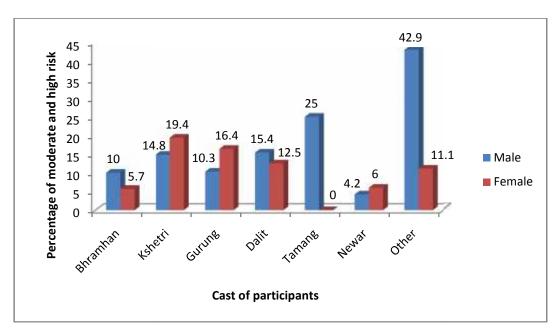


Figure 3: Distribution of moderate and high risk by cast and gender of study subjects

Occupation is related to ten year risk of CVDs (p=0.00). Thirty seven percent of retired participants were in moderate and high risk. Similarly, sixteen percent of homemaker, unemployed and students were in such risk. But, only 1.3 % of self-employed were in risk(table 8). Likewise, among moderate and high risk, 68% of populations were unemployed, homemaker, or student. Whereas, 21% populations were retired and only 11% wereself-employed among the moderate and high risk population (Figure 4).

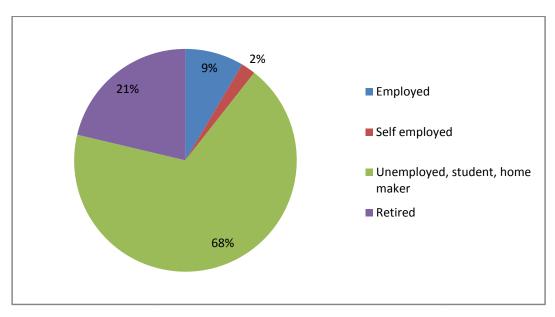


Figure 4: Distribution of moderate and high risk by occupation of study subjects

Percentage of risk was higher among medium income status (14.8%) in comparision to poor income status (13.7%). However, among risk population, 91% were in poor income status and 9% were in medium income status. Amongpoor income status female (15.1%) were in higher risk than male (11.9%). Among medium income status, higher male (18.8%) were in risk in comparision to female (9.1%) (Figure 5-6, table 7).

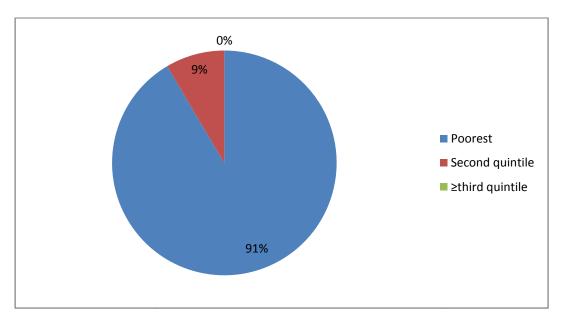


Figure 5:Distribution of moderate and high risk by income level

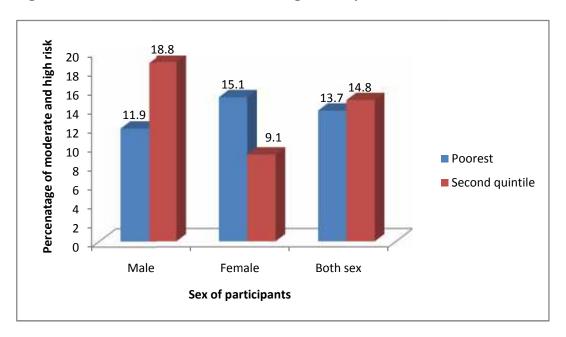


Figure 6: Distribution of moderate and high risk by income status ans gender

Table 8: Distribution of 10 year risk of cardiovascular disease in relation socioeconomic status

Variables	No risk (Mild ten years risk)n=298 n(%)	Risk (Moderate to high)n=47 n(%)	P value
Level of education			
No formal education(n=185)	151(81.6)	34(18.4)	
Primary school level education(n=70)	63(90)	7(10)	0.01*
Secondary and more than secondary	84(93.3)	6(6.7)	0.01
ducation(n=90)			
Cast/ ethnicity [¥]			
Bhramhan(n=75)	69(92)	6(8)	
Kshetri(n=58)	48(82.8)	10(17.2)	
Gurung(n=112)	96(85.7)	16(14.3)	
Dalit(n=21)	18(85.7)	3(14.3)	0.46
Tamang(n=18)	17(94.4)	1(5.6)	
Newar(n=45)	38(84.4)	7(15.6)	
<i>Other(n=16)</i>	12(75)	4(25)	
Occupation [¥]			
Employ(n=40)	36(90)	4(10)	
Self employed(n=78)	77(98.7)	1(1.3)	0.001*
Unemployed, student, homemaker(n=200)	168(84)	32(16)	0.001*
Retired(n=27)	17(63)	10(37)	
Income [¥]			
Poor income status (family income per	271(86.3)	43(13.7)	
person ≤ NRs 8498) (n=314)			
Medium income status (Family income	23(85.2)	4(14.8)	0.72
NRs 8498 - 16294)(n=27)			
High income status (\geq NRs16294)(n=4)	4(100)	0(0)	

Chi-square test was done with p=0.05 as level of significance, *fischer exact test, *=significant

CHAPTER 5: DISCUSSION

Main objective of the study was to estimate the proportion of population who are in moderate and high risk of cardiovascular diseases (fatal and non-fatalmyocardioa infarction and stroke) in next 10 years among those who had not developed clinically menifested cardiovascular diseases. Firstly, individual 10 year risk profile was determined using WHO/ISH chart and then all risk were accumulated to estimated ovarall 10 year risk of the sample.

In current study, moderate risk of cardiovascular diseases was 9.3% and high risk of CVDs was 4.3% among selected rural population. Prevalence of moderate and high risk (>10% risk) for fatal and non-fatal cardiovascular events was 13.6% in our study. This moderate and high risk was higher among female. The result of our study was consistent with STEPS survey 2013 where very high risk was 3.2%among population of 40-69 years of age. However, >10% risk of CVDs was higher than the finding of thestudy conducted in suburban population of Nepal (9.5%)[95]. Similar result was found in a recent study conducted in rural population of India where moderate to higher risk was 16 %[96].

High risk (>20%) was 4.3% in current study. Result was in line with other developing countries using WHO/ISH chart. In Mongolia, Malaysia and Cambodia high risk was 6%, 2.3% and 1.3% respectively[47]. Prevalence of high risk of CVDs was estimated to be less than tenpercent in some Asian countries (10% in China 1.1%, Iran 1.7%, Sri Lanka 2.2%, Cuba 2.8%, Nigeria 5.0%, Georgia 9.6%, Pakistan 10.0%) [97].

In current study, moderate and high risk population was increased to 8.7% after adding hypertensive population having blood pressure >160/100 mm Hg.Thus from this disccusion it can be concluded that >10% risk of CVD was high in the study population.

WHO/ISH chart is easy to use and effective to stratify the high risk population. But the chart may understimate the population under risk because the chart is developed using only six risk factors without incorporating other importance risk factors. As seen in the current study, for example history of taking antihypertensive drug was eighteen percent. In same waydecreased HDL level was seen in half of the respondents and prevalence of obesity was 13% along with high level of triglyceride was in 11.6%. About three fourth of study subjects were not taking adequate amount of fruit and vegetable. In same way one tenth of respondents were involving in low level of physical activities. Similarly, prevalence of family history of CVDs was 7.5% and premature menopause at <40 yrs among female was 10.2%. Higher prevalence of these risk factors indicate that the risk of cardiovascular diseaseswasmore than the risk predicted by the chart alone. Thus, another cohort study is needed to study the effect of these risk factors in setting of rural population of Nepal.

In current study risk of cardiovascular disease was associated with level of education and type of occupation. Risk was higher in lower education levelwhere one fifth of uneducated subjects had moderate and high risk. Risk among uneducated may be related to poor health communication and exposure to unhealthy behavior. Similarly, risk was highest among retired population (37%) may be because of increased age and low level of physical activities. Higher risk among unemployed, student and homemaker group (16%) might be related to less physical activity.

Prevalence of moderate to high risk was higher among tibeto-aryan (14.7%) than among Indo-Aryan (12.3%) in the current study. The result was in line with another study conducted in Nepal where the prevalence of hypertension was 25.3% in the Tibeto-Burmans compared to the 14.0% in the Indo-Aryans[98]. Both of these studiessupport to conclude that indigenous Indo-aryan population had higher risk of cardiovascular diseases. Among the moderate to high risk population, most of the participants were poorest (91%). This current resultmatch the study conducted in Scotland, where socioeconomic deprevation was also responsible for risk of cardiovascular diseases[22]. Therefore, we might conclude that low economic status is also a determinant of cardiovascular risk.

Another aim of the study was to find out the prevalence of cardiovascular diseases risk factors among selected rural community of Nepal at age of 40 to 80 years. In current study, there was high prevalence of smoking, diabetes mellitus, hypertension and total cholesterol. These risk factors are the variables used for predicting 10 yearrisk of cardiovascular diseases using WHO/ISH chart. Other risk factors, like harmful use of alcohol, inadequate intake of fruit and vegetable, low

level of physical activity, obesity, raised triglyceride; raised LDL and decreased HDL were also high.

In current study, 24.8% was smoking currently. Result was slightly higher than the nationwide study in Nepal. According to NCD risk factors STEPS survey 2013 smoking was 18.5 % among 15-65 years of age[28]. However, current result was lower than selected study conducted in urban area. Smoking prevalence was 41.7% among male[99]. But smoking was 28.6% in another study conducted in rural community of Nepal, consistent with our result[100]. Thus, it is reasonable to say that smokers are higher in number in rural population and in age group of current study.

Almost one-fourth (26.2%) subjects were drinking alcohol currently, where half of them were drinking in harmful does. In STEPS survey 2013, 17.4% currently drank alcohol in last 30 days[28]. In comparision to other study, alcohol drinkers were slightly high in our study. Reason for higher prevalence of drinkers might be male gender, indiginous and low cast group participants. As alcohol is required for cultural and ritual program for indigenous and low cast populations and male are more involed in such ritual. Hence, we can conclude that alcholic population was high in study population.

Inadequate intake of fruit and vegetable (<five servings per day) was 72.4 %. It was lower than STEPS survey 2013 where percentage of inadequate intake of fruit and vegetable was 98.9% [28]. Similarly,in another study conducted in peri urban area, prevalence of low intake of fruit and vegetable was 97.9 %[101]. Findings of current study for inadequate intake of fruit and vegetable was lower than these studies. However, fruit and vegetable intake was higher than the finding of STEPS survey 2008(61.9%) [27]. Fruit and vegetable consumption pattern is influenced by sessional availability in the field and low family income. Thus separate study for fruit and vegetable intake in Nepal is essential to conform the result.

Low level of physical activity was 11.6 % in current study. Physical inactivity was higher than the STEPS survey 2013 (2.5%)[28]. In current study, most of the physical activity was related to work in comparision to recreational activities and low level of physical activity was highest among self-employed and homemakers. Where most of the self-employed had retailer shop. That may be the

cause for low level of physical activity in current study in comparision to STEPS survey. However, in another study in peri-urban Nepalese population revealed high prevalence of low physical activity (43.3%)[102]. Geographic condition of the study area, which is hilly and mountainous, might also be responsible to physical activity.

In current study more than two third were hypertensive (40.5%)that was discriminately higher than the finding of nationwidestudy (25.7%)[28]. In another study in eastern Nepal, prevalence of hypertension was 34%[103]. Reason for higher prevalence of hypertension might be higher age ofstudy subjects in current study, because the result was consistent with similar study among the population more than 50 years of age. Prevalence of hypertension was 44.9% among people aged 50 years and more in Banepa Municipality of Nepal[104].

Almost half (46.6%) of the participants had overweight in the current sudy. Similaryobesity was 12.9%. Result of the current study was almost double than the findings of STEPS survey 2013, where overweight was 21.6% [28]. In the same way, overweight was six times the findings of STEPS survey 2008(7.2%) [27]. Likewise, obesity was 4% in 2013 but it was only 1.7% in 2008[27, 28]. Thus, it can be speculated that the trend of overweight and obesity was increasing within five-year interval. That trend might have been reflected in the current study. However, higher discripancy in range of results in different studiesnecessitates separate nationwide study for overweight and obesity.

Prevalence of diabetes mellitus was 16.2% diagnosed by either fasting blood sugar or postprandial blood sugar or history of antidiabetetic drugs in current study. Result was four times higher than STEPS survey 2013. In STEPS survey2013 prevalence of raised fasting blood glucose was only 3.6%. High prevalence of diabetes mellitus was contributed by raised postprandial blood sugar level (8.3%).But, in another study conducted in rural and urban area of kathmanduamong > 60 years of age, prevalence of diabetes was 25.9% [105], which was lower than prevalence in current study. Higher age group, low socioeconomic status, and exposure of multiple risk factors might be responsible for higher prevalence of diabetes in current study. Thus we could conclude that means the prevelence of diabetes was high among rural community.

Raised total cholesterol was 23.2% in current study. Current finding was consistent with STEPS survey 2013, where 22.7% had raised total cholesterol level[28]. Raised triglyceride level more was 27.2% in current study. This result was consistent with STEPS survey 2013 in which prevalence of raised triglycerideswas 25.2% [28]. Decreased high-level lipoprotein was 46.7%. The result was lower than the result of nationwide study where the prevalence of low HDL was 66.9%[28]. High prevalence of overweight and obesity along with higher percentage of use of harmful dose of alchol may be the explanation for these results. Prevalence of different cholesterol was in parrelel to the prevalence of overweight and obesity with male predominance.

Therefore it can be concluded that individual risk factors and risk for development of cardiovascular diseases was high in current study population.Immidiate designation and implementation of necessary intervention is mandatory to control risk factors and to reduce the burden of cardiovascular diseases.

CHAPTER 6: CONCLUSION, RECOMMENDATIONS AND LIMITATIONS

This Chapter includes:

- 6.1 Conclusions
- 6.2 Recommendations
- 6.3 Limitations of the study

6.1 Conclusions

This study tried to assess the risk of cardiovascular diseases in next 10 years in rural community of Nepal. Most of the participants were in mild risk of CVDs. One out of seven people of the total population with age 40 to 80 years was affected by > 10% risk for development of cardiovascular diseases within next10years. One fifth respondent were in moderate to high risk afer including the second grade hypertension.

Higher proportion of female were in moderate and high risk category of CVDs. Risk was increased with increasing age where highest risk was seen in seventies.Low level of education was responsible for CVDs risk. Kshetri, newar, gurung and dalitcast groups had increased risk of CVDs. Retired, homemaker and unemployed was the occupation having high risk of CVDs. Low economic condition was found among moderate and high risk population.

One fourth of respondent were current smoker with similar proportion of participants drinking alcohol currenly. Intake of inadequate amount of fruit and vegatablewas highest. Almost one among ten respondents were not involved in adequate level of physical activities. Hypertension was found in more than one third subjects. Respondents suffering from diabetes mellitus were sixten percent while raised cholesterol level was found in more than one tenth participants.

6.3 Recommendations

On the basis of the study recommendations would be:

- Further large scale study including population of different geographical, socioeconomical condition is recommended to assess 10-year risk of cardiovascular diseases
- Follow up study is recommended to validate and recalibrate the WHO/ISH tool
- Risk assessment program must be conducted in rural and urban population
 of the country in both clinical and public health settings to using evidence
 based tool like WHO/ISH risk prediction chart
- Intervention for primary prevention of moderate risk of fatal and non fatal cardiovascular diseases should be designed and implemented
- Drug intervention should immediately beinitiated to those in high risk population for development of fatal and non fatal coronary heart disease and stroke
- WHO/ISH chart should be incorporated in policy to categorize risk population of cardiovascular disease and target the limited resources to control the risk factors based on risk category

6.2 Limitations of the Study

We are aware of limitations of the study.

- As the study was conducted in selected rural community, population coverage of the study was low. Higher number of indigenous populations and female participants were involved in the study. But it was unintentional and the study was designed in such a way that the sample population was selected using standard method so that they truelyrespresent the study population.
- Patient based knowledge was used to exclude the heart diease or stroke or intermittent claudication. No investigations were used to conform the cardiovascular diseases. This might have overestimated the risk population.
- WHO/ISH risk prediction chart is not validated in current population.

Thus, study finding should cautiously be generalized to rural population.

CHAPTER 7: REFERENCES

- WHO, Global Atlas on Cardiovascular Disease Prevention and Control, ed.
 P.P. Mendis S, Norrving and S. Baba. 2011, Geneva: World Health Organization.
- 2. WHO, Global status report on noncommunicable disaeses 2010. 2011, Geneva: World Health Organization
- 3. WHO. *WHO Global Infobase*. Estimated Proportional Mortality 2013/08/22]; Available from: https://apps.who.int/infobase/Mortality.aspx.
- 4. WHO, Global health risks: Mortality and burden of disease attributable to selected major risks 2009: Geneva.
- 5. Mathers, C.D. and D. Loncar, *Projections of Global Mortality and Burden of Disease from 2002 to 2030.* PLoS Med, 2006. 3(11): p. e442.
- 6. WHO, Non Communicable Diseases Country Profile 2011. 2011: Geneva.
- 7. Centre, S.G.N.H., Shahid Gangalal National Heart Centre Annual Report 2006. 2007.
- 8. Morrish, N.J., et al., *Mortality and causes of death in the WHO Multinational Study of Vascular Disease in Diabetes*. Diabetologia, 2001. 44 Suppl 2: p. S14-21.
- 9. Laing, S.P., et al., Mortality from heart disease in a cohort of 23,000 patients with insulin-treated diabetes. Diabetologia, 2003. 46(6): p. 760-5.
- 10. Levitan, E.B., et al., *Is nondiabetic hyperglycemia a risk factor for cardiovascular disease? A meta-analysis of prospective studies.* Arch Intern Med, 2004. 164(19): p. 2147-55.
- 11. Lewington, S., et al., Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet, 2002. 360(9349): p. 1903-13.
- 12. WHO, Global health risks: Mortality and burden of disease attributable to selected major risks. 2009, World Health Organization: Geneva.

- 13. Doll, R., et al., Mortality in relation to smoking: 50 years' observations on male British doctors. Vol. 328. 2004. 1519.
- 14. Law, M.R., N.J. Wald, and S.G. Thompson, *By how much and how quickly does reduction in serum cholesterol concentration lower risk of ischaemic heart disease?* BMJ, 1994. 308(6925): p. 367-72.
- 15. Berlin, J.A. And G.A. Colditz, *A Meta-Analysis Of Physical Activity In The Prevention Of Coronary Heart Disease*. American Journal of Epidemiology, 1990. 132(4): p. 612-628.
- 16. Wendel-Vos, G.C., et al., *Physical activity and stroke. A meta-analysis of observational data.* Int J Epidemiol, 2004. 33(4): p. 787-98.
- 17. Bazzano, L., M. Serdula, and S. Liu, *Dietary intake of fruits and vegetables and risk of cardiovascular disease*. Current Atherosclerosis Reports, 2003. 5(6): p. 492-499.
- 18. WHO, Global Atlas on Cardiovascular Disease Prevention and Control. 2011: Geneva.
- 19. Wilson, P.W.F., et al., *Prediction of Coronary Heart Disease Using Risk Factor Categories*. Circulation, 1998. 97(18): p. 1837-1847.
- 20. Conroy, R.M., et al., *Estimation of ten-year risk of fatal cardiovascular disease in Europe: the SCORE project.* Eur Heart J, 2003. 24(11): p. 987-1003.
- 21. Julia, H.-C., et al., Derivation, validation, and evaluation of a new QRISK model to estimate lifetime risk of cardiovascular disease: cohort study using QResearch database. BMJ. 341.
- 22. Woodward, M., P. Brindle, and H. Tunstall-Pedoe, *Adding social deprivation* and family history to cardiovascular risk assessment: the ASSIGN score from the Scottish Heart Health Extended Cohort (SHHEC). Heart, 2007. 93(2): p. 172-6.
- 23. World Health Organization, *Prevention of cardiovascular disease: Guidelines for assessment and. management of total cardiovascular risk.* 2007, WHO: Geneva.

- 24. Wilson, P.W. Estimation of cardiovascular risk in an individual patient without known cardiovascular disease. 2015 [cited 2015; Available from: http://www.uptodate.com/contents/estimation-of-cardiovascular-risk-in-an-individual-patient-without-known-cardiovascular-disease.
- 25. Ezzati, M., et al., Estimates of global and regional potential health gains from reducing multiple major risk factors. Lancet, 2003. 362(9380): p. 271-80.
- 26. Bhandari, G., et al., *State of non-communicable diseases in Nepal.* BMC Public Health, 2014. 14(1): p. 23.
- 27. WHO STEPS surveillance:Non Communicable Disease Risk Factors Survey.
 2008, Ministry of Health and Population, Government of Nepal, society for Local Integrated Development Nepal(SOLID Nepal) and WHO.
- 28. Aryal, K.N., S; Mehata, S; Vaidya, A; Singh, S; Paulin, F; Madanlal, RG; Riley, LM; Cowan, M; Guthold, R; Singh, SP; Bhusal, CL; Lohani, GR, *Non communicable diseases risk factors: STEPS Survey Nepal 2013.* 2014: Kathmandu.
- 29. Tunstall-Pedoe H, e., World largest study of heart disease, stroke, risk factors and population trends, 1979–2002. MONICA Monograph and Multimedia Sourcebook, MONICAProject. 2003, World Health Organization: Genava.
- 30. Unal, B., J.A. Critchley, and S. Capewell, *Explaining the decline in coronary heart disease mortality in England and Wales between 1981 and 2000*. Circulation, 2004. 109(9): p. 1101-7.
- 31. World Health Organization, *Prevention of Cardiovascular Diseases: Pocket guidelines for assessment and management of cardiovascular risk* 2007, World Health Organization: Geneva.
- 32. WHO. Global Data Base on Body Mass Index: BMI classification. [cited 2013 Available from: http://apps.who.int/bmi/index.jsp?introPage=intro-3.html.
- 33. World Health Organization, Definition and diagnosis of diabetes mellitus and intermediate hyperglycemia: report of a WHO/IDF consultation, in Geneva: World Health Organization. WHO: Geneva. p. 1-50.

- 34. 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): p. 3143-421.
- 35. WHO. Estimate for 2000-2012: Cause-Specific Mortality 16/08/2014];
 Available at http://www.who.int/healthinfo/global_burden_disease/estimates/en/index1.htm
 1.
- 36. Singh, R.B., et al., *Pathogenesis of atherosclerosis: a multifactorial process*. Experimental & Clinical Cardiology, 2002. 7(1): p. 40.
- 37. WHO. *Cardiovascular diseases*. 2014 [cited 2014 2014/06/30]; Available from: http://www.who.int/topics/cardiovascular diseases/en/
- 38. WHO. *Estimates for 2000-2012: Disease Burden*. 2014/08/16]; Available from:http://www.who.int/healthinfo/global_burden_disease/estimates/en/index 2.html.
- 39. WHO, *Projections of mortality and causes of death*, 2015 and 2030. 2014, World Health Organization.
- 40. WHO, Non Communicable diseases in the South East Asia region: Situation and Response 2011. 2011: Geneva.
- 41. WHO, *Non communicable Diseases country profiles 2011*. 2011, Geneva: World Health Organization.
- 42. Shakya S, S.D., Bhatta YD, *Current Scenario of Heart Diseases in Nepal: At a glance*. Nepalese Heart Journal, 2011. 8(1).
- 43. World Health Organization, *Global status report on noncommunicable disaeses 2010.* 2011, World Health Organisation: Geneva.
- 44. Thom, T., et al., Heart disease and stroke statistics—2006 update a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation, 2006. 113(6): p. e85-e151.
- 45. FHS. *History of the Framingham Heart Study*. 2014 [cited 2014; Available from: https://www.framinghamheartstudy.org/about-fhs/history.php.

- 46. FHS. *Research Milestones*. 2014 [cited 2014; Available from: https://www.framinghamheartstudy.org/about-fhs/research-milestones.php.
- 47. D'Agostino, R.B., Sr., et al., *General cardiovascular risk profile for use in primary care: the Framingham Heart Study*. Circulation, 2008. 117(6): p. 743-53.
- 48. Assmann, G., P. Cullen, and H. Schulte, Simple Scoring Scheme for Calculating the Risk of Acute Coronary Events Based on the 10-Year Follow-Up of the Prospective Cardiovascular Mýnster (PROCAM) Study. Circulation, 2002. 105(3): p. 310-315.
- 49. Ridker, P.M., et al., Development and validation of improved algorithms for the assessment of global cardiovascular risk in women: the Reynolds Risk Score. JAMA, 2007. 297(6): p. 611-9.
- 50. Jackson, R., et al., *Treatment with drugs to lower blood pressure and blood cholesterol based on an individual's absolute cardiovascular risk.* Lancet, 2005. 365(9457): p. 434-41.
- 51. D'Agostino, R.B., Sr., et al., Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. JAMA, 2001. 286(2): p. 180-7.
- 52. Roger, V.L., et al., *Heart disease and stroke statistics--2012 update: a report from the American Heart Association*. Circulation, 2012. 125(1): p. e2.
- 53. Ramachandran, A., et al., *High prevalence of diabetes and cardiovascular risk factors associated with urbanization in India.* Diabetes Care, 2008. 31(5): p. 893-8.
- 54. Pandey, R.M., et al., *Determinants of urban-rural differences in cardiovascular risk factors in middle-aged women in India: a cross-sectional study.* Int J Cardiol, 2013. 163(2): p. 157-62.
- 55. Zaman, M.J., et al., *Socio-economic distribution of cardiovascular risk factors and knowledge in rural India*. Int J Epidemiol, 2012. 41(5): p.1302-14.
- 56. Kar, S.S., et al., Risk factors for cardiovascular diseases: is the social gradient reversing in northern India? Natl Med J India, 2010. 23(4): p. 206

- 57. World Health Organization, and T.F. Initiative, *Building blocks for tobacco control: a handbook*. Tobacco Control, 2004.
- 58. World Health Organization, *The world health report 2002: reducing risks, promoting healthy life. Geneva: WHO; 2002.* 2002.
- 59. Guindon, G.E. and D. Boisclair, *Past, current and future trends in tobacco use.* Tobacco Control, 2003.
- 60. World Health Organization, Global status report on alcohol 2004. 2004.
- 61. *Alcohol*. Comparative Quantification of Health Risks:Global and Regional Burden of Disease Due to Selected Major Risk Factors, ed. Majid Ezzati, et al. 2004, Geneva: World Health Organization.
- 62. Puddey, I.B., et al., *Influence of pattern of drinking on cardiovascular disease* and cardiovascular risk factors--a review. Addiction, 1999. 94(5): p. 649-63.
- 63. Alvear, J., S. Andreani, and F. Cortes, [Fetal alcohol syndrome and fetal alcohol effects: importance of early diagnosis and nutritional treatment]. Rev Med Chil, 1998. 126(4): p. 407-12.
- 64. Faden, V.B., B.I. Graubard, and M. Dufour, *The relationship of drinking and birth outcome in a US national sample of expectant mothers*. Paediatr Perinat Epidemiol, 1997. 11(2): p. 167-80.
- 65. Vuori, I., *Physical inactivity as a disease risk and health benefits of increased physical activity.* The multidisciplinary series of physical education and sport science: Health enhancing physical activity, ed. P. Oja and J. Borms. Vol. 6. 2004.
- 66. Joshi, R., et al., *Prevalence of cardiovascular risk factors among rural population of elderly in Wardha district*. J Cardiovasc Dis Res. 2013 Jun;4(2):140-6. doi: 10.1016/j.jcdr.2013.03.002. Epub 2013 Jun 19.
- 67. Bull F, A.T., Dixon T, Ham S, Neiman A, Pratt M., *Physical inactivity*. Comparative quantification of health risks: global and regional burden of disease attributable to selected major risk factors, ed. L.A. Ezzati M, Rodgers A, Murray C, 2004, Geneva: World Health Organisation.

- 68. Stewart, R., et al., Vascular risk and cognitive impairment in an older, British, African-Caribbean population. J Am Geriatr Soc, 2001. 49(3): p. 263-9.
- 69. Elwood, P.C., et al., *Smoking, drinking, and other life style factors and cognitive function in men in the Caerphilly cohort.* J Epidemiol Community Health, 1999. 53(1): p. 9-14.
- 70. Tunstall-Pedoe, H., Preventing Chronic Diseases. A Vital Investment: WHO Global Report. Geneva: World Health Organization, 2005. pp 200. CHF 30.00. ISBN 92 4 1563001. Also published on http://www.who.int/chp/chronic_disease_report/en. International Journal of Epidemiology, 2006. 35(4): p. 1107-1107.
- 71. World Health Organization, *Diet, nutrition and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation*. Vol. 916. 2003: Diamond Pocket Books (P) Ltd.
- 72. Salmeron, J., et al., *Dietary fat intake and risk of type 2 diabetes in women*. Am J Clin Nutr, 2001. 73(6): p. 1019-26.
- 73. Meyer, K.A., et al., *Dietary fat and incidence of type 2 diabetes in older Iowa women.* Diabetes Care, 2001. 24(9): p. 1528-35.
- 74. Hu, F.B., et al., Dietary fat intake and the risk of coronary heart disease in women. N Engl J Med, 1997. 337(21): p. 1491-9.
- 75. World Health Organization, *Obesity: preventing and managing the global epidemic. Report of a WHO consultation*. World Health Organ Tech Rep Ser. 2000;894.
- 76. World Health Organization, and I.S.o.H.W. Group, 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. Journal of hypertension, 2003. 21(11): p. 1983-1992.
- 77. Chobanian, A.V., et al., *The seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report.* JAMA, 2003. 289(19): p. 2560-2571.
- 78. Williams, B., et al., *British Hypertension Society guidelines for hypertension management 2004 (BHS-IV): summary.* BMJ, 2004. 328(7440): p. 634-640.

- 79. Whitworth, J.A., 2003 World Health Organization (WHO)/International Society of Hypertension (ISH) statement on management of hypertension. J Hypertens, 2003. 21(11): p. 1983-92.
- 80. Lenfant, C., et al., Seventh Report of the Joint National Committee on the Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7): Resetting the Hypertension Sails. Hypertension, 2003. 41(6): p. 1178-1179.
- 81. Wild, S., et al., *Global prevalence of diabetes: estimates for the year 2000 and projections for 2030.* Diabetes Care, 2004. 27(5): p. 1047-53.
- 82. Roglic, G., et al., *The burden of mortality attributable to diabetes: realistic estimates for the year 2000.* Diabetes Care, 2005. 28(9): p. 2130-5.
- 83. World Health Organization, *Screening for Type 2 diabetes: Report of a WHO and International Diabetes Federation meeting*. 2003, WHO Department of ND.
- 84. Morrish, N.J., et al., *Mortality and causes of death in the WHO Multinational Study of Vascular Disease in Diabetes.* Diabetologia, 2001. 44(2): p. S14-21.
- 85. Bell, D.S., Stroke in the diabetic patient. Diabetes Care, 1994. 17(3): p. 213.
- 86. American Diabetes Association, *Diabetic nephropathy*. Diabetes Care, 2000. 23: p. S69.
- 87. Siitonen, O.I., et al., Lower-extremity amputations in diabetic and nondiabetic patients. A population-based study in eastern Finland. Diabetes Care, 1993. 16(1): p. 16-20.
- 88. Economic consequences of diabetes mellitus in the U.S. in 1997. American Diabetes Association. Diabetes Care, 1998. 21(2): p. 296-309.
- 89. Creager, M.A., et al., *Diabetes and Vascular Disease: Pathophysiology, Clinical Consequences, and Medical Therapy: Part I.* Circulation, 2003. 108(12): p. 1527-1532.
- 90. Ezzati, M., et al., Selected major risk factors and global and regional burden of disease. Lancet, 2002. 360(9343): p. 1347-60.

- 91. Bennett, K., et al., *Explaining the recent decrease in coronary heart disease mortality rates in Ireland, 1985-2000.* J Epidemiol Community Health, 2006. 60(4): p. 322-7.
- 92. Franceschini, G., *Epidemiologic evidence for high-density lipoprotein cholesterol as a risk factor for coronary artery disease.* Am J Cardiol, 2001. 88(12A): p. 9N-13N.
- 93. Cullen, P., Evidence that triglycerides are an independent coronary heart disease risk factor. Am J Cardiol, 2000. 86(9): p. 943-9.
- 94. CBS, *National Population and Housing Census 2011*. First ed. Vol. 05. 2014, Kathmandu: Central Bureau of Statistics, Nepal.
- 95. Koju R, G.R., Pant P, Humagain S, Yogol CM, Koju A, Manandhar K, Karmacharya B, Bedi TRS, *Prediction of Cardiovascular Disease in suburban population of 3 municipalities in Nepal*. Nepalese Heart Journal, 2011. 8.
- 96. Ghorpade AG, et al., Estimation of the cardiovascular risk using World Health Organization/International Society of Hypertension (WHO/ISH) risk prediction charts in a rural population of South India. Int J Health Policy Manag, 2015. 4(x): p. 1-6.
- 97. Mendis, S., et al., *Total cardiovascular risk approach to improve efficiency of cardiovascular prevention in resource constrain settings*. J Clin Epidemiol, 2011. 64(12): p. 1451-62.
- 98. Vaidya, A., Is ethnicity an important determinant of high blood pressure in Nepalese population? A community-based cross sectional study in Duwakot, Nepal. Kathmandu Univ Med J (KUMJ), 2012. 10(37): p. 20-3.
- 99. Poudel S, G.D., *Prevalence of smoking and perceived health problems among male population of Dharan municipality*. Journal of Kathmandu Medical College, 2013. 2(5).
- 100. Dhungana, R., et al., *Prevalence of cardiovascular health risk behaviors in a remote rural community of Sindhuli district, Nepal.* BMC Cardiovascular Disorders, 2014. 14(1): p. 92.
- 101. Vaidya A, O.N., Aryal UR, Karki DB, Krettek A, Disparities in fruit and vegetable intake by sociodemographic charecteristics in peri-urban Nepalese

- adults: findings from the Heart Health Associated Research and Dissemination in the Community (HARDIC) study, Bhaktapur, Nepal. Journal of Kathmandu Medical College, 2013. 2(3): p. 3-11.
- 102. Vaidya, A. and A. Krettek, *Physical activity level and its sociodemographic correlates in a peri-urban Nepalese population: a cross-sectional study from the Jhaukhel-Duwakot health demographic surveillance site.* International Journal of Behavioral Nutrition and Physical Activity, 2014. 11(1): p. 39.
- 103. Sharma, S.K., et al., *Prevalence of hypertension, obesity, diabetes, and metabolic syndrome in Nepal.* Int J Hypertens. 2011;2011:821971. doi: 10.4061/2011/821971. Epub 2011 Apr 19., 2011.
- 104. Manandhar K, K.R., Sinha NP, Humagain S *Prevalence and Associated Risk Factors of Hypertension Among People Aged 50 years and more in Banepa Municipality, Nepal.* Kathmandu Univ Med J (KUMJ), 2012. 10(39).
- 105. Chhetri, M.R. and R.S. Chapman, *Prevalence and determinants of diabetes among the elderly population in the Kathmandu Valley of Nepal.* Nepal Med Coll J, 2009. 11(1): p. 34-8.

Annex I: Informed consent form (Nepali)

d~h'/Lgfdf kq

lzif{sM g]kfnsf] b'u{d ;d'bfodfd'6' tyf /StglnhGo /f]usf] hf]lvd cg'dfg .

cg';Gwfgstf{ M 8f= dx]z s'df/ vgfn

cg';Gwfgsf] k|dv pb]Zo M o; ;d'bfodf d'6' tyf /StglnhGo /f]usf] hf]lvdsf] dfqf kQf nufpg' .

hf]lvd cg'dfgsf] ljlwM o; cWoogn] ;'lt{hGo kbfy{ ;]jg, dlb/f ;]jg, kmnkm'n tyf t/sf/L ;]jgsf] dfqf, zf/Ll/s ls|ofsnfk, /Qmrfk, prfO{, tf}n, sDd/sf] gfk, dw'd]xsf] cj:yf, /utdf jf];f]sf] dfqfsf] cfwf/df hf]lvd cg'dfg ug]{5.

cg';Gwfgsf] kmfO{bfM o; cWoogn] ubf{ tkfO{sf] :jf:Yosf] l:yltsf] af/]df hfgsf/L x'g'sf] ;fy} hg:jf:Yo lxtsf nflu sfo{qmd ;~rfng / gLlt lgdf{0f ug{ ;xof]u k'Ug] 5 .

;xeflusf] IhDd]af/L M klxnf] r/0fdf tkfO{n] ;f]lwPsf] k|Zgsf] pQ/ lbg'kg]{ 5 . bf];|f]df tkfO{sf] zf/Ll/s dfk lnOg]5 . To;kl5 tkfO{ /flt !@ 306f ;Dd kflg afx]s c? s]lx gvfO{sg xfldn] tf]s]sf] :yfgdf /ut lbghfg' kb{5 . pSt :yfgdf tkfO{n] yf]/} /ut lbg' kg]{5 . hxfF tkfO{nfO{ &% u|fd Un'sf]h @%) ldln ln6/ kflgdf /fv]/ vfg lbO{g]5 . To;sf] @ 306f kl5 km]/L ! ldlnln6/ /ut lbg' kg]{5 . tkfO{sf] /ut cg'ejk|fKt :jf:YosdL{n] ;+s|d0f /lxt tl/sfn] lgsfNg'x'g]5 .

 $\textbf{xflg gf]S;flg M} o; cg'; Gwfgdf tkfO\{nfO\{ s'g\} /sd lbOg] 5\}g tyf lnOg]klg 5\}g . s; } s; \\ nfO\{ /ut lgsfNbf b'Vg ;S5 . o; jfx]s o; jj]{lf0fdf ;xeflunfO{ s'g} xflg gf]S;flg x'b}g . olb s'g} ;d:of ePdf pkrf/sf] Joj:yf klg ul/Psf] 5 .$

; xeflusf] clwsf/ M tkfO{sf] k|Zgsf] pQ/ cGtjf{tf{stf{ tyf cg;Gwfgstf{ af6 kfpg'x'g]5 . o; cWoogdf tkfO{sf] ; xeflutf :j]IR5s xf] . o;df ; xeflu x'g] gx'g] tkfO{sf] clwsf/ 5 . olb lrQ ga'ßm]df h'g;'s}a]nf cnu x'g ; xeflu chwf/ 5 .

;Defljt	;do	McGtj	t{tt{st]	;do	sl/j #	ŧ) Id	g6st]	x'g]5	. /ut	IgstN	g sl/a	Ps	ldg]6	nfUg]	5 .
o;df x:	tflf/ u	g'{sf] c	Yf{ tkf	O{n]	o; ko	kl9	jf ;'lo	g, ;j} l	h1f;fsf]	pQ/ l	k∣fKt u	/ ;j]·	(IfOfdf	;xeflu	x'g
d~h'/ x	'g'eof	g eGg]	a'lBm0	G5 .											

-==	
	;xeflusf] x:tflf/ jf cf}7f5fk
;xefuLsf] gfd=========	
	d t===================================

Annex 2: Questionnaires (English)

Risk Prediction of Cardiovascular Diseases in selected rural community of Nepal

Survey Information

Locati	on and Date	Response	Code
1.	Ward code		WC
2.	Interviewer name		IN
3.	Date of completion of interview	dd mm year	DI
4.	Respondent name	Bal Maya Gurung	RN
5.	Respondent code		

6.	Respondent Phone No	RP

Step 1 Demographic Information

CORE: Demographic Information						
Questi	on	Response	Code			
7.	Sex (Record Male / Female as observed)	Male 1 Female 2	C1			
8.	What is your date of birth? Don't Know 77 77 7777	known, Go to C4 dd mm year	C2			
9.	How old are you?	Years L_L_I	C3			
10.	In total, how many years have you spent at school or in full-time study (excluding preschool)?	Years	C4			

		No formal schooling	1	
		Less than primary school	2	
	What is the highest level of	Primary school		-
	education you have	completed	3	
	completed?			
		Secondary school	4	
11		completed	7	C5
11.		High school	_	-
		completed	5	
	[INSERT COUNTRY-	College/University		-
	SPECIFIC CATEGORIES]	completed	6	
		Post graduate degree	7	
		Refused	88	
		Bramhan	1	
	What is your <i>ethnic</i>	Kshetri	2	-
12.		Gurung	3	C6
	background?	Dalit	4	-
		Tamang	5	-
		Other	6	-
		Refued Name if other	88	
		Never married	1	
		Currently married	2	_
		Separated	3	
13.	What is your marital status?	Divorced	4	C7
		Widowed	5	_
		Cohabitating	6	
		Refused	88	-
14.	Which of the following best	Government	1	C8

	describes your mainwork	Non-government	2	
	status over the past 12 months?	Self-employed	3	
	1	Non-paid	4	
		Student	5	
		Homemaker	6	
		Retired	7	
		Unemployed (able to	8	
		Unemployed (unable	9	
		Refused	88	
	How many people older than 6			
15.	months, including yourself, live	Number of people		C9
	in your household?			

Question			Response	Code
	Taking the past year , can you tell me what the average	Per week	Go to T1	C10a
16.	earnings of the household have been?	OR per month	Go to T1	C10b
	(RECORD ONLY ONE, NOT ALL 3)	OR per year	Go to T1	C10c
		Refused	88	C10d

Step 1 Behavioural Measurements

Now I am going to ask you some questions about various health behaviours. This includes things like smoking, drinking alcohol, eating fruits and vegetables and physical activity. Let's start with tobacco. Question Response Code

17.	Do you currently smoke any tobacco products , such as cigarettes, cigars or pipes? (USE SHOWCARD)	Yes	2 If No, go to T6	Т1
18.	Do you currently smoke tobacco products daily?	Yes	1 2 If No, go to T6	T2
19.	How old were you when you first started smoking daily?	Age (years) Don't know 77	If Known, go	Т3
	Do you remember how long	In Years	to T5a If Known, go	T4a
20.	ago it was?	OR in Months	If Known, go	T4b
	(RECORD ONLY 1, NOT ALL	OR in Weeks Manufactured cigarettes		T4c
	On average, how many of the following do you smoke each	Hand-rolled cigarettes		T5b
	day?	Pipes full of tobacco		T5c
21.		Cigars, cheroots, cigarillos		T5d
	(RECORD FOR EACH TYPE, USE SHOWCARD) Don't Know 77	Other	If Other, go to T5other, else go to T9	T5e
		Other (please specify):	J	T5othe r

			Go to T9	
22.	Did you try to stop smoking within this year ?	Yes No	2	Т6
23.	Did you get advice from the physician to stope smoking?	Yes	1 2	Т7

1			I	
24.	Did you smoke in the past?	Yes	1	Т8
2		No	2	
25	In the past, did you ever smoke	Yes	1	Т9
25.	daily?	No	2 If No, go to	
26.	How old were you when you	Age (years)	└── If Known, go	T10
20.	stopped smoking daily?	Don't Know 77	to T9	
	How long ago did you stop	Years ago	If Known, go	T11a
	smoking daily?	i cais ago	to T9	TITA
27.	(RECORD ONLY 1, NOT ALL 3)	OR Months ago	L If Known, go to T9	T11b
	Don't Know 77	OR Weeks ago		T11c
20	Do you currently use any	Yes	1	
28.	smokeless tobacco such as	No	2 If No, go to T12	T12
	Do you currently	Yes	1	
29.	usesmokeless tobacco products	No	2 If No, go to T12	T13
	daily?			

CORE: Alcohol Consumption

The next questions ask about the consumption of alcohol.

Questi	on	Res	pon	se	Code
30.	Have you ever consumed an	Yes	1		Ala
30.	alcoholic drink such as beer, wine, spirits, fermented cider or	No	2	If No, go to D1	
	Have you consumed an	Yes	1		
31.	alcoholic drink within the past 12 months?	No	2	If No, go to D1	A1b
	During the past 12 months, how	Daily	1		
	frequently have you had at least one alcoholic drink?	5-6 days per week	2		
32.		1-4 days per week	3		A2
	(READ RESPONSES, USE SHOWCARD)	1-3 days per month	4		
		Less than once a month	5		
33.	Have you consumed an	Yes	1		A3
55.	alcoholic drink within the past	No	2	If No, go to D1	
2.4	During the past 30 days, on how many occasions did you have at	Number			A4
34.	least one alcoholic drink?	Don't know 77	L		

35.	During the past 30 days, when you drank alcohol, on average, how many standardalcoholicdrinks did you have during one drinking occasion? (USE SHOWCARD)	Number Don't know 77	A5
36.	During the past 30 days, what was the largest number of standard alcoholic drinks you had on a single occasion, counting all types of alcoholic drinks together?	Largest number Don't Know 77	A6
37.	During the past 30 days, how many times did you have four or more standard alcoholic drinks in a single drinking occasion?	Number of times Don't Know 77	A7

CORE: Diet

The next questions ask about the fruits and vegetables that you usually eat. I have a nutrition card here that shows you some examples of local fruits and vegetables. Each picture represents the size of a serving. As you answer these questions please think of a typical week in the last year.

Questi	on	Response	Code
38.	In a typical week, on how many days do you eat fruit?	Number of days Don't Know 77 days, go to D3	D1
39.	How many servings of fruit do you eat on one of those days? (USE SHOWCARD)	Number of servings Don't Know 77	D2

40.	In a typical week, on how many days do you eat vegetables? (USE SHOWCARD)	Number of days Don't Know 77 If Zero days, go to D5	D3
41.	How many servings of vegetables do you eat on one of those days? (USE SHOWCARD)	Number of servings Don't know 77	D4

EXPA	EXPANDED: Diet				
		Vegetable oil	1		
		Lard or suet	2		
		Butter or ghee	3		
	What type of oil or fat is most often used for meal preparation in your household? (USE SHOWCARD) (SELECT ONLY ONE)	Margarine	4	D5	
		Other	5 If Other, go to D5 other		
42.		None in particular	6		
		None used	7		
		Don't know	77		
		Other		D5othe r	

		Always	1	DS1
43	How often do you add salt to			
43	your food before you eat it or	Often	2	

	as you are eating it?	Sometimes	3	
		Rarely	4	
	(SELECT ONLY ONE)	Never	5	
	(USE SHOWCARD)	Don't know	77	
44	What is the total amount of salt intake in the family except for the domestic use?	K g/month		DS2

CORE: Physical Activity

Next I am going to ask you about the time you spend doing different types of physical activity in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person.

Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food, seeking employment. [Insert other examples if needed]. In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.

Question		Response		Code
Work				
45.	Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like [carrying or liftingheavy loads, digging or construction work] for at least 10 minutes continuously? [INSERT EXAMPLES] (USE SHOWCARD)	Yes No	1 2 If No, go to P 4	P1

46.	In a typical week, on how many days do you do vigorous-intensity activities as part of your work? How much time do you spend doing vigorous-intensity activities at work on a typical day?	Number of days Hours: minutes	LJ: LJJ:	P2 P3 (a-b)
48.	Does your work involve moderate- intensity activity, that causes small increases in breathing or heart rate such as brisk walking [or carrying light loads] for at least 10 minutes continuously? [INSERT EXAMPLES] (USE SHOWCARD)	Yes	1 2 If No, go to P 7	P4
49.	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days		P5
50.	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours : minutes	hrs mins	P6 (a-b)
Travel	to and from places			
Now I	ext questions exclude the physical activities at would like to ask you about the usual way you for shopping, to market, to place of worship.	ı travel to and	from places. For example	le to
51.	Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places?	Yes No	1 2 If No, go to P 10	P7
52.	In a typical week, on how many days do you walk or bicycle for at least 10 minutes	Number of days	ш	P8
53.	How much time do you spend walking or bicycling for travel on a typical day?	Hours : minutes	hrs mins	P9 (a-b)

CORE: Physical Activity, Continued

Question		Response	Code
Recrea	tional activities		
The ne	xt questions exclude the work and trans	sport activities that you have already mentio	ned.
Now I	would like to ask you about sports, fitn	ess and recreational activities (leisure), [Ins	ert relevant
terms].			
	Do you do any vigorous-intensity	Yes 1	
	sports, fitness or recreational	103	
54.	(leisure) activities that cause large		
	increases in breathing or heart rate		P10
	like [running or football] for at	No 2 If No, go to P 13	
	least 10 minutes continuously?	110 2 19 110, go 10 1 13	
	[INSERT EXAMPLES] (USE		
	SHOWCARD)		
55.	In a typical week, on how many		
	days do you do vigorous-intensity	Nl C. l	D11
	sports, fitness or recreational	Number of days	P11
	(leisure) activities?	Ш	
	How much time do you spend		
56.	doing vigorous-intensity sports,	Hours: minutes	P12
30.	fitness or recreational activities on a	Hours : minutes	(a-b)
	typical day?	hrs mins	
	Do you do any moderate-intensity	Yes 1	
57.	sports, fitness or recreational	103	P13
37.	(leisure) activities that cause a	No 2 If No, go to P16	113
	small increase in breathing or heart		
	In a typical week, on how many		
58.	days do you do moderate-intensity	Number of days	P14
50.	sports, fitness or recreational	Trumber of days	114
	(leisure) activities?		
	How much time do you spend		
59.	doing moderate-intensity sports,	Hours: minutes	P15
39.	fitness or recreational (leisure)	hrs mins	(a-b)
	activities on a typical day?	mis minis	

Sedentary behaviour

The following question is about sitting or reclining at work, at home, getting to and from places, or

with friends including time spent sitting at a desk, sitting with friends, traveling in car, bus, train,					
readin	g, playing cards or watching televisi	ion, but do not include ti	me spent slee	eping.	
[INSERT EXAMPLES] (USE SHOWCARD)					
	How much time do you usually		ا : لـــــــا		P16
60	spend sitting or reclining on a	Hours : minutes			(a-b)
	typical day?		hrs	mins	()
	typical day?		hrs	mins	(a-0

CORE: History of Raised Blood Pressure					
Quest	ion		Res	ponse	Code
	Have you ever had your blood pressure	Yes	1		
61	measured by a doctor or other health worker?	No	2	If No, go to H6	Н1
	Have you ever been told by a doctor or other	Yes	1		
62	health worker that you have raised blood pressure or hypertension?	No	2	If No, go to H6	H2a
63	Have you been told in the past 12 months?	Yes No	1 2		Н2ь

Are you currently receiving any of the following treatments/advice for high bloc prescribed by a doctor or other health worker?					
	Drugs (medication) that you have	Yes	1	НЗа	
64	taken in the past two weeks	No	2	1134	
	Advice to reduce salt intake	Yes	1	H3b	
	Advice to feduce sait intake	No	2	1100	

	Advice or treatment to lose weight	Yes No		Н3с
	Advice or treatment to stop smoking		2	H3d
	Advice to start or do more exercise		2	Н3е
65	Have you ever seen a traditional healer for raised blood pressure or hypertension?	Yes No	2	H4
66	Are you currently taking any herbal or traditional remedy for your raised blood pressure?	Yes	2	Н5

History of Diabetes					
Questio	on	Response	Code		
67.	Have you ever had your blood sugar measured by a doctor or other health worker?	Yes 1 No 2 If No, go to CVD1	Н6		
68.	Have you ever been told by a doctor or other health worker	Yes 1 No 2 If No, go to	Н7а		
69.	Have you been told in the past 12 months?	Yes 1 No 2	H7b		

History	of Diabetes			
	Are you currently receiving any	of the following treatments/advice for diabetes pre	escribed	
70.	70. by a doctor or other health worker?			
	Insulin	Yes 1	Н8а	

History of Menopause (only for female)

		No	2	
	Drugs (medication) that you	Yes	1	
	have taken in the past two weeks	No	2	H8b
	Chariel programmed dist	Yes	1	Н8с
	Special prescribed diet	No	2	пос
	Advice or treatment to lose	Yes	1	H8d
	weight	No	2	пои
	Advice or treatment to stop	Yes	1	Н8е
	smoking	No	2	пъе
	Advice to start or do more	Yes	1	H8f
	exercise	No	2	1101
	Have you ever seen a	Yes	1	
71.	traditional healer for diabetes or raised blood sugar?	No	2	Н9
	Are you currently taking any	Yes	1	
72.	herbal or traditional remedy for your diabetes?	No	2	H10

History of Cardiovascular diseases in the family

	Question	Response		Code
73.	Does your relative have Stroke, Heart attack, peripheral arterial diseases?	Yes No	1 2 If No, go to Mp1	H11
74.	What was the age of starting stroke or heart attack or	Year :		H12

Questi	ion	Response		Code
75	Are you menstruating	Yes	1	H 13
		No	2 If yes, go to M3	11 13
76	When did your menstruation	Age of stoppage of	Ш	
	stop?	menstruation		H14
			years	

Step 2 : Physical Measurement

CORI	E: Height and Weight			
Quest	ion	Response		Code
77.	Interviewer ID			M1
78.	Height	in Centimetres (cm)	<u> </u>	Ht
79.	Weight If too large for scale 666.6	in Kilograms (kg)	ш.ш	Wt
		No	2	
CORI	E: Waist			
80.	Waist circumference	in Centimetres (cm)	ш.ш	WC
CORI	E: Blood Pressure			
81.	Reading 1			Bpla
		Systolic (mmHg)		
		Diastolic (mmHg)		Bp1b
82.	Reading 2	Systolic (mmHg)		Bp2a
		Diastolic (mmHg)		Bp2b
83.	Reading 3	Systolic (mmHg)		Bp3a
		Diastolic (mmHg)		Bp3b
84.	During the past two weeks, have	Yes	1	AH
	you been treated for raised blood	No	2	
	pressure with drugs (medication)			
	prescribed by a doctor or other			
	health worker?			

Hip Circumference						
85.	Hip circumference	in Centimeters	<u> </u>	НС		
		(cm)				

Step 3 Biochemical Measurements

Quest	ion	Resp	ponse	Code
86.	Name of Respondent			
87.	ID of respondants			
88.	Phone no of respodent			
89.	Name of technician			
90.	Time of day blood specimen taken (24 hour clock)	Hours : minutes	hrs mins	Tbt
91.	Fasting blood glucose	mg/dl		FBS
92.	Blood glucose 2 hrs after oral	mg/dl		PPBS
93.	Today, have you taken insulin	Yes	1	AD
	or other drugs (medication) that have been prescribed by a doctor or other health worker for raised blood glucose?	No	2	
94.	Total cholesterol	mg/dl		TC
95.	During the past two weeks,	Yes	1	AC
	have you been treated for raised cholesterol with drugs (medication) prescribed by a doctor or other health worker?	No	2	
96.	Triglycerides	mg/dl		TG

Dhaka University Institutional Repository

97. HDL Cholesterol	mg/dl		HDL
---------------------	-------	--	-----

Annex 3: Questionnaires (Nepali)

d'6' tyf /StglnhGo /f]usf] hf]lvd ca'dfasf] :i]{lf0fsf nflu klZafanl

;j]{lf0f ;Dal	Gw hfgsf/L		
:yfg / ;do		hjfkmx?	sf]
			8
1.	jf8{ -sf]8_		W
			С
2.	cGt/jftf{st		IN
	f{sf] gfd		
3.	cGt/jftf{		DI
	k"/f ePsf]	lbgdlxgfjif{	
	ldlt	;do	
		306f ldg]6	
4.	pQ/bftfsf]		R
	gfd y/	=======================================	N
		============	
5.	pQ/bftfsf]		R
	klxrfg g+		C
6.	pQ/bftfsf]		RP
	kmf]g g+	=======================================	
		===	

r/0f–! hg;f ^a \lVos laa/0f				
k Zgx?		hjfkmx?		sf]8
7.	InË	k'?if	!	C1
		dlxnf	@	
8.	tkfO{sf] hGd ldlt eGg'xf];\ .			C2
		lbg dlxgf	jif{	
9.	k"/f ePsf] pd]/	jif{		C3

10.	tkfO{n] ;du df cf}krfl/s lzlffsf nfuL slt jif{ latfpg'eof] < -k"j{ k fylds tx nfO{ ;dfj]z gug]{_	jif{		C4
11.	tkfO{n] k"/f ug'[{ ePsf] dflyNnf]	cgf}krfl/s lzlff	!	C5
	txsf] lzlff s'g xf]<	k fylds eGbf sd	@	
		k fylds tx	#	
		dfWolds tx	\$	
		k lj0ftf jf pRr lzlff	%	
		sn]h jf ljZj ljBfno k"/f ePsf]	٨	
		kf]i6 uf h'P6 jf ;f] eGbf	&	
		dfly		
		pQ/ lbg grfx]sf]	**	
12.	tkfO{sf] hft s'g xf] <	a Dx0f	!	C6
	-hflto al{us/0f sf8{ k of]u ug]{_	lf]qL	@	
		u'?Ë	#	
		blnt	\$	
		tfdfË	%	
		cGo hflt	٨	
		pQ/ lbg grfx]sf]]	**	
		cGo ePdf gfd	-	C6a
13.	tkfO{sf] xfn j}jlxs l:ylt s] xf] <	cljjflxt	!	C7
		ljjflxt	@	
		5'l6Psf]	#	
		ljwjf jf ljw'/	\$	
		pQ/ lbg grfx]sf]	**	
14.	ljut !@ dlxgf b]lv tkfO{ d'Vo	;/sf/L hflu/	!	C8
	s'g k]zfdf ;+nUg x'g'x'G5<	u}/;/sf/L hflu/	@	
		cf^g} :jfldTjsf]	#	

		a]tnaL	\$	
		ljBfyL{	%	
		3/fo;L sfd	۸	
		cjsf; k fKt	&	
		j]/f]huf/ -sfd ug{ ;Sg]_	*	
		j]/f]huf/ -sfd ug{	(
		g;Sg]_		
		pQ/ lbg grfx]sf]]	**	
15.	tkfO{sf] 3/df tkfO{ nufot ^ dlxgf dflysf slt hgf ;b:ox? x'g'x'G5 <	;b:ox?sf]; ^a \Vof		C9
16.	ut jif{sf] cfwf/df tkfO{sf] cf};t			C10
	sdfO{ slt x'G5 atfpg ;Sg'x'G5	k lt dlxgf LLLL		a
	<- s'g} Ps pQ/ dfq_	k ltjif{ LLLLLL		C10
			,	b
_		pQ/ glbPdf	**	C10
		F = 3.2. 2.		c

r/0f ! Jojxfl/s dfkg

	;'lt{hGo kbfy{ ;]jg				
ca d tkfOnfO{ ;'lt{hGo w'd kfg ;DalGw s]lx k Zgx? ;f]Wg uO/x]sf] 5' .					
k Zgx?		hjfkmx?		sf]8	
17.	tkfO{n] xfn s'g} ;'lt{hGo	u5'{	!	T1	
	w'd kfg ;]jg ug'{x'G5<	ul{bgF	@ ,olb ul{b{gF eg] T8	-	
	-h:t} la8L, r'/f]6, tdfv', x'Ssf jf		df hfg]		
	cGo:yflgo?kdfpTkfbgx'g]				
	;'It{thGo kbfy{x?_				
	;f] sf8{ k of]u ug]{				
18.	s] clxn]b}lgs w'd kfg	u5'{	!	T2	
	ug'{x'G5<	ul{bgF	@		
19.	s] tkfO{nfO{ ofb 5 slt jif{sf]	jif{		Т3	
	pd]/df z'? ug'{ ePsf] lyof] <				
20.	s] tkfO{ ;Demg'x'G5 slt ;do	jif{df		T4a	

Dhaka University Institutional Repository

cuf8L z'? ug'{ePsf] lyof]<	jf dlxgfdf	T4b
Ps hjfkm dfq eg'{xf];\		

		ofb gePdf	&&&	
21.	lgDg pNn]lvt w'd kfg lbgdf jf		b}lgs	
	xKtfdf cf};t slt ;]jg ug'{x'G5< -k To]s k sf/sf] w'd kfg /]s8{	pTkfbg ul/Psf] r'/f]6		T5a
	ug]{_ yfxf gePdf &&&	xftn] a]/]sf] r'/f]6		T5b
	ym ger ar ada	kfOk -x'Ssf, Irlnd cflb		T5c
		l;uf/		T5d
		cGo		T5e
		cGo eP	olb cGo ePdf T5 other / T6 df hfg]	
		gfdv'nfpg]		T5other
22.	ljutsf] !@ dlxgfdf tkfO{n]	lyP	!	Т6
	w'd kfg TofUgsf nfuL sf]lz; ug'{ePsf] lyof] <	lyPg	@	
23.	ljutsf] !@ dlxgfdf tkfOnfO{	lyP	!	T7
	8fS6/ jf :jf:YosdL{n] w'd kfg TofUgsf nfuL ;Nnfx lbPsf	lyPgg\	@ T12 df	
	lyP <	:jf:YosdL{sf]df uPsf] 5}g	# hfg]	
24.	tkfO{n] ljutdf slxNo}} w'd kfg	5	!	
	ug'{ePsf] 5< ;f] sf8{ k of]u ug]{	5}g	@olb 5}g eg] T12 df hfg]	T8
25.	ljutdf tkfO{n] s'g} klg k sf/sf] w'd kfg b}lgs ?kdf k of]u	uy]{	!	Т9
	ug'{x'GYof]<	ul{bgy]+	@	

26.	tkfO{n] w'd kfg ug{{ 5f8\bf	k"/f ePsf] pd]/-		T10
	tkfO{ slt jif{sf] x'g'x'GYof]<	jif{df_	T12 df hfg]	
		ofb gePdf	&&	
27.	tkfO{n] b}lgs w'd kfg ug{{	jif{ cuf8L	olbofb ePdf	T11a
	5f8\]sf] slt ;do eof]<		T12 df hfg]	
	-Ps hjfkm dfq eg'{xf];_	jf dlxgf cuf8L	olbofb ePdf	T11b
			T12 df hfg]	
		<i>jf</i> xKtf cuf8L		T11c
		ofb gePdf	&&	T11d
28.	tkfO{n] xfn w'Fjf/lxt ;"lt{ h:t}	5	!	T12
	-;'lt{, a]6]n, u'6\sf, kfg d;nf,			
	v}gL cflb_ sf] ;]jg ug'{ePsf]	5}g	@5}g eg] Aladf hfg]	
	5<			
29.	tkfO{n] xfn s'g} w'Fjf/lxt	u5'{	!	T13
	;"It{hGo kbfy{ b}lgs ;]jg	ulb{g	@olb ul{bgF eg] A1a	
	ug{'x'G5<		df hfg]	

	dlb/fhGc	kbfy{ ;]jg		
casf k	Zgx? dlb/fhGo kbfy{ ;]jg ; u ;DalGwt	5g\ .		
30	tkfO{n] hLjgdf slxNo} dBkfg	5	!	Ala
	ug'{ePsf] 5<-h:t} ljo/ ,jfOg,x'l:s	5}g	@ olb 5}g eg] D1	
	,uf'p3/d} agfPsf] /S;L, hf+8, tf]Daf_		df hfg]	
	;f] sf8{sf] k of]u ug]{			
31	tkfO{n] !@ dlxgf leqdf s'g} dlb/fhGo	5	!	A1b
	kbfy{sf]	5}g	@olb 5}g eg] D1	
	;]jg ug'{ePsf] 5 <		df hfg]	
32	ljt]sf] !@ dlxgf leqdf slt ;dosf] cGt/df	b}lgs	!	A2
	tkfO{n] dlb/fhGo k]o kbfy{ lkpg'ePsf]			
	lyof] <	%^ lbg/ xKtf	@	
	;f] sf8{sf] k of]u ug]{	!_\$ lbg/ xKtf	#	
		· v ibg/ xitti	TT .	
		!-#lbg/dlxgf	\$	
		dlxgfdf Psk6s	%	
		eGbf sd		
33	tkfO{n] ljt]sf] ! dlxgfdf -#) lbgdf_ s'g}	5	!	A3
	k sf/sf] dBkfg ug'[{ ePsf] 5 <	5}g	@,olb 5}geg] D1	
			df hfg]	
34	ljt]sf] ! dlxgfdf -#) lbgdf_ slt lbg	lbg ;+Vof		A4
	tkfO{n] -slDtdf Ps k6s_ dBkfg	yfxf 5}g	&&	
	ug'{eof] <			
35	ljt]sf] ! dlxgfdf -#) lbgdf_ tkfO{n]	:6ofG88{		A5
	dBkfg u/]sf] lbgdf cf};tdf slt :6fG88{	l8«+S;sf] ;+Vof		
	l8«S; Ing'eof] <	yfxf 5}g	&&	
	;f] sf8{sf] k of]u ug]{			
36	ljt]sf]! dlxgfdf -#) lbgdf_ tkfO{n]	:6ofG88{		A6
	;a}eGbf a9L lkPsf] lbg slt :6fG88{	I8«+S;sf] ;+Vof		
	I8«S; lkpg'eof] <	yfxf 5}g	&&	
37	ljt]sf] ! dlxgfdf -#) lbgdf_ tkfO{n]	slt lbg		A7
	<pre>\$ jf \$ eGbf w]/}</pre>	ofb gePdf	&&	
	:6fG88l8«+S; slt lbg lkpg'ePsf] lyof]	oib goi di	~~	
	<			

cfxf/

csf]{ k|Zgx? tkfO{n] b}lgs ?kdf vfPsf] kmnkm'n / ;fu;lAhx?sf] af/]df ;f]lwg]5 . oxf' d kf]if0ftTj ;+alGw :yflgo kmnkm'n / ;fu;lAhx?sf] s]lx pbfx/0fx? tkfO{nfO{ b]vfp'5' . k|To]s lrqn] lnOPsf] dfqf OlGut u5{ . s[kof tkfO{n] of] k|Zgsf] pQ/ ;f]r]/ lbg'xf];\ .

k Zgx?		hjfkmx?		sf]8
38.	tkfO{ xKtfdf slt lbg kmnkm'n vfg'x'G5	lbgsf] ;+Vof		D1
	<	ofb gePdf	&& ,olb z'Go	
	;f] sf8{sf] k of]u ug]{		lbg ePdf D3 df	
			hfg]	
39.	tL lbgdWo] Pslbgdf slt ;le{a kmnkm'n	InPsf] ;+Vof		D2
	vfg'x'G5	ofb gePdf	&&	
	;f] sf8{sf] k of]u ug]{			
40.	tkfO{ xKtfdf slt lbg xl/of] t/sf/L	lbgsf] ;+Vof		D3
	vfg'x'G5 <;f] sf8{sf] k of]u ug]{			
		ofb gePdf	&& olb z'Go lbg	
			ePdf D5 df hfg]	
41.	It lbg dWo] Ps lbgdf slt ;le{a	InPsf] ;+Vof		D4
	vfg'x'G5<;f] sf8{sf] k of]u ug]{			
		ofb gePdf	&&	

42.	tkfO{ cfkm\gf] 3/df vfgf ksfpgsf] nflu	jg:klt t]n- tf]/L,	!	D5
	k foh;f] s:tf] k sf/sf] t]n jf l3psf] k of]u	e6df;, ;'o{d'lv,		
	ug'{x"G5 <	cflb_		
	;f] sf8{sf] k of]u ug]{	af];f]	@	
	-Ps hafkm dfq eg'{xf];_	gf}gL jf £o'	#	
		rfprfpsf] t]n	\$	
		8fN8f - jg:klt £o'_	%	
		h'g;'s}	۸	
		ofb gePdf	&&	
		cGo -gfd v'nfpg]_	=======	D5a
			=======	
			=======	
			==	

vfg] g'	g			
43.	tkfO{n] s'g} klg vfgf vfg' cl3 vfgfdf	;w}+ vfG5 '	!	DS1
	g'g yk]/ vfg' x'G5 <	k fo vfG5'	@	
	-Ps hafkm dfq eg'{xf];_	slxn]sflx vfG5'	#	_
		cfSsnem'Sns	\$	
		vfG5'		
		slxn] klg vflVbg	%	-
		ofb gePdf	&&	
44.	tkfO{sf] kl/jf/n] slt dfqfdf g'gsf] k of u ug'[x'G5 < - ufO{j:t'nfO{	ls=uf ÷dlxgf		DS2
	k of]u ug]{ jfx]sPp6f dfq ljsNk	ofb gePdf	&&	
	/f]Hg]_			

zfl//Ls ls|ofsnfk

ca d tkfO{nfO{ cf^gf] xKtfel/sf] ;do s] s:tf] zfI//Ls ls|ofsnfk ul/ latfpg'x'G5< eGg] af/]df ;f]Wg rfxG5' .

 $s[kof\ tkfO\{n]\ tn\ ;f]lwPsf]\ k|Zgsf]\ hjfkm\ lbg'xf]nf\ .olb\ tkfO\{n]\ cfkm'nfO\{\ zfl//Ls\ ?kdf\ blf\ g7fGg'ePsf]\ ePklg,\ klxn]\ ;f]Rg'xf];\ cfkm'n]\ slt\ ;do\ sfd\ u/]/\ latfpg'x'G5<\ sfd\ eGgfn]\ Hofnfbf/L\ jf$

a]tnla sfd, 3/]n'sfd, k9fO jf tflnd, v]ltkflt, df5fdfg]{ jf lzsf/ ug]{, /f]huf/ vf]Hg] . al9 kl/>d kg]{ ls|ofsnfk eGgfn] To:tf] ls|ofsnfk h;df zfl//Ls an k|of]u x'G5, h;sf] sf/0f Zjf; jf d'6'sf] w8sg al9 dfqfdf a9\5 . dWod vfnsf] kl/>d kg]{ ls|ofsnfk eGgfn] To:tf] ls|ofsnfk h;df yf]/} an k|of]u x'G5 / h;sf] sf/0f Zjf; jf d'6'sf] w8sg dWod tl/sfn] a9\5 .

	k Zgx?		hjfkmx?	sf]8
45.	tkfO{n] sfd ubf{ slDtdf !) ldg]6;Dd Zjf; / d'6'sf]	u5{'	!	P1
	w8sg a9\g] ul/ -al9 kl/>d kg]{ ls ofsnfk			
	k of]u x'g]ul/_ sfd ug'{x'G5< -h;df ef/L ;fdfg	ulb{g	@, olb ulb{g eg]	
	af]Sg', vGg' jf lgdf{0f ug'{ cflb_		P4df hfg]	
	;f] sf8{sf] k of]u ug]{			
46.	tkfO{n] xKtfdf slt lbg al9 kl/>d kg]{ ls ofsnfk	lbgsf]		P2
	ug'{x'G5<	;+Vof		
47.	Pslbgdf;/b/ 3/ jf aflx/ sfd ubf{ slt;do al9 kl/>d	306f		P3a
	kg]{ ls ofsnfk ug'{xG5M<	ldg]6		P3b
		L		
48.	tkfO{n] sfd ubf{ slDtdf !) ldg]6;Dd Zjf; / d'6'sf]	u5'{		
	w8sg dWod tl/sfn] a9\g] ul/ -dWod vfnsf]		!	P4
	kl/>d kg]{ ls ofsnfkk of]u x'g]ul/_ sfd			
	$ug'\{x'G5 < -h;df lx+8\g], ;fdfg p7fpg], n'uf w'g],$			
	kf]5f nufpg], dn af]Sg],Ogf/af6 kfgL tfGg],	ulb{g	@, olb ulb{g eg]	
	wf/faf6 kflg Nofpg] cGg lkGg], au}+rfsf] :ofxf/		P7df hfg]	
	;';f/ ug{]{,v]t uf]8\g], cflb_			
	;f] sf8{sf] k of]u ug]{			
49.	tkfO{n] xKtfdf slt lbg dWod vfnsf] kl/>d kg]{	lbg		P5
	ls ofsnfk ug'{x'G5<			
50.	Pslbgdf;/b/ 3/ jf aflx/ sfd ubf{ lbgdf slt;do	306f		P6a
	dWod vfnsf] kl/>d kg]{ ls ofsnfk			
	ug'{xG5M<		ldg]6	P6b

lx8\8'n -zf/l/s lqmofsnfk;+alGw_				
dflysf k Zgx? zfl//Ls ls ofsnfk;uF ;DalGwt lyP h'g tkfO{n] cl3g} k'/f ug'{ePsf] 5 . clxn] d				
$tkfO\{nfO\{\ k foh;f]\ tkfO\{n]\ lx+88'n\ ubf\{\ s]\ ;fwgsf]\ k of]u\ ug'\{x'G5\ -h:t\}\ sfd\ ug\{\ hfFbf,\ lsgd]n\ ug\{nfO\{nfO\{\ k foh;f]\ tkfO\{nfO\{nfO\{nfO\{nfO\{nfO\{nfO\{nfO\{nfO\{nfO\{n$			l]n ug{	
hfFbf, ahf/ hfFbf, k"hfug{ hfFbf cflb_ To; ;u ;D	hfFbf, ahf/ hfFbf, k"hfug{ hfFbf cflb_ To; ;u ;DalGwt k Zgx? ;f]Wg rfxG5' .s[kof tkfO{n] tn			
;f]lwPsf] k Zgsf] hjfkm lbg'xf]nf .				
51. tkfO{n] cfjthfjtsf nflu slDtdf !) ldg]6	u5'{	!	P7	

	lx+8\g' jf ;fOsnsf] k of]u ug'{x'G5<	ulb{g	@ olb ul{b{g eg] P10 df hfg]	
52.	xKtfdf tkfO{n] slt lbg slDtdf !) ldg]6 cfjthfjtsf nflu lx+8\g' jf ;fOsnsf] k of]u ug{'x'G5<	lbgsf] ;+Vof		P8
53.	tkfO{n] lbgdf slt ;dolx+88'n jf ;fOsn rnfP/ latfpg'x'G5	306	ldg]6	P9a P9b

dgf]/~	hgfTds ls ofsnfk			
dflysf k	k Zgx? lx8\8'n / sfd;uF ;DalGwt lyP h'g	tkfO{n] cl3g} k'/f ug	'{ePsf] 5 . clxn] d tkf0	O{nfO{
v]ns'b	Jofod / dgf]/~hgfTds ls ofsnfkx? -vfnL ;	dodf ul/g] ls ofsnfk	_sf af/]df ;f]Wg rfxG5	5' .
s[kof tl	dO{n] tn ;f]lwPsf] k Zgsf] hjfkm lbg'xf]n	f .		
54.	s] tkfO{n] slDtdf !) ldg]6;Dd s'g}	u5{'	!	P10
	zl//nfO{ an kg]{ vfnsf Joofd jf vfnL			
	;dodf dgf]/~hgfTds lsofsnfk	ul{bgF	@ olb ulb{g eg]	
	ug'{x'G5 <h;n] a9fpg],d'6'sf]<="" th="" zjf;=""><th></th><th>P13 df hfg]</th><th></th></h;n]>		P13 df hfg]	
	w8\sg a9fpg] u5{, h:t} bf}8g],			
	km'6an v]Ng] cflbal9 kl/>d kg]{			
	Joofd_			
	;f] sf8{sf] k of]u ug]{			
55.	tkfO{n] xKtfdf slt lbg al9 kl/>d kg]{	lbgsf] ;+Vof		P11
	Joofd ug'{x'G5<			
56.	tkfO{n] pQm lbgdf slt ;do al9 kl/>d		306f	P12
	kg]{ Jofofd ug'{xG5M<	ldg]6		a
				P12
				b
57.	s] tkfO{n] slDtdf !) ldg]6;Dd s'g} sd		!	
	an kg]{ Jofofd jf vfnL ;dodf	u5{'		P13
	df]/~hgfTds ls ofsnfk ug'{x'G5 <h;n]< th=""><th>ulb{g</th><th>@</th><th></th></h;n]<>	ulb{g	@	
	Zjf; sd a9fpg],d'6'sf] w8\sg sd		olb ulb{g eg] P16	
	a9fpg] u5 {h:t}M ;fOsn rnfpg], kf}8L		df hfg]	
	v]Ng], elnan v]n cflb . -dWod kl/>d			
	kg]{ Joofd_			
	;f] sf8{sf] k of]u ug]{			

58	tkfO{n] xKtfdf slt lbg dV kg]{ Joofd ug'{x'G5<	/od kl/>d	lbgsf] ;+Vof		P14
59	. tkfO{n] pQm lbgdf slt ;d kl/>d kg]{ Jofofd ug'{		306f]	P15 a P15
6/6 !!					b
cf/fdk	ofoL Is ofsnfk				
tnsf k	Zgx? tkfOn] slt ;do 3/df jf	sfddf cf/fd u/]/ jf ysfO d]6]/ latfpg	g'x'G5 eGg] s'/f;uF ;Da	alwt
5g\ h	dfF – 8]:sdf a:g], ;fyL;uF a:	g], sf/df e d0	fug]{, /]ndf r9\g], k9	\g], tf; v]Ng] jf l6 le x]	g]{ t/
;'t]/ ;c	lo latfpg'nfO{ eg] ;dfj]z u	ıl/+b}g .			
60	tkfO{n] lbgdf slt ;do	306f			P16
	a;]/ latfpg'x'G5<				a
		ldg]6			P16
					b

	a9\bf] pRr/Qmrfksf] Oltxf;				
	k Zgx?	hjfl	kmx?	sf]8	
61	tkfO{n] slxNo} cf^gf] /Qmrfksf] hfFr	5	!	H1	
	8fS6/ jf cGo :jf:Yo sdL{af6 u/fpg' ePsf] 5<	5}g	@ olb 5}g eg] H6 df hfg]		
62	tkfO{nfO{ s'g} klg ;dodf 8fS6/ jf	5	!	H2a	
	:jf:YosdL{n] pRr /Qmrfk 5 eGg'ePsf] 5<	5}g	@ olb 5}g eg] H6 df hfg]		
63	tkfO{nfO{ lautsf] !@ dlxgfsf pRr /Qmrfk 5 eGg'ePsf] 5<	5	!	H2b	
	ŭ .	5}g	@ olb 5}g eg] H6 df hfg]		

	dw'd]xsf] Oltxf;						
k Zgx?		hjfkmx?		sf]8			
64.	tkfO{n] slxNo} dw'd]xsf] -;'u/_ hfFr 8fS6/ jf	5	!	Н6			
	:jf:YosdL{af6_u/fpg' ePsf] 5<	5}g	@,olb 5}g eg]				
			CVD1 df hfg]				
65.	tkfO{nfO{ 8fS6/ jf :jf:YosdL{n] dw'd]x /f]u 5	5	!	Н7а			
	eg]/ eGg'ePsf] 5 .	5}g	@, olb 5}g eg]				
			CVD1 df hfg]]				
66.	tkfO{nfO{ lautsf] !@ dlxgfdf dw'd]x /f]u 5	5	!	H7b			
	eGg'ePsf] 5<	5}g	@ olb 5}g eg]				
			CVD1 df hfg]				
67.	tkfO{nfO{ dw'd]x /f]u nfu]sf] slt eof] <	jif{		Н7с			
		dlxgf		H7d			

68.	tkfO{n] xfn} 8fS6/ jf :jf:YosdL{af6 tn lbOPsf pRr/Qmrfk;DalGw pkrf/ jf ;Nnfx k fKt ug'ePsf] 5<						
	kl5Nnf] @ xKtfb]lv cf}iflw	5	!	НЗ			
	vfg lbg' ePsf] 5<	5}g	@	a			
		5	!	НЗ			
	g'gsf] dfqf sd ug{ lbg' ePsf] 5 .	5}g	@	b			
	jhg 36fpgsf nflu;Nnfx	5	!	НЗ			
		5}g	@	c			
	w'd kfg 5f]8\gsf nflu;Nnfx	5	!	НЗ			
		5}g	@	d			
		cfjZos gePsf]	*				
	zfl//Ls Jofofd z'? ug]{ jf	5	!	НЗ			
	klxn]eGbf al9 ug{	5}g	@	e			
69.	tkfO{n] cf^gf] pRr/Qmrfksf]	5	!	H4			
	36fpgsf nflu cfo'j]{lbs cf}ifwL, 3/]n' cf}ifwL h8La'6L cflb vfO/xg' ePsf] 5 <	5}g	@				

70.	tkfO{n] clxn] 8fS6/ jf :jf:YosdL{sf] ;Nnfx adf]lhd tn lbOPsf ;Nnfx tyf pkrf/ lnO/xg'						
	ePsf] 5<						
	OG;'Ing	5	!				
		5}g	@	Н8			
				a			
	vfg] cf}ifwL -h'g	5	!	Н8			
	tkfO{n] uPsf] @	5}g	@	b			
	xKtfb]lv lnO/fVg'ePsf]						
	5_						
	8fS6/4f/f vfg elgPsf]	5	!	H8			
	cfxf/	5}g	@	c			
	8fS6/4f/f jhg 36fpg	5	!	H8			
	lbOPsf];Nnfx	5}g	@	d			
	8fS6/4f/f w'd kfg	5	!	H8			
	TofUg lbOPsf] ;Nnfx	5}g	@	e			
	zfl//Ls Jofofd ug]{	5	!	H81			
	af/]df lbOPsf] ;Nnfx	5}g	@				
71.	slxNo} k/Dk/fut	5	!	Н9			
	:jf:YosdL{af6 dw'd]x - ;'u/_ sf] hfFr	5}g	@				
	u/fpg'ePsf] 5<						
72.	s] clxn] tkfO{n] dw'd]x	5	!	H1			
	-;'u/_ /f]usf] nflu	5}g	@	0			
	k/Dk/fut :jf:YosdL{af6	-13					
	pkrf/ tyf cf}ifwL						
	InO/xg'ePsf] 5<						

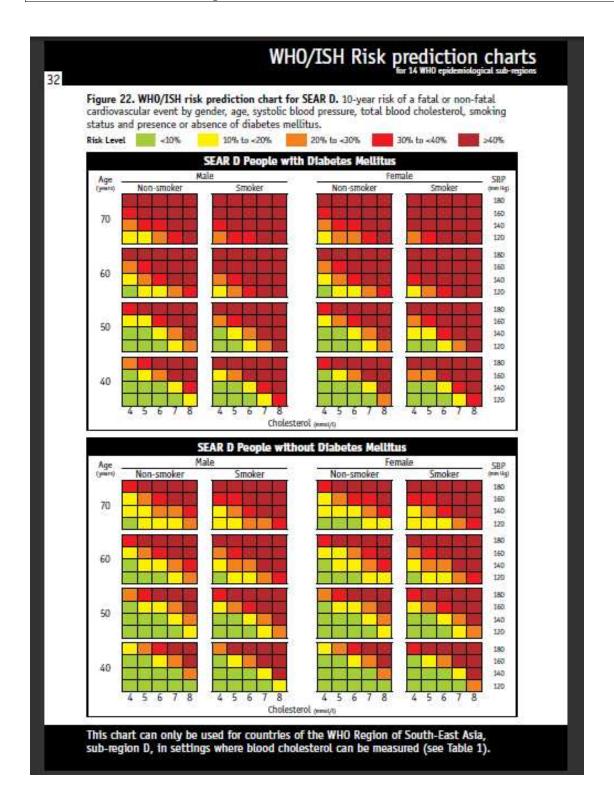
k/Ljf/df d'6' tyf /utsf] /Stgln ;DalGw /f]usf] Oltxf;						
k Zgx?		hjfkmx?		sf]8		
73.	tkfO{sf] a'jf cfdf bfO{ lblb sf]lx d'6'sf]	5	!	CV		
	/f]u, klff3ft, /StglnhGo /f]u nfu]sf]] 5 <	5}g	@,olb 5}g eg] Mp1	D1		
			df hfg]			
74.	slt jif{sf] pd]/df /f]u nfu]sf] lyof] <		1	CV		
		ji		D2		

	clGtd dlxgfjf/L ;DalGw- dlxnfx?sf] nflu dfq_						
k Zgx?		hjfkmx?		sf]8			
75.	tkfO{sf] dlxgfjf/L eO{/fv]sf] 5 ls /f]lSs ;Sof] <	e}/fv]sf] 5	!	Mp1			
		/l]lsoL	@				
		;Sof]					
76.	olb /f]lSs ;Sof] eg] , slt jif{sf] pd]/df /f]lsPsf]	jif{	========	Mp2			
	xf] ofb 5 <		====				

		r/0f @M	zf/Ll/s dfkg						
prfO{/j	hg								
k Zgx?			hjfl	kmx?				sf]	8
77.	7. k fljlws÷cGtjf{tf{stf{sf]							M1	
	cfO{ I8								
78.	prfO{		;]IG6ld6/df -		1		1 -	Ht	ı
			;]=ld=_						ı
79.	jhg		lsnf]u fddf -				1	Wt	·
	If too large for scale 666.6		s]=u f_] []
sDd/sf]	gfk		I.						
80.	sDd/sf] rf}8fO -;] dL_	;]IG6ld6/df	-l;=Pd=_					Wa	
/Qmrfk		I							
81.	/Qmrfk -gfk !_	l;:6f]lns (m	mHg)					BP	1a
		8fo:6f]lns	(mmHg)					BP	1b
82.	/Qmrfk -gfk @_	l;:6f]lns (m	mHg)					BP	2a
		8fo:6f]lns	(mmHg)					BP	2b
83.	/Qmrfk -gfk #_	l;:6f]lns (m	mHg)					BP.	3a
		8fo:6f]lns	(mmHg)					BP.	3b
84.	kl5Nnf] @ xKtf leqdf	InPsf] 5'		!				AH	[
	tkfO{n] pRr/Qmrfksf]	In Date (I to)							
	36fpgsf nflu 8fS6/n]	InPsf] 5}g		@					
	lbg'ePsf] cf}iflw								
	Ing'ePsf] 5 <								
lxksf] g	fk			_	•	_			
85.	lxksf] df]6fO	;]IG6ld6/df	-l;=Pd=_				1 [НС	Í
									İ

IS	Snlgss	f n	flu		
ı			r/Of #N	l afof]s]ldsn dfkg	
	pQ/bf	tfs	=======================================		=======
	f] gfd	y/	======		
	pQ/bf	tfs			
	f] klxrl	fg			
	g+				
	pQ/bf	tfs	=======================================		==
	f] kmf	lg			
	g+				
	k fljlw:	ss			
	f] gfd				
	/ut				
	InPsf]		lbgdlxgfjif{		
	;do		;do		
			306f ldg]6		
	91.	vf	nL k]6df lnPsf] /utdf lrgLsf]		FBS
			fqf	mg/dl	
	92.	U	n'sf]h vfPsf] @ 306f kl5 lnPsf]		PPBS
		/u	ıtdf Irlgsf] dfqf	mg/dl	
	93.	cf	h tkfO{n] OG;'Ing of cGo s'g}	5 !	AD
÷		81	fS6/n] lbg'ePsf] cf}iflw	5}g @	
		In	g'ePsf] 5<		
	94.	hl	Ddf sf]n]:6]/f]n		TC
				mg/dl	
	95.	kl	5Nnf] @ xKtf leqdf tkfO{n]	5 !	AC
		cf	kgf] sf]n]:6]/f]nsf] nflu 8fS6/n]	5}g @	
		lb	g'ePsf] cf}iflw Ing'ePsf] 5 <	0)9	
	96.	6	«fOUnfO;]/fO8		TG
				mg/dl	
	97.	Р	r= I8= Pn= sf]n]:6]/f]n		HDL
				mg/dl	

Annex IV: WHO/ISH risk prediction chart



Annex V: Kish Household Coversheet

List the sex and age of all adults in the house aged 40-80 years in the empty table below. To complete the Rank column, order all adults in the list by: Example:

• males in order of decreasing age (oldest to youngest) females in order of decreasing age (oldest to youngest)

Sex	Age	Rank
M	45	1
F	47	3
M	28	2
F	35	4

In the **Kish Selection Table** find the square whose column heading matches the last digit of the Household Number and whose row heading matches the total number of eligible persons in the household. The person whose <u>Rank</u> matches this number is the selected participant for this household.

List all persons age 40-80 in household

Sex	Age	Rank	Selected Respondent

Full physical household address:

Household Number

Number of		Last Digit of Household Number									
Eligible Persons in Household	0	1	2	3	4	5	6	7	8	9	
Kish Selection	Гаble:	1	1	1	1	1	1	1	1	1	
2	1	2	1	2	1	2	1	2	1	2	
3	3	1	2	3	1	2	3	1	2	3	
4	1	2	3	4	1	2	3	4	1	2	
5	1	2	3	4	5	1	2	3	4	5	
6	6	1	2	3	4	5	6	1	2	3	
7	5	6	7	1	2	3	4	5	6	7	
8	1	2	3	4	5	6	7	8	1	2	
9	8	9	1	2	3	4	5	6	7	8	
10	9	10	1	2	3	4	5	6	7	8	

mmol/l

mmol/l

Annex VI: Participants Feedback Form

ISnIgs sf8{ tyf k|ItIs|of kmf/d

	IdItM ========	:========
;xeflusf] gfdM		
=======================================	=======================================	=========
;xeflusf] I. D gDj/		
;xeflusf] df]jfO{n gDj/M=======	.======================================	:====
xflb{s wGojfb . of] ;j]{lf0fn] ;xe	f]lvd cg'dfg ;DalGw ;j]{lf0fdf tkfO{s flusf] pd]/, lnË, ;'lt{hGo kbfy{sf] ;]jg dfqfs]f cfwf/df d'6' tyf /QmglnhGo /	g, /Strfksf] cj:yf,
	ISnlgs	
:yfg========		
	;Dks{	
ldlt======		========
;do==========	=======================================	:=======
	=====	
tkfO{sf] zf/Ll/s dfk, ;'u/ tyf jf];f]s	sf] dfk o; k sf/ /x]sf] 5 .	
Blood Pressure	Systolic mmHg	
	DiastolicmmHg	
Blood Sugar	Fasting	mmol/l
	2 hr post Prandial after glucose	mmol/l

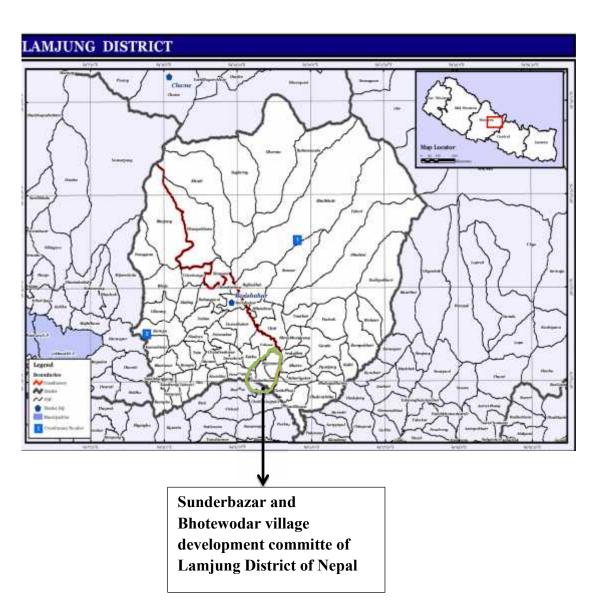
Total cholesterol

HDL

Triglyceride	mmol/l
--------------	--------

s[kof ISnlgs cfpFbf of] sf8{ Nofpg ge'Ng'xf]nf

Annex VII: Map of study site/Geographical Location



Annex VIII: Work plan

Work plan													
	July	August		epte ber	October			November				De c- Feb	Feb - Sep
Activities	W k 4	W k 4	W K 3	W K 4	W K 2	W K 3	W K 4	W K 1	W K 2	WK 3	W K4		
Proposal development and ethical clearance													
Literature Review													
Training for Data Collector													
Community mobilization													
Pilot testing													
Data collection													
Data Management and Analysis													
Draft Report Writing													
Final Report Submission													