RISK PREDICTION AND FACTORS ASSOCIATED WITH CARDIOVASCULAR DISEASES AMONG WORKERS AND THEIR SPOUSES IN TWO BEVERAGE PROCESSING INDUSTRIES IN RWANDA

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Risk Prediction and Factors Associated with Cardiovascular Diseases among Workers and their Spouses in Two Beverage Processing Industries in Rwanda

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DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

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DEDICATION

To the powerful Father of wisdom.

To my beloved wife Lilian and my two daughters: Eden Norah and Eden Naelle.

To my beloved parents, brothers, and sisters.

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ABREVIATIONS AND ACRONYMS

ASSIGN Assessment Scottish Intercollegiate Guidelines Network

ASTDR Agency for Toxic Substances and Disease Registry

BP Blood pressure

CAD Coronary Artery Disease

CDC Center for Disease and control

CHD Coronary Heart Disease

CI Confidence Interval

CNS Centre Nerve System

CRP C Reactive Protein

CSLA Cooperative Study on Lipoprotein and atherosclerosis

CVD Cardiovascular Disease

CVI Content Valid Index

CVPD Cardiovascular and Pulmonary diseases

DALYs Day Adjusted life years

DIP Drinks Processing Industry

DM Diabetes mellitus

DORICA Dyslipidemia, Obesity, and Cardiovascular Risk

FGRS Framingham General Risk score

FRS Framingham Risk Score

GDP Gross Domestic Product

HB Hemoglobin

HB1A3 Glycated Hemoglobin

HBM Health Believe Model

HDL-C High Density Lipoprotein-Cholesterol

HL Hosmer lemeshow

HPA Hypothalamic-pituitary-adrenal axis

HTN Hypertension

IHD Inflammatory Heart Disease

ILO International Labor Organization

KUTH Kigali University Teaching Hospital

LDL-C Low-density Lipoprotein-Cholesterol

LMICs Low- and middle-income countries

MI Myocardial Infarction

NCD Non communicable Disease

NCEP National Centers for Environmental Prediction

NICE National Institute for Health and Care Excellence

NYHA New York Heart Association

OSH Occupational safety and Health

PBT Planned behavior theory

PROCAM Prospective Cardiovascular Munster

QRISK Cardiovascular Risk Prediction algorithm

RHD Rheumatoid Heart Disease

SAM Sympathetic-adrenal medullary axis

SCORE Systematic Coronary Risk Evaluation

TC Total Cholesterol

TG Triglycerides

TRA Theory of reasoned action

UNPA United Nation Population Fund

USEPA United States Environmental Protection Agency

WHO World Health Organization

WHO/ISH World health Organization/International Society of Hypertension

YLD Years Lost due to a Disability

YLL Years of Life Lost due to premature deaths

DEFINITION OF OPERATIONAL TERMS

Atherosclerotic disease Refers to an intricated pathological phenomenon that undergoes many years to evolve into the inner layer of the vessels. It is within the lumen of medium and large-sized blood vessels that fatty and or adipose matter made by cholesterol accumulate to form the plaques. Therefore, the formed plaques reduce the vessel's caliber and flexibility due to the created irregularities. Thus, blocking the normal blood flow.

Behavior /Practices Is a series of repetitive actions and mannerisms expressed by organisms, systems, or artificial entities in return to their animate or inanimate environmental factors. It is the reaction of the system or organism to different inner or outer catalysts or inputs, whether deliberately or not deliberately, observably, or not observably, and awarely, or unawarely (Daniel, 2009).

Cardiovascular Diseases Refers to the general term that describes a variety of disease and condition, which affect heart and vessels. They include coronary heart disease, brain vessels diseases, Peripheral heart diseases including hypertension and heart failure (Yuling, 2009; Olvera, 2020). Rheumatic and congenital heart diseases were not taken into consideration in model-based risk prediction (Bengt, 2015).

Cardiovascular risk Is the probability of developing a heart and vessels diseases within a defined period. The 10 years is taken into consideration for the inbuilt model of prediction, as well as analyzing several risk factors simultaneously.

Health behaviors	Are the performed activities with the aim to prevent or diagnose illnesses or for heightening the quality of health and well-being. It is also explained as health maintenance, restoration, and enhancement-oriented gestures, activities, performance, and habits due to health impairing. Health impairing behaviors negatively affect health or otherwise make individuals susceptible to the disease. Some impairing behaviors are inactivity, tobacco use, high fat, and alcohol consumption. On the other hand, the person's promotion of health-enhancing behaviors creates health benefits and protects individuals from the disease (Corner, 2002).
Hypertension prevalence	Refers to the proportion of hypertension disease cases existing in study participants at a specific point in time (Ward, 2013; Spronk, 2019).
Incidence	Refers to the number of new cases of a disease over a period divided by the population at risk.
Prevalence	It is the result of the division of existing illness cases by the total population at a point in time.
Risk factors	Is any attribute, characteristic, or exposure of an individual that increases the likelihood of developing a disease or injury. Besides, it is explained as, variables, features, or hazards that will cause the development of certain illnesses to everyone who presents it in the entire population (Tora, 2016).

ABSTRACT

Cardiovascular diseases are responsible for 30% of all deaths worldwide and assume 80% of the burden in low and middle-income countries. Although they affect people at large, a big proportion of these diseases afflict people of working age with a great negative impact on premature death, dependencies, and loss of working days. Rwanda in 2008 was in the top region countries with high blood pressure prevalence in the whole African region. This study's aim was to determine factors associated with cardiovascular diseases predicted risk among workers and spouses of two beverage-processing plants in Rwanda and was conducted under a cross-sectional quantitative research design. The sample size of this study was 440 study participants calculated by the Cochran formula from 822 target population. The study used proportionate stratified random sampling for the sample size of the study where each subgroup was adequately represented. The instruments of this study were the WHO standardized questionnaire and cardiovascular diseases risk prediction models: WHO/ISH and FGRS (Framingham general risk score). The Data was analyzed by SPPS version 22, where a descriptive, bivariate, and multivariate analysis, C-statistic, and Kappa test with 95%CI were applied. The significance was set at $p<0.05$. Overall risk prediction (<10%) by FGRS and WHO/ISH score were 74.5%, 95.4%, respectively while the CVD elevated risk (>=10%) was 25.5%, 4.6%, respectively. FGRS CVD risk (>= 10%) was 16.1% of males versus 9.3% of females while 2.7% of males versus 1.5% of females classified by WHO/ISH. CVD risk increases in both models with age but very much in FGRS. 8.4% of employees versus 5.2% of spouses are classified as having the risk of 10-20% by FGRS while WHO/ISH classify 2.5% of employees and 0.9% of spouses as having the risk of 10-20%. FGRS classified 11.7% of all participants as having absolute cardiovascular diseases risk above 20% while WHO/ISH classify only 1% as having absolute cardiovascular diseases risk above 20%. Two models' kappa agreement level was fair or minimal interrater reliability with 0.25 with a p-value < 0.001 and the correlated ROC curve of FGRS and WHO/ISH of 0.887 AUC, 0.847AUC all with a p-value <0.001, respectively. Night shift dominated other working conditions with AOR=2.41(1.27-4.58), p=0.007, and a high level of sedentary (>10hrs) also dominated to be associated with metabolic diseases with crude OR=8.196(2.07-32.3) while its association to CVDs was: OR=3.777(1.7-8.2), hypertension prevalence was 32.27% in the previous classification while it was 61.81% in updated classification, metabolic syndrome prevalence was 38.2%. The use of PPE for Noise and chemicals was negatively correlated with cardiovascular disease risk for both models after Kendall's tau_b -0.218 p<0.001, -0.157 p=0.004, respectively, and spearman's rho test-0.244, p<0.001, -0.175, p=0.004, respectively. Noise and vibration and radiation were significantly associated with cardiovascular diseases by Framingham risk score with p<0.05. The three unchangeable factors (Age, Gender, and family history) were associated with CVD risk, p<0.05. The workplace risk factors such as radiation and high stress with AoR=0.36(0.15-0.86), p=0.02 and AoR=21.398(2.65-172.59),p=0.004. The FGRS showed that eight modifiable factors were associated with CVDs while it was only four factors for WHO/ISH. The most prominent factors were diabetes, hypertension, low physical inactivity (<600MET/Week), times exceeded alcohol standards, low fruits, and vegetable intake, and tobacco use. The two novel risks (CRP and HBA1C) were four to fivefold linked with cardiovascular disease risk, p<0.05. Male employees dominated other groups for 10 risk factors. The study conclusion demonstrated a fair agreement between the two models and suggested the usage of FGRS for proactive management of cardiovascular disease risk. Additionally, the employees presented a high cardiovascular disease risk with more novel risk factors and traditional risk factors than the remaining groups. Moreover, a culturally based strategy, evidence-based preventative program, workplace, and community policies would be advised to establish a safe world of work in the industrial environment and for the community at large. Hence, a reduction of CVDs direct and indirect costs and improvement of quality of health and production.

CHAPTER ONE

INTRODUCTION

1.1 Background of the study

Cardiovascular diseases and their epidemiology did not exist in 1946. Then Ancel Keys and his group (Medical Marco Polos) established the laboratory of physiological hygiene regarding cardiovascular diseases (Frederik, 1996). Including the Evidence-based clinical and preventative public health approach (Blackburn & Darwin, 2012).

The birth of the Framingham heart study, on the idea of Joseph Mountin in 1946 was the cardiovascular epidemiology milestone. This was influenced by White Dudley, who was the presidential medical doctor in the US during World War II. The Framingham heart study started after the death of Franklin Delano Roosevelt due to a stroke caused by hypertension and stress (Mahmood, 2013). This death happened two months following WWII aftermaths management meeting with allied country presidents, Joseph Stalin and Winston Churchill at the Black Sea, Crimea today (Bitton, 2010).

In addition, The US showed that cardiovascular diseases were the most economic burden on businesses including death and disability waged by these medical conditions (CDC, 2004; American Heart Association., 2005; Rohan, 2020).

The global burden of disease has drastically changed from infectious diseases, perinatal, maternal, and nutritional causes, to non-communicable diseases (NCDs) (Fuster, 2014). In India alone, two-thirds of the total morbidity burden were attributed to NCDs for the entire population. Around 53% of total mortality (40.4% in 1990 to 59% in 2015) were caused by the abrupt change in the country's society and lifestyle (WHO, 2013).

A study carried out by Dumper in 2007 showed that after the Primitive drink-processing maneuver, which started in Mesopotamia, Iraq today earlier in 2500 years

BC, the new working conditions and technologies potentially produce risks to the workforce. Some of those working conditions involved hazardous substances, where over one million chemicals were identified by the international labor organization (ILO). Moreover, thousands more are created every year and can harm the heart health of the workers (Elisa, 2010).

The change that marked the last century was a typical example of economic development, industrialization, broad urbanization, and market globalization (Fuster, 2011). Thus, the significant shift in food patterns, type of diet, and inactive lifestyle endangered the current society toward the increase of cardiovascular diseases (Santosh, 2020).

Heart disease and stroke fatality were forecasted to increase to more than 24 million by 2030 and be the major global population cause of death and disability by 2020 (Mozaffarian, 2008; Fuster, 2011). These deadly killers are currently burdening the low and middle-income countries, which harbor 80% of global deaths related to cardiovascular diseases in comparison to the western countries (Fuster, 2014).

In the United States, cardiovascular diseases were high, where 82 million people had one or more types of CVDs, 76 million had high blood pressure, 16 million had CHD, 7 million had Stroke in 2007 and 800,000 people died in 2009 (Roger, 2011). In addition, heart conditions consumed 116.3 billion dollars in 2011 and were projected to be 1,208 billion dollars from 2015 to 2030 (Mozaffarian, 2015).

A study carried out by Goldhaber in 1983 showed that carbon tetrachloride and carbon disulfide were the sources of exposure for more than five hundred employees in the United States in 1983. Hence, they noted an increase in heart issues in settings with exposure to carbon disulfide (Chung, 2017). The rayon industry employees presented 2.5 times the death rate among men than in other industries due to carbon disulfide exposure for more than 10 years. This was due to the workplace hazards brought into the industrial world by new technology. Currently, technology is an important component, especially in the brewery industry. The change of primitive drinks processing maneuver, which started in 2500 BD in Mesopotamia, Iraq Today, to modern technology is an obvious hazard. This exposes many people of working

age to Workplace hazards (Stress, Sound, vibration, Cold, chemicals) (Elisa, 2010). Whereas the international labor organization at the World Day of Health and safety in 2013 emphasized safety and health in the use of chemicals. Thus, prevention of occupational diseases which can jeopardize the health of workers (ILO, 2014).

The workplace provides a large audience to cardiovascular diseases, where workers in 2012 were 2.9 billion and projected to be 3.5 billion in 2030 around the world (MGI, 2012). An estimated 25% to 30% of companies' medical costs per year were spent on employees with the major cardiovascular risk factors in the USA (Carnethon, 2009, Elizabeth, 2018; Rohan, 2020).

Besides sleep time, work is a reality that globally consumes more time in human life. Furthermore, it obliges many people to stay at the worksite. The workplace is a necessary environment for the adult world. It consists of elements that can improve health as well as elements that can harm it. This happens in the case of the presence of cardiovascular risk factors in the working environment (Carnethon, 2009). In addition, the worksite is an appropriate setting to provide various opportunities to promote the adoption and maintenance of healthy lifestyle behaviors (WHO/WEF, 2008).

Observational and nutritional research was carried out in Massachusetts at EMC Corporation. This study included employees and their spouses to tackle factors of cardiovascular diseases such as Obesity, not eating fruits, and sedentarity, showed good results in their nutritional approach. They also included spouses to show that workplace is tied with home regarding the nutritional issues (Thomas et al., 2008).

The shared social-economic factors such as high industrial wages and beverage donations from the brewery, behaviors, and a common environment among spouses are the major contributors to similar health outcomes. As found in research done in US communities for estimating the association of hypertension status to spouses. Being married to a hypertensive person was taken as an exposure to develop High blood pressure which is a major risk of cardiovascular diseases (DeMarco et al., 2011).

The involvement of spouses in this study is of great importance to better understand work-home stress spillover. Where stress experienced in one domain results in stress in another domain of life. In other words, stress crossover when experienced at work can be transmitted to the spouses at home (Bolger, 1989; Chong, 2016). Moreover, type two diabetes, coronary heart disease, and stroke were found associated with workplace stress (Mika & Ichiro, 2015).

A study done on Korean couples highlighted the crossover effect of spouse weekly working hours on an estimated 10-years risk of cardiovascular diseases. The Comparison showed that the partners with spouses who worked 30 hours per week, developed higher cardiovascular risk than partners with spouses who worked a few hours per week (Kang, 2017).

1.2 Statement of the problem

Cardiovascular diseases exasperate the working-age people (Wolfgang, 2007) and are responsible for 30% of all deaths worldwide. CVDs assume 80% of the burden in Low- and Middle-Income Countries (Jabaris, 2014). Especially in sub-Saharan Africa, wherein 2017, a ten-year retrospective study done on heart failure scope in three rural districts of Rwanda demonstrated that 719 patients who managed to get to the clinic were transferred with confirmed heart failure. Females were 72% of the adult majority and 78% were farmers, 39.7% of the patients had dilated cardiomyopathy, and 26.8% had rheumatoid heart diseases. Hypertensive heart diseases were 13% and only 42.8% were retained and alive, 29.5% of their deaths were documented, and 23.9% were lost to follow up, which highly explains the undocumented death due to missing in their rural environment without any health care (Eberly et al., 2019).

In 2012/13, a STEPS study was carried out in Rwanda as stated in NCDs policy. This study showed the shared risk factors for non-communicable diseases and cardiovascular diseases. Where 12.9% were tobacco users, 99.7% could not eat fruit per day. A proportion of the 91.1% could not eat enough vegetables, 78.6% were not engaged in low-level physical activity, 41.3% were alcohol drinkers and 23.5 were episodic binge drinkers (WHO, 2015).

Cardiovascular diseases are an impediment in the life of the labor force, increase dependencies, and lost working days (Stephen et al., 2003). Moreover, WHO statistical profile, 2015 has shown that Rwanda has lost around 300 DALYs due to only cardiovascular diseases and diabetes.

One Cardiac Surgery Hospital serves 120,000 People, in North America, while it is 33 million per Cardiac Surgery Hospital in Sub-Saharan Africa (Jabaris, 2014). The stagnant of patients due to a lack of health professionals increases the morbidity and mortality in Rwanda and in the region without knowing the cause. Mucumbitsi stated that in sub-Saharan Africa including Rwanda 2.5 million heart patients (5-16yrs) have been diagnosed and 300,000 deaths have been recorded per year in 2007.

Cardiovascular diseases contributed to more than 13% of overall mortality in Kenya. Myocardial infarction was Obvious, while rheumatic heart disease was rare (Ogeng, 2011). Hospital mortality by cardiovascular diseases was high reaching 9.2% in Cameroon and 21.9% in Tanzania (Mocumbi, 2012), and 14% of all deaths in Rwanda in 2016 (WHO, 2018). In addition, a heavy burden on global employees' health was 50% of all causes of death and at least 25% of work disability (Tsutsumi, 2015).

According to the beverage industry absenteeism record in 2013, cardiovascular diseases were one of the three major causes of workplace absenteeism (153 days lost for CVDs). This shows only to what extent cardiovascular disease burdens life and the indirect cost that weighs on the industry. It is also necessary to fight this emerging problem in Rwanda and in the industrial arena where many risk factors are higher than in other industries. As some studies have shown that Arterial hypertension prevalence among workers in the soft drink industry was 27% and 26% in the São Paulo City hospital complex. However, it was 24.7% among workers at an iron and steel company (Cavagioni, 2012).

WHO statistical profile, 2015 showed that Rwanda in 2008 was counted in the top region countries with high blood pressure prevalence (43.6% among males; 40.2% among Females). Whereas the whole African region's high blood pressure prevalence was 38.1% among males, and 35.5% among females. Around 10% of cardiovascular

diseases equaled to the low respiratory infectious diseases which were the first killer diseases in Rwanda. A recent multicenter stroke study in Rwanda showed a worse stroke burden. Where around 2.1% of all received patients were due to stroke and about 61% of stroke patients died and 14.3% were tremendously disabled (Nkusi, 2017).

1.3 Justification of the study

The industrial manufacturing process produces hazards, which can endanger the employees and the community as well. The Rwanda industrial survey in 2013 by establishment census carried out in 2011 showed that around 97% of all Rwandan manufacturing industries were micro institutions. In addition, around 2.2% were small and medium industries, whereas big industries were from 0.2% to 0.8%. The beverage industry was ranked as one of the big industries in terms of workers number and production as well (Kamarudeen, 2014).

This study of cardiovascular diseases prediction and associated factors in beverage manufacturing industry served as a unique watertight evidence-based and model-based prediction approach. In addition, this paramount approach is significant, in occupational clinics at the worksite and a cost-effective health program to fight heart diseases. The proactive and reactive program should also rely on the research findings as corroboration and nationalization of international theory on the matter.

There are various external and internal factors affecting cardiovascular health. Apart from working conditions, the worker's spouses in the drink processing industries would be affected by the availability of financial resources. This is high in beverage industries compared to other employers in society. Their economical level and easy accessibility to beer and soft drinks by bonuses from their companies may expose them on sedentarity, mechanized transport, and high BMI due to change of diet and lifestyle.

Therefore, the change in working nature in many organizations has pushed us to undertake this study in the manufacturing workplace. Where the working way has been radically transformed due to the growth of industrial technology hazards.

Especially in the drink processing industries, the exposure was influenced, and dictated by the presence of multifactorial issues related to cardiovascular diseases. It is in this working environment that daily hazard such as alcohol and soft drink may be available. Hence, irresponsible consumption may be a source of the overweight, high blood sugar, and heart muscle weakness if not moderated (Zahran, 2017; Romina, 2020). Working in a stressful workplace and with much sound area (Zamanian, 2013). And staying longtime in the chamber with refrigeration and cooling systems, ice, and stress were associated with CVDs and the Reynaud phenomenon in causing peripheral vascular diseases (Plissonneau, 2015). Carbon dioxide exposure in brewing, fermentation using yeast and malt (Smallegange, 2010), and carbonated drinks manufacturing (Cable, 2004). The presence of Sodium hydroxide (NaOH) used in bottle washing and NO2(nitrogen dioxide), Ammonia gazes are daily hazards. These hazards may cause less severe lesions of the heart, lungs, and skin to workers in the production of soda if not protected. The metal intoxication of the drinks was assessed three decades ago when cobalt was used in American and Canadian breweries. This study showed its serious effects on the heart as stated by Alexander in 1972 and Goldhaber in 1983. However, a recent study showed a lack of negative effects of cobalt on the heart (Lantin, 2013). Today's mechanized transport, sedentarity and new brewing technology effect are in such worksite where adult at working age can meet those factors. All these risk factors can be more found in this workplace than being found alone outside in the community.

The linkage of the workplace, community, and cardiovascular diseases explained the purpose of showing the riskier and unsafe working conditions. Additionally, the strategy to address, protect and advise the workforce, employers, community, and policymakers. Hence, developing a safe working environment where a part of society spends most of their time.

Additionally, the significance of this study is to reduce the cost spent on cardiovascular diseases and improve the health of the brewery workforce and their spouses in Rwanda. Hence, this generated a positive impact and more benefits in increasing awareness and behavior change toward CVDs Risk Factors. Therefore, a reduction of the direct cost due to treatment and indirect cost due to low productivity,

presenteeism, workers' compensation, absenteeism, and low quality of health. Furthermore, this study's significance is to help reduce the deaths, disabilities, and dependencies due to cardiovascular diseases. Hence, reducing the cost of doing business.

1.4 Objectives

1.4.1 General objective

To determine factors associated with cardiovascular diseases predicted risk among workers and spouses of Kicukiro soft drink plant and Rubavu brewery plant and their spouses.

1.4.2 Specific Objectives

i. To predict the 10-year cardiovascular diseases risk and compare two prediction models among the study participants in the study area
ii. To determine the behavioral factors associated with cardiovascular diseases among the study participants in the study area
iii. To determine the Working condition factors associated with cardiovascular diseases among workers in the study area.
iv. To determine the proportion of people with Awareness of cardiovascular diseases factors and using personal protective equipment among the study participants in the study area.
v. To determine the proportion of people with biological risk factors among the study participants in the study area.
vi. To determine the differentials between groups for novel risk, traditional risk factors, and cardiovascular disease risk levels among the study participants in the study area.

1.5 Research questions

i. What is the 10-year cardiovascular diseases Risk and the agreement level between two models among study participants in the study area?

ii. What are the behavioral, and lifestyle factors associated with cardiovascular diseases among study participants in the study area?
iii. What are the work condition factors associated with cardiovascular disease among workers in the study area?
iv. What are the proportion of people with awareness of cardiovascular diseases and using Personal protective equipment among study participants in the study area?
v. What are the proportions of people with biological factors associated with cardiovascular diseases among study participants in the study area?
vi. What are the differentials between groups for novel risk, traditional risk factors, and cardiovascular diseases risk levels among the study participants in the study area?

1.6 Variable of the study

The dependent variable in the study was the cardiovascular diseases risk, defined as a predicted risk level of fatal and non-fatal cardiovascular diseases (heart failure, cerebrovascular diseases, coronary heart disease, and peripheral vascular disease). The independent variables were eight predictors (age, gender, total cholesterol, HDL, smoking, blood pressure, treated and untreated hypertension, and diabetes). The cofactors such as working conditions, and behavioral and biological factors were also the contributory independent variables. The moderating variables were the awareness of cardiovascular diseases risk factors and using personal protective equipment.

CHAPTER TWO

LITERATURE REVIEW

2.1 Cardiovascular diseases of the study interest for risk prediction

Cardiovascular diseases represent a constellation of diseases that affect the heart and the vessels (Yuling, 2009; Olvera, 2020). The subject of this study was atherosclerotic cardiovascular disease (CVD) events, such as coronary heart disease, cerebrovascular disease, peripheral vascular disease, and heart failure (D'Agostino, 2008). In addition, many others like congenital; rheumatoid; congestive; Arrhythmias heart diseases, and Aortic aneurism were not studied in this research. Although they are all heart and vessel pathologies and are typically categorized into structurally, electrically, and circulatory aspects but not necessarily associated with atherosclerotic process (Bengt, 2015; Thiriet, 2018; WHO, 2021).

2.1.1 Coronary heart disease

Coronary heart disease is the illness of the blood vessels supplying the heart muscle (Coupland, 2017). It is one common type of heart disease, which is sometimes referred to as coronary artery disease (Janet, 2009). It consists of a number of diseases due to atheromatous changes in coronary vessels (Ashley, 2004). It was considered to be a simple, ineluctable process of reducing the artery caliber. It consequently causes Ischemia (Oxygen deficiency in the heart muscle cells) or finally ended by completely blocking the blood vessels, which leads to myocardial infarction (heart attack) (Janet, 2009). The discovery of the existence of plaque in the entire coronary spectrum changed the explanatory pattern. It showed the plaques phases toward rupturing, where the stable phase was composed of the thick fibrous and poor lipid cap. The unstable phase was composed of the lipid-rich and thin fibrous cap. The rupturing of unstable plaque makes it much more unstable and much more prone to future rupture. Hence, the additional vasoconstrictive and prothrombin secretion raise the probability of obstruction of the entire artery (Peter & Pierre, 2005). Furthermore, the prognostic outcome at the rupture site was determined by the thrombolytic pathways and the body's prothrombotic balance. The transitory

blockage leads to pain and ischemia; permanent obstruction leads to transmural Myocardial Infarction (Ashley, 2004; Taqueti,2018).

2.1.2 Heart Failure

Heart failure is a chronic condition with cardiac reduced longevity. It is characterized by a group of signs and symptoms of the inability of the heart to execute its usual function of pumping blood, which marks the four-stage of cardiac dysfunction.

To set up a diagnosis of heart failure, the European Society of Cardiology guidelines warrant the presence of signs and symptoms and objective-based evidence of cardiac dysfunction preferably by echocardiography. Hence, a favorable response to treatment was established for heart failure (Arend, 2007). The clinical definition establishment of heart failure was based on several studies and provided various views. However, the universal definition demonstrated heart failure as a clinical constellation of signs or symptoms due to functional and structural heart abnormality. This is confirmed by the high levels of the natriuretic peptide with evidenced pulmonary systemic congestion. Heart failure has been divided into four levels(A-D) based on individual signs and symptoms accumulation. Although reduced ejection fraction <40% was not considered, it hence, suggested classifying the heart failure. (Coronel, 2001; Bozkurt, 2021).

2.1.3 Stroke

A stroke is the turmoil of the blood provision into the brain. This may be caused by either obstruction (ischemic stroke) or a burst of a blood vessel (hemorrhagic stroke) (Coupland, 2017). Moreover, it is a significant cause of death and disability. A stroke is a neurological deficiency due to a central nervous system (CNS) focal injury.

It starts with a vascular cause, cerebral infarction, and intracerebral hemorrhage (ICH). In addition, it includes the subarachnoid hemorrhage (SAH). Hippocrates circa firstly used this condition as apoplexy in 400 BC. Furthermore, William Cole introduced the word stroke in medicine in 1689 as stated by Adams in 1939 (Sacco, 2013).

2.1.4 Peripheral vascular diseases

Peripheral vascular diseases (PVDs) are the affections of all blood vessels outside the heart and brain due to atherosclerotic vascular impairment. Besides, the distant veins, arteries, and lymphatic vessels of legs and arms, PVDs attack the vessels that supply the blood into the organs below the stomach.

Peripheral vascular diseases (PVD) showed up as inadequate tissue perfusion due to the early deposit of atherosclerosis (Thukkani & Kinlay, 2015). This is associated with either embolus or thrombus (Suzuki, 2015).

Peripheral vascular disease (PVD) is the universal term, which consists of venous, vasospastic, and lymphatic diseases. The disease of veins is subdivided into two categories where, the first category is regarding the Obstruction of the vein due to a blood clot or thrombosis. There are also two types of thrombosis (Deep vein thrombosis (DVT) and superficial thrombophlebitis (ST)). The second category is the insufficiency of drainage of the veins. It is caused by either a blood clot or inherited vein wall abnormality. The classification of the second category can also fall into deep thrombosis, with chronic venous insufficiency, and superficial thrombophlebitis with varicose veins (Joshua, 2002; Gul, 2022).

The different meanings of words justified the preference for utilization of the general term" disease" rather than utilizing “stenosis". For instance, the general term such as "renal artery disease could be utilized in the place of "renal artery stenosis" instead. Because the artery occlusion and artery stenosis may present similar clinical manifestations. Hence, the term" disease" is suitable for covering all conditions (Mark et al., 2008). Peripheral vascular diseases have different types of subdivisions due to the significant manifestations of various organic and functional dimensions. The arteries diseases caused by atherosclerosis are having one general term, which is "Atherosclerotic vascular disease".

Peripheral vascular diseases apart from Heart and cerebral vascular diseases

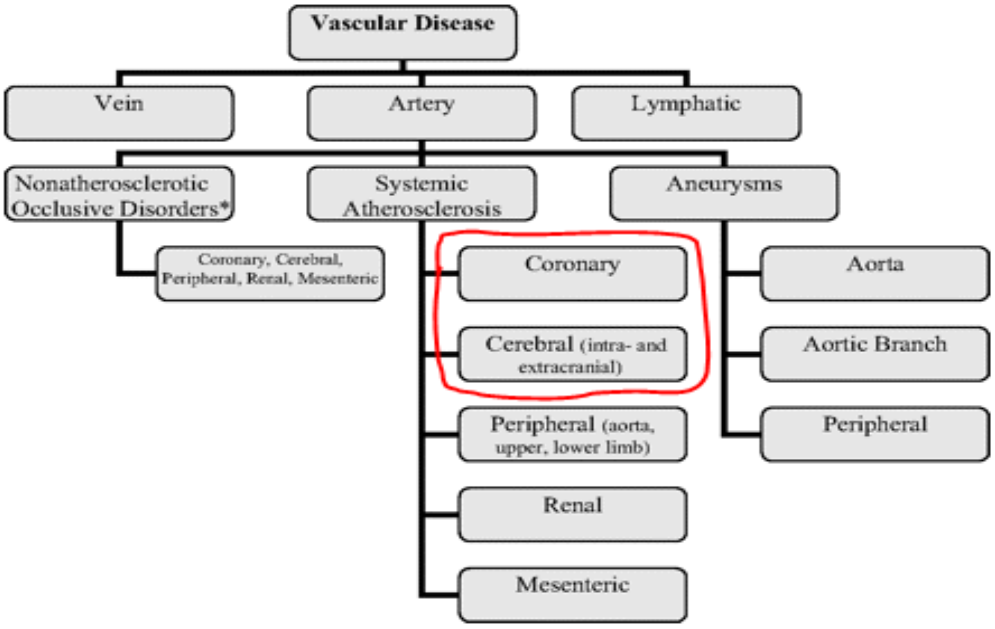

Figure: 2.1: Peripheral vascular diseases

(William, 2008)

2.2 Cardiovascular diseases Risk

2.2.1 Risk prediction

Risk prediction is a crucial element in cardiovascular diseases prevention and global health as well. The predicted risk level for cardiovascular diseases development in a defined time was neatly studied to proactively understand and prevent the factors associated with the diseases (Yang, 2020). The severity, exposure, and probability risk assessment model (SEP model) is mostly used in a workplace incident and accident risk assessment. However, this study used the pre-established models (WHO/ISH and FGRS) to predict the 10-year cardiovascular diseases level for proactive cardiovascular diseases management. The model for risk prediction refers to mathematical equations that used patients' information as predictors to generate the probability of a patient to develop the disease. Hence, the risk level was generated for a defined period (Xia, 2019).

There are many cardiovascular prediction models such as FGRS, PROCAM (Prospective Cardiovascular Munster), ASSIGN, QRISK1, QRISK2, and SCORE (Systematic Coronary Risk Evaluation) (Selvarajah et al., 2014). The Reynolds Score and WHO/ISH (Stephan, 2015).

However, four of them are cardiovascular risk prediction models calculated at the time of renal transplant, which are the Framingham Risk Score and the European Systematic Coronary Risk Evaluation (SCORE) equation. The REGICOR Registre Giron´ı delcor (Gerona Heart Registry), and the DORICA (Dyslipidemia, Obesity, and Cardiovascular Risk). The last two models were adapted from the Framingham equation for Spanish population characteristics (Mansell, 2014),

There are various ways of model development using mathematical equations, among others, logistic and Cox regressions (Stuart, 2018).

2.2.2 Cox regression formula

$$\Rightarrow \hat{p} = 1 - s_o(t)^{exp(\sum_{i=1}^{p} ßiXi - \sum_{i=1}^{p} ßi\bar{X}}$$

$\hat{p}$ = Cardiovascular risk

p= denotes the number of risk factors.

$S_o(t)$= denotes the baseline survival at follow-up time t (here t=10 years)

ßi=is the estimated regression coefficient (log hazard ratio)

Xi=is the log-transformed value of the *i*th risk factor, (if continuous)

$\bar{X}$ =is the corresponding mean.

The survival or failure time data was processed through cox or proportional hazards regression.

The original Cox regression importance was not to measure the mean and another measure of location. However, it was to model the hazard function, which is sometimes called the intensity function or the mortality force. The hazard function is the likelihood of estimating the death of alive people in a certain little unity of time. A conditional hazard was proposed to be modeled as the product of an arbitrary baseline hazard λ0(t) by the cox model (Cox, 1972). In addition, it is a linear exponential form in an explanatory variable (Andersen, 1991; Zhang, 2018).

The cox model was explained as the multiple linear regression of hazard logarithm on the variables xi, with an intercept being the baseline hazard, which could vary with time (Cox, 1972; Stuart, 2018).

The hazard is multiplicatively affected by the covariates at any point in time. Hence, it generates the key assumption of the proportional hazard model, which is that "the hazard of the event in any group is a constant multiple of the hazard in any other" (Nihal & Tekin, 2007). In addition, the hazard curves for the group must be proportional without crossing, which is the implication of the above-mentioned assumption. The quantities exp (bi) were called hazard ratios due to proportionality implication.

Furthermore, the covariate value (ith) grows with the event hazard. it is, therefore, the result of the symmetrical increase of (bi) value and hazard ratio higher than zero and one, respectively. Hence, the extent of survival period decreases" (Clark & Bradburn, 2003). The event likelihood is positively associated with the covariate when a hazard ratio is superior to one. The higher the covariates and the hazard ratio, the higher the probability of the event happening. Hence, the length of survival is negatively impacted. In other words, survival is reduced. The Cox model is mathematically exhibited as:

$$h(t) = h_0(t) \times \exp\{b_1 x_1 + b_2 x_2 + \cdots + b_p x_p\}$$

It is noted that the use of hazard formula h(t) depends on p covariates (x1, x2, y, xp) whereby its influence may be calculated based on the size of its coefficients (b1, b2,

y, pb). In addition, the h0 is named the baseline hazard on the condition that all xi's are equal to zero. In the same way, it was well clarified that the hazard may change from time to time if we use the function "t" in h(t) (Bradburn, 2003; Zhang, 2018).

2.2.3 Ankle brachial index

The Ankle-branchial index (ABI) is the cardiovascular health gauge, which predicts cardiovascular risk and indicates the systemic atherosclerosis establishment in the human body. The ABI levels under 0.90 were associated with cardiovascular disease occurrences and eventual mortality (Matthew, 2018). The ankle-branchial index is a useful benchmark, which informs the establishment of peripheral arterial diseases (PAD) at the lower-extremities (Lange, 2007).

It is an easy measure to perform and is a cost-efficient gauge that can serve the primary health care facilities to discover peripheral arterial stenosis. In addition, it can detect the patients with arterial injury of the lower extremities after a certain disease or accidental penetrating or blunt trauma.

More than 50% of lower-extremities arterial stenosis was associated with the ABI gauge under 0.90 with high specificity and sensitivity of 98%, and 90%, respectively (Ouriel, 1982). Another study showed similar results, where the sensitivity and specificity of ABI<0.90 were exceeding 87% and 97%, respectively. It also executed for detecting the lower-extremity arterial injury in an accidental emergency setting (Johansen, 1991; Casey, 2019). The ABI interpretation was executed by ranges creation to facilitate the readings and decisions.

The range of 0.00-0.40, is the status capable of causing pain at rest and even gangrene, 0.41-0.90, is the peripheral artery diseases (PAD), which is capable of causing claudication. The level between 0.91-1.30: is the normal range that indicates the normal peripheral cardiovascular health. Finally, the level >=1.31: was associated with severe vessel calcification (Chan, 2015). A study done by Carmo in Brazil showed that measuring the ABI using a stethoscope was a practical method to discover the PAD. In addition, the mean stethoscope ABI was 1.01± 0.15, and the mean Doppler ABI was 1.03± 0.20 with p=0.04, which showed a good relationship.

The gold standard test comparison showed a specificity of 91%, 95% CI (81.5-96.6), and a sensitivity of 71.4%, 95%CI (41.9-91.6). Hence, the Receiver Operating Curve (ROC) was 0.895, 95% CI, (0.804–0.986), $p < 0.001$, (Carmoa, 2008). However, ABI was not used here because it predicts only the PAD risk (Casey, 2019).

2.3 Cardiovascular diseases risk factors

The risk factors concept (RF) was turned into the foundation for the prevention of morbidity and mortality from cardiovascular diseases and other non-communicable diseases. In addition, it was largely used in clinical and public health practice (Petrukhin & Lunina, 2012). Moreover, the risk factor can be based on finding out in the workplace what is the major source of cardiovascular diseases. This is perfomed for improving the employees' life in changeable working environmental exposures, as adults people spend most of their working time at work (McEachan, 2008). Hence, it makes the workplace ideal for providing health promotion education (WHO, 2012).

The classification of cardiovascular risk factors in three different constellations is paramount to the prevention of cardiovascular diseases (CVDs). Because they indicate the modifiable risk factors to target before and after the establishment of the disease. Moreover, those factors are necessary for every onset of CVDs. The **reversible** risk factors are the changeable risk factors to prevent the non-established or reverse the established condition. These factors are among other sedentarity, alcohol abuse, stress, smoking, obesity, arterial hypertension, and hyperlipidemia. The **irreversible** risk factors are the unmodifiable factors, which are age, family history, genetic, and gender. The **partially reversible** factors are the factors that you can partially modify, which are diabetes and menopause.

One or more cardiovascular risk factors could be prevented from individuals who annually develop stroke and heart attack. We can find among others: hypertension, diabetes, smoking, physical inactivity, high-calorie, and saturated fat diet, obesity, and high stress (Roger, 2011).

2.3.1 Social-demographic risk factors

In the study done in Bangladesh concerning the risk of developing heart and vessel diseases in relation to the social demographic factors. They found that regional residence such as urban was onefold associated with CVDs than rural residence (AOR=1.32). The old age (≥70 years) (AOR=2.87) was twofold (AOR=2.87) associated with CVDs while being aged (55-69 years) was onefold AOR=1.95) or ≥70 years than 35-54 years. Urban residence and old age were significantly associated with higher CVD risk (Rahman, 2015). In Canada, the period from 1994 to 2005 was marked by a heart disease increase of 19% for men and 2% for women with 1.29 million patients in 2005.

The lowest-income countries showed a major increase of 27% in heart disease, 37% in lower-middle-income countries, and finally 12% in middle-income countries. However, the highest income countries showed a slight increase of 6% (Douglas et al., 2009).

2.3.1.1 Age

Age is an unchangeable factor, which acts as an independent factor in cardiovascular disease. The aging-induced alterations on the heart. Such alteration in the heart and in arteries decreased the elasticity, and the atherosclerosis process decreased compliance of heart activity. Hence, heart mass increased, the fibrosis, and calcification follow. In addition, this previous aging process increases the resistance to the heart-pumping action. Therefore, it hampers the work of the heart to deliver blood to different parts of the body. Moreover, it leads to cardiovascular diseases and events such as increased blood pressure, angina pectoris, heart attack, and heart failure. Aging is, hence, an independent risk factor for cardiovascular diseases (Shlomo, 2003; Jennifer, 2019).

2.3.1.2 Gender

The misperception of women's protection in reproductive age based on endogenous estrogen that trivializes the risk of women suffering CVDs. it was also shown that

CVDs evolve in women, seven to ten years after their development in men (Maas & Appelman, 2010).

Coronary heart disease (CHD) occurrence in men compared with women was ≈3 times higher and mortality was ≈5 times higher with an obvious gender difference in a middle-aged population. Moreover, men presented two to fivefold more coronary heart diseases than women. However, the difference remains obvious between populations. Biological differences and gene expressions for sex hormones are the major causes of differential risk factors. This is shown by the remarkable difference in vascular function, signaling level, and myocardial remodeling under stress, or metabolism of drugs by sex-specific cytochrome expression (Vera, 2015). Although more cardiovascular risk factors were auspicious to females, age increase reduces drastically the level of risk factor gender difference. The risk of CHD increases either for men or for women with age. However, females have a higher risk of cardiovascular diseases than age-matched men (Jennifer, 2019).

2.3.1.3 History of family diseases

Family history of cardiovascular diseases can differently affect the family members with dependence on the age and position of first-degree relatives. The offspring of the patient with cardiovascular diseases have the CVD risk from 60% to 75%, which is relatively high than a CVD risk of 40% for the siblings. The true attributable risk approximate could be conferred by the steady premature CVD definition (Michael, 2014). A person whose one parent has CVD presents a double 8-year risk for males and a rise of CVD risk to 70% for females, which is significant epidemiological evidence for family history and cardiovascular diseases association (Christopher, 2014).

2.4 Behavioral risk factors

Behavioral risk factors are tangible and changeable risk factors for people in every community to prevent CVDs. Inadequate physical activity, sedentarity, poor diet, tobacco use, and alcohol abuse are the core factors of CVDs. In addition, their

association with CVDs was found in a study carried out in Russia, the USA, and in Rwanda (Zabina, 2001; Nahimana, 2018).

2.4.1 Smoking

Smoking affects all processes of atherosclerosis as a significant hazard. It can trigger the rise of inflammation, oxidative stress, and thrombosis by the endothelial dysfunction towards acute clinical events. Hence, this mechanism continues until smoking causes cardiovascular dysfunction (Ambrose, 2004).

Different tobacco products use had been the source of an astonishing morbidity and mortality burden in the United States (Catherine, 2014). Although the period from 2000 to 2011 marked a reduction in cigarette smoking, cigars and cigarillo consumption had doubled during the same period, which was seriously bad. In addition, it was found that the cigar contained in the binder or wrapper and filler, a high level of tobacco. However, the cigar without filter or not premium brand users were described as cigarillo smokers but were all taken as consuming increased levels of tobacco (Perelman, 2011). Cigarette smoking was considered to cause 30% of coronary heart diseases and the double risk of ischemic stroke with increased peripheral vascular diseases in the USA. Smoking cessation from heavy smokers or former smokers has great benefits for even people who have already suffered tobacco use-oriented illnesses. In addition, smoking cessation can reduce 50% of total mortality, abrupt cardiac death, and risk of reinfarction for already coronary heart disease diagnosed patients (Cole, 2019).

2.4.2 Physical inactivity

Physical inactivity epidemiology was born after the weight and coronary heart disease mortality rate comparison of drivers who must collect the fares after climbing stairs and others who stayed behind the wheel in London. The 2008 guideline was supported by a systematic evidence review, where the expert panel opinion showed that people who performed much more physical activities both men and women had a lower risk of CHD development. The median risk reduction was from 30% to 35% for those who are physically active. The current recommended weekly physical

activity dose is 150min/week for moderate-intensity aerobic physical activity (PA) while it is 75min/week for vigorous-intensity aerobic physical activity (Eric, 2010; Mohammad, 2018).

Another study had shown a positive impact of physical activities to reduce the triglyceride, the apolipoprotein B, and triggering the rise of the HDL. In addition, physical activity can increase the tissue plasminogen activator activity; change the low-density lipoprotein particle size. Hence, decreases the coronary artery calcium (Haitham, 2012).

2.4.3 Poor dietary habits

Diet is paramount for cardiovascular diseases evolvement and prevention. In a prospective cohort study, baseline exposure for sodium intake was assessed and then examined in relation to subsequent health outcomes (cardiovascular events). In some analyses of these studies, a part of the usual sodium intake with a defined range between 3000 to 5000 mg/day was assessed. They found that either low or high intake of salt was associated with elevated cardiovascular disease risk (Mary, 2016). A study carried out in SSA revealed that hypertension and cardiovascular diseases could be enormously controlled and managed by a dietary salt reduction in countries where the prevalence is currently rising, particularly in Sub-Saharan Africa (Noubiap, 2015).

The selection of dietary carbohydrates is crucial to reducing the risk of type 2 diabetes and cardiovascular diseases. On the one hand, the concomitant use of whole-grain, dietary fiber, and drugs could provide excellent results in diabetes management; on the other hand, the only dietary measures could protect people against prediabetic and diabetic status (Dagfinn, 2016). Hence, their selected intake reduces the risk of coronary heart disease and other CVDs (Lorene, 2015; Dagfinn, 2017).

A better way to prevent CVDs would be tied to the selection of vegetables, fruits, fibers, and whole grains, which are the excellent source of cardioprotective

components and good carbohydrates with non-starch polysaccharides (NSP) (Mann, 2007; WHO, 2020).

Relationship of lifestyle risk factors and established diseases to cardiovascular diseases

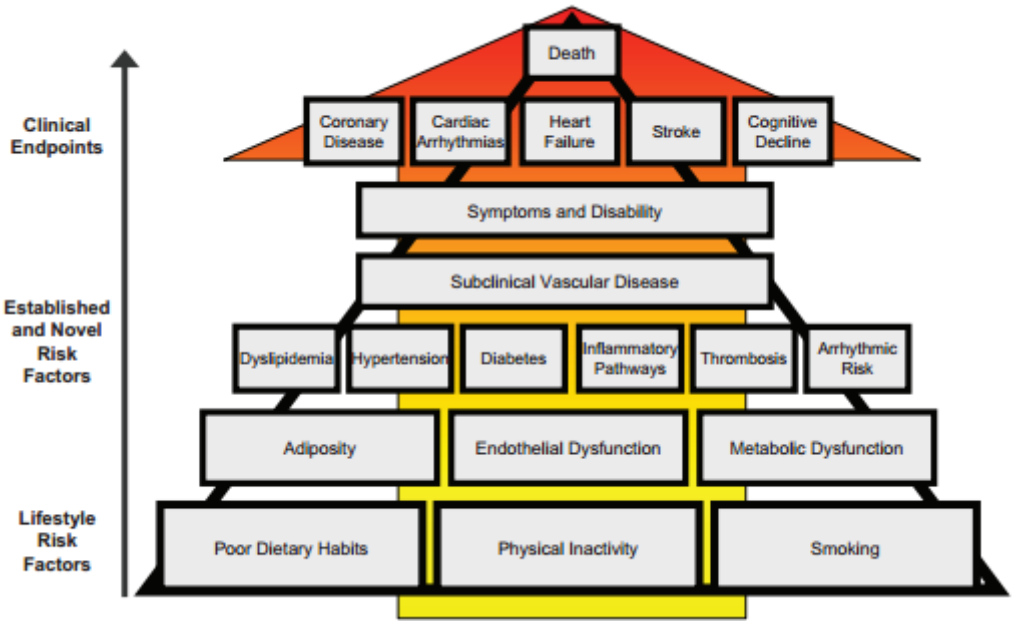

Figure 2.2: Bottom-up concentration of risk factors to cardiovascular diseases

(Mozaffarian, 2008).

Lifestyle has a markable link with the established and non-established novel risks and CVDs.

In the best way of optimistic results of scientific studies, performance measures, practice guidelines, and treatment must significantly target hypertension, diabetes, and dyslipidemia. This creates a successful healthcare and society health improvement. The lifestyle examples that influence cardiovascular diseases are smoking, inactivity, and bad dietary behaviors, which may increase adiposity due to excessive calories. Bad lifestyle behaviors may trigger the risk of illness by being the impetus of underlying inflammation/oxidative stress, thrombolytic, and arrhythmia

factors. Hence, the accumulation of lifestyle-related risk factors pushes the speed of cardiovascular diseases occurrence (Mozaffarian, 2008).

2.4.4 Alcohol consumption

Studies showed contradictory and controversial results concerning the alcohol effect has on adult morbidity and mortality. A Russian study suggests that the high levels of binge drinking in Russia translated into increased cardiovascular disease mortality (Leon, 2010). Outside heavy irregular or binge drinking, there has been a long tradition of considering the optimal health promotion with moderate alcohol intake, which was first empirically demonstrated. In addition, recently many studies associated the moderate intake of alcoholic beverages with positive effects of antioxidants and blood pressure reduction. Moreover, the polyphenol contents exerted a positive effect on the coagulation system and on the human lipid profile. It has also anti-inflammatory action by the inhibition of inducible nitric oxide synthase (iNOS). Furthermore, it causes the inhibition of the activity of cyclooxygenase 1(COX-1), which may explain the reduction in the risk of cardiovascular disease (Arranz, 2012). However, Ethanol metabolism produces free radicals and reduces the levels of glutathione. It is the major cellular protection against oxidative stress, which was related to coronary heart disease (Covas, 2004; Luc, 2009; WHO, 2016).

2.4.5 Cardiovascular diseases and soft drink

Cardiovascular diseases and obesity were linked to excessive sugar-sweetened beverages (Fung, 2009; Malik, 2010).

A study that followed 40,000 participants, men, for two decades, was performed on sugary drinks. This study showed that men who rarely consumed sugary beverages had a 20% lower risk of heart attack morbidity and mortality. However, the risk increases for men who regularly consumed an average of one can per day (DeKoning, 2012).

Another follow-up study for two decades was carried out for around 90,000 women participants. It showed that women who rarely took sugary beverages developed a 40% lower risk of heart attack morbidity and mortality. However, the risk increases

for women who regularly took superior or equal to three servings of sugary drinks (Fung, 2009). Sugary beverages' high glycemic content was attributed to increasing cardiac disease risk due to their effect on blood sugar and inflammatory factors. In addition, some cardiovascular disease risks may also be attributed to the metabolic effects of High fructose corn syrup (HFCS). Moreover, it was found to be produced at a lower cost. It has markedly become an attractive choice of sweetener in the commercial industry than sucrose. HFCS is industrially processed in two forms: HFCS-42 (42% fructose) and HFCS-55 (55% fructose), from cornstarch, which is initially converted to glucose. Hence, it then undergoes isomerization to fructose (Bray, 2004). Several studies have shown that High fructose corn syrup and sucrose have been found to increase adiposity (Bantle, 2000), insulin resistance (Elliott, 2002), (D'Angelo, 2005), uric acid, and hypertension (Farah, 2006; Brown, 2008). However, Dagfinn in his study of soft drinks, aspartame, and the risk of cancer and cardiovascular disease, did not find any association between aspartame and CVDs but with NHL (Non-Hodgkin lymphoma) (Dagfinn, 2017).

A study of 155,000 participants women showed that the caffeine consumed alone as coffee was less likely to be associated with high blood pressure than the added caffeine in soft drinks (Winkelmayer, 2005). In addition, caffeine is a constituent of many soft drinks, with concentration doses ranging from 10mg to 15mg per 100 ml (Frary, 2005). Another study conducted by Dullo in 1986 showed that Caffeine was much lower than the dosages that have been shown to acutely elevate blood pressure. However, it can potentiate the thermogenic effects of sympathetic stimulation induced by other substances. The modification of ingested sugar autonomic responses by the soft drink added caffeine was attributed to causing the acute cardiovascular dysregulation. In addition, it was found to be highly concentrated in energy drinks with 32mg per 100ml than 10mg to 15mg in regular soft drinks (Brown, 2008; Lugasi, 2015).

2.5 Workplace condition risk factors to cardiovascular diseases

2.5.1 Psycho-social risk factors

Several studies showed that the stress was a cause of 1/4 of the overall mortality increase in people of working age in Russia (Brainerd, 2004). A study carried out by Friedman in 1958 showed that workplace stress in the accountant group was linked to increased serum cholesterol and blood clotting (Cooper, 2013).

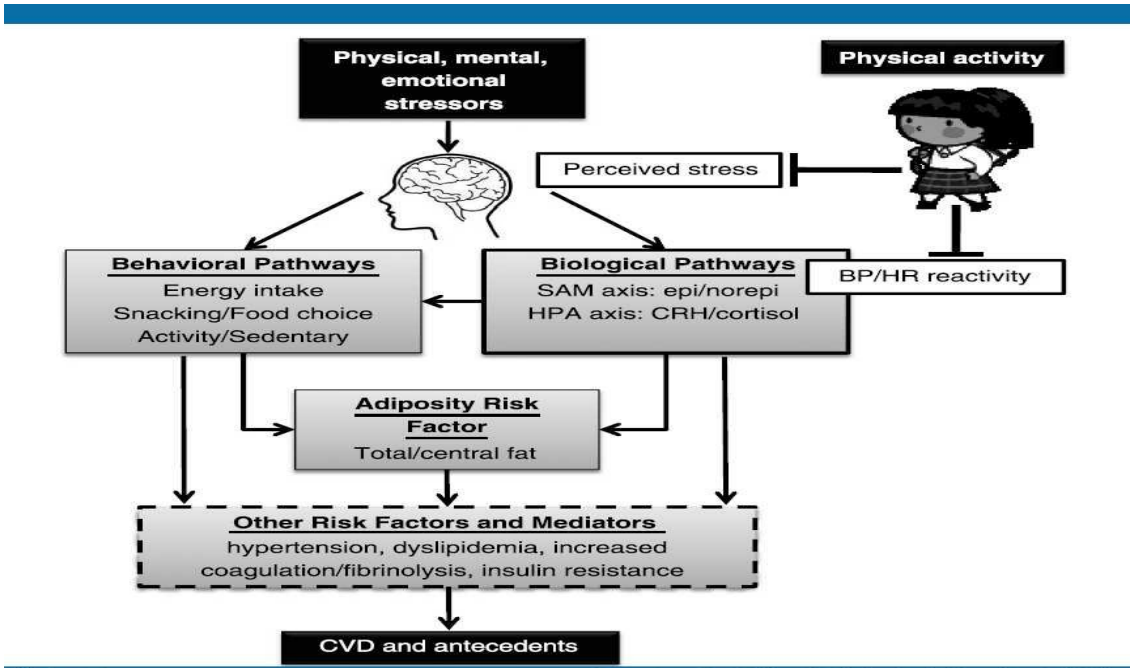

Figure 2.3: Relationship of stress and cardiovascular diseases

(James, 2014)

Stress was defined as the body's awkward response to the stressor, which could bring adverse health outcomes if stressors are exaggerated. Threatening internal and external environmental demands can create a total reaction from the body, which is termed stress reaction behavior. In addition, it is physiological and behavioral expressed where physiological reaction implied the neuroendocrine response and transformed the defense system function. Two axes of the autonomous nervous system (ANS) work together to create stress. The Sympathetic nervous system (SNS) and Parasympathetic Nervous system (PNS) are sources of short-term and long-term

stress (James, 2014). The SNS acts through the sympathetic-adrenal-medullary (SAM) axis by triggering the secretion of epinephrine and norepinephrine. Hence, the direct sympathetic action and the circulating catecholamines generate the physiological short-term responses. This creates in turn, the flight or fight response and encourage the coping mechanism by SAM (Bitsika, 2014). The PNS acts through the hypothalamic-pituitary-adrenal (HPA) axis by controlling and exerting influences on the secretagogues and all related hormones to create long-term stress responses. We can find among the secreted hormones, corticotropin-releasing factor (CRF) known as a corticotrophin-releasing hormone, cortisol, and adrenocorticotropic hormone (Sivan, 2016).

The effect of cortisol on physiology and behavior makes it a stress hormone. It generates some physiologic changes such as lipolysis, gluconeogenesis, inflammatory reaction, and finally proteolytic reaction. Cortisol affects behavior by influencing vigilance, arousal, and cognition (Cohen, 2007). Hence, the balance of this hormone can help to cope with the situation and homeostasis re-establishment (Bitsika, 2014).

The potential contributor was a researcher named Karasek who coined the "job strain model". The researcher stated that the low job decision level with the excessive physiological occupational request could create job strain. Hence, the generation of a deleterious factor, which is social isolation. However, the high demand coupled with high occupational decisions generates a climate of better learning and coping with the situation. Therefore, promote health outcome improvement (Nehal & Kanwal, 2011).

Workplace stress measurement has followed many different models such as the "effort reward-model". This model was used to assess the occupational imbalance between the effort and the reward and its impact on employees (Colby & Karen, 2014). A study named the Whitehall expressed that in around 5.3 years employees developed a double risk of coronary heart disease (CHD) for people who faced high effort with low reward. A high psychological burden was observed for work, which requires a high level of vigilance to prevent workplace disasters. The assessment

showed that sea pilots, air traffic controllers, and finally professional drivers were among those types of jobs.

Shift work was found to be associated with workplace factors to cardiovascular diseases. The definition of shift work was not conventionally explained. However, the shift pattern was organized as fixed activities at night for certain employees in a certain workplace. In addition, the night shift workers are currently rising. Wedderbun showed that around 18% of employees carried out their activities at night and about 25% of total hours are executed outside of the normal working hours. The occurrence of metabolic syndrome was 15.9% for night shift workers versus 10.3% for daily shift workers (Abu, 2018).

Studies currently showed that the night shift is a workplace factor that is associated with cardiovascular diseases. Eradicating the night shift as a cardiovascular disease factor is impossible due to the growing work requirement. The current work schedule requires working even at night. Hence, the strategic maneuver must be applied to the night shift for preventing associated cardiovascular diseases by modifying the night impact for employees on night roster. The discrepancy between circadian rhythms, behavioral changes, and social disruption for people who work at night and the normal daily workers, marks the source of cardiovascular diseases. Hence, lennermas in 1994 stated that those changes create eating patterns and metabolism disturbances and finally increase cholesterolaemia (Anne, 2004). Moreover, the early morning was associated with myocardial infarction rates and angina pectoris (Marianna, 2018).

2.5.2 Physical hazards

2.5.2.1 Temperature extremes

The workplace with extreme heat or cold factors exposure was linked with an elevated risk of acute cardiovascular events, usually where workers presented pre-existing CVDs. The heatstroke or heat exhaustion was due to the magnitude of heat stress. Hence, this exposure causes cardiac ischemia in people with existing CVDs (Anne, 2004).

The cold was found to be present at home and in the industrial workplace. Hence, it is a prominent environmental risk factor. Ice factories and breweries were found in a working cold environment. Working in a cold microclimate can lead to health problems, decreased performance, and absenteeism because of related sickness. The worst-case working in cold conditions may be linked to deaths due to accidents related to cold or because of an acute event occurring in a pre-existing condition (Florin, 2011).

Research showed that approximately 4% of the general population developed cardiovascular symptoms such as chest pain and arrhythmias when exposed to the cold (Ikäheimo, 2018). Exposure to cold causes vasoconstriction, which increases peripheral resistance and central blood volume. Hence, increase the load of heart activity. In addition, the increased pressure in the left ventricle at the end of the diastole and increased filling volumes, lead to stroke. Raynaud's phenomenon (FR) is marked by common clinical disorders, which are shown by recurrent vasospasm in fingers and toes (Fujii, 2020). They are often associated with exposure to cold temperatures or emotional stress. Aggravated vasoconstriction in response to cooling, may lead to decreased job performance. Therefore, cause a failure of thermoregulation in patients with Renal Failure (Nia, 2010; Ikäheimo, 2018).

According to Kim, men who work around one-third (about 3 h/day) of the total work time in a cold environment (-20 to -50ºC), develop asymptomatic hypertensive episodes at work compared with employees working in a hot environment (Kim, 2012). A study of 102 participants in Poland exposed to cold temperatures from -26 to 20ºC in deposits measured the physiological responses (cold pressure test, blood pressure monitoring). It was found that systolic and diastolic blood pressure during the day and at night was significantly higher in those working in cold (0-10ºC). However, it was lower for those who worked in an environment less cold (10-14ºC) with an increased response in women (Florin, 2011).

2.5.2.2 Noise

Noise is an important occupational hazard worldwide, which may lead to hypertension as a well-known risk factor for cardiovascular disease worldwide

(Hahad, 2019). It is currently the greatest cause of disability in retirement. Biomedical evidence of the association between noise exposure and the non-hearing effect was able to show a higher level of stress among individuals exposed to 55 dBA. Therefore, above 50dB(A), every 10dB(A) road traffic noise increase, creates an increase of 8% in coronary heart disease incidence due to sleep fragmentation, hormone disturbance, and oxidative stress (Hahad, 2019).

Another study showed that the association between hypertension and noise could be explained by the biochemical changes related to the mechanisms of stress (Babisch, 2003; Hahad, 2019). Therefore, the increased heart rate increased arterial blood pressure and peripheral vasoconstriction. These effects are profoundly expressed in response to the stress caused by noise due to elevated blood concentrations of cortisol, adrenaline, and noradrenaline (Ising, 2004).

A study carried out by Tatiana on noise exposure and hypertension showed the investigation of a silent relationship using the ≤75 dB (A) as the reference category. The risk increment was detected by the increase of noise measure were, 75-85 dB (A) and >=85 dB (A) showed the OR: 1.56, 95% CI (1.13-2.17) and OR 1.58, 95% CI (1.10-2.26), respectively. Other parameters such as BMI, gender, and age were independently linked with hypertension (Tatiana, 2015).

The relationship of industrial noise exposure at borderline exposure measure was significantly linked to ST-segment depression in ambulatory ECG monitoring (Anne, 2004; WHO, 2004; OSHA, 2020).

2.5.2.3 Vibration

Exposure to the vibration of the hand can cause a variety of disorders collectively named hand-arm vibration syndrome (VHA). The neurovascular component is represented by a white finger (VWF), which appears to users of vibrating tools or machinery such as chain saws, and pneumatic hammers (Florin, 2011). The vibration exposure can reach a certain part of the body, which is qualified as segmental vibration such as hand-arm vibration (HAV). This can even affect the whole-body part in case of Whole-body vibration (WBV) for truck machine operation such as

Forklift. Both hand-arm and whole-body vibration have an adverse effect on the arterial intima. Hence, this leads to a cardiovascular negative outcome (Anne, 2004; HSE, 2005; Oluseyi, 2019).

2.5.3 Chemical hazards

Exposure to the various occupational chemicals was linked to specific cardiovascular conditions with the strongest evidence at a high level of exposure (Bulka, 2019).

2.5.3.1 Carbon monoxide (CO)

Some combinations of organic materials that occurred at the workplace are the source of intoxication for employees. Employees may breathe the fumes generated by a complex mixture of gases. The sources are enormous where; carbon monoxide, polycyclic aromatic hydrocarbons, hydrogen cyanide, nitrosamines, and oxides of nitrogen constitute the hazardous materials.

Certain studies showed that exposure to a high level of Carbon monoxide could increase cardiovascular diseases risk. The low-level exposure to carbon monoxide could also generate heart ischemia in people with existing coronary heart diseases. A cigarette smoking generates an elevated level of carboxyhemoglobin around 5-15%, which is more than the level generated by workplace hazards. While the worksite hazards may generate a level between 2%-8% of exposure. An incremental level may jeopardize the health of employees with existing CVDs and Pulmonary diseases. Moreover, People with existing cardiovascular diseases and chronic pulmonary diseases could not tolerate a carboxyhemoglobin of 5% (Anne, 2004).

Carbon monoxide reduces the oxygen delivery to tissues and cardiac muscles by competitively binding on hemoglobin. Although it is rarely happening in the workplace, the carbon monoxide exposure increment could cause a 25% carboxyhemoglobin blood concentration. This may only happen by using diesel engines in confined spaces and in firefighting environments. Hence, cause myocardial infarction, arrhythmias, and even sudden death (Gonullu, 2011).

2.5.3.2 Carbon disulfide (CS2)

The absorption of carbon disulfide could pass through the skin and through inhalation to intoxicate people at the workplace. Those hazardous materials may occur during manufacturing chemicals, solvents, and viscose rayon. The exposure beyond the acceptable level, which is lower than 4 parts per million (ppm) for eight hours shifts and 12 ppm for short-term, could cause adverse health outcomes. The level of 20-60 ppm was reportedly associated with cardiovascular diseases (Anne, 2004).

Research conducted to assess the cardiotoxicity in 1992 at the workplace showed an increment from two to five-fold in mortality due to cardiovascular diseases for CS2 exposed employees in 1968. However, the exposure reduction to CS2 reduced the risk of cardiovascular diseases. Furthermore, the CS2 effect on cardiovascular diseases was reversible (Chung, 2017).

The higher toxic effect could cause cardiovascular diseases. The adverse effects caused by this toxicity increment were the increase of low-density lipoprotein, decreased fibrinolysis, hypertension occurrence, and microaneurysms. In addition, the appearance of a negative inotropic effect and direct ECG Changes. This happened after multilateral cascaded reactions to produce dithiocarbamates. The last was formed after the reaction of amine and amino acids. The reaction was observed in the pyridoxine coenzyme, zinc, and copper. Hence, all these reactions caused the inhibition of the enzymatic systems and cause cardiovascular diseases. Moreover, a safe workplace was associated with reduced exposure and eventual reduced cardiovascular diseases (Schramm, 2016). A study done for workers with carbon disulfide cumulative exposure index of 128.2 ppm and exposure of ≥10 ppm for some workers and below 10 ppm. This study has shown that the prevalence of hypertension was 69.2%, 13.9% for coronary artery disease. In addition, around 24.8% had cerebral vascular disease, and 1.3% had diabetes prevalence among the exposed workers (Chung, 2017).

2.5.3.3 Carbon dioxide (CO2)

The brewers, carbonated beverage employees including miners, and grain elevator employees were mostly exposed to carbon dioxide. According to the Canadian Centre for Occupational Health and Safety (CCOHS) in 2005, CO2 engaged an active replacement of O2 on hemoglobin. Hence, according to Nelson in 2000 and Tox review in 2005, the CO2 released by yeast caused poisoning hazards at the brewer's workplace in the process of alcohol fermentation (Permentier, 2017).

The CO2 could silently cause problems in the workplace due to its odorless, colorless state, and non-flammable gas. It is generated by the breathing of cells and fossil fuel burning. Its molecular weight is 44.01g/mol as depicted by National Institute for Occupational Safety and Health (NIOSH) in 1976). In addition, it was shown that the environmental rise in C02 pollutants, aggravated the illness and jeopardized the quality of health of existing cases of cardiopulmonary illness (Rice, 2014). Moreover, the mixture of CO2 and other gaseous substances with Particle matters (PM) can harm humans. Those Particle matters have an aerodynamic diameter of 10, 2.5, 1, or < 1 μm. They caused 3.7 million deaths in 2012 and 29% were caused by cardiac disease and stroke (Lee, 2014).

Although the atmosphere contains CO2, it is present at a very low level of 0.035% that cannot harm humans as stated by CCOHS in 2005. The CO2 permissible exposure limit (PEL) for workplace safety was established by the occupational safety and health administration (OSHA) to ensure worksite safety. The PEL was set to 5,000 ppm for the 8-hours workday, the level that is equivalent to 0.5% by volume of air. This level is exceeding 0.015% of the atmospheric CO2 content by volume of air. Similarly, the American Conference of Governmental Industrial Hygienists (AACGIH) also established the threshold limit value (TLV) of 5,000 ppm for an 8-hour workday. In addition, they established a 30.000 ppm of ceiling exposure limit (CEL). This stand for 0.3% by volume of air, for a period of 10-minute adjusted on the basis of acute inhalation as depicted by the Massachusetts Department of Public Health (MDPH) in 2005. The increased concentration of CO2 from 0.5% to 5% in the sports rooms was substantially linked to the rise in cardiac frequency and systolic blood pressure after the 3-minute exercise (Liu, 2015).

2.5.3.4 Methylene chloride

Workplace exposure to methylene chloride can increase carboxyhemoglobin. The carboxyhemoglobin could be very much increased again when the liver metabolizes the methylene chloride into part of carbon monoxide. Hence, this shows the utility of carboxymeter for exposure monitoring (Hoang, 2021).

2.5.3.5 Nitrate esters

Inhalation and skin passages are the intoxication pathway of nitrate esters for human people. Although hand chemical manipulation is currently being replaced by automation, occupational exposure is providing elevated blood concentration than the therapeutic mechanism. A study carried out by Anne in 2004 showed that 1 mg tablet of nitroglycerine or glyceryl trinitrate (GTN) through sublingual route taking produced 5.7 nmol/l. However, in the factory of gun production exposure produced 98.1 nmol/l of median measurement (Anne, 2004). Hence, in 2011 the scientific committee for occupational exposure limits set the GTN time-weighted average (TWA) to 0.01 ppm (0.095 mg/m3) for eight-hour TWA. They also set occupation exposure level to 0.02 ppm (0.19 mg /m3) for short-term, exposure limits (STEL) (15 minutes).

The GTN is normally used to treat coronary stenosis, peripheral artery resistance, peripheral ischemia, and diabetic peripheral neuropathic pain by creating vasodilatation and blood pressure reduction. However, it was also found to create methemoglobinemia and insulin resistance (Sean, 2012).

The much-known exposed industry employees to GTN and ethylene glycol dinitrate are munition, explosive, and construction industrial employees. The exposure increases as long as they handle dynamite in construction, cartridge, and explosives mixing.

After World War II, an epidemic of chest pain and sudden death occurred. Studies showed that the attacked people were employees in munition industries and the issue occurred in three days around 36 to 72 hours after exiting from munition industries. In addition, they found that the nitrate esters were able to cause withdrawal

syndrome in workers. Moreover, chronic exposure to nitrate ester triggers the renin-angiotensin system to produce an opposing effect. This can balance the vasodilatation effect caused by nitrate ester. Hence, any withdrawal leaves the body with more compensating vasoconstrictors. This leads to heart vessels spasm, coronary stenosis, chest pain, angina, myocardial infarction, and sudden death. Furthermore, the effect name was coined and named "Monday morning angina" (Anne, 2004; Münzel, 2011).

2.5.3.6 Cobalt

Cobalt had been added to beer in the form of cobaltous chloride at a dosage of about 1 ppm by several breweries in Belgium from 1959 on. This was added to prevent beer from gushing, to stabilize and improving the appearance of its foam. Some breweries ceased the addition of cobalt to beer in the middle of 1965 after a change in the Belgian law on food additives. All breweries stopped adding cobalt to beer in March 1967, after the possible toxic influence of this agent became apparent. The disease was called "alcoholic peri-myocardiopathy" It was thought to be due to a direct influence of alcohol, although another toxic origin was not excluded. It was by the correspondence of Professor Morin of the Laval University of Quebec to enlighten the darkness of the unknown and showed the toxicity of beer containing Cobalt (Parker, 2016; Gessner, 2019).

In Quebec, a total of 48 patients studied, 20 died, and in Omaha 11 of 28 patients died. Most patients died in profound shock. Multiple arterial emboli were seen. Shock is a known complication of the intravenous administration of cobalt. It was found in the dog that the intravenous administration of 3 mg of cobalt/kg lowers the blood pressure to between 50 and 70% of the initial value. This is not due to a direct action of cobalt on the heart, as its action in this concentration was shown to be positively inotropic but was found to cause cardiomyopathy (Parker, 2016).

It may be assumed that beer drinkers consumed an average of 6 mg of cobalt a day. Much larger doses of cobalt, however, have been given in the treatment of anemia without cardiotoxicity being reported as a result of the therapy. Doses of between 20 and 35 mg of cobalt a day were given for several months, and no heart disease was

described. Although hypothyroidism and thyroid hyperplasia have developed. Cobalt has been given in a dosage of 75 mg a day for 6 weeks in cases of sickle cell anemia, nerve deafness, and tinnitus, but no cardiac toxicity was reported. Cobalt has also been given in a dosage of about 20 mg a day for three months to 78 pregnant women. The anemia of pregnancy was prevented in this group, and no toxic manifestations were observed.

Studies have not found any association between heart toxicity with cobalt miners or cobalt purification professionals. Cobalt was even used for therapy in cases of hypertension at a dosage of 6 to 8 mg a day, and no toxic effects have been noted. Around 22 Doses of cobalt of 2.5 mg/kg/day have been given to experimental animals for a period of 6 months. At autopsy, nothing special was found. In a dosage of 5 mg of cobalt/kg/day, no animal died after 5 months. At autopsy, some myocardial edema without degeneration was observed. In a dosage of cobalt of 25 mg/kg/day the animals died after 1 month and massive pericardial effusion was noted. The myocardium was edematous; degeneration and swelling of the myocardial fibers were present with clarification and vacuolization of the cytoplasm. Thus, very high concentrations of cobalt seem to be necessary to produce toxicity. This may occur in both humans and experimental animals, and even in the cobalt production worksite (Linna, 2020; Zhang, 2020). Some study results showed that cardiomyopathy was brought about by the combination of cobalt, excessive alcohol consumption, and malnutrition (Anne, 2004). However, a recent study proved contrary to the cobalt effect on cardiomyopathy (Lantin, 2013).

2.5.3.7 Arsenic/Arsine

Contaminated beer caused subacute arsenic poisoning has been linked to cardiomyopathy and cardiac failure. With the unclear mechanism, 70 deaths have been recorded from an epidemic that touched 6000 people in Manchester (National Research Council, 1977; Moon, 2012).

The recurrent ventricular fibrillation and abnormal ECG were observed with acute arsenic intoxication. Heavy intoxication with arsine gas can generate cascade adverse health outcomes, which start from hemolysis of red blood cells, heart failure, and

eventually hypercalcemia. The arsenic occupational intoxication was observed in arsenic ore processing in metallurgical industries and in agricultural vineyards exposure by spraying the arsenical insecticides (Anne, 2004; Assadi, 2017). A study carried out in Italy to detect the three heavy metals in beer contents (arsenic, lead, and cadmium) showed that nine out of nineteen beer samples were below the limits of detection. They found that the quantifiable residue level of lead was noted in 52.6% of samples, three for cadmium and none for arsenic. Furthermore, the Italian regulation for drinking water found that arsenic was beyond the legal limit for nine samples. Additionally, the Tukey post-hoc test indicated that arsenic was found at a significantly higher level than lead and cadmium. A significant correlation at the 0.034 level (Pearson's R =0.489) was found between cadmium and lead. Also, a highly statistically significant correlation (Pearson's R =0.620) at the 0.01 level was found between % Alcohol by Volume (ABV) (Donadini, 2008) and total arsenic content, (Bengt, 2015).

2.5.3.8 Lead

Studies showed that people exposed to lead developed hypertension. The lead intoxication can pass through the lungs by inhalation of environmental lead, exhaust fumes, and water intoxicated by leaded petrol and lead pipes. One millimeter and 0.6 mm increase in systolic and diastolic blood pressure were associated with a 2-fold blood concentration of lead. According to steenland in 2000, the physio-pathologic phenomenon was that Lead creates an effect on vascular smooth muscle and enhances sympathetic stimulation (Navas-Acien, 2007). Hence, it interferes with calcium metabolism (Bengt, 2015).

2.5.3.9 Solvents and arrhythmias

Exposure to solvents chemicals has been linked with heart rhythm troubles (Bradycardia, atrial ventricular block). Arrhythmias were observed for some chemicals among others methylene chloride, methyl chloroform, bromofluorocarbons, and trichloroethylene. However, Glue sniffing was found to be

intentional exposure. The concerned exposure employees were found in chemical manufacturing industries, dry cleaning, painting, and finally degreasing chemicals.

It was noted in the literature that abuse and workplace solvent chemical exposure caused several cases of sudden death. In addition, solvent exposures increase the sensitivity of the heart to catecholamines, where a lower dose of adrenaline can generate ventricular tachycardia and fibrillation. Moreover, exposure to halogenated hydrocarbons could exert a negative effect on the heart (Anne, 2004; Assadi, 2017).

A study carried out to examine the effect of hazardous chemicals with epinephrine and norepinephrine showed that toluene, styrene, and xylene exposure induced cytochrome P-450 isoenzymes disturbances (Bulka, 2019). Hence, the metabolism of cholesterol disturbance inevitably follows for the exposed group (Ki-Woong, 2012).

2.5.3.10 Cardiovascular disease and workplace

In Brazil, a study done in a carbonated beverage industry has shown that 83% was with a sedentary lifestyle, and 63% with obesity. Around 28% presented systematic hypertension while 45% were with pre-hypertension. In addition, 49% of the study participants were with impaired blood glucose, and 7% and 11% presented hypercholesterolemia in the study participants, respectively (Cassani, 2009).

The study done on the 123 workers of são Paulo state distillery (Liquor factory), showed that 26% presented Cerebral vascular accidents, 27.6% presented Diabetes mellitus, and 65.9% reported alcohol intake. In addition, 11% had systolic hypertension and 12.2% with diastolic hypertension, and finally, levels one and two of obesity were 27.6% (Simao, 2002).

A study carried out in the united brewery of America, union in New York revealed 25 deaths over two years' experience due to heavy drinkers' workers. This was estimated without considering other industrial factors in their working environment. However, the mortality rate was rising in breweries and distilleries according to the organization of life insurance and the official statistic for occupational mortality (Kiran, 2022).

Recently industrial workers in the US showed that people of advanced age were suffering from CHD and stroke. In addition, the risk increased in the people out of the labor force by 6.3% while unemployed people searching for jobs presented 2.5%. Moreover, employed adults' people beyond 55 years presented a lower history for CHD and stroke at a level of 1.9. This study was the baseline of CHD and stroke prevention programs in adult people (Luckhaupt & Geoffrey, 2014).

The mega industrial corridor Nepal study revealed that the prevalence of cardiovascular diseases was 13.8% in 494 industrial employees. The aged people were prone to have cardiovascular diseases, where the Odd of developing the cardiovascular disease for people with more than 45 years was 2.72. In addition, it was 1.9, 2.47 and 4.32 times more likely to develop CVD for people with hypertension in family history, lack of fruit consumption, and tobacco use, respectively. Moreover, the higher the LDL $\geq$ 130mg/dl, the higher the risk of developing cardiovascular disease (Schnall, 2000). Furthermore, the risk of suffering CVDs increased to OR= 3.03 for such people (Pyakurel, 2016).

A case-control study concerning the alcohol intake impact on coronary heart disease was conducted on Indian employees and their family members. This study has declined the positive impact of alcohol to prevent coronary heart disease (CHD). This happened after finding out that many cardiovascular diseases risk factors were elevated in the drinkers than in the abstainers.

Therefore, coronary heart diseases were less likely to attack non-alcohol users at a level of 2.4% while it was 3.3% for alcohol users. The fasting blood glucose and hypertension levels were higher for alcohol users (98.7±30.5 mg/dl), (128.7±17.6 mmHg/80.1±11.3mmHg). However, the FBG and blood pressure levels were lower for the non-alcohol users (96.6±26.0 mg/dl, $p < 0.01$), (126.9±15.9 mmHg/79.5±10.3mmHg, $p < 0.01$), respectively (Roy, 2010).

Another study done in India in the industrial workplace for the NCDs profile risk factors has revealed the overall risk factors profile of the study subjects. The study showed a universal prevalence of inferior 500gms daily intakes of vegetables and

fruits, followed by 65.7% and 65.5% prevalence of High blood pressure and high BMI, respectively.

The proportion of 72.7% of the high waist to hip ratio and 32.3% of high abdominal circumference were observed in the increment of abdominal obesity. The prevalence of alcohol intake, smoking, and inactivity was 5%, 31.4%, and 17.3, respectively.

The three factors such as diabetes 19.1%, hypertension with 38.2%, and cholesterolemia with 40.5% were found in 34.1% of the study participant with high risk (Mehan, 2006).

A South African study revealed that the intake of sugar-sweetened beverages (SSBs), has been linked with increased high BMI and risk of stroke. They forecasted a reduction of around 550,000 stroke-related-adjusted life years and 72,000 stroke deaths. Hence, a reduction of healthcare costs of around $400 million if only they impose an SSB tax over 20 years. Moreover, the prevalent and incident cases may be reduced to approximately 13,000 and 85,000, respectively (Manyema, 2012).

Another study concluded that, although artificially sweetened beverages and fruit juice also showed positive associations with the incidence of type 2 diabetes and adiposity, the bias was more likely to be found in the findings (Imamura, 2015).

A study conducted in the UK for three cardiovascular factors (high blood pressure, alcohol intake, and metabolic syndrome) in comparison to non-drinkers showed a reduction in CHD risk over 10 years with the alcohol consumption increase. The risk was greatly reduced for women with consumption between 1-7 units/week, OR=0.9, (95% CI: 0.72–0.87). However, the alcohol consumption between 15-21 units/week, increased the prevalence of hypertension with OR = 1.68, (95% CI: 1.14–2.46) (Nanchahal, 2000).

A study carried out in Korea to assess the impact of a Worksite Multiple cardiovascular Disease Risks Reduction Program (WMCVDRRP) revealed a positive effect. It has presented 40% behavior improvement in the study subjects after six months of implementation. The improved factors are diet and stress management, physical activity, and finally sticking to medication. Although the improvement in

drinking behaviors did not show statistical significance, 21% of the participants changed in alcohol consumption and 21% quit smoking. Eight physical indicators including systolic and diastolic blood pressure, total cholesterol, triglyceride, body mass index, waist-hip ratio, body fat, and muscle weight improved significantly (Huang, 2013).

The workplace self-reported study that was carried out in United Kingdom for occupational diseases from 2011 to 2012 showed that 2.3 million were work-related illnesses (WRI). In addition, 33 million days were lost due to the WRI. Hence, they estimated around 80,000 CVD prevalence due to WRI or made worse by the work during the period of the study year. The average number lost days in a year due to sickness was 23 days for each reported person with WRI. In addition, all lost days were equal to 1.84 million days due to work-related cardiovascular disease. Moreover, the associated cost was around £120 million (Jones, 2002).

A study carried out in Rwanda, on the cardiovascular disease changeable risk factors in university employees, has revealed that 36 participants were hypertensive, with a significant age correlation. Cardiovascular diseases were associated with factors such as smoking, sedentary, lack of physical activities, diabetes mellitus, high BMI, and alcohol intake. About 28% of study participants were physically inactive and 41% of the participants were overweight (Phillips & Banyangiriki, 2015).

2.6 Biological risk factors to cardiovascular diseases risk

2.6.1 Metabolic syndrome

Metabolic syndrome (MetS) is a group of clinical, physiological, biochemical, and metabolic disorders that create a state of a complex of interrelated factors. It is subsequent to low-level inflammation, which may increase type 2 diabetes, cardiovascular disease risk, and death (Grundy, 2005). The MetS are formed by various factors among others elevated blood pressure, impaired fasting glucose, central obesity, atherogenic dyslipidemia, and endothelial dysfunction (Jaspinder, 2014). In addition, genetic vulnerability, high coagulability, and prolonged stress are the core factors of the MetS (Kazim, 2014).

Having metabolic syndrome is very critical to the extent of increasing three to fourfold myocardial infarction development, two to four-fold of stroke increased risk, and finally two-fold the risk of death occurrence than people without metabolic syndrome (Alberti, 2005). These morbidity and mortality risks happen to people without any cardiovascular event history (Olijhoek, 2004).

The qualification of atherothrombotic complications could be based on the development of the metabolic syndrome as a first-line risk factor. Presenting or not presenting the MetS could be a gauge of long-term risk. However, some other benchmarks of the short-term risk such as REGICOR (Registre Gironí del Cor) and Framingham could be used to ascertain the risk between five and ten years (Grundy, 2006).

2.6.2 Hypertension

High Blood pressure was evaluated as one of the significant causes of the global disease burden, which caused 9.5 million deaths each year and 16.5% of all deaths. Hypertension was considered the twin risk factor to cardiovascular diseases and is therefore presenting a global rising prevalence and occurrence (Lim, 2012).

High blood pressures were approximated to be 30% of the adults' population in the European WHO region while it was 23% in the Americas WHO region as stated by the World health organization in 2008. However, the 2019 study showed that the rate would rise from 30 to 45% in the general population (Giuseppe, 2013).

Hypertension control and management showed a remarkable reduction of 40% in mortality due to cerebrovascular diseases and a reduction of 20% in coronary heart diseases. This makes it a major risk factor and an important target to prevent and fight cardiovascular disease burden in the global population (Kaplan, 2002; Flávio, 2019). Hence, continuous practical behavior studies and treatment are necessary (Kaplan, 2010).

A joint effort of three entities, which are the Colombia Earth Institute and Harvard School of public, and finally the millennium village's project on the prevalence of hypertension. The study showed that in three villages selected in various countries,

hypertension prevalence was 22.8% in one village in Rwanda, 15.9% in one village in Malawi, and 26.8% in also one village in Tanzania. These prevalence's were not negligible and as taken in small and sporadic villages, the real burden could be high (Stewart, 2010).

2.6.3 Diabetes

Diabetes development is critically linked to the development of cardiovascular development. Hence, it is an independent risk factor for cardiovascular diseases. Diabetes damage peripheral and extremity small vessels, which is the source of diseases such as nephropathy, retinopathy, and eventually peripheral vascular diseases. Moreover, the degenerative destruction of vessels in the brain and in the heart causes stroke and coronary artery diseases (CAD). Therefore, the heart muscle develops infarction, reduction of ejection fraction. Then, diastolic, and systolic disorders start, which lead to heart failure (Dokken, 2008).

The impairment of fasting glucose (IFG) is one of the major causes of metabolic syndrome. Diabetic patients without effective control of metabolic gauge develop macrovascular and microvascular degeneration. This causes irreversible diabetic complications. Hence, these complications lead to worldwide Morbi-mortality and economic burden. In 2004, the deaths due to diabetes in the population older than 65 years in the USA were associated with 16% of stroke and 68% of other cardiovascular diseases (Alessandra, 2013). The American heart association showed that in around 65% of deaths due to cardiovascular diseases, diabetes was a major and an independent risk factor. Stamler in his study in 1993, showed that patients with diabetes presented increased mortality due to stroke by three-fold than no diabetic patients (Martín-Timón, 2014). In addition, diabetes causes the occlusion of small arteries. Some prospective studies showed that patients with diabetes have an increased risk and development of CVD events when other independent risk factors (smoking, hypertension, high serum cholesterol) accumulate. However, the diabetic health improvement by only drugs administration cannot change the cardiovascular risk increment if other factors are not controlled (Martín-Timón, 2014).

2.6.4 Dyslipidemia

The lipid metabolism turmoil creates a disorder of lipid deposits in cardiovascular pathways, which is the source of heart diseases and other cardiovascular diseases. A study done by Carlson, 1979 showed that a 1% increase in total cholesterol was expected to cause a 2-3% incidence of coronary heart disease (Mee, et al., 2017). Another study done by Law, 1994 showed a strong relationship with an increase of 38% of the risk of coronary mortality after an increase of 10% in total cholesterol. Therefore, various studies described that the increase of triglyceride-rich lipoproteins (Quilomicra and VLDL) has a direct and significant correlation with coronary heart disease incidence (Mee et al., 2017). In addition, the study showed that the proportion of 37% of women and 13% of men suffered cardiovascular diseases due to high triglycerides (Telmo, 2012).

People with HDL-cholesterol, have also a low risk of coronary heart disease. Hence, every increase of 1 mg/dl of HDL-Cholesterol was linked to the reduction of 2 to 3% of coronary heart diseases as shown by Gordon in 1989 (Rajagopal, 2012). High-density lipoprotein is a part of the reverse transport of cholesterol and can carry out the work of inflammation prevention and eventually ensure the blocking action against the oxidation caused by the LDL-Cholesterol (Ansell, 2004). On the other hand, people with a decreased level of HDL are having concomitant hypertriglyceridemia, obesity, a sedentary lifestyle, and active tobacco intoxication (Cui, 2001). In addition, decreased glucose tolerance and an increased occurrence of cardiovascular events were noted for levels of HDL-cholesterol below 40 mg/dl (1.0 mmol/L) in men. This phenomenon was also noted for women with less than 46 mg/dl (1.2mmol/L) of HDL-c (UK HDL-Consensus Group., 2004). Moreover, HDL-Cholesterol is of great importance to be determined as a parameter of cardiovascular health. HDL-c can better translate the risk of cardiovascular mortality than LDL cholesterol, and express more accurately the lipoprotein atherogenicity (Telmo, 2012).

2.6.4.1 Lipid’s metabolism

Lipids constitute an essential source of energy storage, represented by triglycerides, and were made by a very heterogeneous group of compounds. The lipid influence on metabolism goes far beyond the misdeeds attributed to it. They are transported by apoproteins, which constitute the protein fraction of lipoproteins. In addition, lipids form a part of the brain (17% of its dry weight), hormones, and lipoproteins. Ultimately, lipids play a substantial role in the creation of bile acids, vitamins, and the structure of cell membranes.

Relying on the composition, type, size, function, and density, lipoproteins are oftentimes classified into six groups. Those groups are: Quilomicra, VLDL (very low-density lipoproteins), IDL (intermediate density lipoprotein), LDL (low-density lipoprotein), HDL (high-density lipoprotein), and Lipoprotein (a).

The large lipoprotein particles (ApoB48, ApoAI, and ApoAIV) and chylomicron aid the transportation of the diet fat rich in triglyceride from the intestine into the bloodstream (Hussain, 2014).

By the way, other elevated quantities of lipoproteins such as Very low-density lipoprotein (VLDL) and triglycerides were ultimately produced by the liver alone. Hence, VLDL and triglycerides are finally broken down by the extracellular enzyme (Lipoprotein-lipase) and the degraded free fatty acids are deposited in tissues. The low-density lipoprotein binds to peripheral or liver receptors after being produced from Intermediate-density lipoprotein. This was also formed after hydrolysis of lipoproteins by hepatic-lipase. Thus, in another cycle of reverse cholesterol transport, HDL particles pick up cholesterol deposited in the arterial wall. Therefore, they ensure its transportation to the liver, where it is subsequently excreted in the bile (Eckardstein, 2005). The equilibrium of a complex metabolism formed by the interaction of lipoproteins with an elevated number of enzymes, transport proteins, and receptors, is determined by intrinsic and extrinsic factors. Hence, the prominent clinical consequences were manifested due to the pathological process of atherosclerosis caused by the system disequilibrium (Silva, 2003; Rafieian-Kopaei, 2014).

2.6.4.2 Atherogenesis and cardiovascular diseases risk-atherosclerotic plaque

The course of events takes place in the lumen of the arterial wall until the clinical devastating manifestation to the person. The arterial wall is the inner portion of our vessels, which can be compared to a thin membrane that carpets the blood vessels. Thus called the vascular endothelium. The maintenance of several potentially unstable equilibrium requires the fundamental integrity of the vascular endothelium. However, endlessly blood and other circulating factors are closely alternating defense and aggression with Nitric Oxide as the key protector. Therefore, many interactions are produced in the vicinity of endothelium produced such as vasodilation/ vasoconstriction, anti-thrombotic/pro-thrombotic, anti-inflammatory/ pro-inflammatory. In addition, around ten lipidic factors were involved to explain this phenomenon. Those factors are lipoprotein a, lipoprotein remnants, HDL subspecies, small and dense LDL, and apolipoprotein A-1. Besides, the aforementioned factors we can include, the apolipoprotein B, C reactive protein, homocysteine, interleukin-6, cell adhesion molecule-1, and finally the selection-CD40. Moreover, metabolic postprandial hyperinsulinemia, and coagulation factors such as fibrinogen, Von Willebrand factor, factor VII, and plasminogen activator inhibitor (PAI-1). Hormonal: Loss of estrogen production (menopause); and neurotransmitter, enzymes, and chemicals released in the blood after the psychological/behavioral events. Those events are: alcoholism, depression, social isolation, loss, and social support, low socioeconomic status as stated by the International lipidic information bureau (ILIB) in 2003 (Linton, 2019). The relative dominance of each of these interacting factors could determine the final maintenance of endothelial integrity or, conversely, its dysfunction, and destruction (Houston, 2002; David et al., 2004; Chen, 2015; Houston, 2018).

The initial phase starts with the endothelial dysfunction of atherosclerosis, once the endothelial barrier is compromised; the process of lipid flooding starts in the vascular wall. Hence, it mobilizes the inflammatory cells and influences the chemotactic factors and multiplication of smooth muscle and connective tissue.

The vascular changes and calcification due to the lipid streak-atherosclerosis process marked the histologic consequence, which generates the vascular stenosis (Silva, 2003; Rafieian-Kopaei, 2014).

The atherosclerotic plaque stability is thus due to the type of plaque. Vasoconstrictor triggers, hypertension, and sympathetic activity could not sometimes disrupt the plaque with a thin lipid core and small inflammation. This plaque is less vulnerable due to the though and thick outer layer. In contrast, plaques with a rich lipid core, inflammatory activity (Ridolfi & Hutchins, 1977), and a significantly weak fibrous cap will present a higher risk of fracture and exposure to their internal contents (Telmo, 2012).

2.6.5 Overweight, obesity, and central obesity

The definition of obesity was universally expressed as a surplus of body fat in relation to height. It is actually calculated by the weight in kilograms over height in meters squared. The usual and general benchmark is called Body mass index (BMI).

Many prospective studies have reported a J-shaped curve between BMI and mortality/morbidity (Despres, 2012). Overweight and obesity were found to be associated with cardiovascular diseases versus underweight (AOR=1.80) (Rahman, 2015). Obesity especially produces visceral fat deposition. This is associated with low-grade inflammation, which plays a role in the pathogenesis of diabetes. Moreover, both diseases are associated with a significant increase in morbidity and mortality due to CVD (Alessandra, 2013).

Obesity, with the common benchmark, is defined as having a BMI>=30kg/m2. Hence, it creates several long-term detrimental health consequences of surplus weight. This condition leads to premature atherosclerosis, increased risk of myocardial infarction, and heart failure. Moreover, it reduces the survival time due to early cardiovascular death for people with morbid obesity (Berrington, 2010). Through a complex phenomenon, obesity interconnects with different factors, which lead to cardiovascular diseases. We can find among those factors; atherogenic risk factors, insulin resistance, metabolic disorders, dyslipidemia, and hypertension. Moreover, it causes detrimental heart muscle change, heart hypertrophy, the reduction of ejection fraction with a systolic and diastolic impediment, and vascular endothelial disorders.

Furthermore, this complex phenomenon generates an adverse effect on the rise of sympathetic tone, pulmonary venous, and artery hypertension, and early coronary artery diseases. Finally, the damages extend to the right-sided heart and the creation of arrhythmias (Poirier, 2011).

The hepatic adipose particles produce constant circulating pro-inflammatory and inflammatory factors such as cytokines, which leads to various consequences. Therefore, cardiovascular diseases are manifested after the litany of issues such as insulin resistance, the activation of plaque, and enlargement of the heart muscle.

In fact, it is the pathogenic-adipose-cardiovascular complex that generates systematic arterial disorders due to observable body traits adversely changing (Apovian, 2008).

BMI represents a crucial element for the overall risk forecast. However, there are numerous factors to consider for clinically estimating the occurrence. The body fat percentage, fat distribution, and the quality of adipose tissue are also other factors used in combination with BMI to estimate the overall risk (Apovian, 2012).

Table 2.1: Gender and age-specific waist circumference cut-offs

Country/Ethnic group	**Waist circumference cut-off**	
	Male(cm)	**Female(cm)**
Europids	≥94	≥80
In USA the ATPIII values (102cm male and 88 cm for female) are likely to continue to be used for clinical purposes		
South Asian	≥90	≥80
Based on Chinese, Malay and Asian Indian population		
Chinese	≥90	≥80
Japanese	≥90	≥80
Ethnic south and central Americans	Use south Asian's recommendations until more specific data are available	
Sub-Saharan Africans	Use European data until more specific data are available	

Eastern Mediterranean and Middle East (Arabs) population	Use European data until more specific data are available

(Jaspinder, 2014)

2.6.5.1 Body mass index (BMI) and cardiovascular diseases

Body mass index (BMI) is the gauge of low, normal, and surplus weight which uses the weight in kg divided by the squared height in meters (KG/m2) (Berrington, 2010).

The measure beyond the normal or overweight range (BMI ≥25 kg/m2 and ≥30 kg/m2) of BMI has been linked with an increase in hypertension, diabetes, and cardiovascular diseases. Hence, the combination of such diseases increases mortality (Robert, 2014).

BMI is frequently used as a surrogate measure of fatness in children and adults (Poirier et al., 2006). Although studies stated that it measures surplus weight rather than surplus fat, its results correspond to body fat gauges like dual-energy x-ray absorptiometry and underwater weighing.

Table 2.2: Body mass index relation to waist circumference as reference to central adiposity

Disease Risk Relative to Normal weight and Waist Circumference			
	Body mass index Kg/m^2	**Men, ≤ 102cm; Women, ≤88cm**	**Men, >102; Women,>88cm**
Underweight	<18.5		
Normal	18.5-24.9		
Overweight	25.0-29.9	Increased	High
Obesity, class			
I	30.0-24.9	High	Very high
II	35.0-39.9	Very high	Very high
III			
Extreme obesity	≥40	Extremely high	Extremely high
Disease risk for type 2 diabetes, Hypertension, and Cardiovascular diseases			

(Poirier, 2006)

2.6.5.2 Free fat in the body

Some studies showed that regional fat distribution was not the key element to assess its linkage with adverse health outcomes and complications. The pattern of fat accumulation like gynoid or android was also involved. The waist-to-hip ratio (WHR) or the ratio of waist to hip circumference is the crucial benchmark. This was more associated with metabolic consequences and cardiovascular adverse outcomes than the BMI. Moreover, the recent evidence declared that the elevated level of WHR served as a good predictor of elevated hypertension, diabetes, dyslipidemia, and cardiovascular disease risk (Despres, 2012).

Fat maldistribution process

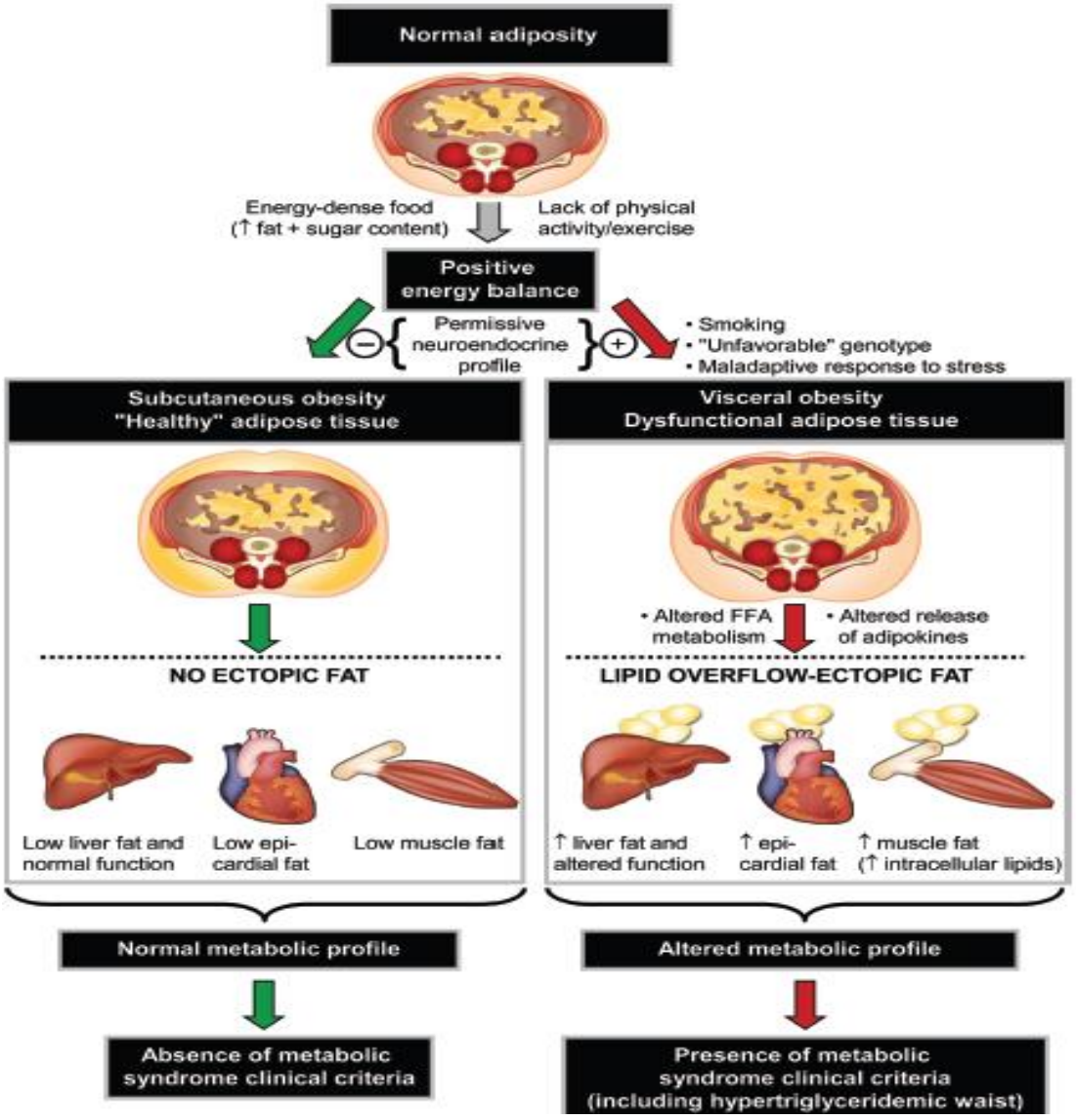

Figure 2.4: The lipid overflow-ectopic fat model

(Despres, 2012).

2.6.5.3 Excess visceral adiposity

Adipose tissue dysfunction is marked by fat maldistribution with central obesity. The storage and inappropriate concentration of fatty acid due to excess energy production impede and harm the liver metabolism. Therefore, it implicates systemic inflammation, glucose intolerance, insulin resistance, and high triglyceridemia. Besides those factors, the body generates apolipoprotein B, atherogenic

dyslipidemia, and a reduction of high-density lipoprotein-Cholesterol follow (Björntorp, 1990; Longo, 2019).

The capacity of the body to cope and adapt itself to sedentary and the consumed energy (plenty of calories) balances the metabolic syndrome occurrence. In addition, the maladaptive response to stress and the way the body fights smoking and genetic effect. Therefore, the excess triglycerides could be deposited or stored in unneeded body places like the heart, the liver, skeletal muscles, and the abdomen. Thus, called the ectopic fat deposition phenomenon (Bergman, 2006; Palikaras, 2017).

The fat surplus increases saturated subcutaneous adiposity, which generates excessive intrahepatic and pericardial fat. This serves as a marker of heart incapacity to manage the excessive fat. Therefore, the increment of using free fatty acids as a surrogate and the excretion of cytokine/adipokine caused by excessive fat liver-heart mechanism; creates many adverse health outcomes. Furthermore, this excessive fat liver mechanism generates vasodilatation deterioration, diastolic impairment, and eventually heart failure (Iozzo, 2011).

A larger waistline for a given BMI would predict a greater accumulation of visceral adipose tissue. It can be taken as an index of total adiposity, which is influenced by abdominal adiposity. Waist circumference was used to distinguish visceral from subcutaneous adiposity when coupled with fasting hypertriglyceridemia (Despres, 2012).

Fat Percentage

The percentage of Body Fat (PBF) is the benchmark for measuring body fat and it is calculated by the body fat mass of an individual divided by the total mass times 100. The percentage of body fat breaks down more the body parts' fat composition than BMI. However, both gauges increment indicated an increase in cardiovascular disease risk. Different formulae use different parameters to calculate the PBF for men, women, young males, and females (Qiang, 2012).

2.7 Novel risk factors to cardiovascular diseases

2.7.1 Fasting blood glucose

A study carried out in Korea showed that ischemic heart disease (IHD) and ischemic stroke were linked to grade two impaired fasting glucose (IFG). The IFG was undependably linked to cardiovascular diseases for men and was defined as blood glucose concentration >=100mg/dl and <126mg/dl. In addition, type 2 diabetes was potentially associated with CVDs and IHD (Hong-Kyu, 2013). Besides, cardiovascular diseases, coronary heart disease, and stroke, they found intermittent claudication to be associated with hyperglycemia (James, 2002). For the understanding of the different levels of plasma glucose concentration in contrast of IFG, the Impaired glucose tolerance (IGT) was explained as an elevated glucose plasma concentration after 2 hours of taking 75g of oral glucose that is comprised between >=140 mg/dl and <200mg/dl (Genuth, 2003). The oral glucose tolerance test (OGTT) could be processed after finding out a fasting plasma glucose (FPG) concentration superior to 126mg/dl (David, 2007; Sakaguchi, 2015).

2.7.2 Serum Uric Acid

Since the 19th century, the linkage between cardiovascular disease and hyperuricemia was observed (Luis & César, 2012). The elevated level of serum uric acid was positively associated with cardiovascular diseases. This novel risk was also linked with other CVD risk factors such as triglycerides, hypertension, and glycosylated hemoglobin. In addition, BMI, waist circumference, FPG, and finally two-hour postprandial plasma glucose were all significantly associated with hyperuricemia with $p<0.05$ (Li, 2014).

High uric acid was potentially used as a tangible predictor of acute and chronic heart failure morbidity and mortality (Luis & César, 2012). Other studies confirmed the increased uric acid and hypertension development (Daniel, 2008).

2.7.3 C reactive protein

Inflammation is the core factor in the initiation and progression of atherothrombosis, which leads to cardiovascular disease. Clinical studies have linked chronic inflammation to future cardiovascular events. In addition, the emerging biomarker of inflammation reveals the overshadowed identification of asymptomatic patients (Omair, 2013).

Studies revealed C reactive protein (CRP) to be a predictor of recurrent ischemia and death for patients with stable and unstable angina. In addition, it is used for patients that are undergoing percutaneous angioplasty and patients who are suffering acute coronary syndromes in the emergency department (Ridker, 2003). The hs-CRP is a very sensitive gauge of inflammation, which is measuring the trace amounts of CRP in the blood between 0.5 to 10 mg/L. It is therefore accurate, and reliable with high precision, whereas the CRP is measured within the range of 10 to 1000 mg/dl (Pearson, 2003).

The hs-CRP is classified into three categories where the first range is inferior 1mg/L is low risk, the range between 1 to 3 mg/L is classified as average risk, and the range >3mg/L is high risk (Hong, 2008). Finally, the hs-CRP which is >10mg/dl was associated with plaque rupture, which leads to thrombosis (Michelle, 2015).

2.8 Cardiovascular diseases prevention

The need for effective prevention strategies is necessary for the working area where adult people spend most of their time. The extension of preventive strategies in their family should be taken into account to maintain and improve the acquired good results. The American heart association had set the strategic goal to ensure a 20% improvement in American cardiovascular health by ensuring the reduction of 20% of cardiovascular diseases and stroke mortality by 2020. However, this ambitious goal requires great collaboration efforts. The multilateral effort from experts to lower-level health professionals such as cardiologists, clinicians, nurses, pharmacists, and nutritionists. In addition, the family medicine, pediatrics, and Exercise science involvement is of great necessity in all stages of prevention throughout people's

lifespan. Although some studies targeted high-risk old people for behavioral risk modification, the risk factors, majority stroke, and CVD events were also found in people with averaged years and with mildly elevated factors. Hence, preventive strategies should be extended to people of all ages and of both workplace and community populations.

2.8.1 Pre-employment and redeployment after CVD event experience

Heart disease has a negative effect on work. Its diagnosis may also have an impact on different ways capacity and manners of carrying out the assigned duties. Therefore, it is important to get evidence of this impact in a broad spectrum of the workplace.

Besides, the Hippocratic procedure in regard to patient consultation, the emphasis on the history taking that requires questioning patients in regard to five hazardous dimensions. In the 17^{th} century, Ramazzini proposed the involvement of the following five-dimension inpatient treatment, which are physical, biological, chemical, ergonomic, and psychosocial hazards. These factors must be documented to ensure the complete evaluation and proposition of strategic preventive and curative treatment (Carnevale, 2014). The pre-employment is governed by the occupational service plan for employee health, working conditions, and safety history. Then, considering the workplace exposure effect on the health of employees and improvement of health practice. The employees with congenital health conditions should be known to help their adaptability and orientation to safe carrier vis a vis their health conditions (Anne, 2004; Reibis, 2019).

2.8.2 Redeployment at work after the heart disease development

Studies showed that returning to work after the start of the cardiac disease requires the consideration of different factors such as occupational requirement profile, their heart function, psychosocial factors, and physical fitness (Reibis, 2019).

2.8.2.1 Nature of the person

It is more crucial to evaluate the psychosocial factors than only relying on the clinical factors for predicting the probability of employees returning to work after a cardiac event. In addition, it is necessary to consider the period of being sick, place of occurrence of cardiac event, type of work, personal capacity, and finally the lack of appropriateness of redeployment.

Many other factors were considered, where the employer feared the workplace event recurrence and linked litigation. This is also due to the attitude of the employer and his adaptability, education, and personality. The existence of a "cardionoxious" workplace, low employer motivation, lack of consideration of rehabilitation period, and risk acceptability. Moreover, the lack of safety procedures for related medical issues could negatively create the lassitude of returning to work than considering the illness prognosis (Anne, 2004; Reibis, 2019).

2.8.2.2 Functional capacity of a person after heart disease

The examination of the functional capacity of an employee who suffered a cardiac event should be carried out before returning to his usual or any other job. People with coronary artery disease and hypertension must pass the exercise stress test to get useful information for permission of returning to work. However, further investigation should be done on people with cardiac failure (Anne, 2004).

2.8.2.3 Cardiovascular event nature: root cause which let to stop the work

Drugs alone or angioplasty and coronary artery bypass count in the treatment of coronary heart disease. Myocardial infarction and angina could also be treated in the same way.

Coronary heart disease can take different forms. However, the crucial aspect for getting back to work is relying on the evaluation of the persistence of chest pain during exercise, the risk of arrhythmia, and the level of left ventricular function. Especially if this may affect exercise capacity.

It is also crucial to heighten the careful consideration for high-risk people to detect silent ischemia. It is interesting that patients who underwent coronary artery bypass surgery (CABG) patients and angioplasty have the same long-term employment expectation. However, the patients that underwent angioplasty have a quick return to work (Anne, 2004). Another study showed that one-fifth get early retirement while four-fifth returns to work (Kirsten, 2014).

The work redeployment must be considered after ensuring the effectiveness of treatment regimens. In addition, it is paramount to control physical and psychological factors to avoid their negative effect on the pre-redeployment employee's health status. Some symptoms such as dizziness, headache, general malaise, and syncope may drastically and negatively affect the patient health. Moreover, non-controlled hypertension can worsen the situation. Furthermore, the pre-examination is necessary to evaluate the relapse indicator, which is shown by syncope or and general malaise. It is finally important to note that higher heart failure treatment effectiveness, higher chances of returning to work. Because the treatment improvement demonstrated that more people with cardiac failure can return to work in whatever condition (Anne, 2004).

2.8.2.4 The prognosis of the causative cardiovascular diseases

Prognostic indicators were well documented for most cardiological problems. It was found that people with poor prognoses presented a high risk of recurrence. Hence, the return to work may be inappropriate and create unrealistic expectations for the patient and his family (Anne, 2004).

2.9 Cardiovascular diseases and the community

A third of cardiovascular disease deaths occurred in the United States each year. In addition, coronary heart disease and stroke account for most of those deaths (Sara, 2014). A study called The Global Burden of Disease (GBD) demonstrated that Ischaemic stroke was 11.6 million cases, whereas low and middle-income countries (LMICs) sustained 65% in 2010. Moreover, it was 5.3 million hemorrhagic strokes (80 % in LMICs) occurred worldwide in 2010. Sixty-four percent of the disability-

adjusted life years (DALYs) were caused by ischaemic stroke and 86 % of DALYs due to hemorrhagic stroke were lost in LMICs (Krishnamurthi, 2013).

The relevant strategies to prevent cardiovascular diseases and deaths are articulated to accessibility and use of information on global cardiovascular risk factors and exposure. In addition, the knowledge improvement concerning the CVD factor's effect on exposed people can help to curb these silent killers. The Global comparative risk assessment study showed that a high number of CVD-related mortality was attributed to cardiovascular risk factors. High blood glucose, hypertension, high cholesterol, and smoking were the first factors to cause cardiovascular disease burden. In addition, High BMI, excessive alcohol use, sedentarity and environmental exposure were also among such CVD factors. Hypertension is the strongest cardiovascular risk factor, which caused more CVD deaths than any other risk factor. In addition, the BMI and other accumulated obvious risk factors have caused around 9.7 million annual cardiovascular disease mortality (Tzoulaki, 2016).

The nine modifiable traditional risk factors were attributed to the CVD population risk of CVD morbidity and mortality. The nine factors are smoking, sedentarity, unhealthy diet, obesity, history of hypertension or diabetes; harmful alcohol consumption raised blood lipids, and psychosocial factors (Gersh, 2010). Around 61% of worldwide cardiovascular disease mortality was attributed to eight risk factors. These factors are: (harmful alcohol use, hypertension, smoking, Obesity, sedentarity, unhealthy diet, diabetes, and high levels of cholesterolemia). The low and middle-income countries sustained about 84 % of the total CVDs worldwide burden. Therefore, studies demonstrated that the reduction of exposure to those CVD risk factors could enhance the improvement of worldwide life expectancy by almost 5 years (Francesco, 2016).

The active people in industrialized regions were vulnerable to cardiovascular diseases. This vulnerability is currently increasing in developing countries. Around 15% to 20% of active people were registered with CVDs in industrialized countries. The risk increase was observed with age, where 1/3 of men and 1/4 of women that suffered CVDs were people between 45 to 64 years (Florin, 2011).

Cardiovascular diseases account for 57 percent of all deaths in Russia in 2012 (Petrukhin & Lunina, 2012). Cardiovascular disease hit adults in their most productive years. For example, in South Africa, 41% of deaths due to cardiovascular disease occur in adults between 35 and 64 years old, compared to 12% in the United States (Leeder, 2014).

The potential costs of this cardiovascular disease epidemic for African countries are staggering. United States cardiovascular disease burden estimate was tremendously elevated to the level of the entire African continent's gross domestic product. It was around 300 billion United States dollars annually. Therefore, if this happens in Africa, it can drastically harm the full African economy and hamper the African development trend. In this way, the growing CVD epidemic in Africa will increase unacceptable levels of inequity in access to health care services (Jamison, 2006).

A study carried out in Tunisia showed the estimates of cardiovascular disease risk factors. Where 36% were android obesity, 28% were general obesity, and 21% of tobacco use. In addition, the risk factors were 19% and 10% for hypertension and diabetes respectively. Recently the prevalence of cardiovascular risk factors was 50.5% for hypertension, 18.2% for diabetes, 44% for dyslipidemia, 24.4% for obesity, and 24% for smokers (Jemaa, 2020). Moreover, the diet intake showed 2,483 kilocalories with 67%, 18%, and 15% for carbohydrates, protein, and fat respectively. Currently, a decrease in high-fiber diet coupled with an increase in the high-fat diet was noted in sub-Saharan city dwellers. The rural population with a traditional lifestyle showed a lower mean level of serum cholesterol than urban people (de Groot, 2019).

Another study was done in Nigeria on the Prevalence of cardiovascular disease risk factors among a Nigerian adult population, to assess the relationship between income level and accessibility to CVD risks screening. This study showed that females were 64.7% while the males were 35.3% for 422 participants with a mean age (±SD) of 42.9 ± 20.7 and 38.3 ± 20.5 years, successively.

The study also showed an elevated level of central obesity of 52.2%, hypercholesterolemia (38.1%), hypertension (35.7%), hypertriglyceridemia (23.2%),

low HDL (17.8%). It has also showed a low prevalence of prediabetes (4.9%), diabetes (5.4%) (Oguoma, 2015).

A study carried out in Gabon demonstrated that the ongoing epidemiological transition in Sab-Saharan Africa increased the prevalence of clinical and subclinical CVD in this community. This study showed that 13.3% were identified as having CVDs with highly prevalent hypertension up to 47.7% and 53.7% in men and women aged 50 to 60 years, respectively. Among the 382 participants with hypertension, 19.4% were treated and only 5.8% had controlled blood pressure (Ngoungoua, 2012).

According to Rhonda, in his research of an overview of cardiovascular risk factor burden in sub-Saharan African countries, demonstrated that SSA intricated factors are the sources of cardiovascular diseases increase.

The increasing prevalence of cardiovascular diseases in SSA is due to some changeable lifestyle risk factors such as an unhealthy diet, sedentarity, and smoking. Some lifestyle factors were considered gender-oriented; some are salient for women and others for men. Obesity was a predominant risk factor for women compared to men, but smoking remained mostly a risk factor for men.

The prevention and treatment efforts to fight these silent killers are blocked by insufficient healthcare facilities, poor planning, or lack of government programs such as surveillance. In addition, Poverty, promiscuity coupled with rising urbanization are also the underlying factors to the issue (Rhonda, 2009). An epidemiologic study has shown that ischemic heart diseases will rise in the two next decade due to the rising prevalence of risk factors. Especially hypertension, diabetes, obesity, physical inactivity, increased tobacco use, and dyslipidemia.

Ischemic heart disease was projected to grow by 25% in women and 27% in men for the entire African continent in 2015. It was forecasted to rise from 74% to 70% age-standardized mortality for women and men by 2030 (Lukwiya, 2013).

2.10 Conceptual framework

The conceptual framework of this study was presented in the schematic illustration of variables' interaction and relationship. In this study, the factors that affect the development the cardiovascular disease risk were classified into two categories (Predictor and cofactor variables) and were termed independent variables. The dependent variable is made of the predicted fatal and non-fatal cardiovascular disease risk (Cerebrovascular diseases, peripheral vascular diseases, heart failure, and coronary heart disease).

The prediction was carried out by two models using the different predictors among the eight predictors. The WHO/ISH model used six predictors while the FGRS used eight predictors (WHO, 2007; D'Agostino, 2008).

The predictor variables were:

1. Age
2. Gender
3. Cholesterol
4. High-density lipoprotein
5. Smoking
6. Blood pressure
7. Treated and untreated hypertension
8. Diabetes

The cofactor variables were:

1. Working condition factors
2. Behavioral factors
3. Novel risk and biological factors

The two moderating variables were the awareness of study participants toward cardiovascular disease factors and the use of personal protective equipment.

The choices of the predictor variables were based on the evidence brought out at the end of the Framingham heart study. Moreover, cross-validation studies were carried out to prove the effects of these variables on the outcome (Kuklina, 2010; Chia, 2015).

The correlation between predictor variables and cardiovascular disease risk cofactors was evaluated to determine those which were associated with a high risk in the study participants.

The cofactors (behavioral, biological, and working conditions) were deeply learned to assess their contribution to cardiovascular disease development for workers and spouses separately. Workplace hazards to cardiovascular disease development such as cold and Reynaud phenomenon (Peripheral vascular disease), (Plissonneau, 2015) and stress were also learned (Mika et al., 2002). Besides, a comparison between groups concerning cardiovascular disease risk was carried out.

This study sought to predict the cardiovascular disease risk, compare the prediction models, and determine factors associated with cardiovascular diseases risk for employees and spouses in two beverage manufacturing industries in Rwanda. Figure 2.5 illustrates the predictor and independent cofactor variables and the dependent variable which is cardiovascular disease risk of fatal and non-fatal cardiovascular diseases.

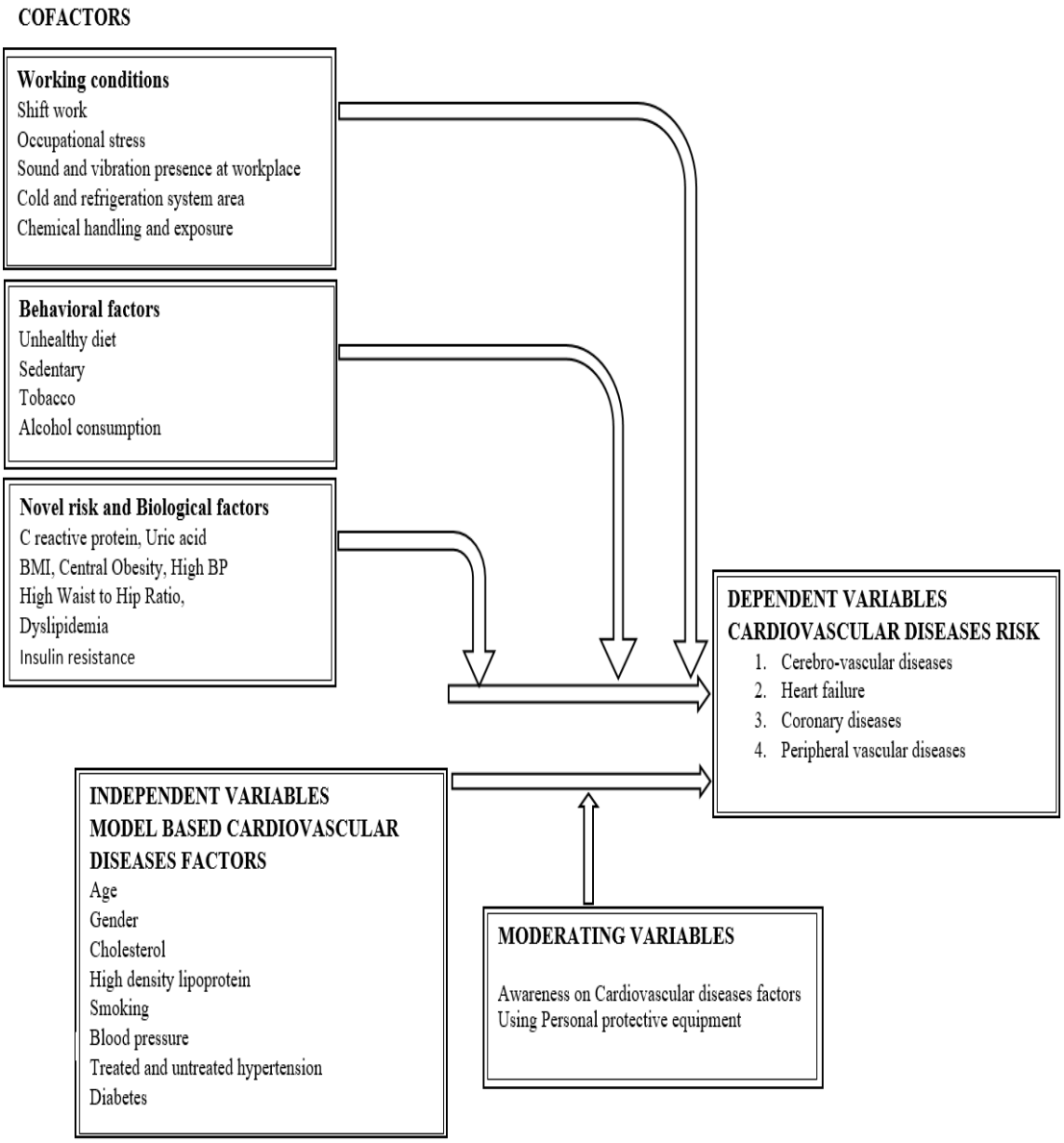

Figure 2.5: Conceptual framework

2.11 Assumption of the study

The study design was a cross-sectional where the interview and blood samples were collected at the industrial environment facility.

It was assumed that all study participants would be screened to exclude those with established cardiovascular diseases and respect the selection criteria to avoid the introduction of confounding or other biases in the study.

It was assumed that the blood samples would be taken the following day after the interview, in the morning without taking breakfast by considering eight to twelve hours of fasting. It was also assumed that the study participant who would not respect the eight to 12 hours of fasting, the blood sample collection would be postponed the next day.

CHAPTER THREE

MATERIALS AND METHODS

3.1 Study site

Kicukiro site

Kicukiro plant site started in 1973 in Kicukiro district with soft drink processing. Kicukiro district is in the middle south of Kigali town of Rwanda. Its population is predominantly urban with 87.9% (279,941 inhabitants) that reside in urban areas and 12.1% live in rural areas.

Rubavu site

Rubavu plant worksite started in 1957 in Rubavu with beer processing. Rubavu district is a rural district in northwest Rwanda. The population of Rubavu District is predominantly rural, where 63% of the resident population (254,453 inhabitants) lives in rural areas vs. 37% in urban.

3.2 Study design

An analytical quantitative cross-sectional study was conducted from May to December 2018 in the beverage manufacturing industry. Its aim was to determine factors associated with cardiovascular diseases predicted risk among workers and spouses of the Kicukiro soft drink plant and Rubavu brewery plant and their spouses.

3.3 Study limitation

Although this study has predicted the cardiovascular disease risk for all the study participant. The risk has been limited to four cardiovascular diseases (Cerebrovascular diseases, heart failure, coronary heart diseases and peripheral vascular diseases). The congenital heart diseases and rheumatoid heart diseases were not involved in this study.

As this study is a cross sectional design has learned the exposure and the effect at the same time. The study has predicted the cardiovascular disease risk based on the model inbuilt cox regression but has not done survival analysis with hazard ratio as for cohort study.

3.4 Study population

The target population of this study was composed of 822 population of regular employees and their spouses of two worksites. Kicukiro worksite and Rubavu worksite study population are described in the table below.

Table 3.1: Target population of the study

Study site	Study population	Employees	Spouses
Rubavu	336	204	132
Kicukiro	486	299	187
Total	822	503	319

3.4.1 Inclusion criteria

i. To be a worker in Kicukiro soft drink plant or in Rubavu brewery plant.
ii. Participant age must be ≥ 30 years to 75 years.
iii. To be a spouse of the worker of Kicukiro soft drink plant or Rubavu brewery plant.

3.4.2 Exclusion criteria

i. Anyone selected and who didn't want to join the study.
ii. Visitors and casual workers have not been selected for the study.
iii. Participants with clinically established cardiovascular disease were excluded due to bias of elevating the CVD risk.

3.5 Sample size determination

In the following formula for populations that are large, a sample for proportion was used to estimate sample size (Anokye, 2020).

3.5.1 Cochran formula for general sample size determination

The $n = \frac{z^2 p\, q}{e^2}$

Where

n is sample size

e is a standard error and assumed to be 0.05

z is normal deviation assumed to be 1.96

p is the proportion of workers and spouses with cardiovascular diseases, assumed to be 50% due to non-exact prevalence of CVDs in Rwanda.

q is the proportion of workers and spouses without cardiovascular diseases

p=50%= 0.5

q= 1-0.5=0.5

$n = \frac{1.96^2 x 0.5x\ 0.5}{0.05^2} = \frac{0.9604}{0.0025} = 385$ Hence, after the sample size adjustment for the 12% of non-response bias, the formula is as follow:

n1=385, the adjustment allowance was based on the addition of 10-20% individuals to take care of missed data, non-response rate and withdrawals (Suresh, 2012). The non-response rate is assumed to be 12% or 0.12. The final adjusted sample size was **n**=n1/ (1-0.12). Hence, 385/ (1-0.12) = 437.5 ≈ **440**.

Participants were proportionately allotted according to the size of workers and spouses in each site (Anokye, 2020).

3.5.2 Proportionate stratification formula for stratum sample size

The: $nh = \left({}^{Nh}/_{N}\right) * n$

Where

nh=Sample size for stratum h

Nh= the population size for stratum h

N= is the total population

n=is the total sample size

3.6 Sampling techniques

Stratified Random sampling techniques were mixed with simple random sampling where the first technique was used to select participants from their different organizational plants. This was performed to ensure the representativity of all sites' employees who met the inclusion criteria and the second was used to select participants in each status-based stratum.

Table 3.2: The proportions of the sample by workers and spouses in each drink processing plant

Drink processing plant	Target Population	Mean age	Gender	Formula	Sample Size
Kicukiro plant workers	299	39,5	F:25 M:135	(299/822)*440	160
Kicukiro plant worker's spouses	187	37,1	F:89 M:10	(187/822)*440	99
Rubavu plant workers	204	42,5	F:11 M:99	(204/822)*440	110
Rubavu plant worker's spouses	132	39,6	F:66 M:5	(132/822)*440	71
Total	822	39,6	F:191 M:249	Cochran+12% for Sample adjustment.	440

Employees and spouses list Jan 2017.

3.7 Data collection Instruments

3.7.1 Research Tool: Questionnaires

The tools consisted of three parts: A standardized questionnaire with Clinical and anthropometric measures form and a laboratory form for biochemical samples. The WHO/ISH risk prediction chart and FGRS were used to determine the cardiovascular disease risk.

i. Who stepwise standardized and semi-structured interview questionnaire. Health, and Safety Executive management standards indicator tool regarding the demographic and behavioral factors. In addition, the work conditions and lifestyle cardiovascular risk factors were addressed to the participants.
ii. The clinical inquiry and anthropologic measurement were filled on the designed form to record the blood pressure, height, weight, and abdominal circumference.
iii. Biochemical specimens were taken for prediction analysis purposes. The taken samples were fasting blood sugar, High-density lipoprotein (HDL) Low-density lipoprotein (LDL), and total cholesterol. In addition, C reactive protein, HB1AC, and Uric Acid were taken. Moreover, the venous samples were all taken after a certain fasting period. National Cholesterol Education Program (NCEP) explained that the fasting lipid profile data of adult people ≥ 20 years old could be taken once every five years. The values of HDL-C and Total cholesterol (TC) could be used alone without any other implication if fasting samples were not obtained (NCEP, 2001). However, 440 study participants followed the requested requirement of this study sampling and testing.

3.7.2 Research Materials and procedures for clinical and anthropometric measures

3.7.2.1 Research materials

i. **Stethoscope**: was used in clinical examination to eliminate the established CVDs with pathologic modification of heart sounds (WHO, 2007).

ii. **Scale for Height in Cm and weight in Kg measurement:** Scale Name: Seca mod:220, Max=150Kg Min=5kg, d=0,1kg e=0,1kg. The BMI was calculated after using the above-mentioned (height and weight) data by considering the World Health Organization (WHO) - Anthro Plus 2007 program.

iii. **Sphygmomanometer for Blood pressure measurement in mmHg: Medical tensiometer**: Beurer GmbH, Söflinger str.218, 89077. 652.10 Typ: Bm 20. The researcher took blood pressure three times at 10 minutes intervals. Then record the mean value of blood pressure to eliminate the bias of white coat hypertension by using the mean blood pressure after 3 times measurements (Mion, 2006).

iv. **Waist tape Tool for Waist circumference measurement in Cm: Waist tape tool:** It was used to measure the waist circumference at the central point between the last rib and iliac crest.

3.7.2.2 Procedure for data collection

This study data was collected in three-fold by 2 trained interviewers and 1 medical laboratory scientist for each site and supervised by the principal investigator. Firstly, a standardized questionnaire interview started. Then, clinical, and Anthropometric measurements followed and lastly biological measurements. In addition, the condition of blood sugar measures relied on the fasting blood sugar taken in the morning.

Biological measurements were based on for Cardiovascular risk prediction to predict the cardiovascular diseases risk. The prediction relied on mostly three biological variables HDL, total cholesterol, and fasting blood glucose among eight predictor variables. Those predictors are: (Age, gender, HDL, smoking, taking or not taking hypertension medicine, diabetes, systolic blood pressure, and finally total cholesterol). The calculation was also based on the mean of risk factors averaged in 10 years of event occurrences based on the specific sub-region countries for the WHO/ISH model and Framingham general risk score model (WHO, 2007). Hence, after the prediction, the 10-year cardiovascular diseases risk level was classified into different categories: >40, 40-30%, 20-30%, 10-20% and <10%.

3.7.3 Laboratory methods and quality

Apart from semi-structured questionnaires, anthropometric and clinical measurements used the following laboratory method. The measurement processes were executed with accuracy, reliability, and consistency to the national laboratory measurement regarding the calibration, and quality control of measurements:

Biochemistry measurements: The total cholesterol, high-density lipoprotein (HDL), low-density lipoprotein (LDL), C reactive protein (CRP), glycated hemoglobin (HBA1C), fasting blood sugar, and Acid uric: were all measured by:

Machine: Humalyser 3500, Human GmbH, Max Pbg Ring 21, 65205 Miesbaden Germany ref: 16800 Vers: 2014. The blood samples were taken between 8 to 12 hours of fasting. The used procedure to collect blood 440 samples was venipuncture in the ante-cubital region (Lorene, 2015).

The conversion formula had only regarded the total cholesterol, HDL, and blood sugar. The total cholesterol and high-density lipoprotein were converted from mg/dl to mmol/l by dividing 38, 67(Rugge, 2011). The value can also be multiplied by 0.0259 (Balder, 2017). The blood sugar values were converted from mg/dl to mmol/l by dividing the value by 18.01 (Ming, 2000; Jennifer, 2013).

3.7.4 Prediction models

3.7.4.1 WHO/ISH Cardiovascular risk prediction charts

Risk prediction chart for AFR E. with cholesterol measurement

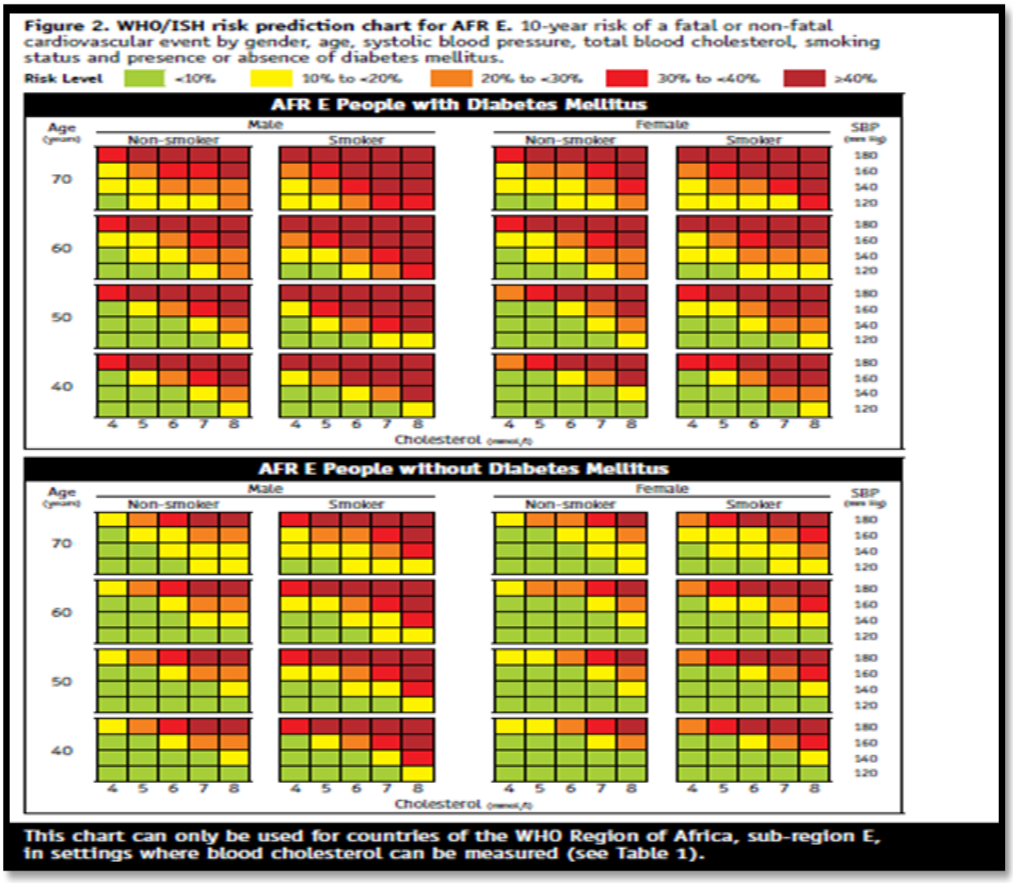

Figure 3.1: Region total blood cholesterol can be measured

This is the World Health Organization/international society of hypertension chart used to predict the 10-year risk of fatal and non-fatal cardiovascular events. This chart is only applied in Africa subregion E, in a setting where blood cholesterol can be measured. Its adoption relied on because Rwanda is mentioned in this subregion. It uses six predictors among others, gender, age, total blood cholesterol, smoking status, systolic blood pressure, and absence, or presence of diabetes Mellitus (WHO, 2007).

Risk prediction chart for AFR E. without cholesterol Measurement

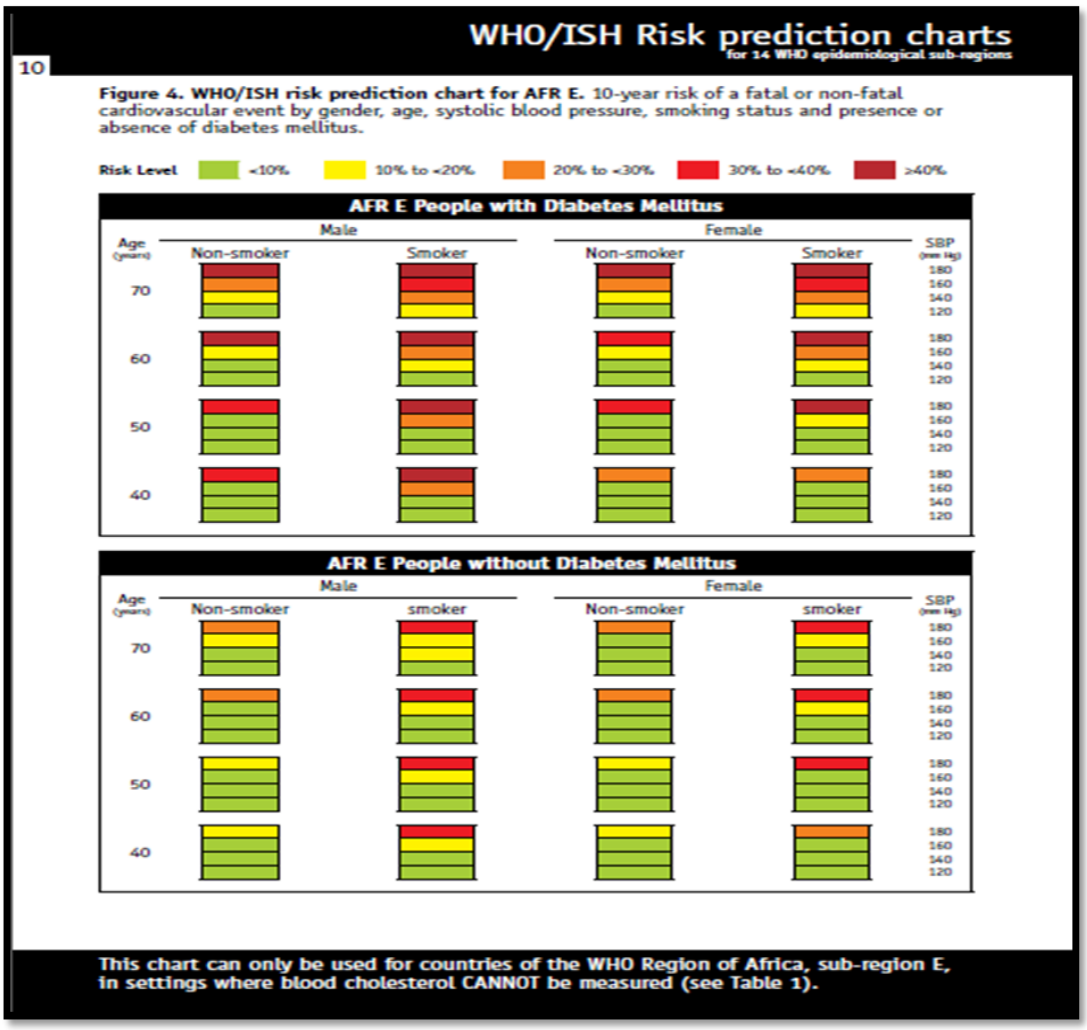

Figure 3.2: Region total blood cholesterol cannot be measured

This is also the World health organization/international society of hypertension chart and was used to predict the 10-year risk of fatal and non-fatal cardiovascular events. It was also applied in Africa subregion E. Contrary to the previous chart. This was applied in a setting, where blood cholesterol cannot be measured and used five predictors among others, gender, age, smoking status, systolic blood pressure, absence, or presence of diabetes Mellitus (WHO, 2007).

3.7.4.2 Framingham general cardiovascular diseases risk score model

Table 3.3: Cardiovascular diseases score sheet for man

Points	Age,Y	HDL	Total Cholesterol	SBP not treated	SBP Treated	Smoker	Diabetic
-2		60+		<120			
-1		50-59					
0	30-34	45-49	<160	120-129	<120	No	No
1		35-44	160-199	130-139			
2	35-39	<35	200-239	140-159	120-129		
3			240-279	160+	130-139		Yes
4			280+		140-159	Yes	
5	40-44				160+		
6	45-49						
7							
8	50-54						
9							
10	55-59						
11	60-64						
12	65-69						
13							
14	70-74						
15	75+						
Points allotted							

(D'Agostino et al., 2008)

SBP: Indicate Systolic Blood Pressure
HDL: indicate High density lipoprotein

This table 3.3 illustrates a score sheet of six predictors with points to attribute to each factor, whether present or not. This score sheet is only reserved for men to predict the 10-year risk for a cardiovascular event (D'Agostino, 2008).

Table 3.4: Cardiovascular points toward the risk percentage for man

Points	Risk, %
≤-3 Or less	< 1
-2	1.1
-1	1.4
0	1.6
1	1.9
2	2.3
3	2.8
4	3.3
5	3.9
6	4.7
7	5.6
8	6.7
9	7.9
10	9.4
11	11.2
12	13.2
13	15.6
14	18.4
15	21.6
16	25.3
17	29.4
18+	≥30

(D'Agostino, 2008)

The table 3.4 illustrates how after scoring the men's points in accordance with the presence or absence of predictors. The points obtained are matched to the risk from the lower risk to the higher risk of cardiovascular events (D'Agostino, 2008).

Table 3.5: Cardiovascular diseases score sheet for women

Points	Age,Y	HDL	Total Cholesterol	SBP not treated	SBP Treated	Smoker	Diabetic
-3				<120			
-2		60+					
-1		50-59			<120		
0	30-34	45-49	<160	120-129		No	No
1		35-44	160-199	130-139			
2	35-39	<35		140-149	120-129		
3			200-239		130-139	Yes	
4	40-44		240-279	150-159			yes
5	45-49		280+	160+	140-149		
6					150-159		
7	50-54				160+		
8	55-59						
9	60-64						
10	65-69						
11	70-74						
12	75+						
Points allotted							

(D'Agostino et al., 2008)

SBP: Indicate Systolic Blood Pressure

HDL: Indicate High Density Lipoprotein

Table 3.5 illustrates a score sheet of six predictors with points to attribute to each factor, whether present or not. This score sheet is only reserved for women to predict the 10-year risk for a cardiovascular event (D'Agostino et al., 2008).

Table 3.6: Cardiovascular points toward the risk percentage for woman

Points	Risk, %
≤-2	< 1
-1	1.0
0	1.2
1	1.5
2	1.7
3	2.0
4	2.4
5	2.8
6	3.3
7	3.9
8	4.5
9	5.3
10	6.3
11	7.3
12	8.6
13	10.0
14	11.7
15	13.7
16	15.9
17	18.5
18	21.5
19	24.8
20	28.5
21+	>30

(D'Agostino, et al., 2008)

Table 3.6 illustrates how after scoring the women's points in accordance with the presence or absence of predictors. The points obtained are matched to the risk from the lower risk to the higher risk of cardiovascular events (D'Agostino, 2008).

3.8 Validity and reliability of research instruments

The validity and reliability of the instruments relied on the pre-validation of the models (D'Agostino, et al., 2008). Including the WHO steps standardized questionnaire with longtime scientific performance.

3.8.1 Validity

Questionnaire validity

The WHO stepwise approach for non-communicable diseases, standardized and validated questionnaire was applied. This was performed with the aim to emphasize the facilitation of comprehension and relevance to the intended topics (WHO, 2008; WHO, 2018). In addition, it helps to raise the effectiveness of useful information delivery and enhance the degree to which the questions were interpreted and understood by different individuals (Sarma, 2019).

Model validity

This study adopted two prediction models: Framingham general risk score (FGRS) and the World Health Organization/International Society of Hypertension (WHO/ISH) risk prediction chart. The models' validity is based on their global capacity, useful research, and WHO recommendations for Its application in the African population. Their validity (Calibration, Discrimination, and clinical usefulness) has been tested and accepted by the World health organization in the sub-region of Africa (Kuklina, 2010).

The multi-ethnic Asian population study revealed a good calibration of the Framingham general CVD risk score. In addition, the Hosmer-Lemeshow test showed a good result with $\chi2=3.25$, $p=0.78$ (chia, 2015). CVD prediction models have also presented a good calibration where the $\chi2$ for men was $\chi2=13.48$ and $\chi2=7.79$ with excellent goodness of fit. Moreover, the study expressed an AUC of 0.763, 95%CI (0.746 to 0.780) for men, and an AUC of 0.793, 95% CI (0.772 to 0.814) for women. Hence, this was a C statistic with good discrimination (D'Agostino, 2008).

The clinical importance and usefulness of risk function were gauged by decision curve analysis, sensitivity, specificity, and finally the net benefit fraction. It was, therefore, resulted by the recommendations in Iran's population, which was taken as a screening tool (Davood, 2012).

The revised Framingham function has a good performance and ability for accurately predicting the total CVDs versus the risk function evolved specifically from the individual cohorts' data. Hence, resulted in good validity (Kuklina, 2010). The predicted total CVDs are Coronary heart disease, Cerebrovascular diseases, Peripheral vascular diseases, and Heart Failure (Peter, 2016). In addition, after recalibration, considering the different prevalence of risk factors and underlying rates of developing CHD, the Framingham functions worked well. Moreover, the Framingham prediction function showed a good discrimination ability between CHD patients and safe persons than non-Framingham cohorts (D'Agostino, 2001).

Predicting cardiovascular diseases with one, multiple variable risk function was represented by the Framingham general CVD risk score (D'Agostino, 2008). The ''WHO/ISH'' risk prediction model was used regarding the sub-region as recommended by WHO and has shown a good agreement with FRS (WHO, 2007; Norhayati, 2013).

3.8.2 Reliability

The reliability and validity of instruments were based on the adoption of a pre-validated WHO stepwise-standardized questionnaire. Moreover, the researcher conducted the instruments simulation on 40 participants chosen before the study. The simulation analysis for the item of the instruments showed a significant Cronbach alpha > 0.8. We followed the WHO guideline on a stepwise approach to ensure understanding and valid and reliable application.

3.9 Data management and analysis

The analysis of this study data was conducted using SPSS software version 22, according to respective objectives. Bivariate and multivariate analyses were used to

determine the factors associated with high-risk categories and underlying risk factors correlated to predictor variables.

The prediction was applied to the first objective. This was based on the inbuilt cox hazard regression model (Framingham general cardiovascular risk score) and WHO/ISH risk prediction chart to determine the 10-year CVD risk in the population of the study. The comparison of FGRS and WHO/ISH was facilitated by four procedures. These procedures were: binary categorization of cardiovascular diseases risk level, predictive probability generation of the two models by binary status, multilevel categorization, and status correlation. Finally, the performance of AUC comparison with predictive probability by a correlated status.

Cohen Kappa test for model's agreement and ROC curve classification performance (NCEP, 2001). Therefore, this test was used to determine the level of risk prediction agreement between the two models (Mary, 2012).

The Anova model and X^2 were used to compare the group participants and determine the relationship between variables in two different areas regarding cardiovascular risk factors differences. The analysis helped to express the factors' interaction between exposure and outcome for explaining what factors could be focused on to minimize the cardiovascular disease risk. The significance level was set at a P-value less than 0.05 at 95% CI.

Confounding management

The twisting bias that is risen when similar causal factors interact with the exposure and effect phenomenon is termed confounding (Wallach, 2020). Wayne explained it as a contortion or an alteration bias. Hence, this bias occurred when other factors interact with the exposure effect to render a null, positive, or negative influence on the outcome; different from what would happen if such confounding was managed (Wayne, 2016). For this study's management of cardiovascular factors confounding was managed by the following interventions.

- The researcher has done the restriction of people with established CVDs. This was done to prevent that, one of the independent factors

would be accounted for the high strength of being associated with the CVDs while it is not. Alternatively, when the potential common causes of exposure factors were not involved in predictors. Restriction of older people of >=75yrs who may also have the CVDs (Wallach, 2020).

- After the identification of known confounders and their classification, we have used multivariate analysis to measure and adjust their level of association (Taravatmanesh, 2017).
- The stratum divisions were created to only analyze the workplace-based cardiovascular factors alone and common community cardiovascular factors alone (Wallach, 2020).

3.10 Ethical consideration

The researcher got a letter of authorization from the Board of postgraduate studies. In addition, ethical clearance was received from the Rwanda National Ethical Committee (RNEC) in the Ministry of Health. The Beverage processing worksite Company also gave permission to collect data. Trained data collectors filled the translated data collection form during permitted or convenient time before proceeding to the Physical examination and biochemical sample taking.

Moreover, each participant had the freedom to participate voluntarily after his consent and approval in the study without coercion. The participant had a right and a possibility to withdraw from the investigation whenever they became uncomfortable or do not want to continue. Participants' comfortability and protection were ensured during the data collection to avoid psychological and physical harm. The results were confidentially and anonymously used for the purpose of the study only.

CHAPTER FOUR

RESULTS

4.1 Demographic characteristics of the study participants

The analysis results for demographic characteristics were processed on the total study participants sample (N=440). 270 employees were 61.8% of all participants and 170 of their spouses were 38.6% of all participants in Rwandan Beverage Company. They all consented to participate in the study where 58.9% were in Kigali plant and 41.1% were in Rubavu plant. The median age was (45 Years, IQR: 14). Other demographic findings are tabulated in table 4.1.

Table 4.1: Socio-demographic characteristic of respondents

Variables	n (440) Subjects	Rate (%)
Location		
Kicukiro	259	58.9
Rubavu	181	41.1
Age Group		
<35	84	19.1
35-49	73	16.6
40-44	65	14.8
45-49	86	19.5
50-54	89	20.2
55-59	30	6.8
>=60	13	3
Gender		
Males	249	56.6
Females	191	43.4
Marital status		
Single	36	8.2
Married	401	91.1
Living together	0	0
Divorced	0	0
Widow	3	0.7
Place of birth		
Kigali city	30	6.8
Eastern province Rwanda	34	7.7
Western Province Rwanda	98	22.3
Northern province Rwanda	58	13.2

Variables	N (440) Subjects	Rate (%)
Southern province Rwanda	104	23.6
DRC	90	20.5
Burundi	10	2.3
Uganda	13	3
Tanzania	2	0.5
Peru Latina America	1	0.2
Religion		
Roman catholic	293	66.6
Protestant	102	23.2
Muslim	16	3.6
7th day Adventist	15	3.4
Witness of Jehovah	9	2
Branham believer	1	0.2
Others	4	0.9
Participant status		
Employees	270	61.4
Spouses	170	38.6
Education		
None	2	0.5
Primary	70	15.9
A3 post primary certificate	21	4.8
Secondary	155	35.2
Diploma	36	8.2
Bachelor's degree	145	33
Master's degree	11	2.5
Experience for employees		
<=4	33	7.5
5-9	56	12.7
10-14	67	15.2
15-19	8	1.8
20-24	86	19.5
25-29	12	2.7
>=30	8	1.8

Researcher, 2019

4.2 Levels of the 10-year cardiovascular diseases risk predicted among the study participants of Kicukiro soft drink plant and Rubavu Brewery plant

4.2.1 The 10-year cardiovascular diseases risk prediction by Framingham risk score and WHO/ISH

The prediction of cardiovascular diseases risk was processed by using the Framingham general risk score and WHO/ISH model score chart and the Cox regression formula for only Framingham general risk score. Among the eight predictors' findings, gender was composed of 56.6% of males and 43.4% of females in the total sample (N=440). Treated systolic blood pressure findings showed that 17% were under treatment among 32% of respondents with systolic blood pressure, (N=440). Out of the sample of 440(100%), the smokers were 6.8% versus 93.2% of non-smokers. The diabetic respondents were 11.1% versus 88.9% of non-diabetic respondents. The mean age was 44.92 years. The presentation of the mean value of the lipide profile (Total cholesterol, triglyceride, and high-density lipoprotein) is tabulated in Table 4.2.

Table 4.2: Distribution of cardiovascular diseases risk predictors

Variables	Proportion (%)
Gender	
Male	56.6
Female	43.4
Treated SBP	
yes	17
No	83
Smoking	
Yes	6.8
No	93.2
Diabetic	
Yes	11.1
No	88.9
Age	44.92
Total Cholesterol(mg/dl)	164.4
High density lipoprotein (mg/dl)	49.35
Triglyceride (mg/dl)	145.5

The cardiovascular diseases risk prediction showed that the Framingham general risk model predicted more people (25.5%) with elevated cardiovascular diseases risk (>10%), than 4.6% by WHO/ISH model. The risk increased by age with dominance in male respondents than in female respondents (N=440). Table 4.3 shows the comparative risk prediction levels by age and gender.

Table 4.3: Cardiovascular diseases risk stratification by age and gender for two models.

Model	**N**	**Male**			**Female**		
	440	<40yrs	40-50yrs	>50yrs	<40yrs	40-50yrs	>50yrs
FGRS							
Low risk (<10%)	328	90	53	35	62	59	29
2nd level risk (10-20%)	60	2	12	19	2	14	11
rd level risk (20-30%)	28	0	5	13	1	4	5
4th level risk (30-40)	24	0	3	17	0	1	3
5th level of risk (>40)	0	0	0	0	0	0	0
Total	440	92	73	84	65	78	48
WHO/ISH	440	<40yrs	40-50yrs	>50yrs	<40yrs	40-50yrs	>50yrs
Low risk (<10%)	420	91	69	76	65	72	47
2nd level risk (10-20%)	15	0	2	6	0	6	1
3rd level risk (20-30)	1	0	0	1	0	0	0
4th level risk (30-40)	3	1	1	1	0	0	0
5th level of risk(>40)	1	0	1	0	00	0	0
Total	440	92	73	84	65	78	48

This study findings were based on the comparison of cardiovascular diseases risk between employees(n=270) and spouses(n=170), (N=440). The findings showed that employees presented higher cardiovascular risk than spouses with $x^2=2.152$, $p<0.001$. Figure 4.1 shows the pyramid of comparative risk levels between employees and spouses.

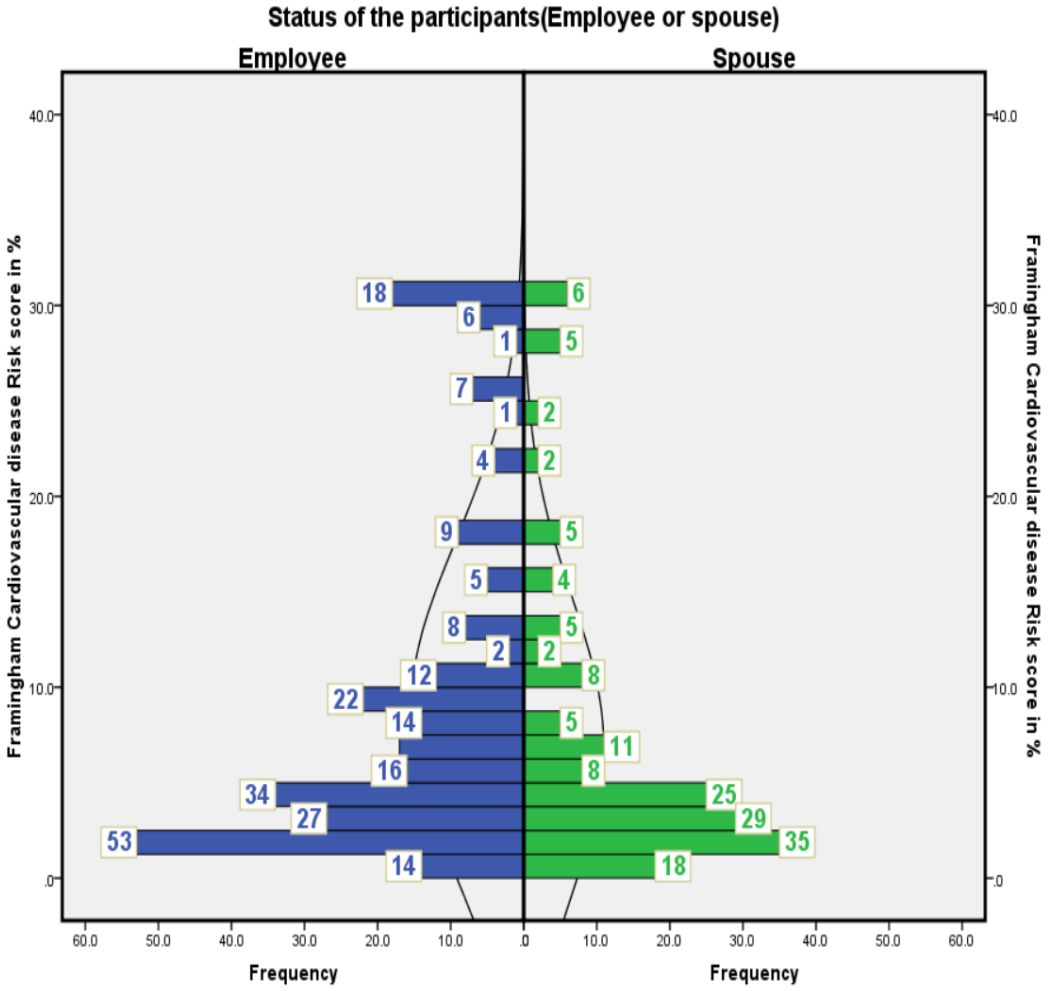

Figure 4.1: Cardiovascular diseases risk pyramid by status of the participants

4.2.2 Framingham general risk score and WHO/ISH model's comparison

The comparison of the model's performance was conducted by means of generated predictive probabilities in logistic regression with elevated risk (>10%) and low risk (<10%). The comparison of the model's discriminatory capacity by the receiver-operating characteristic showed a perfect performance with AUC above 0.847 for both models, $p<0.001$, (n=440). The comparative performance of the two models were depicted in Figure 4.2.

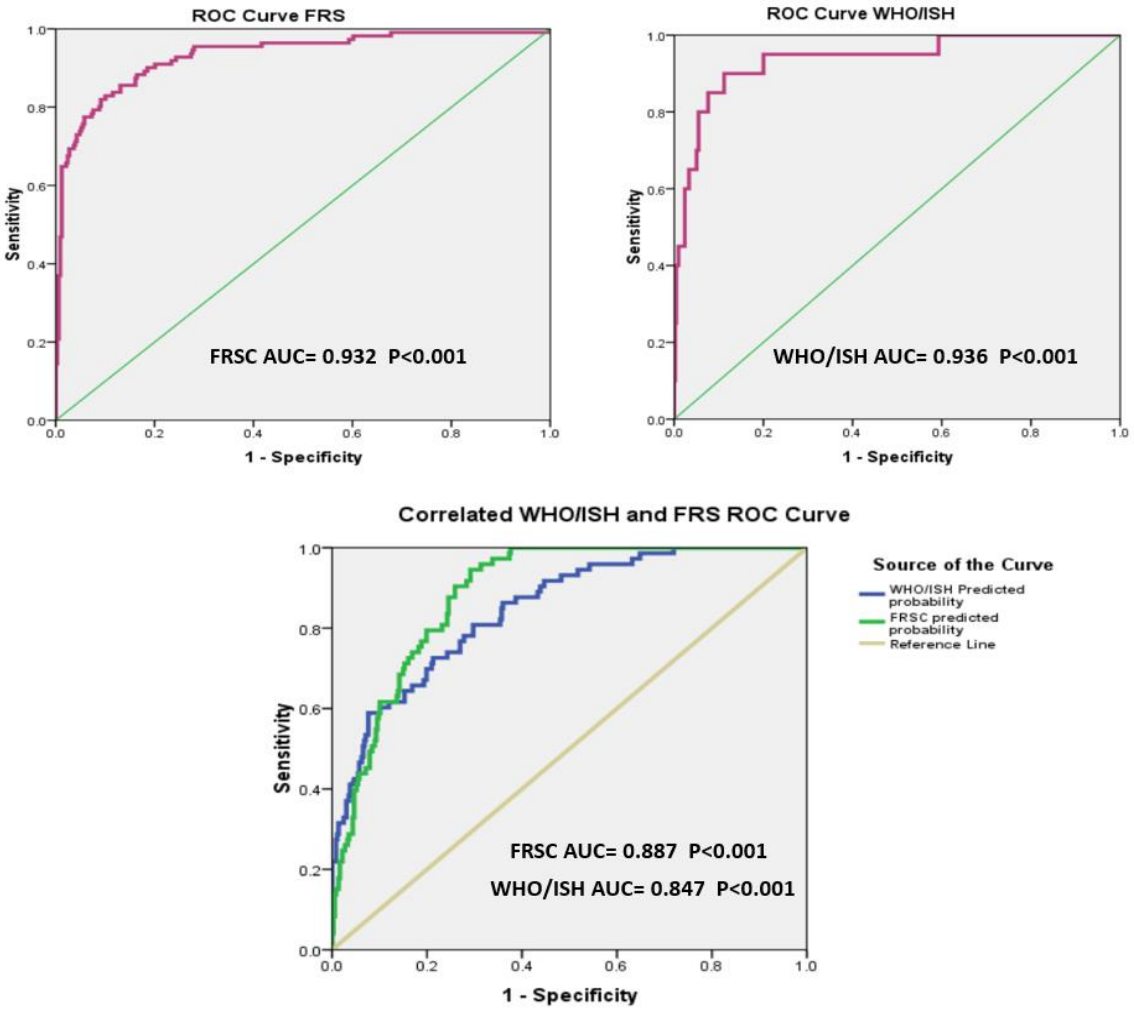

Figure 4.2: Performance comparison of the area under the curve (AUC) of two prediction models WHO/ISH and FGRS

The cardiovascular diseases prediction risk model's agreement comparison was performed by using the Kappa test inter-rater reliabilities, $p<0.05$. The comparison of the Framingham general risk score chart and the WHO/ISH score chart by the kappa test showed a minimal value of 0.25, $p<0.001$. The comparative agreement level is portrayed in Table 4.4.

Table 4.4: Level of agreement of Framingham general risk prediction model and World Health organization/International Society of hypertension model by Cohen kappa

Symmetric Measures of model agreement					
		Value	Asymp. Std. Error[a`]	Approx. T[b]	Approx. Sig.
Measure of Agreement	Kappa	.250	.047	7.928	.000
N of Valid Cases		440			

a. Not assuming the null hypothesis.

b. Using the asymptotic standard error assuming the null hypothesis.

4.3 Proportion of behavioral factors associated with cardiovascular diseases among the study participants of Kicukiro soft drink plant and Rubavu Brewery plant

The second study objective was to determine the proportion of behavioral factors associated with cardiovascular diseases risk for the total sample size(n=440). All the seven factors were modifiable behavior risk factors among the study participants.

4.3.1 Level of smoking behavior risk factor to cardiovascular diseases

The findings of this study showed that the level of smoking among the study respondents, was ranged from 6.8% of current smokers, 6.6% of daily smokers, and 3.2% of respondents smoking 1 to 5 cigarettes a day. Second-hand smoking was 4% at the workplace while it was 15.8% at home. The proportion levels are portrayed in Table 4.5.

Table 4.5: Proportion of people with smoking behavior in the study participants

Variables	Subjects (n)	Rate (%)
Currently smoke any tobacco products: Cigarettes, Cigars, Pipe		
Yes	30	6.8
No	410	93.2
Currently smoke tobacco daily		
Yes	29	6.6
No	411	93.4
How old were you when you start smoking		
<20 Years	10	2.2
20-30 Years	10	2.2
31-40 Years	5	1.1
41 years and above	4	0.9
Average daily tobacco products smoked: manufactured and hand-rolled cigarette, pipe, cigar, cigarillos or others		
1-5 Tobacco Products	14	3.2
6-10 Tobacco Products	3	0.7
11-20 Tobacco Products	2	0.5
21-30 Tobacco Products	3	0.7
31&+ Tobacco Products	1	0.2
Other tobacco product specification		
Shisha	5	1.1
Cannabis	3	0.7
Ever smoke in the past		
Yes	9	2
No	397	92.2
Age in the past when stop smoking		
<40 Years	5	1.1
>40 Years	4	1.8
Time spent after stop smoking		
1-5 Years	3	0.7
6-10years	4	1.1
11-15years	1	0.2
16Years and more	1	0.2
Current use of any smokeless tobacco, betel		
Yes	12	2.7
No	428	97.3
Current daily use of smokeless tobacco products		
Yes	12	2.7
No	428	97.3
Average times a day of using smokeless tobacco products		
1-59 Min	8	1.8
1-2 Hours	4	0.9
Past ever use smokeless tobacco, betel, snuff, chewing tobacco		
Yes	8	1.8
No	432	98.2
How many days in the past 7 days someone smoke around you at home		
1 day	17	3.9
2 days	25	5.7
3 days	7	1.6
4 days	3	0.7
5 days	7	1.6
6 days	1	0.2
7 days	10	2.3
How many days in the past 7 days someone smoke around you at work		
2 days	2	0.5
3 days	3	0.7
4 days	4	0.9
5 days	3	0.7
6 days	2	0.5
7 days	3	0.7

Bivariate analysis was processed using the chi-square, where male respondents showed elevated smoking levels versus females regarding the daily smokers (n=29) and current smokers (n=30), where (N=440), $p<0.05$. The association findings are tabulated in table 4.6.

Table 4.6: Bivariate analysis of smoking by gender

Variable	N(440)	Male	Female	Statistical test X^2 (df); *P*
Currently smoke any tobacco products: cigarettes, Cigars, Pipe				
Yes	30(6.8)	24(5.5)	6(1.4)	
No	410(93.2)	225(51.1)	185(42.0)	
Total	440(100)	249(56.6)	191(43.4)	7.182 (1); .005
Currently smoke tobacco products daily				
Yes	29(6.6)	23(5.2)	6(1.4)	6.523(1); .008
No	411(93.4)	226(51.4)	185(42.0)	
Total	440(100)	249(56.6)	191(43.4)	

4.3.2 Alcohol consumption behavior of the study participants

These alcohol consumption behavior findings were presented regarding the type of Alcohol intake: beer 5%, wine 12%, liquor 40%, and their daily consumption standards.

The findings of this study about the behavioral alcohol consumption as portrayed in Table 4.7 showed the general alcohol intake and the levels of alcohol intake in the study respondents. The majority of people have ever consumed alcohol with (n=355) while (n=303) of respondents took alcohol in the past 30 days (N=440).

Table 4.7: Proportion of behavioral alcohol consumption in the study participants

Variables	Subjects (n)	Rate (%)
Ever consumed alcoholic drink (Beer, wine, spirits, fermented cider or local)		
Yes	355	80.7
No	85	19.3
Alcoholic consumption in the past 12 months		
Yes	303	68.9
No	137	31.1
Frequency of alcoholic consumption in the past 12 months		
None	137	31.1
<1 day per month	1	0.2
1-2 days per month	2	0.5
2-3 days per month	13	3
3-7 days per month	4	0.9
7-10 days per month	2	0.5
10-15 days per month	1	0.2
4 days per week	160	36.4
5-6days a week	13	3
Daily	107	24.3
Alcohol consumption within the past 30 days		
Yes	303	68.9
No	137	31.1
How many occasions at least one occasion of alcoholic drink for 30 days		
None	137	31.1
1-5 Occasion	57	13.0
6-10 Occasion	43	9.8
11-15 Occasion	36	8.2
16-20 Occasion	48	10.9
21-25 Occasion	17	3.9
26-30 Occasion	101	23.0
Alcohol intake at workplace for employees		
NA	153	80.1
Never	8	4.2
Seldom	11	5.8
Sometimes	18	9.4
Often	1	0.5

This study's findings on the 5% beer consumption were processed in terms of average beer 5% standard drink in 30days. Where, 9oz=10gr of pure alcohol/266ml, 9-12oz=10-14gr of alcohol in the 266-350ml glass. Glasses were counted based on the reported bottles consumed on different occasions in 30 days by the study respondents, where 67.5% consumed 5% beer, (n=440). The presentation of 5% beer intake was carried out in line with the status (employees, spouses) and gender. Male employees(n=234), Female employees(n=36), Male spouses(n=15), female spouses(n=155), with the total sample of 440. The 5% beer intake levels were depicted in table 4.8.

Table 4.8: Level of beer 5% intake, 12Oz glass of 354ml=1Standard with 14gr of pure alcohol consumption in the study participants by status and gender.

Variables	N(440)	Employees		Spouses	
		Male	Female	Male	Female
Average beer 5% standard drink in 30days 9oz=10gr of pure alcohol/266ml, 9-12oz=10-14gr of alcohol in 266-350ml glass (Deborah, 2003)					
None	143	52	13	3	75
1-5 Glasses	22	7	4	1	10
6-10 Glasses	22	13	3	0	6
11-15Glasses	50	29	5	1	15
16-20 Glasses	14	7	1	0	6
21-25 Glasses	30	14	2	3	11
26-30 Glasses	5	3	0	0	2
31-60 Glasses	39	27	3	0	9
61-90 Glasses	42	30	3	1	8
91-120 Glasses	30	27	0	2	1
121-150 Glasses	13	4	2	2	5
151-180 Glasses	13	8	0	2	3
181-210 Glasses	10	7	0	0	3
211-240 Glasses	5	5	0	0	0
241-270 Glasses	1	1	0	0	0
271-300 &+ Glasses	1	0	0	0	1
Total	440	234	36	15	155

This study's findings on the 12% wine consumption were processed in terms of the average 12% vine standard drink in 30days. Where, 5oz=147ml/glass which contains 14gr of pure alcohol. Glasses were counted based on the reported glasses consumed on different occasions in 30 days by the study respondents where 41% consumed vine (n=440). The presentation of 5% beer intake was carried out in line with the status (employees, spouses) and gender. Male employees(n=234), Female

employees(n=36), Male spouses(n=15), female spouses(n=155), with the total sample of 440. The 12% wine intake levels were portrayed in Table 4.9.

Table 4.9: Level of alcohol consumption in 143ml of wine 12% in 30days amongst the study participants by status and gender

Variables	**N(440)**	**Employees**		**Spouses**	
		Male	Female	Male	Female
Average Wine 12%					
None	260	121	27	3	109
1-2 Glasses	63	43	2	4	14
3-4 Glasses	51	27	3	4	17
5-6 Glasses	22	16	0	3	3
7-8 Glasses	13	6	2	2	3
9-10 Glasses	18	13	0	4	1
11-12 Glasses	10	7	2	0	1
13-14 Glasses	3	1	0	0	2
Total	440	234	36	15	155

The study findings portrayed the 40% liquor consumption for 30 days. The standard liquor drink was measured as 1.5 oz equated to 44ml per glass containing 14gr of pure alcohol. Around 23.9% consumed 40% liquor in different occasions for 30 days, among others male employees (n=84), female employees(n=5), male spouses(n=3), female spouse(n=13), where (N=440). The 40% liquor intake levels were portrayed in Table 4.10.

Table 4.10: Level of alcohol in 44ml of liquor consumption in 30 days amongst the study participants by status and gender

Variable	N(440)	Employee		Spouse	
		Male	Female	Male	Female
Average liquor 40% standard drink of alcohol in 30days					
None	335	150	31	12	142
1-2 Shot glasses	40	33	3	0	4
3-4Shot glasses	30	20	2	2	6
5-6 Shot glasses	18	16	0	1	1
7-8 Shot glasses	13	12	0	0	1
9-10 Shot glasses	3	2	0	0	1
11-12 Shot glasses	0	0	0	0	0
13-14 Shot glasses	0	0	0	0	0
15-16 Shot glasses	0	0	0	0	0
17-18 Shot glasses	0	0	0	0	0
19-20 & more Shot glasses	1	1	0	0	0
Total	440	234	36	15	155

The mean value differences between gender and the different level of age groups on the largest alcohol intake on one occasion were processed by the F test and standard deviation. The variation between the age groups of females was statistically significant, $p<0.001$. However, the variation was not significant in groups of males alone. The total gender groups showed a significant difference within age groups, $F=3$, $p=0.03$ for only drinker respondents (n=303). The mean value and standard deviation were portrayed in Table 4.11.

Table 4.11: Largest standard alcohol intake mean value on one occasion of alcohol drinking by age and gender for only drinker in last 30 days

Largest alcohol intake on one occasion (Mean value ± SD)				
	n=303			
Age group		Male	Female	Total
<40Years	97	4.96(4.653)	2.07(1.668)	4.09(4.206)
40-50Years	97	6.58(4.829)	3.64(2.775)	5.31(4.307)
>50Years	109	5.78(4.969)	5.29(4.644)	5.62(4.851)
Total	303	5.72(4.841)	3.75(3.507)	5.03(4.513)
Statistical test				
F=		*1.738*	*7.541*	3.269
df=		*2*	*2*	2
p=		*0.179*	*0.001*	0.039

The mean value differences between gender and the different level of age groups on the excess of 5 drinks for men and 4 drinks for women were processed by the F test and standard deviation. The variation between the age groups of females was statistically significant, p=0.006. However, the variation as not significant in groups of males alone. The total gender groups showed a significant difference within age groups, F=5, p=0.007, for only alcoholic drinker respondents (n=303). The mean value and standard deviation were portrayed in Table 4.12.

Table 4.12: Excess of Alcohol standard intake mean value of times taken 5 and more standard drinks for a man, 4 and more standard drink for a woman by age and gender for only drinkers for 30 days

Five drinks and more for men/Four drink and more for women (Mean value ± SD)				
	n=303			
Age group		Male	Female	Total
<40Years	97	2.63(4.350)	0.28(0.841	1.93(3.819)
40-50Years	97	4.44(5.249)	3.24(5.226)	3.92(5.245)
>50Years	109	3.47(4.603)	3.23(4.305)	3.39(4.491)
Total	303	3.45(4.738)	2.42(4.314)	3.09(4.613)
Statistical test				
F=		*2.233*	*5.364*	5.005
df=		*2*	*2*	2
p=		*0.110*	*0.006*	0.007

The findings on the relationship of exceeding 5 drinks for men and 4 drinks for women by gender and age groups were conducted using the chi-square. The relationship was not significant for the gender and the level of excess drinks in the study respondents. Whereas, the relationship was significant with age group, x^2=14, p=0.02, (n=123). The level of this relationship was tabulated in Table 4.13.

Table 4.13: Bivariate analysis of times consumed four or more alcoholic drinks for women and five or more for men with gender and age.

Variable	n(123)	Male	Female		Statistical test
					$X^2(df);P$
Times exceeded 4 drinks for female and 5 drinks for male in 30 days.					
1-4 Times	32(26.0)	20(16.3)	12(9.8)		4.562(3);.207
5-10 Times	60(48.8)	47(38.2)	13(10.6)		
11-15 Times	28(22.8)	18(14.6)	10(8.1)		
16+ Times	3(2.4)	3(2.4)	0(0.0)		
Total	123(100)	88(71.5)	35(28.5)		
Variable	**n(123)**	**<40Years**	**40-50 Years**	**>50Years**	**Statistical test**
					$X^2(df);P$
Times exceeded 4 drinks for female and 5 drinks for male in 30 days.					
1-4 Times	32(26.3)	15(12.2)	8(6.5)	9(7.3)	14.249(3);.027
5-10 Times	60(48.8)	10(8.1)	20(16.3)	30(24.4)	
11-15 Times	28(22.8)	5(4.1)	14(11.4)	9(7.3)	
16+ Times	3(2.4)	1(0.8)	1(0.8)	1(0.8)	
Total	123(100)	31(25.2)	43(35.0)	49(39.8)	

4.3.3 Level of fruits intake and servings amongst the study participants

The level of fruit intake behavior findings was presented regarding the weekly and quantity of servings intake for the entire study respondents (N=440).

In a typical week, the study findings on the fruit intake in terms of daily servings for employees were (male: n=234, female: n=36). The spouse's findings were (Male: n=15, female: n=155) showed that 53.3% took fruit for only one day while 24.8% (n=109) did not (see Table) 4.14.

Table 4.14: Proportion of fruits intake by status and gender

Variables	N(440)	Employees		Spouses	
		Male	Female	Male	Female
How many days you eat fruits in a typical week					
None	109(24.8)	63(14.3)	14(3.2)	4(0.9)	28(6.4)
One day	235(53.3)	138(31.4)	17(3.9)	8(1.8)	72(16.4)
Two days	85(19.3)	30(6.8)	5(1.1)	3(0.7)	47(10.7)
Three days	11(3.5)	3(0.7)	0(0.0)	0(0.0)	8(1.8)
Four days	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Total	440(100)	234(53.2)	36(8.2)	15(3.4)	155(35.2)
Number of fruits servings on one day					
None	109(24.8)	63(14.3)	14(3.2)	4(0.9)	28(6.4)
Under one serving	183(41.6)	95(21.6)	13(3.0)	8(1.8)	67(15.2)
One serving	143 (32.5)	74(16.8)	8(1.8)	3(0.7)	58(13.2)
Two servings	5(1.2)	2(0.5)	1(0.2)	0(0.0)	2(0.5)
Total	440(100)	234(53.2)	36(8.2)	15(3.4)	155(35.2)

4.3.4 Level of vegetable's intake: weekly and servings for participants in the study area

The level of vegetable intake behavior findings was presented regarding the weekly and quantity of servings intake for the study respondents (N=440).

In a typical week, the study findings on the vegetable intake in terms of daily servings for employees (male: n=234, female: n=36) and spouses (Male: n=15, female: n=155). The findings showed that 56.6%(n=249) took fruit for only two days while 2.8% (n=12) did not (see Table) 4.15.

Table 4.15: Proportion of vegetable's intake by status and gender

Variables	N (440)	Employees		Spouses	
		Male	Female	Male	Female
How many days you eat vegetables in a typical week					
None	12(2.8)	6(1.4)	2(0.5)	0(0.0)	4(0.9)
One day	60(13.7)	46(10.5)	1(0.2)	2(0.5)	11(2.5)
Two days	249(56.6)	132(30.0)	25(5.7)	11(2.5)	81(18.4)
Three days	96(21.8)	41(9.3)	5(1.1)	2(0.5)	48(10.9)
Four days	18(4.2)	6(1.4)	2(0.5)	0(0.0)	10(2.3)
Five days	5(1.1)	3(0.7)	1(0.2)	0(0.0)	1(0.2)
Total	440(100)	234(53.2)	36(8.2)	15(3.5)	155(35.3)
Number of vegetables servings on one day					
None	12(2.8)	6(1.4)	2(0.5)	0(0.0)	4(0.9)
Under one serving	240(54.5)	137(31.1)	19(4.3)	11(2.5)	73(16.6)
One serving	88((20.0)	71(16.1)	11(2.5)	2(0.5)	65(14.8)
Two servings	33(11.8)	19(4.3)	4(0.9)	2(0.5)	8(1.8)
Three servings	6(1.3)	1(0.2)	0(0.0)	0(0.0)	5(1.1)
Total	440(100)	234(53.2)	36(8.2)	15(3.5)	155(35.3)

4.2.5 Level of oil intake amongst the study participants in the study

The level of oil intake behavior findings was presented regarding the type of oil, the source of oil, nutrients and lipid content, and smoking temperature label for the users at the time of data collection (N=440). The labels were recorded after the recognition of the type of oil used by the respondent at the time of the interview.

The findings of this study in terms of oil consumption showed that 38.4% of consumed oil was sunseed oil while most of the source oil consumed was palm with 41.6% (N= 440). The findings showed that 93.4% of oil contents were unmarked for trans-fat content. In addition, 74.1% didn't mark the heating smoke as a point level when cooking the oil. The remaining findings were tabulated in Table 4.16.

Table 4.16: Proportion of source of oil, type of oil, nutrient content, and temperature markings for oil usage in the study participants

Variable	Subjects (n)	Rate (%)
What type of oil or fat used for meal		
Sunseed Oil	169	38.4
Mukwano Oil	78	17.7
Golden oil	55	12.5
Dyanas oil	32	7.3
Neo oil	29	6.6
Crystal oil	22	5.0
Palm oil	12	2.7
Sabroso (olive oil)	11	2.5
Canola oil	9	2.0
Butter or ghee	6	1.4
Zahabu oil	4	0.9
Rafael Salgado	4	0.9
Slite oil	3	0.7
Jambo oil	2	0.5
Yonca oil	2	0.5
Viking oil	1	0.2
Karanga oil	1	0.2
Source of oil		
Palm	183	41.6
Sunflower	179	40.7
Soybean	43	9.8
Olive fruits	15	3.4
Rapeseed	9	2.0
Fat of Milk	6	1.4
Soybean and Palm olein	4	0.9
Corn, Palm and blended vegetables	1	0.2
Total fat in 100gr of oil		
Unmarked	191	43.4
99.9g/100g	169	38.4
100g/100g	32	7.3
14g/Serving	29	6.6
14g/15ml (sab)	11	2.5
91g/100ml(Y&J)	4	.9
100g/100g(Rs)	4	.9
Cholesterol content in oil		
Unmarked	228	51.8
Free cholesterol (Marked)	147	33.4
0.0 mg/100g	34	7.7
0 mg	27	6.1
0 mg (Y&J)	4	.9
Monounsaturated fat		
Unmarked	370	84.1
42.7g/100g	32	7.3

Variable	Subjects (n)	Rate (%)
3.08g/14g (cs)	16	3.6
10g/15ml(Sab)	11	2.5
31g/100ml (Y&J)	4	0.9
75g/100g(Rs)	4	0.9
3.80g/14g(Csun)	3	0.7
Polyunsaturated fat		
Unmarked	341	77.5
10.7g/100g	33	7.5
Omega 6(1488mg/serv) & Omega 9(5891mg/serv)	28	6.4
8.54g/14g cs	16	3.6
1.5g/15ml(sab)	11	2.5
51g (Y&J)	4	0.9
10g/100g(Rs)	4	0.9
8.4mg/14g(Csun)	3	0.7
Saturated fat		
Unmarked	343	78.0
46.6g/100g	32	7.3
6g/serv	27	6.1
2.38g/14g (cs)	15	3.4
2g/15ml (sab)	11	2.5
15g/100g(Rs)	5	1.1
9g (Y&J)	4	0.9
1.8g/14g Csun)	3	0.7
Hydrogenated oil		
Unmarked	431	98.0
Marked	9	2.0
Trans fat amount		
Unmarked	411	93.4
0 mg/serving	27	6.1
Trans fat free	2	0.5
Heating smoke point		
Unmarked	326	74.1
Not overheat	114	25.9

4.3.6 Physical activities for the study participants based on frequency, duration, and intensity of energy expenditure

The analysis results for physical activities were processed regarding the vigorous and moderate intensity spent while at work (n=270). Sport, fitness, and leisure for recreational activities, going to and from places by feet or by bicycles for study participants were also considered for the entire study participants (n=440).

The relationship between physical activities with vigorous and moderate intensity spent at work, and gender was not significant. Almost 98.5% of the employees didn't

do vigorous-intensity physical activity for at least 10min in a typical week (n=270) as shown in Table 4.17.

Table 4.17: Bivariate analysis of physical activity with vigorous and moderate intensity spent at work by gender

Variable	n(270)	Male	Female	Statistical test $X^2(df);P$
Work vigorous intensity at least 10min				
Yes	4(1.5)	4(1.5)	0(0.0)	0.625(1);.429
No	266(98.5)	230(85.2)	36(13.3)	
Total	270(100)	234(86.7)	36(13.3)	
How many days you do vigorous intensity work a week				
None	266(98.5)	230(85.2)	36(13.3)	0.625(3);.891
One day	1(0.04)	1(0.4)	0(0.0)	
Two days	1(0.4)	1(0.4)	0(0.0)	
Three days	0(0.0)	0(0.0)	0(0.0)	
Four days	2(0.7)	2(0.7)	0(0.0)	
Total	270(100)	234(86.7)	36(13.3)	
How much time(Min) you spend do Vigorous-intensity a day				
None	266(98.5)	230(85.2)	36(13.3)	0.625(2);.732
10-20 Min	2(0.7)	2(0.7)	0(0.0)	
21-30 Min	0(0.0)	0(0.0)	0(0.0)	
31-40 Min	0(0.0)	0(0.0)	0(0.0)	
41-50 Min	0(0.0)	0(0.0)	0(0.0)	
51-60 Min	2(0.7)	2(0.7)	0(0.0)	
Total	270(100)	234(86.7)	36(13.3)	
Your work involves moderate-intensity activity with breathing increase at least 10min				
Yes	34(12.6)	31(11.5)	3(1.1)	0.685(1);.408
No	236(87.4)	203(75.2)	33(12.2)	
Total	270(100)	234(86.7)	36(13.3)	
How many days do u do moderate-intensity activity at work				
None	236(87.4)	203(75.2)	33(12.2)	
One day	5(1.9)	5(1.9)	0(0.0)	
Two days	5(1.9)	4(1.5)	1(0.4)	3.272(5);.658
Three days	2(0.7)	2(0.7)	0(0.0)	
Four days	11(4.1)	9(3.3)	2(0.7)	
Five days	11(4.1)	11(4.1)	0(0.0)	
Total	270(100)	234(86.7)	36(13.3)	
How much time(Min) you spend do Moderate-intensity a day at work				
None	236(87.4)	203(75.2)	33(12.2)	1.488(5);.914
10-20Min	11(4.1)	10(3.7)	1(0.4)	
21-30Min	15(5.5)	13(4.8)	2(0.7)	
31-40Min	3(1.1)	3(1.1)	0(0.0)	
41-50Min	2(0.7)	2(0.7)	0(0.0)	
51-60Min	3(1.1)	3(1.1)	0(0.0)	
Total	270(100)	234(86.7)	(36(13.3)	

The results of gender-based relationship with physical activities: going to and from places on feet or bicycle showed that 9.1% of males walked or used bicycles for at least 10 min to get or from places. Whereas it was only 3.9% of females who did so with p=0.02. Other variables were not significant. This relationship is tabulated in Table 4.18.

Table 4.18: Bivariate analysis of physical activity: going to and from places on feet or bicycle by gender

Variable	N (440)	Male	Female	Statistical test X^2(df);*P*
Do walk or use bicycle for at least 10min to get to or from places				
Yes	57(13)	40(9.1)	17(3.9)	4.919(1);.027
No	383(87)	209(47.5)	174(39.5)	
Total	440(100)	249(56.6)	191(43.4)	
How many days you walk or bicycle at least 10Min continuously to get to or from places				
None	383(87)	209(47.5)	174(39.5)	6.546(6);.365
One day	1(0.2)	1(0.2)	0(0.0)	
Two days	8(1.8)	5(1.1)	3(0.7)	
Three days	4(1.0)	2(0.5)	2(0.5)	
Four days	7(1.6)	5(1.1)	2(0.5)	
Five days	25(5.7)	19(4.3)	6(1.4)	
Six days	12(2.7)	8(1.8)	4(0.9)	
Total	440(100)	249(56.6)	191(43.4)	
How much time(Min) spend walking or bicycling to travel on a typical day				
None	383(87)	209(47.5)	174(39.5)	6.774(4);.148
10-30	28(6.3)	20(4.5)	8(1.8)	
31-60	25(5.6)	16(3.6)	9(2.0)	
61-90	1(0.2)	1(0.2)	0(0.0)	
91-120	3(0.7)	3(0.7)	0(0.0)	
Total	440(100)	249(56.6)	191(43.4)	

The analysis results of the relationship between gender and physical activity with vigorous intensity spent on sport, fitness, and leisure for recreation were conducted. The results showed a significant relationship between doing the sport, the number of days in a typical week, and the time in minutes spent doing physical activity. Male

respondents were more than female respondents for recreational physical activity with $p<0.05$. These results were tabulated in Table 4.19.

Table 4.19: Bivariate analysis of physical activity with vigorous intensity spent by sport, fitness, and leisure for recreational by gender

Variable	**N(440)**	**Male**	**Female**	**Statistical test**
	n(%)	**n(%)**	**n(%)**	X^2(df);*P*
Vigorous-intensity sport, fitness, recreational or leisure with large increase of breathing				
Yes	30(6.8)	25(5.7)	5(1.1)	9.376(1);.002
No	410(93.2)	224(50.9)	186(42.3)	
Total	440(100)	274(56.6)	191(43.4)	
How many days you do vigorous intensity sport, fitness, leisure for recreational				
None	410(93.2)	224(50.9)	186(42.3)	10.405(2);.034
One day	23(5.2)	18(4.1)	5(1.1)	
Two days	5(1.1)	5(1.1)	0(0.0)	
Three days	1(0.2)	1(0.2)	0(0.0)	
Four days	1(0.2)	1(0.2)	0(0.0)	
Total	440(100)	274(56.6)	191(43.4)	
How much time(Min) you spend do Vigorous-intensity a day for sport, fitness, leisure				
None	410(93.2)	224(50.9)	186(42.3)	9.160(3);.021
10-30	14(3.2)	12(2.7)	2(0.5)	
31-60	14(3.2)	11(2.5)	3(0.7)	
61-90	2(0.5)	2(0.5)	0(0.0)	
Total	440(100)	274(56.6)	191(43.4)	

The analysis results of the relationship between gender and physical activity with moderate intensity spent on sport, fitness, and leisure for recreation were conducted. The results showed a non-significant relationship between doing the sport, the number of days in a typical week, and the time in minutes spent doing physical activity. These results were tabulated in Table 4.20.

Table 4.20: Proportion of physical activity with moderate intensity spent by sport, fitness, and leisure for recreational by gender

Variable	N(440)	Male	Female	Statistical test
	n(%)	n(%)	n(%)	X^2(df); *P*
You do moderate intensity sports, fitness, leisure all recreational at least 10 min				
Yes	66(15.0)	42(9.5)	24(5.5)	1.569(1);.210
No	374(85.0)	207(47.0)	167(38.0)	
Total	440(100)	249(56.5)	191(43.5)	
How many days you do moderate intensity sport, fitness; leisure for recreational				
None	374(85.0)	207(47.0)	167(38.0)	8.947(df);.111
One day	24(5.5)	11(2.5)	13(3.0)	
Two days	21(4.8)	13(3.0)	8(1.8)	
Three days	13(3.0)	11(2.5)	2(0.5)	
Four days	7(1.6)	6(1.4)	1(0.2)	
Five days	1(0.2)	1(0.2)	0(0.0)	
Total	440(100)	249(56.5)	191(43.5)	
How much time (Min) you spend do Moderate intensity a day for sport, fitness, leisure				
None	374(85.0)	207(47.0)	167(38.0)	9.260(4);.05
10-30	24(5.5)	10(2.3)	14(3.2)	
31-60	36(8.1)	27(6.1)	9(2.0)	
61-90	5(1.1)	4(0.9)	1(0.2)	
91-120	1(0.2)	1(0.2)	0(0.0)	
Total	440(100)	249(56.5)	191(43.5)	

The analysis results for the levels of metabolic equivalent for the task (MET) were processed and presented in Table 4.21. The classification of the MET followed the number of MET expenditures. Given that one MET equals the quantity of energy spent while sitting. This implies consumption of approximately 3.5 milliliters of oxygen per kg of body weight per minute. The classification of weekly energy expenditure was based on the intensity of physical activity executed per week. Therefore, The low-intensity physical activity (<4MET), moderate-intensity physical activity (MET=4), and high-intensity physical activity (MET=8) per minute.

The energy>= 1500MET was classified as high weekly energy expenditure. The energy between 600-1500 MET was classified as moderate weekly energy expenditure. The energy <600 MET was classified as low weekly energy expenditure. The findings showed the level of energy expenditure with a 95% confidential interval, where 1.8% of the respondents were classified as having high energy expenditure >=1500 MET per week. (N=440), (Table 4.21).

Table 4.21: Levels of metabolic equivalent of study participants (MET) by age, location, occupation status and gender

Metabolic equivalence classification 8Met/Min Vigorous intensity PA: >=1500MET/WEEK, 4MET moderate intensity PA for MET/min/Week:600-1500MET/week, Low MET:<600MET/Week				
Variables	**N (%)**	**Low % (CI, 95%)**	**Moderate % (CI, 95%)**	**High % (CI, 95%)**
Overall	440(100%)	89.8(86.8-92.7)	8.4(5.6-11.1)	1.8(0.5,3.1)
Age category				
<40 Years	157(35.7)	30.5(23.3-37.7)	3.9(0.8-6.9)	1.4(-0.4-3.2)
40-50 Years	151(34.3)	31.8(24.3-39.2)	2.5(0.0-4.9)	0.0(0.0-0.0)
>50Years	132(30.0)	28.0(20.3-35.6)	2.0(-0.3-4.3)	0.0(0.0-0.0)
Field location				
Kicukiro	259(58.9)	56.2(50.1-62.2)	1.8(0.1-3.4)	0.7(-0.3-1.7)
Rubavu	181(41.1)	33.9(27.0-40.8)	6.6(2.9-10.2)	0.7(-0.5-1.9)
Status of the participants				
Employees	270(61.4)	53.0(47.0-58.9)	7.0(3.9-10.0)	1.4(0.0-2.8)
Spouses	170(38.6)	37.3(30.0-44.5)	1.4(-0.3-3.1)	0.0(0.0-0.0)
Gender				
Male	249(56.6)	48.9(42.6-55.1)	6.4(3.3-9.4)	1.4(-0.06-2.8)
Female	191(43.4)	41.4(34.4-48.3)	2.0(0.0-3.9)	0.0(0.0-0.0)

4.3.7 Sitting time /sedentarity for the study participants

The analysis results were processed by classifying the level of sedentarity as a prominent risk factor for cardiovascular diseases. The low level of sedentarity was

classified as a low level of sitting time inferior to 6 hours, the moderate sedentarity was classified as a moderate level of sitting time between 6 to 10 hours. The high sedentary was classified as long sitting time >10hours. The results showed that 6.6%(n=29) of respondents were classified as having a high level of sedentarity while 38.2%(n=168) were classified as having metabolic syndrome (Table 4.22).

Table 4.22: Prevalence of sedentary and metabolic syndrome

Variables	Frequency	Percentage%
Sedentarity levels		
Short sitting time (<6hrs)	316	71.8
Moderate sitting time (6-10hrs)	95	21.6
Long sitting time (>10hrs)	29	6.6
Metabolic Syndrome (MetS)		
No MetS	272	61.8
With MetS	168	38.2

The analysis results showed the association of sedentarity levels and metabolic syndrome with crude odd ratios (95%CI) N=440. The findings showed the higher the sedentarity levels, the higher the likelihood to develop the metabolic syndrome (Table 4.23).

Table 4.23: Association of sitting time with metabolic diseases

Variables	df	OR (95%CI)	P-value
Sedentary levels			
Short sitting time (<6hrs)	2	Reference	
Moderate sitting time (6-10hrs)	1	2.686(1.42-5.06)	0.002
Long sitting time (>10hrs)	1	8.196(2.07-32.3)	0.003
BMI	1	1.207(1.11-1.30)	<0.001

The analysis results showed that after the classification of the sedentary levels and evaluation of their association to other cardiovascular risk factors such as metabolic syndrome; the moderate and high levels of sedentary were three-fold associated with cardiovascular disease elevated risk (Table 4.24).

Table 4.24: Association of sitting time with cardiovascular diseases risk (FGRS)

Variables	df	OR (95%CI)	*P*-value
Sedentary levels			
Short sitting time (<6hrs)	2	Reference	
Moderate sitting time (6-10hrs)	1	3.238(2.238-6.050)	<0.001
Long sitting time (>10hrs)	1	3.772(1.718-8.285)	0.001

Bivariate analysis results portrayed the association between sitting time and components of metabolic syndrome which were all significantly associated with a high level of sedentary (Table 4.25).

Table 4.25: Association of sitting time with metabolic syndrome components

Variables	N(440)	Sedentary levels			Statistical test X^2(df);*P*
Impaired Fasting Glycose for Metabolic Syndrome Classification FPG >= 100mg/dl(5.6mmol/l) or treatment (Rx)		Short sitting time period	Moderate sitting time period	High sitting time period	
IFG	174(39.6)	62(14.1)	83(18.9)	29(6.6)	187.691(2);.001
Normal	266(60.4)	254(57.7)	12(2.7)	0(0.0)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
Triglyceridemia for Metabolic Syndrome Classification (High level Triglyceride>=150mg/dl(1.7mmo/dl) or ttt for high TG					
High Triglycerides>=150mg/dl	157(35.7)	85(19.3)	46(10.5)	26(5.9)	54.151(2);.001
Normal<150mg/dl	283(64.3)	231(52.5)	49(11.1)	3(0.7)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
High Blood Pressure for Metabolic Syndrome Criteria HBP>= 130/85mmgh or Treatment of HBP+					
HLBP for MetS>=130/85mmgh	254(57.7)	162(36.8)	70(15.9)	22(5.0)	19.227(2);.001
LLBP for MetS<130/85mmgh	186(67)	154(35.0)	25(5.7)	7(1.6)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
HDL for Metabolic Syndrome Classification (low level men <40mg/dl(1mmol/l), low level women <50mg/dl(1.3mmol/l) or ttt for low HDL					
Normal	267(60.6)	206(46.8)	53(12.0)	8(1.8)	16.958(2);.002
Abnormal (Low levels)	173(39.3)	110(25.0)	42(9.5)	21(4.8)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
Waist Circumference					
Central Obesity: F>88Cm,M>102Cm	73(37)	96(21.8)	49(11.1)	18(4.1)	22.411(2);.002
Normal: F<88Cm, M<102Cm	277(63)	220(50)	46(10.5)	11(2.5)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	

The findings result portrayed the bivariate analysis of the association of sitting time with other factors of cardiovascular diseases risk. Only gender was not linked to sedentarity levels (N=440), (Table 4.26).

Table 4.26: Bivariate analysis of sitting time and other factors to cardiovascular diseases risk

Variables	N(440)	Sedentary levels			Statistical test
					X^2(df);*P*
FGRS		Short sitting time	Moderate sitting time	High sitting time	
Low risk (<10%)	328(74.6)	261(59.3)	61(13.9)	6(1.4)	105.552(6);.001
2nd level risk (10-20%)	60(13.7)	35(8.0)	19(4.3)	6(1.4)	
3rd level risk (20-30%)	28(6.3)	14(3.2)	9(2.0)	5(1.1)	
4th level risk (30-40%)	24(5.5)	6(1.4)	6(1.4)	12(2.7)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
Gender					
Male	249(56.6)	182(41.4)	52(11.8)	15(3.4)	.542(2);.7
Female	191(43.5)	134(30.5)	43(9.8)	14(3.2)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
Age Group					
<40 years	249(35.6)	123(28.0)	29(6.6)	5(1.1)	11.292(4);.02
40-50Years	132(30)	84(19.1)	38(8.6)	10(2.3)	
>50 Years	151(34.4)	109(24.8)	28(6.4)	14(3.2)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	
Status of participant					22.411(2);.001
Employees	270(61.4)	206(46.8)	50(11.4)	14(3.2)	
Spouses	170(38.6)	110(25.0)	45(10.2)	15(3.4)	
Total	440(100%)	316(71.8)	95(21.6)	(29(6.6)	
Waist Circumference					
Central Obesity: F>88Cm,M>102Cm	73(37)	96(21.8)	49(11.1)	18(4.1)	7.102(2);.02
Normal: F<88Cm, M<102Cm	277(63)	220(50)	46(10.5)	11(2.5)	
Total	440(100%)	316(71.8)	95(21.6)	29(6.6)	

4.4 Proportion of working condition factors associated with cardiovascular diseases among workers of Kicukiro soft drink and Rubavu Brewery plant

4.4.1 Workplace condition factors for workers in the study area

4.4.1.1 Organizational hazard (Shift workers and Night workers)

The analysis findings presented the shift levels including the regular night shift. The regular shift work (n=125) with (N=270) of the total employees. The regular night shift work (n=118), with (N=270) of the total employees. The findings showed that the higher the age, the lower the regular night shift work (Table 4.27).

Table 4.27: Distribution of regular shift work and regular night shift

Variables	Frequency	Percentage %
Regularly Doing shift work in current position		
Yes	125	46.3
No	145	53.7
Regularly doing Night shift Work		
Yes	118	43.7
No	152	56.3
Regularly doing night shift work		
Gender		
Male Yes	115	97.4
Female Yes	3	2.5
Age		
<40 years	48	40.6
40-50 Years	38	32.2
>50Years	32	27.1

The analysis findings painted the bivariate analysis on the association of work occupation with night shift. The technical (n=76) and logistic (n=17) departments were on regular night shifts with $p<0.001$, (N=270) of the total employees (Table 4.28).

Table 4.28: Bivariate analysis of regular night shift and occupation

Variables	Frequency			Test X^2(df),*P*
Occupation & working Dpt for Employees	Regularly doing night Shift work		Total	
	Yes	No		
Commerce	13(4.8)	43(15.9)	56(20.7)	58.17(6);<.001
Marketing	1(0.3)	10(3.7)	11(4.0)	
Human Resource	2(0.7)	14(5.1)	16(5.9)	
Logistic	17(6.2)	11(4.0)	28(10.3)	
Finance	1(0.3)	18(6.6)	19(7.0)	
Technical Direction	76(28.1)	41(15.1)	117(43.3)	
General Management	8(2.9)	15(5.5)	23(8.5)	
Total	118(43.7)	152(56.2)	270(100)	

The analysis findings painted the bivariate analysis on the association of night shift work(n=119) and elevated cardiovascular diseases risk>10% by the Framingham general risk score model (N=270), (Table 4.29).

Table 4.29: Bivariate analysis of night shift and cardiovascular diseases risk (FGRS model)

	n(270)	**Low level risk <10%**	**Elevated risk (10-40%)**	**Statistical Test.**X^2(df);*P*
Regular doing night shift				
Yes	119(44.1)	94(34.8)	25(9.3)	3.92(1);.03
No	151(55.9)	103(38.1)	48(17.8)	
Total	270(100%)	197(73.0)	73(27.0)	

4.4.1.2 Physical hazards for industrial worker status participants

The analysis findings described the prevalence of physical hazard conditions. The cold room (n=31), vibration exposure >1.15m/s for WBV and 2.5m/s for HAV(n=66), much noise exposure>85dB(n=139), radiation(n=48) were the physical hazards with (N=270) for total workers. The results showed that 11.5% of employees worked in cold chambers (Cold rooms), 24.4% of employees were exposed to a high level of vibration at work, 51.5% were exposed to a high level of

noise at work, and 17.8% of employees were exposed to whether gamma or X rays (Table 4.30).

Table 4.30: Prevalence of physical working hazards concerning working conditions of the study participants

Variables	Frequency	Percentage (%)
Regular working in cold chamber or refrigeration		
Yes(-4ºC)	31	11.5
No	239	88.5
Days working in cold chambers		
None	239	88.5
A day a week	13	4.8
2days a week	12	4.4
3 days a week	1	.4
5days a week and more	5	1.9
Much vibration exposure at work		
Yes(>1.15m/s)	66	24.4
No	204	75.6
Much noise exposure at work		
Yes(>85dB)	139	51.5
No	131	48.5
Gamma ray exposure at work		
Yes	48	17.8
No	222	82.2
X ray exposure at work		
Yes	48	17.8
No	222	82.2

The analysis findings portrayed bivariate analysis which showed the association between physical hazards and gender (Table 4.31), (N=270). Physical hazards and gender relationships were insignificant, with 11.5% of male-dominated and 0.4% of female, p=0.07. The exposure to much vibration (WBV>1.15m/s for 8 hours| HAV>2.5m/s) was 23.3% for males and 1.1% for females with a significant p=0.01. Males were more exposed to noise (>85dB) with 49% than females with 2.5% with a significant p<0.001. In addition, 17.8% of males were exposed to gamma and X rays with 17.8% with p=0.003. This was due to few women in the industrial working environment (Table 4.31).

Table 4.31: Bivariate analysis between physical hazard factors to cardiovascular diseases risk and gender

Variables	n(270)%	Male	Female	Statistical test X^2(df);*P*
Regular working in cold chamber or refrigeration				
Yes(-4^{o}C)	31(11.5)	30(11.1)	1(0.4)	3.096(1);.07
No	239(88.5)	204(75.6)	35(13.0)	
Total	270(100%)	234(86.7)	36(13.3)	
Days are you working in cold chambers				
None	239(88.5)	204(75.6)	35(13.0)	
A day a Week	13(4.8)	13(4.8)	0(0.0)	3.538(4);.4
2days a Week	12(4.4)	11(4.1)	1(0.4)	
3days a week	1(0.4)	1(0.4)	0(0.0)	
4days a week	0(0.0)	0(0.0)	0(0.0)	
5days& more	5(1.9)	5(1.9)	0(0.0)	
Total	270(100)	234(86.7)	36(13.3)	
Much vibration Exposure at work				
Yes(>1.15m/s)	66(24.4)	63(23.3)	3(1.1)	5.838(1);.016
No	204(75.6)	171(63.3)	33(12.2)	
Total	270(100)	234(86.7)	36(13.3)	
Much Noise exposure at work				
Yes(>85Db)	139(51.5)	132(49)	7(2.5)	17.069(1);.001
No	131(48.5)	102(37.7)	29(10.8)	
Total	270(100%)	234(86.7)	36(13.3)	
Gama Rays Radiation exposure				
Yes	48(17.8)	48(17.8)	0(0.0)	8.981(1);.003
No	222(82.2)	186(68.9)	36(13.3)	
Total	270(100%)	234(86.7)	36(13.3)	
X rays radiation Exposure				
Yes	48(17.8)	48(17.8)	0(0.0)	8.981(1);.003
No	222(82.2)	186(68.9)	36(13.3)	
Total	270(100%)	234(86.7)	36(13.3)	

A bivariate analysis of physical hazards and age (Table 4.32), indicated for total workers(N=270). Around 11.5%(n=31) of workers were working in cold chambers (Cold rooms: -4oC), and 25.9%(n=66) of workers were exposed to high-level

vibration (WBV>1.15m/s, 8 hours). Around 51.5%(n=139) of workers were exposed to much noise(>85dB) and 17.8% of workers were exposed to radiation. This was not significantly related to age structure (Table 4.32).

Table 4.32: Bivariate analysis of physical hazards factors to cardiovascular diseases and age

Variables	N(270)	<40 Years	40-50Years	>50Years	Statistical test, X^2(df); *P*
Regular working in cold chamber or refrigeration					
Yes	31(11.5)	11(4.1)	11(4.1)	9(3.3)	0.622(2);.733
No	239(88.5)	89(33.0)	69(25.6)	81(30.0)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	
Days are you affected in cold chambers					
None	239(88.5)	89(33.0)	69(25.6)	81(30.0)	8.827(8);.357
A day a Week	13(4.9)	4(1.5)	5(1.9)	4(1.5)	
2days a Week	12(4.4)	7(2.6)	2(0.7)	3(1.1)	
3days a week	1(0.4)	0(0.0)	1(0.4)	0(0.0)	
4days a week	0(0.0)	0(0.0)	0(0.0)	0(0.0)	
5days& more	5(1.8)	0(0.0)	3(1.1)	2(0.7)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	
Much vibration Exposure at work					
Yes	66(25.9)	26(11.1)	19(7.0)	21(7.8)	0.212(2);.899
No	204(74.1)	74(25.9)	61(22.6)	69(25.6)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	
Much Noise exposure at work					
Yes	139(51.5)	53(19.6)	45(16.7)	41(15.2)	2.086(2);.352
No	131(48.5)	47(17.4)	35(12.9)	49(18.1)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	
Gama Rays Radiation exposure					
Yes	48(17.8)	20(7.4)	13(4.8)	15(5.6)	0.542(2);.763
No	222(82.2)	80(29.6)	67(24.8)	75(27.8)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	
X rays radiation Exposure					
Yes	48(17.8)	20(7.4)	13(4.8)	15(5.6)	0.542(2);.763
No	222(82.2)	80(29.6)	67(24.8)	75(27.8)	
Total	270(100)	100(37.0)	80(29.6)	90(33.3)	

The analysis results portrayed bivariate analysis which showed the association between physical hazards and departments. The results showed that the technical department presented more exposed people and all the physical hazards were

significantly linked with the technical department except the days working in cold rooms (N=270). The technical department dominated other departments in all studied physical hazards aspects. This was due to the fact that a high proportion of technical department workers around 10.4% were exposed due to regular working in the cold chamber (Cold room with t<-4oC) or refrigeration p<0.001. Thus, 19.6% were exposed to vibration with p<0.001, 26.7% were exposed to noise with p<0.001, and 17% were exposed to radiation with p<0.001 (Table 4.33).

Table 4.33: Analysis of physical hazards factors by Workstations

Variables	N(270)%	Sales	Marketing	HR	Logistic	Finance	Techn	General M	Statistical test X^2(df);*P*
Regular working in cold chamber or refrigeration									
Yes	31(11.5)	0(0.0)	1(0.4)	0(0.0)	1(0.4)	0(0.0)	28(10.4)	1(0.4)	32.585(6);<.001
No	239(88.5)	56(20.7)	10(3.7)	16(5.9)	27(10)	19(7.0)	89(33.0)	22(8.1)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	
Days are you working in cold chambers									
None	239(88.5)	56(20.7)	10(3.7)	16(5.9)	27(10.0)	19(7.0)	89(33.0)	22(8.1)	35.146(24);.06
day a Week	13(4.8)	0(0.0)	0(0.0)	0(0.0)	1(0.4)	0(0.0)	12(4.4)	0(0.0)	
2days a Week	12(4.5)	0(0.0)	1(0.4)	0(0.0)	0(0.0)	0(0.0)	10(3.7)	1(0.4)	
3days a week	1(0.4)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	1(0.4)	0(0.0)	
4days a week	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	
5days & more	5(1.9	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	5(1.9)	0(0.0)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	
Much vibration Exposure at work									
Yes	66(24.5)	5(1.9)	0(0.0)	0(0.0)	5(1.9)	0(0.0)	53(19.6)	3(1.1)	52.010(6);<.001
No	204(75.5)	51(18.9)	11(4.1)	16(45.9)	23(8.5)	19(7.0)	64(23.7)	20(7.4)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	
Much Noise exposure at work									
Yes	87(32.3)	4(1.5)	0(0.0)	0(0.0)	8(3.0)	0(0.0)	72(26.7)	3(1.1)	88.084(6);<.001
No	183(67.7)	52(19.3)	11(4.1)	16(5.9)	20(7.4)	19(7.0)	45(16.7)	20(7.4)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	
Gama Rays Radiation exposure									
Yes	48(17.8)	0(0.0)	1(0.4)	0(0.0)	1(0.4)	0(0.0)	46(17.0)	0(0.0)	66.214(6);<.001
No	222(82.2)	56(20.7)	10(3.7)	16(5.9)	27(10.0)	19(7.0)	71(26.3)	23(8.5)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	
X rays radiation Exposure									
Yes	48(17.8)	0(0.0)	1(0.4)	0(0.0)	1(0.4)	0(0.0)	46(17.0)	0(0.0)	66.214(6);<.001
No	222(82.2)	56(20.7)	10(3.7)	16(5.9)	27(10.0)	19(7.0)	71(26.3)	23(8.5)	
Total	270(100)	56(20.7)	11(4.1)	16(5.9)	28(10.4)	19(7.0)	117(43.3)	23(8.5)	

4.4.1.3 Psychological hazards amongst the workers

4.4.1.3.1 Stress levels

The analysis results portrayed the prevalence of role and performance-based stress levels (N=270). The study participants were comfortable concerning clear objectives. The mean stress level of the job process was very low for Kicukiro and Rubavu study areas, respectively. However, the mean stress level was high for unclear duties and unachievable deadline items. The stress levels in two manufacturing plants were classified based on the Likert scale levels as shown in Table 4.34 below.

Table 4.34: Role and performance-based stress level amongst workers in the study area

Variables	Subjects (n)	Rate (%)	Mean	Interpretation
Likert scale stress score				
Level 4			3.40 -5.0	Very high
Level 3			2.60-3.40	High
Level 2			1.80-2.60	Low
Level 1			1.0 -1.80	Very Low
Unclear objectives				
Kicukiro				
Never	79	49.4	1.62	Very low
Seldom	65	40.6		
Sometimes	14	8.8		
Often	1	0.6		
Always	1	0.6		
Total	160	100		
Rubavu				
Never	54	49.1	1.58	Very low
Seldom	48	43.6		
Sometimes	8	7.3		
Often	0	0.0		
Always	0	0.0		
Total	110	100		
Unclear duties				
Kicukiro				
Never	1	0.6	4.11	Very high
Seldom	3	1.9		
Sometimes	3	1.9		
Often	123	76.9		
Always	30	18.8		
Total	160	100		
Rubavu				
Never	0	0.0	4.1	Very high
Seldom	1	0.9		
Sometimes	5	4.5		
Often	82	74.5		
Always	22	20.0		

Variable	Subjects (n)	Rate (%)	Mean	Interpretation
Total	110	100		
Don't know job process				
Kicukiro				
Never	4	2.5	2.08	Low
Seldom	143	89.4		
Sometimes	10	6.2		
Often	2	1.2		
Always	1	0.6		
Total	160	100		
Rubavu				
Never	1	0.9	2.0	Low
Seldom	108	98.2		
Sometimes	1	0.9		
Often	0	0.0		
Always	0	0.0		
Total	110	100		

The analysis results presented the prevalence of pressure and workload-based stress levels (N=270). The stress levels in two manufacturing plants (Kicukiro and Rubavu) were classified based on the Likert scale levels. The four items of pressure-based stress, among others working very intense, task negligence, long pressure hours, and being unable to take sufficient breaks were all very high. The mean stress level indicated that workplace stress was very high for Kigali and Rubavu employees The findings are tabulated in Table 4.35 below.

Table 4.35: Pressure or workload-based stress amongst workers participants

Variables	Subjects (n)	Rate (%)	Mean	Interpretation
Unachievable deadline				
Kicukiro				
Never	0	0.0	3.4	Very high
Seldom	18	11.2		
Sometimes	80	50.0		
Often	36	22.5		
Always	26	16.2		
Total	160	100		
Rubavu				
Never	0	0.0	3.9	Very high
Seldom	1	0.9		
Sometimes	49	44.5		
Often	20	18.2		
Always	40	36.4		
Total	110	100		
Having to work very intensively				
Kicukiro				
Never	0	0.0	4.0	Very high
Seldom	4	2.5		
Sometimes	26	16.2		
Often	81	50.6		

Variable	Subjects (n)	Rate (%)	Mean	Interpretation
Always	49	30.6		
Total	160	100		
Rubavu				
Never	0	0.0	4.0	Very high
Seldom	4	3.6		
Sometimes	24	21.8		
Often	41	37.3		
Always	41	37.3		
Total	110	100		
Having negligence of some task because of much to do				
Kicukiro				
Never	4	2.5	3.2	Very high
Seldom	32	20.0		
Sometimes	71	44.4		
Often	25	15.6		
Always	28	17.5		
Total	160	100		
Rubavu				
Never	0	0.0	3.5	Very high
Seldom	22	20.0		
Sometimes	44	40.0		
Often	4	3.6		
Always	40	36.4		
Total	110	100		
Pressured to work long hours				
Kicukiro				
Never	2	1.2	3.8	Very high
Seldom	6	3.8		
Sometimes	52	32.5		
Often	47	29.4		
Always	53	33.1		
Total	160	100		
Rubavu				
Never	0	0.0	3.9	Very high
Seldom	1	0.9		
Sometimes	43	39.1		
Often	28	25.5		
Always	38	34.5		
Total	110	100		
Being Unable to take sufficient Break				
Kicukiro				
Never	0	0.0	3.6	Very high
Seldom	9	5.6		
Sometimes	82	51.2		
Often	26	16.2		
Always	43	26.9		
Total	160	100		
Rubavu				
Never	0	0.0	3.7	Very high
Seldom	5	4.5		
Sometimes	58	52.7		Very high
Often	11	10.0		
Always	36	32.7		
Total	110	100		

The analysis results presented the prevalence of workplace behavior-based stress levels (N=270). The stress levels in two manufacturing plants (Kicukiro and Rubavu) were classified based on the Likert scale levels. Two behaviors related to stress items: bullying at the workplace was very low for all employees. This means that respect between employees was perfect. However, the last three items (no opportunity to question the manager at work, unable to talk to the manager about an annoying thing, unable to get line manager Encouragement) mean stress level was very high. The findings are tabulated in Table 4.36 below.

Table 4.36: Workplace behavior-based stress

Variables	Subjects (n)	Rate (%)	Mean	Interpretation
Being subject to Bullying at Work				
Kicukiro				
Never	113	70.6	1.5	Very low
Seldom	7	4.4		
Sometimes	35	21.9		
Often	4	2.5		
Always	1	0.6		
Total	160	100		
Rubavu				
Never	58	52.7	1.9	Low
Seldom	6	5.5		
Sometimes	45	40.9		
Often	1	0.9		
Always	0	0.0		
Total	110	100		
Don’t receive due respect from colleagues				
Kicukiro				
Strongly Disagree	126	78.8	1.3	Very low
Disagree	26	16.2		
Neutral	3	1.9		
Agree	3	1.9		
Strongly Agree	2	1.2		
Total	160	100		
Rubavu				
Strongly Disagree	95	86.4	1.1	Very low
Disagree	12	10.9		
Neutral	2	1.8		
Agree	1	0.9		
Strongly Agree	0	0.0		
Total	110	100		
No opportunity to question manager at work				
Kicukiro				
Strongly Disagree	1	0.6	3.9	Very high
Disagree	12	7.5		
Neutral	23	14.4		
Agree	87	54.4		
Strongly Agree	37	23.1		
Total	160	100		
Rubavu				
Strongly Disagree	0	0.0	3.9	Very high
Disagree	5	4.5		
Neutral	6	55		
Agree	85	77.3		
Strongly Agree	14	12.7		
Total	110	100		
Unable to talk to manager about annoying thing				
Kicukiro				
Strongly Disagree	2	1.2	3.9	Very high
Variable	**Subjects (n)**	**Rate (%)**	**Mean**	**Interpretation**

Disagree	10	6.2		
Neutral	26	16.2		
Agree	83	51.9		
Strongly Agree	39	24.4		
Total	160	100		
Rubavu				
Strongly Disagree	0	0.0	3.9	Very high
Disagree	2	1.8		
Neutral	10	9.1		
Agree	89	80.9		
Strongly Agree	9	8.2		
Total	110	100		
Unable to get line manager Encouragement				
Kicukiro				
Strongly Disagree	11	6.9	3.6	Very high
Disagree	15	9.4		
Neutral	23	14.4		
Agree	76	47.5		
Strongly Agree	35	21.9		
Total	160	100		
Rubavu				
Strongly Disagree	27	24.5	2.8	High
Disagree	15	13.6		
Neutral	14	12.7		
Agree	51	46.4		
Strongly Agree	3	2.7		
Total	110	100		

4.4.1.3.2 Stress relationship with department, gender, age, and cardiovascular risk by both models

The analysis results presented the bivariate analysis of the association of stress levels prevalence and gender, with a significance of $p<0.05$. The level of stress in the workplace after a Likert scale-based classification, the results showed that the severe form of stress was 30%, moderate stress was 64.4%, and mild stress form was 5.2%. Males highly dominated women in all forms of stress levels with a significant gender relationship $p=0.03$. Stress increased with age where employees with age <40 Years were 5.2% with a severe form of stress, 40-50 years were 8.9% with a severe form of stress, >50 years were 15.9% with a severe form of stress with an age significant relationship $p<0.001$. The findings are tabulated in Table 4.37 below.

Table 4.37: Prevalence of stress and relationship by gender and age among the employees

Variable	Subjects (n)	Rate (%)	
Stress levels categorized according Renis likert,1932			
Mild stress	15	5.6	
Moderate stress	174	64.4	
High level of stress (Severe form)	81	30.0	
Stress levels relationship with gender and age			
Variables	**Subjects(n)**	**Rate (%)**	**Statistical test** X^2(df);*P*
Gender			
Male			
Mild stress	14	5.2	6.47(2);.03
Moderate stress	144	53.3	
High level stress	76	28.1	
Female			
Mild stress	1	0.4	
Moderate stress	30	11.1	
High level stress	5	1.9	
Age			
<40 years			
Mild stress	12	4.4	34.52(4);<.001
Moderate stress	74	27.4	
High level stress	14	5.2	
40-50 Years			
Mild stress	1	0.4	
Moderate stress	55	20.4	
High level stress	24	8.9	
>50Years			
Mild stress	2	0.7	
Moderate stress	45	16.7	
High level stress	43	15.9	

The analysis results portrayed the prevalence and association of stress levels and the organization departments. Three departments presented high levels than others, starting with the technical department with 26.6% in moderate stress and 14% in high stress. Commerce (Sales) with 12.5% in moderate stress, 6.6% in high stress and at last logistic department with 7.0% in moderate stress and 7% in high stress. The high stress-level was more predominant in technical department than the remaining departments (N=270). The findings are presented in Figure 4.3 below.

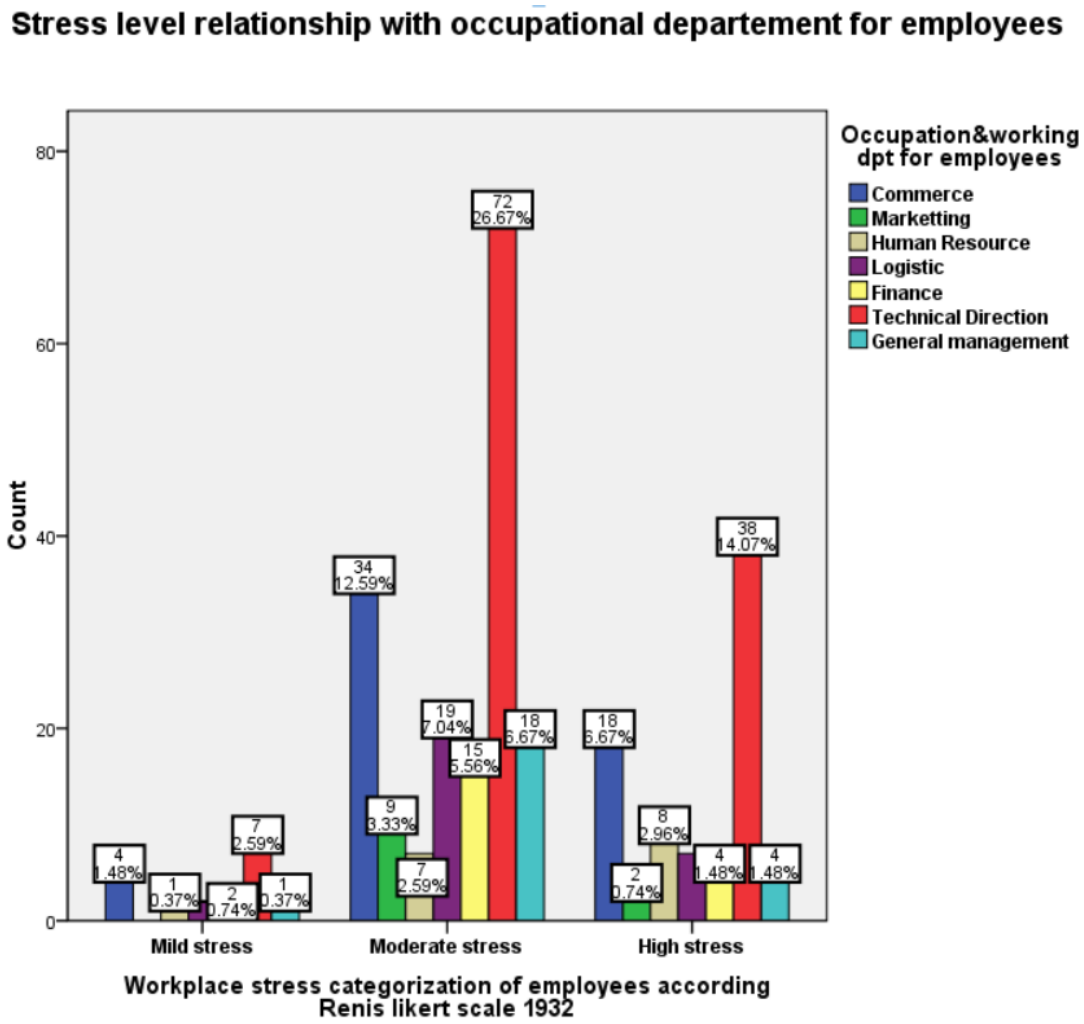

Figure 4.3: Relationship of stress and organizational department

The analysis results portrayed the association of stress levels and cardiovascular diseases with elevated levels>10% by Framingham general risk score model. The results showed that the high-level stress was associated with elevated cardiovascular diseases risk with $p<0.001$, (N=270). The findings are presented in Figure 4.4 below.

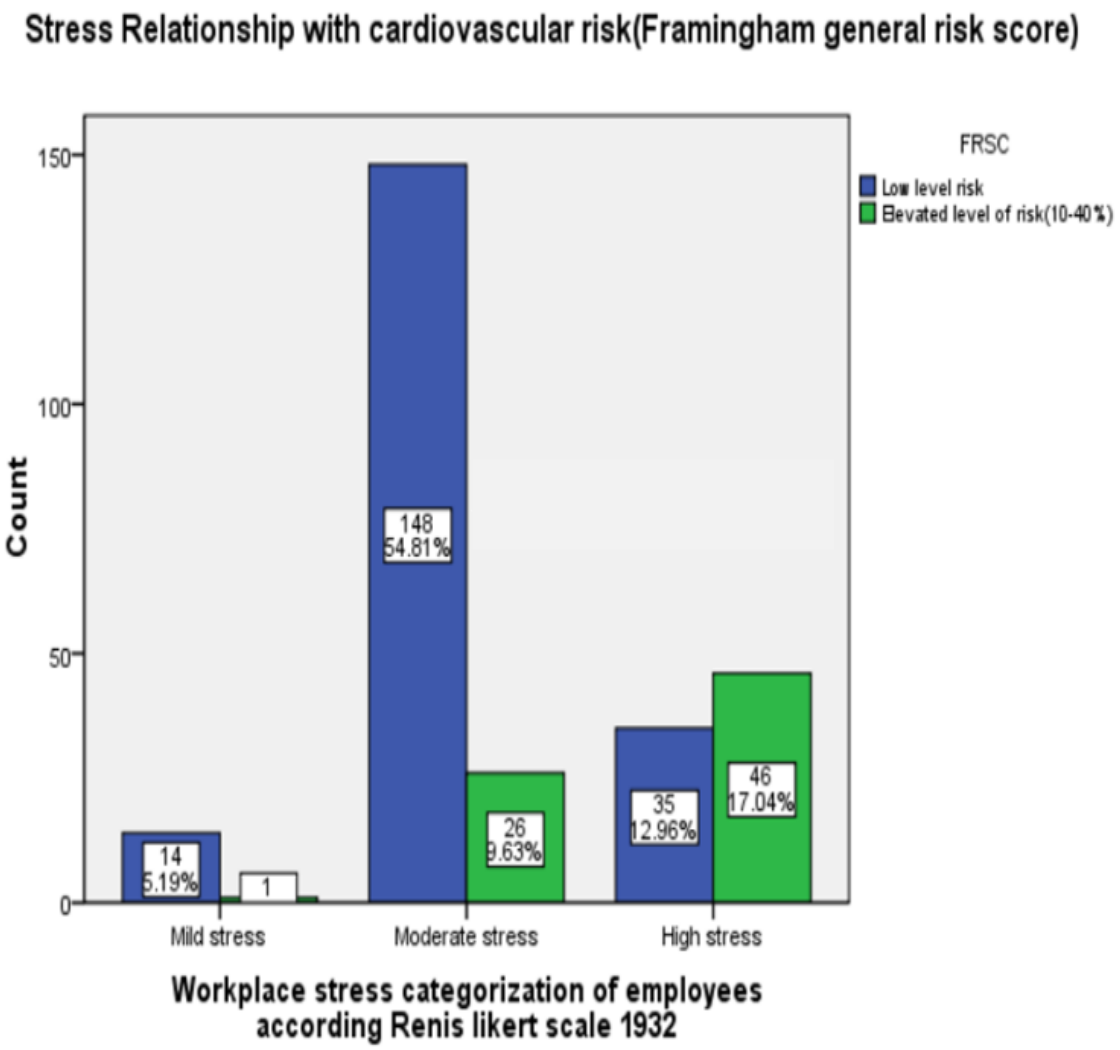

Figure 4.4: Relationship between Stress levels and cardiovascular disease's risk according to Framingham general risk score

The analysis results portrayed the association of stress levels and cardiovascular diseases with elevated levels>10% by the Framingham general risk score model. The results showed that the higher the stress level, the higher the likelihood of association with cardiovascular diseases elevated risk with 95%CI, $p<0.05$. The findings are presented in Table 4.38 below.

Table 4.38: Association between Stress levels and cardiovascular disease's risk by FGRS

Variable	Odds ratio (CI: 95%) reduced Model	*P*-value
Working conditions		
Low stress level	Reference	
Moderate stress level	2.459(0.310-19.513)	0.394
High stress level	18.400(2.308-146.671)	0.006

The analysis results portrayed the association of stress levels and cardiovascular diseases with elevated levels>10% by the WHO/ISH score chart. The results showed no association with each type of stress (N=270). The findings are tabulated in Table 4.39 below.

Table 4.39: Stress levels association with cardiovascular disease's risk by WHO/ISH score chart

Variable	Odds ratio (CI: 95%) reduced Model	*P*-value
Working conditions		
Low stress level	Reference	
Moderate stress level	7.785(-)	0.999
High stress level	1.770(-)	0.998

4.4.1.4 Chemical hazards to workers

The analysis results portrayed the prevalence of 132 chemical hazards in various departments. The analysis was processed regarding the chemical hazards encountered or handled by the employees in their working stations. All those department are: technical department (n=118), logistic(n=28), human resource (n=16), general management(n=23), sales(n=56), (N=270). The technical department handled more chemical hazards than other departments in both beverage manufacturing industries plants. technical department employees encountered and or

handled many more chemical hazards than other departments. Four chemical hazards (SpectrusOX, Cortrol IS 2015, Optiperse PQ517.6, and Continum AT 4505) were more handled or encountered to a level of 84.7% of all technical department employees and 40.7% of all employees. The three hazardous substances lowly handled or encountered were Gentian violet, Safranin, and Spectrus NX 1102, at the level of 3% for all employees. Only the marketing department has never handled nor encountered any chemical hazardous material. The General management, sales, and human resources departments were the least exposed departments to hazardous substances, where dust dominated other chemicals. Technical and logistic departments have encountered or handled more chemical hazards than other departments. The findings are tabulated in Table 4.40 below.

Table 4.40: Distribution of hazardous chemicals by the area of handling/ encountering hazardous substances

Variables	N:270	TD	Logistic	HR	GM	Sales
	n	118	28	16	23	56
	Ex	51826	195	46	12	8
Carbon Monoxide	32(11.9)	28(23.7)	2(7.1)	0(0.0)	0(0.0)	2(3.6)
Carbon Dioxide	86(31.9)	76(64.4)	7(25.0)	0(0.0)	0(0.0)	3(5.4)
Ammonia(NH3)	24(8.9)	23(19.5)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Nitric Acid	28(10.4)	28(23.7)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Kieselguhr(DE/Sio2*nH2O	26(9.6)	25(24.4)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Caustic Soda	48(17.8)	46(39.0)	2(7.1)	0(0.0)	0(0.0)	0(0.0)
Ethanol	10(3.7)	8(6.8)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Methanol	10(3.7)	8(6.8)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Sulfuric acid	20(7.4)	17(14.4)	1(3.6)	2(12.5)	0(0.0)	0(0.0)
Hydrochloric acid(HCL)	9(3.3)	9(7.6)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
VB13	9(3.3)	9(7.6)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
VA4(Super dulac H3PO4,HNO3)	21(7.8)	21(17.8)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
VT5	21(7.8)	21(17.8)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Divo 100	10(3.7	10(8.5)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Gentian violet	3(1.1)	1(0.8)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Safranin	3(1.1)	1(0.8)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Spectrus NX 1102	3(1.1)	3(2.5)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Methyl Orange Indicator (C14H12N3NaO3S)	12(4.4)	12(10.2)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Methyl red sodium salt indicator	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Phenolphthalein indicator	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Bromocresol green indicator	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Eriochrome Black indicator	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Murexide	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Methylene Blue	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
2-Tolidine	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Methyl Violet	15(4.8)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Diethyl ether extra pure	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Toluene extra pure	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Mucasol	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Dichloromethane	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Carbon tetrachloride	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Toluidine	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Methyl isobutyl ketone	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Acetone	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Denatured Alcohol	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Sodium Thiosulfate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ammonia buffer solution	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Variables	**N:270**	**TD**	**Logistic**	**HR**	**GM**	**Sales**
Ethylenediaminetetraaceticacid(EDTA)	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Crown crock corrosion test solution	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Calcium chloride 2-hydrate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Zinc sulfate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ammonium ferrous sulphate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Anhydrous Sodium carbonate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Copper sulphate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Potassium Dichromate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Potassium Chloride	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Copper Chloride	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium Arsenate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Potassium Permanganate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ammonium Chloride	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Hexamethylenetetramine	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)

Hydraziniumsulfate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Tri-potassium citrate monohydrate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium metabisulfite	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ammonium Molybdate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ammonium heptaMolybdate tetrahydrate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Potassium di-hydrogen orthophosphate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Potassium hydrogen phthalate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Hydroquinone	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium acetate trihydrate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium Disulphite	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium Acetate anhydre	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sabouraud Dextrose Agar	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Plate count Agar	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
MacConkey Broth	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Ortho-Phosphoric acid 85% (H3PO5)	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Phosphoric Acid(H3PO4)	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Oxalic Acid	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Citric acid monohydrate	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Potassium Hydroxide	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Manganese	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Chloride	15(5.6)	13(11.0)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Chloride test	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
N,N-Diethyl-p-phenylenediamine sulfate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Alminium test	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Cyanuric Acid test	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
THM Reagent 1,2,3,4(Total trihalomethanes)	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
3 phosphate reagent	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ascorbic Acid	13(4.8)	13(11.0)		0(0.0)	0(0.0)	0(0.0)
Buffer powder Citrate for Manganese	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Molybdate reagent and Molybdate 3 reagent	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Acid reagent for silica	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Citric Acid	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Amino Acid reagent	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ca and Mg indicator solution	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Alcalinity for Ca and Mg	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
EGTA solution(Egtazic acid or Aminopolycarboxylic acid)	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Sodium Peliodate	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
PAN indicator solution0.1%	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Ferric Ion solution	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Mercuric Thiocyanate solution	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Alkaline Cyanide Reagent	13(4.8)	13(11.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Iron Reagent	22(8.1)	20(16.9)	0(0.0)	2(12.5)	0(0.0)	0(0.0)
Speedloob(VL9)	72(26.7)	72(61.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Acifoam VF10	72(26.7)	72(61.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Safefoam VF9	86(31.9)	72(61.0)	14(50)	0(0.0)	0(0.0)	0(0.0)
DivoLEVF92	86(31.9)	72(61.0)	14(50)	0(0.0)	0(0.0)	0(0.0)
Divo AI(VB93)	86(31.9)	72(61.0)	14(50)	0(0.0)	0(0.0)	0(0.0)
Divo brite Y-81-S	82(30.4)	68(59.6)	14(50)	0(0.0)	0(0.0)	0(0.0)
Divsan Osan vs 37	89(33.0)	75(63.6)	14(50)	0(0.0)	0(0.0)	0(0.0)
Fosfree G VB11	93(34.4)	79(67.5)	14(50)	0(0.0)	0(0.0)	0(0.0)
Videojet ink/Solvent/Cleaning solution/Makeup	96(35.6)	82(69.5)	14(50)	0(0.0)	0(0.0)	0(0.0)
Greases& oils/Lubricants	87(32.2)	86(72.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Triphosphates	95(35.2)	95(80.5)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Aluminium sulphate	95(35.2)	95(80.5)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Calcium Hypochlorite	101(37.4)	100(84.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
SpectrusOX	110(40.7)	100(84.7)	10(35.7)	0(0.0)	0(0.0)	0(0.0)
Cortrol IS 2015	110(40.7)	100(84.7)	10(35.7)	0(0.0)	0(0.0)	0(0.0)
Optiperse PQ517.6	110(40.7)	100(84.7)	10(35.7)	0(0.0)	0(0.0)	0(0.0)
Continum AT 4505	110(40.7)	100(84.7)	10(35.7)	0(0.0)	0(0.0)	0(0.0)
Glycol Water	103(38.1)	101(85.6)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Ion Chloride	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Chemical oxygen demand (COD) cell test(Inlet)/Effluent	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Phosphate tests	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Variables	**N:270**	**TD**	**Logistic**	**HR**	**GM**	**Sales**
Nitrogen(Total) tests	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Methane	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Butane	28(10.4)	27(22.9)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Freon R134a,R404a	34(12.6)	33(28.0)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Argon(inert gas of group 18(Noble gas)	34(12.6)	33(28.0)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Isooctane	56(20.7)	55(46.6)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
2-Phenylethanol	56(20.7)	55(46.6)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Cycloheximide (Actidione)	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Ortophenylnediamine	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Ninhydrin	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Potassium Iodate(KI03)	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Formazine	103(38.1)	102(86.4)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Potassium di-hydrogenophosphate(KH2PO)	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Di-sodium hydroenophosphate (Na2HP04)	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
D(-)-Fructose	103(38.1)	100(84.7)	1(3.6)	2(12.5)	0(0.0)	0(0.0)
Glucose	103(38.1)	100(84.7)	1(3.6)	2(12.5)	0(0.0)	0(0.0)
Glycine	49(18.1)	48(40.7)	1(3.6)	0(0.0)	0(0.0)	0(0.0)
Dust	56(20.7)	19(16.1)	20(71.4)	2(12.5)	12(52.2)	3(5.4)

The analysis results portrayed the chemical hazards exposure/handling prevalence (n=156) and the bivariate analysis of the association of chemical handling with age and gender. The only significance was found in gender due to few women in the industrial workplace (N=270). Around 57.8% of workers handled chemical hazards

and males were 37.8% among workers with p=0.01 for gender relationships. Whereas the age-chemical handling relationship was not significant with p=0.1. The findings are tabulated in Table 4.41 below.

Table 4.41: Prevalence of workplace chemical hazard exposure (Chemical handling, Dust, Fumes, or reagents) and relationship with gender and age

Variables	**Frequency**	**Percentage (%)**	
Workplace chemical exposure			
Yes	156	57.8	
No	114	42.2	
Relationship of chemical exposure with gender and age			
Variable	Frequency	Percentage (%)	Statistical test
Chemical exposure			X^2(df);*P*
Gender			
Male Yes	102	37.8	
Female Yes	8	3.0	5.9(1);.01
Age			
<40 years	43	15.9	4.43(2);.1
40-50 Years	38	14.1	
>50Years	29	10.7	

The bivariate analysis findings showed the association between chemical exposure to the working department. A high proportion of 32.9% of technical department employees handled chemical substances whereas all other departments didn't with p<0.001. The findings are presented in Figure 4.5 below.

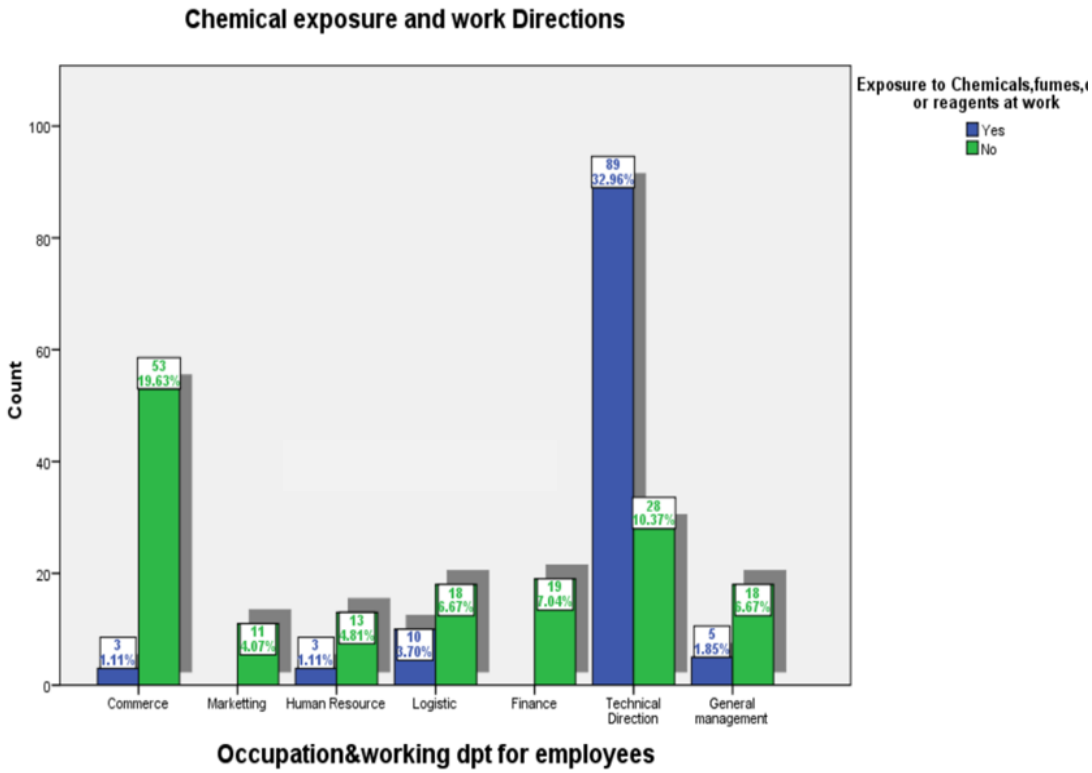

Figure 4.5: Proportion of Chemical hazard exposure versus worksite department

4.4.2 Association of working conditions to cardiovascular diseases risk

The analysis results were processed by a logistic regression, where the multivariate analysis by the Framingham general risk score model showed that three variables. These variables: being a worker in human resources, exposed to much noise>85dB, and night-shift workers) were associated with cardiovascular diseases elevated risk by full model. Whereas one variable (Night shift) was significantly associated to CVDs risk by reduced model. The results are tabulated in Table 4.42.

able 4.42: Association of working conditions to cardiovascular diseases risk by Framingham general risk score model

Variables	Full model (OR) CI95% (Risk>=10%)	p-value	Reduced model (AOR) CI 95% (Risk>=10%)	*P*-value
Commerce(dpt)	Reference			
Marketing	0.669(0.12-3.50)	0.634	-	-
Human resource	3.52(1.08-11.48)	0.037	-	-
Logistic	1.40(0.47-4.1)	0.536	-	-
Finance	1.62(0.52-5.03)	0.397	-	-
Technical	0.95(0.36-2.47)	0.923	-	-
General mgt	1.49(1.22-2.22)	0.761	-	-
Shift workers	0.362(0.08-1.51)	0.163	-	-
Night shift workers	4.257(1.03-17.44)	0.044	2.41(1.27-4.58)	0.007
Cold chambers	1.66(0.54-5.13)	0.3125	-	-
Vibration	2.11(0.81-5.13)	0.125	-	-
Much sound	0.209(0.071-0.61)	0.004	0.54(0.28-1.04)	0.06
Chemical handlers at work	1.89(0.74-4.79)	0.17	-	-

The analysis results were processed by a logistic regression. The multivariate analysis by the WHO/ISH score chart showed that three variables (shift work, night shift work, and vibration) were significantly associated with CVDs elevated risk by the full model (N=270). The findings are presented in Table 4.43.

Table 4.43: Association between working conditions and cardiovascular diseases risk by WHO/ISH score chart model

Variable	Odds ratio (CI: 95%) reduced Model	*P*-value
Working conditions		
Shift work (1)	0.156(0.31-0.784)	0.024
Night shift work (1)	14.227(2.533-79.913)	0.003
Vibration (1)	0.289(0.088-0.950)	0.041

4.5 Level of awareness of traditional cardiovascular diseases Risk factors and use of Personal protective equipment among the participants

4.5.1 Level of Awareness amongst participants in the study

The analysis results for the prevalence of awareness regarding the three prominent cardiovascular risk factors (hypertension, diabetes, and dyslipidemia) were processed on two-item questions. The respondents were asked if they have been told by Dr or a health worker about the factor or if the respondents are aware that the control of the factor could prevent cardiovascular diseases (N=440). Only 30.7% were told by health workers to have hypertension. A proportion of 13% was told by health professionals to have diabetes. A proportion of 6.8% was told to have dyslipidemia.

Table 4.44: Level of awareness among participants concerning hypertension and diabetes as prominent risk factors to cardiovascular diseases

Variables	Frequency	Percentage%
People ever told by Dr. or health worker to have high BP		
Yes	135	30.7
No	305	69.3
People aware that controlling BP reduces Heart and vessels diseases		
Yes	218	49.5
No	222	50.5
People ever told by Dr. or Health worker to have diabetes		
Yes	57	13
No	383	87
People aware of Diabetes control reduces heart and vessels diseases		
Yes	190	43.2
No	250	56.8
People ever told by Dr. or health worker to have dyslipidemia		
Yes	30	6.8
No	410	93.2
People Aware of reducing bad fat reduces heart and vessels diseases		
Yes	180	40.9
No	260	59.1

4.5.2 Level of PPE wearing and correlation to cardiovascular diseases risk

The analysis results were processed regarding the frequency of wearing personal protective equipment against the noise (Earmuffs, Ear Plugs) and chemical (Mask, Gloves, Boots, Gown, Goggles) exposure for only workers (N=270). The findings are tabulated in Table 4.45 below.

Table 4.45: Proportion of workers using Personal protective equipment (PPE) against prominent worksite hazards exposure (noise and chemical handling by department)

Variables	Frequency	Percentage%
Wearing PPE (Earmuffs, Ear Plugs) with noise exposure(>85dB)		
NA	131	48.5
Yes	133	49.3
No	6	2.2
Frequency of wearing PPE(Earmuffs, Ear plugs) when working with noise exposure(>85Db)		
NA	131	48.5
Never	13	4.8
Seldom	15	5.6
Sometimes	30	11.1
Often	46	17
Always	35	13
Wearing PPE(Mask, Gloves, boots, Gown, Goggles) with chemical exposure		
NA	114	42.2
Yes	117	43.3
No	39	14.4
Frequency of wearing PPE with Chemical exposure		
NA	114	42.2
Never	43	15.9
Seldom	18	6.7
Sometimes	38	14.1
Often	40	14.8
Always	17	6.3

The analysis results of this study were processed regarding the correlation between the frequency of wearing PPE and cardiovascular disease risk. The analysis used the non-parametric correlational test with a two-sided level of 95CI, $p<0.01$, for the total employees (N=270). The two prediction models were used (Framingham general risk score and WHO/ISH model). The two non-parametric tests were also used

(Spearman's Rho and Kendall's tau b) to generate the correlation level. The frequency of putting PPE against much noise was -0.218 p<0.001, frequency of putting PPE against chemical exposure was -0.157 p=0.004 for Kendall's tau_b. The listwise (N=270) for total workers. The findings showed a negative correlation for both tests. The results are tabulated in Table 4.46 below.

Table 4.46: Distribution of non-parametric correlation test statistic for cardiovascular disease and PPE usage for all employees (total employees:TE)

Non-parametric Correlation test statistic

			FRSC	WHO/ISH	How often you Put PPE/ear plugs when Much noise	How often you put PPE when chemicals	Wearing PPE when working with much noise	Wear PPE when Chemicals, fumes, dust
Kendall's tau_b	FRSC	Correlation Coefficient	1.000**	.412**	-.218**	-.157**	-.048**	-.019**
		Sig. (2-tailed)	.	.000	.000	.004	.408	.745
	WHO/ISH	Correlation Coefficient	.412**	1.000	-.059**	-.033**	.011**	-.009**
		Sig. (2-tailed)	.000	.	.276	.544	.844	.875
	Put PPE/ear plugs when Much noise	Correlation Coefficient	-.218**	-.059**	1.000**	.775**	.342**	.350**
		Sig. (2-tailed)	.000	.276	.	.000	.000	.000
	How often you put PPE when chemicals	Correlation Coefficient	-.157**	-.033**	.775**	1.000**	.328**	.228**
		Sig. (2-tailed)	.004	.544	.000	.	.000	.000
	Wearing PPE when working with much noise	Correlation Coefficient	-.048**	.011**	.342**	.328**	1.000**	.882**
		Sig. (2-tailed)	.408	.844	.000	.000	.	.000
	Wear PPE when Chemicals, fumes, dust	Correlation Coefficient	-.019**	-.009**	.350**	.228**	.882**	1.000**
		Sig. (2-tailed)	.745	.875	.000	.000	.000	.
Spearman's rho	FRSC	Correlation Coefficient	1.000**	.412**	-.244**	-.175**	-.050**	-.020**
		Sig. (2-tailed)	.	.000	.000	.004	.409	.745
	WHO/ISH	Correlation Coefficient	.412**	1.000	-.066**	-.037**	.012**	-.010**
		Sig. (2-tailed)	.000	.	.277	.545	.844	.876
	Put PPE/ear plugs when Much noise	Correlation Coefficient	-.244**	-.066**	1.000	.857**	.498**	.490**
		Sig. (2-tailed)	.000	.277	.	.000	.000	.000
	How often you put PPE when chemicals	Correlation Coefficient	-.175**	-.037**	.857**	1.000	.450**	.356**
		Sig. (2-tailed)	.004	.545	.000	.	.000	.000
	Wearing PPE when working with much noise	Correlation Coefficient	-.050**	.012**	.498**	.450**	1.000	.902**
		Sig. (2-tailed)	.409	.844	.000	.000	.	.000
	Wear PPE when Chemicals, fumes, dust	Correlation Coefficient	-.020**	-.010**	.490**	.356**	.902**	1.000
		Sig. (2-tailed)	.745	.876	.000	.000	.000	.

**. Correlation is significant at the 0.01 level (2-tailed).
a. Listwise N = 270=TW

The results of this study regarding the correlation between the frequency of wearing PPE and cardiovascular disease risk were processed by using a non-parametric correlational test. The two-sided significance level was 95CI, p<0.01, (n=117), where (N=270). The two prediction models were used (Framingham general risk score and WHO/ISH model) including two non-parametric tests (Spearman's Rho and Kendall's tau b). The findings showed a negative correlation for both tests. The negative correlation results for both tests were -0.211, p=0.022, and -0.182 p=0.049, regarding the frequency of Putting PPE/earplugs when exposed to much noise>85dB. Moreover, the item about how often you put PPE when handling chemicals by the WHO/ISH Model and a correlation test was -0.560 p<0.001, and -0.459, p<0.001. This was done on the two respective factors by Framingham's general risk score model. The listwise (n) stand for technical department employees (n=117). The results are tabulated in Table 4.47 below.

Table 4.47: Correlation for non-parametric test for CVD risk and PPE Wearing by Noise and chemical

Non-parametric Correlation test statistic for most exposed employees=117						
Test	variables		Put PPE/ear plugs when Much noise	How often you put PPE when chemicals	WHO/ISH	FRSC
Kendall's tau_b	Put PPE/ear plugs when Much noise	Correlation Coefficient	1.000	.497**	-.192**	-.508**
		Sig. (2-tailed)	.	.000	.023	.000
	How often you put PPE when chemicals	Correlation Coefficient	.497**	1.000	-.165**	-.414**
		Sig. (2-tailed)	.000	.	.049	.000
	WHO/ISH	Correlation Coefficient	-.192**	-.165**	1.000	.450**
		Sig. (2-tailed)	.023	.049	.	.000
	FRSC	Correlation Coefficient	-.508**	-.414**	.450**	1.000
		Sig. (2-tailed)	.000	.000	.000	.
Spearman's rho	Put PPE/ear plugs when Much noise	Correlation Coefficient	1.000	.570**	-.211**	-.560**
		Sig. (2-tailed)	.	.000	.022	.000
	How often you put PPE when chemicals	Correlation Coefficient	.570**	1.000	-.182**	-.459**
		Sig. (2-tailed)	.000	.	.049	.000
	WHO/ISH	Correlation Coefficient	-.211**	-.182**	1.000	.450**
		Sig. (2-tailed)	.022	.049	.	.000
	FRSC	Correlation Coefficient	-.560**	-.459**	.450**	1.000
		Sig. (2-tailed)	.000	.000	.000	.

**. Correlation is significant at the 0.01 level (2-tailed).
a. Listwise n = 117=TDE

4.6 Proportion of people with biological factors among the study participants in the study area

4.6.1 Hypertension

Table 4.48 compares the mean values of systolic blood pressure and diastolic blood pressure by gender and age. The F test was calculated for each gender subdivision (Male, female) and total parts versus the age structure. The age-related blood pressure levels for men are superior to women's levels which explains the high CVDs risk for men. There is a significant difference in all age groups by gender in BP mean values (systolic BP Male p = 0.002, female p<0.001, the total mean SBP p<0.001, Diastolic BP male p<0.001, female p<0.001, the total mean DBP p<0.001).

Table 4.48: Distribution of mean systolic and diastolic blood pressure (mmHg) by age and gender

		Systolic BP (Mean ± SD)			Diastolic BP (Mean ± SD)		
Age group	N= 440						
		Male	Female	Total	Male	Female	Total
<35	84	130.32 ± 11.27	122.25 ± 9.89	127.63±11.43	74.39±9.97	70.57±8.94	73.12±9.75
35-39	73	132.19 ± 12.97	126.78 ± 11.61	129.45±12.51	75.33±13.71	74.70±11.42	75.01±12.52
40-44	65	132.55 ± 16.20	130.03 ± 12.32	131.23±14.25	79.16±12.85	78.82±11.90	78.98±12.27
45-49	86	137.33 ± 16.16	133.23 ± 14.77	135.23±15.51	84.10±13.92	83.25±10.95	83.66±12.42
50-54	89	137.24 ± 13.58	136.71 ± 11.75	137.01±12.77	82.84±12.76	79.11±11.87	81.25±12.46
55-59	30	141.14 ± 11.36	139.25±8.65	140.63±10.60	86.27±8.40	89.62±8.12	87.17±8.32
>=60	13	144.82 ± 11.65	141.00±1.41	144.23±10.74	89.91±5.68	91.00±0.00	90.08±5.20
Total	440	135.06 ± 14.03	130.83±13.09	133.22±13.78	80.22±12.78	78.47±11.91	79.46±12.43
Significance test	*F*	*3.663*	*5.604*	*8.488*	*6.496*	*6.321*	*12.324*
	df	*6*	*6*	*6*	*6*	*6*	*6*
	p	*<0.002*	*<0.001*	*<0.001*	*<0.001*	*<0.001*	*<0.001*

Table 4.49 compares the levels of blood pressure regarding previous versus updated classifiers and bivariate analysis of blood pressure levels with age. These levels moderate the cardiovascular risk association (N=440) and were all significant to age increment, p<0.05. The results showed a significant difference in prevalence within blood pressure classifiers and gender group by age group. The previous systolic classification for men with x^2 = 34.215, p=0.001 and for women with x^2 = 41.494

P=0.001. Previous Diastolic classification for men with x^2 = 41.345 p=0.001 and women with x^2 = 45.377, P<0.001. The updated systolic classification for men with x^2 =40.746, P=0.002 and for women with x^2 = 46.162, P<0.001. Update diastolic classification for men with x^2 =31.814, P=0.001 and for women with x^2 = 38.100, P<0.001. The updated classifier shifts several patients from normal and pre-hypertension of the previous classifier into stages 1 and 2 of the updated classifiers. More patients were shifted in systolic BP than in diastolic BP according to the above-displayed p-values while there is no significant difference in age groups by previous and updated blood pressure classification.

Table 4.49: Hypertension prevalence of previous and update classification of blood pressure by gender and age group among study participants

Variable		Age group of the study participants							Significance X^2(df),*P*
Previous_S	n	<35	35-39	40-44	45-49	50-54	55-59	>=60	
Men									
Normal	29	6 (20.6)	5 (17.2)	7 (24.1)	4 (13.7)	5 (17.2)	2 (6.8)	0 (0.0)	34.2(18);.001
Pre-HTN	129	42(32.5)	22(17.0)	13(10.0)	18(13.9)	22(17.0)	7(5.4)	5(3.8)	
HTN-1	82	7(8.5)	8(9.7)	10(12.1)	18(21.9)	21(25.6)	13(15.8)	5(6.0)	
HTN-2	9	1(11.1)	1(11.1)	1(11.1)	2(22.2)	3(33.3)	0(0.0)	1(11.1)	
Women									
Normal	29	8(27.5)	7(24.1)	4(13.7)	7(24.1)	3(10.3)	0(0.0)	0(0.0)	
Pre-HTN	109	20(18.3)	24(22.0)	25(22.9)	19(17.4)	18(16.5)	3(2.7)	0(0.0)	41.4(18);.001
HTN-1	50	0(0.0)	6(12.0)	4(8.0)	17(34.0)	16(32.0)	5(10.0)	2(4.0)	
HTN-2	3	0(0.0)	0(0.0)	1(33.3)	1(33.3)	1(33.3)	0(0.0)	0(0.0)	
Previous_D									
Men									
Normal	139	41(29.4)	23(16.5)	20(14.3)	19(13.6)	28(20.1)	7(5.0)	1(0.7)	41.3(18);.001
Pre-HTN	52	10(19.2)	7(13.4)	7(13.4)	10(19.2)	7(13.4)	8(15.3)	3(5.7)	
HTN-1	40	3(7.5)	5(12.5)	3(7.5)	6(15.0)	12(30.0)	5(12.5)	6(15.0)	
HTN-2	18	2(11.1)	1(14.2)	1(14.2)	7(38.8)	4(22.2)	2(11.1)	1(14.2)	
Women									
Normal	102	23(22.5)	25(24.5)	18(17.6)	19(18.6)	17(16.6)	0(0.0)	0(0.0)	
Pre-HTN	54	5(9.2)	7(12.9)	12(22.2)	12(22.2)	13(24.0)	5(9.2)	0(0.0)	45.3(18);<.001
HTN-1	28	0(0.0)	4(14.2)	2(7.1)	10(35.7)	8(28.5)	2(7.1)	2(7.1)	
HTN-2	7	0(0.0)	1(14.2)	2(28.5)	3(42.8)	0(0.0)	1(14.2)	0(0.0)	
Updated-S									
Men									
Normal	29	6(20.6)	5(17.2)	7(24.1)	4(13.7)	5(17.2)	2(6.8)	0(0.0)	40.7(18);.002
elevated	68	24(35.2)	12(17.6)	9(13.2)	11(16.1)	11(16.1)	1(1.4)	0(0.0)	
HTN stage1	61	18(29.5)	10(16.3)	4(6.5)	7(11.4)	11(18.0)	6(9.8)	5(8.19)	
HTN stage2	91	8(8.7)	9(9.8)	11(12.0)	20(21.9)	24(26.3)	13(14.2)	6(6.5)	
Women									
Normal	29	8(27.5)	7(24.13)	4(13.7)	7(24.13)	3(10.3)	0(0.0)	0(0.0)	46.1(18);<.001
elevated	77	16(20.7)	20(25.9)	16(20.7)	14(18.1)	9(11.6)	2(2.5)	0(0.0)	
HTN stage1	32	4(12.5)	4(12.5)	9(28.1)	5(15.6)	9(28.1)	1(3.1)	0(0.0)	
	n	<35	35-39	40-44	45-49	50-54	55-59	>=60	
HTN stage2	53	0(0.0)	6(11.3)	5(9.4)	18(33.9)	17(32.0)	5(9.4)	2(3.7)	
Updated-D									
Men									
Normal	139	41(29.4)	23(16.5)	20(14.3)	19(13.6)	28(20.1)	7(5.0)	1(0.7)	31.8(12);.001
HTN-1	53	10(18.8)	7(13.2)	7(13.2)	10(18.8)	7(13.2)	8(15.0)	4(7.5)	
HTN-2	57	5(8.7)	6(10.5)	4(7.0)	13(22.8)	16(28.0)	7(12.2)	6(10.5)	
Women									
Normal	102	23(22.5)	25(24.5)	18(17.6)	19(18.6)	17(16.6)	0(0.0)	0(0.0)	38.1(12);<.001
HTN-1	54	5(9.2)	7(12.9)	12(22.2)	12(22.2)	13(24.0)	5(9.2)	0(0.0)	
HTN-2	35	0(0.0)	5(14.2)	4(11.4)	13(37.1)	8(22.8)	3(8.5)	2(5.7)	

Table 4.50 depicts a comparison of the blood pressure levels by the previous and update classifiers for employees and spouses in two plants (Rubavu and Kicukiro)

(N=440). The normal systolic blood pressure participants decreased from 238(54.1%) to 58(13.2%) and an increase in BP from pre-hypertension of 58(13.2%) to elevated BP of 145(33.0). The reduction of stage 1 and an increase of stage 2 from 12(2.7%) to 144(32.7) by the updated systolic classifier (USC). Whereas the diastolic classifier increased the 39(8.6%) to stage 1 and 67(15%) to stage 2 of diastolic hypertension. A high proportion of SBP and DBP were found in the Kicukiro plant where PSC 91(20.7%), PDC 63(14.3%) and USC 147(33.4%), UDC 130(29.5%) were employees and spouses. Whereas PSC 53(12%), PDC 30 (6.8%) and USC 90(20.4%), UDC 69(15.6%) were Rubavu plant employees and spouses, respectively.

Table 4.50: Prevalence of Hypertension by site, and status of study participants

Variable	N=440	Kicukiro worksite			Rubavu worksite		
		Employee	spouse	Total	Employee	spouse	Total
Previous SBP							
Normal	238(54.1)	83(18.9)	40(9.1)	123(28.0)	64(14.5)	51(11.6)	115(26.1)
Pre-HTN	58(13.2)	21(4.8)	24(5.5)	45(10.2)	9(2.0)	4(0.9)	13(3.0)
HTN stage1	132(30.0)	52(11.8)	35(8.0)	87(19.8)	31(7.0)	14(3.2)	45(10.2)
HTN stage2	12(2.7)	4(0.9)	0(0.0)	4(0.9)	6(1.4)	2(0.5)	8(1.8)
Total	440(100)	160(36.4)	99(22.5)	259(58.9)	110(25.0)	71(16.1)	181(41.1)
Updated SBP							
Normal	58(13.2)	21(4.8)	24(5.5)	45(10.2)	9(2.0)	4(0.9)	13(3.0)
Elevated	145(33.0)	45(10.2)	22(5.0)	67(15.2)	37(8.4)	41(9.3)	78(17.7)
HTN stage1	93(21.1)	38(8.6)	18(4.1)	56(12.7)	27(6.1)	10(2.3)	37(8.4)
HTN stage2	144(32.7)	56(12.7)	35(8.0)	91(20.7)	37(8.4)	16(3.6)	53(12.0)
Total	440(100)	160(36.4)	99(22.5)	259(58.9)	110(25.0)	71(16.1)	181(41.1)
Previous DBP							
Normal	241(54.8)	84(19.1)	45(10.2)	129(29.3)	69(15.7)	43(9.8)	112(25.5)
Pre-HTN	106(24.1)	36(8.2)	31(7.0)	67(15.2)	20(4.5)	19(4.3)	39(8.9)
HTN stage1	68(15.5)	29(6.6)	21(4.8)	50(11.4)	11(2.5)	7(1.6)	18(4.1)
HTN stage2	25(5.7)	11(2.5)	2(0.5)	13(3.0)	10(2.3)	2(0.5)	12(2.7)
Total	440(100)	160(36.4)	99(22.5)	259(58.9)	110(25.0)	71(16.1)	181(41.1)
Updated DBP							
Normal	241(54.8)	84(19.1)	45(10.2)	129(29.3)	69(15.7)	43(9.8)	112(25.5)
HTN stage1	107(24.3)	36(8.2)	31(7.0)	67(15.2)	21(4.8)	19(4.3)	40(9.1)
HTN stage2	92(20.9)	40(9.1)	23(5.2)	63(14.3)	20(4.5)	9(2.0)	29(6.6)
Total	440(100)	160(36.4)	99(22.5)	259(58.9)	110(25.0)	71(16.1)	181(41.0)

The analysis results were processed to compare the levels of blood pressure for spouses and employees, where the normal blood pressure, hypertension, and isolated

blood pressure levels changed. The proportion of respondents with blood pressure was increased in the updated classifier where, spouses (n=170), and employees (n=270) with the total sample size (N=440). The findings are tabulated in Table 4.51 below. In the previous blood pressure classifier, the normal blood pressure of employees and spouses was reduced from 298(67.22%) to 168(38.18%) by the updated blood pressure classifier. Whereas full hypertension was increased from 82(18.63%) to 37.72%). The isolated systolic hypertension was increased from 50(11.36%) to 73(16.59%) while the isolated diastolic hypertension was also increased from 10(2.27%) to 33(7.50%). The total additional hypertension was from 142 (32.27%) to 272(61.81%) with a total difference of 130(29.54%). The increase was subdivided among 270 employees, from 97(35.92%) to 176(65.18%), and among 170 spouses, from 45(26.47%) to 96(56.47%). The percentage of hypertension among employees is slightly 5.8% superior compared to the spouses.

Table 4.51: Distribution of normal blood pressure (BP), hypertension, systolic and diastolic isolated hypertension by status of participants

Status of participants	N=440	Normal BP	Hypertension	Isolated S HTN	Isolated D HTN
Previous-C					
Employees	270(61.36)	173(64.07)	56(20.74)	35(12.96)	6(2.22)
Spouses	170(38.64)	125(73.52)	26(19.29)	15(8.82)	4(2.35)
Total	440(100%)	298(67.7)	82(18.63)	50(11.36)	10(2.27)
Update-C					
Employees	270(61.36)	94(34.81)	100(37.03)	60(22.22)	16(5.92)
Spouses	170(38.64)	74(43.52)	66(38.82)	13(7.64)	17(10.00)
Total	440(100%)	168(38.18)	166(37.72)	73(16.59)	33(7.50)
Gender					
Male	249(56.6%)	155(62.2)	53(21.3)	35(14.1)	6(2.4)
Female	191(43.4%	143(74.9)	29(15.2)	15(7.9)	4(2.1)
Total	440(100%)	298(67.7)	82(18.6)	50(11.4)	10(2.3)

The analysis results depict the comparison of the drugs used by the hypertensive study respondents (N=72), where employees (n=49), and spouses(n=23). Most of the respondents used dual therapy for hypertension treatment. Out of the overall

percentage of used combinants anti-hypertensive employees were 68.1% and followed by 31.9% of spouses. The findings are tabulated in Table 4.52 below.

Table 4.52: Distribution of hypertensive patients in comparison with combinants anti-hypertensive therapy

Variables (SD)	**Overall %**	**Mono therapy %**	**Dual therapy%**	**3-therapy %**	***P*-value**
Status of Participants					
Employees	49(68.1)	16(22.2)	32(44.4)	1(1.4)	
Spouses	23(31.9)	15(20.8)	8(11.1)	0(0.0)	<0.03
Total	72(100)	31(43.1)	40(55.6)	1(1.4)	

Figure 4.6 shows the comparison of predicted mean risk intergroup differences within the previous and updated blood pressure classification. The figure presented cardiovascular diseases risk reduction for the updated blood pressure classifier while it was high for the previous blood pressure classifier (N=440). The previous classification was slightly showing a high mean risk of 0.06% prehypertension, 0.17% hypertension stage 1, 0.40% hypertension stage 2. Whereas the updated classification marked a reduction of mean risk of 0.04% on elevated SBP, 0.09% hypertension stage 1, 0.19% hypertension stage 2. All the blood pressure classifications showed intergroup significant differences with $p<0.001$.

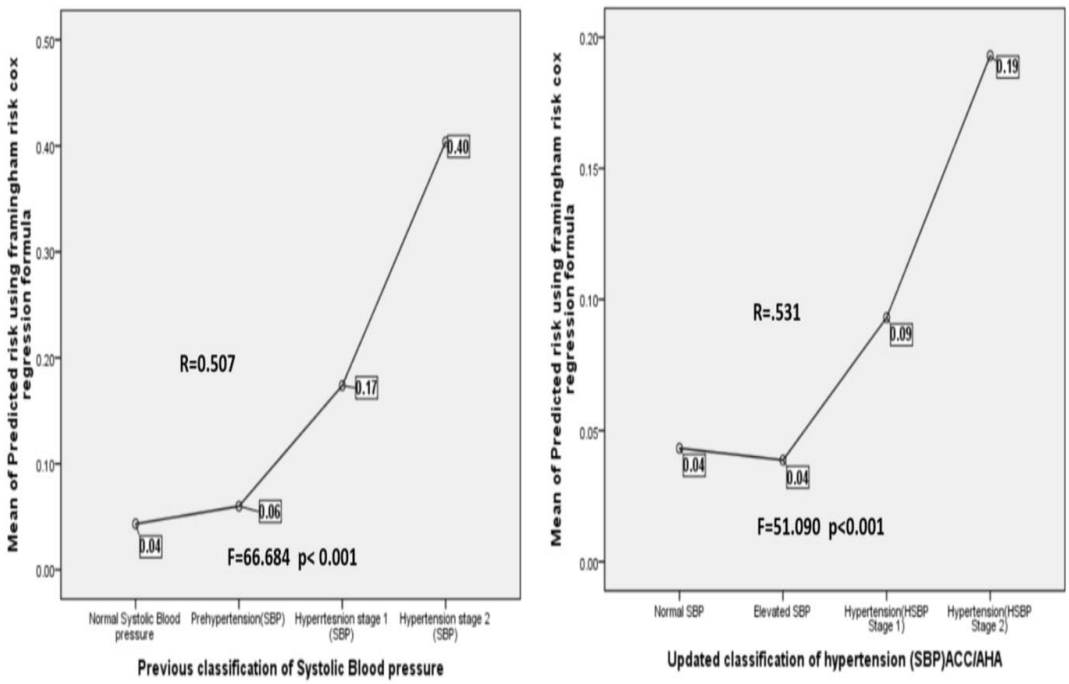

Figure 4.6: Distribution of predicted cardiovascular risk mean by 100(Framingham risk cox regression) by previous and updated SBP classification

The analysis results for the multivariate analysis for the association of modifiable and non-modifiable risk to hypertension were processed. The non-modifiable risk factors such as being male and family history of hypertension were significantly associated with hypertension. The modifiable factors such as smoking, central obesity, stress, and diabetes were also significantly associated with hypertension with, $p<0.05$, (N=440). The odds of being hypertensive were higher among the male subjects with (AOR: 0.736) and (AOR: 0.205). The eldest age group of >50 years with (AOR: 3.787) and (AOR: 3.383), being employees (AOR: 0.229) and (AOR: 0.316), Family history with hypertension (AOR: 0.314) and (AOR: 0.498). The odds of being hypertensive for alcohol intake within 30 days were (AOR: 0.541) and (AOR: 0.792), with moderate central obesity (AOR: 2.063) and (AOR: 2.958). The odds of being hypertensive with high waist circumference were (AOR: 1.235) and (AOR: 6.964), being diabetic (AOR: 0.719) and (AOR: 1.328). Being moderately stressed (AOR: 0.206) and (AOR: 0.267), smoking (AOR: 0.282) and (AOR: 0.2418) by previous and update hypertension classification, respectively. All p-values were

significant with a p-value < 0.05 except for males, with a high waist circumference and being diabetic in previous hypertension classification reduced model. Mild stress, being diabetic, and taking alcohol within 30 days presented a p-value > 0.05 in the updated hypertension classification. The findings are presented in Table 4.53.

Table 4.53: Association of modifiable and non-modifiable risk factors to hypertension

Variables	**Odds ratio (CI 95%)**	**p value**	**Odds ratio**	***P*-value**
	Reduced model previous_htn		**Reduced model update_htn**	
Gender				
Female	1.0 (reference)		1.0 (reference)	
Male	0.736(0.345- 1.568)	0.42	0.205(0.092-0.458)	<0.001
Age category				
<40	1.0 (reference)		1.0(Reference)	
40-50	2.710(1.429-5.140)	0.002	1.416(0.841-2.382)	0.190
>50	3.787(1.985-7.224)	<0.001	3.383(1.884- 6.074)	<0.001
Status of participants				
Spouse	1.0(Reference)		1.0(Reference)	
Employee	0.229(0.121- 0.435)	<0.001	0.316(0.122- 0.815)	0.017
Family history				
Without HTN	1.0(Reference)		1.0(Reference)	
With HTN	0.314(0.190- 0.518)	<0.001	0.498(0.303-0.819)	0.006
Alcohol intake in 30days				
No	1.0 (reference)		1.0 (reference)	
Yes	0.541(0.310- 0.944)	0.031	0.792(0.486-1.289)	0.348
Central obesity				
Normal WC	1.0(Reference)		1.0(Reference)	
Moderate WC	2.063(1.068-3.984)	0.031	2.958(1.588-5.508)	0.001
High WC	1.235(0.592-2.576)	0.572	6.964(3.456-14.029)	<0.001
Diabetes				
Normal	1.0 (Reference)		1.0 (Reference)	
Diabetes	0.719(0.342-1.514)	0.386	1.328(0.582-3.029)	0.500
Stress level				
Low stress	1.0 (Reference)		1.0 (Reference)	
Mild stress	0.112(0.013-0.947)	0.044	0.458(0.235-0.126)	0.235
Moderate stress	0.206(0.109-0.389)	<0.001	0.267(0.124-0.574)	<0.001
High stress	-	-	-	-
Smoking				
No	1.0 (Reference)		1.0 (Reference)	
Yes	0.282(0.111-0.715)	0.008	0.2418(0.075- 0.773)	0.017

4.6.2 Diabetes

Diabetes is a prominent risk factor for cardiovascular diseases risk. The analysis of the diabetes data was carried out for all layers of diabetes such as blood sugar levels, glycosylated hemoglobin, and diabetes treatment.

The analysis results of this study presented the categorization of blood sugar as normal blood sugar, prediabetes, and diabetes or treated diabetes (N=440). Non-diabetics were 59.6%, followed by 26.8% of prediabetics and 13.6% of diabetics. Bivariate analysis showed no significant association with gender. The findings are presented in Table 4.54.

Table 4.54: Bivariate analysis of blood sugar categorization/normal, prediabetes, and diabetes by gender

Variable	N(440)	Male	Female	Statistical Test X^2(df);*P*
Type 2 Diabetes Mellitus				
No diabetes: BS<100mg/dl	262(59.6)	145(32.9)	117(26.5)	0.697(1),.247
Prediabetes:BS=100-125md/dl	118(26.8)	73(16.5)	45(10.2)	
Diabetes: BS>125mg/dl or treated	60(13.6)	31(7.0)	29(6.5)	
Total	440(100)	249(56.5)	191(43.5)	

Table 4.55 portrayed the blood sugar and glycosylated hemoglobin mean value and standard deviation comparison by age and gender. Women presented slightly elevated levels of both blood sugar and glycosylated hemoglobin. The mean value of blood sugar by age was only significant for males with different levels of <40 years: 94.89 ± 15.52, 40-50 years: 108.92 ± 51.85,>50 years: 107.22 ± 32.82, p=0.018. Whereas it was not the case for females with different levels of <40 years: 101.75 ± 44.08, 40-50 years: 101.27 ± 28.60,>50 years: 108.70 ± 32.15, p=0.475. There was also a significant difference in age levels, p=0.043. There was also an increase in glycosylated hemoglobin levels by age.

Table 4.55: Distribution of mean blood sugar mg/dl and glycosylated hemoglobin (HBA1C %) by age and gender

Age group	N=440	**Blood sugar mg/dl (Mean ± SD)**			**Glycosylated Hemoglobin% (Mean ± SD)**		
		Male	Female	Total	Male	Female	Total
<40	157	94.89 ± 15.52	101.75 ± 44.08	97.73±30.81	4.653±0.72	4.97±1.41	4.78±1.07
40-50	151	108.92 ± 51.85	101.27 ± 28.60	104.97±41.54	5.27±1.89	5.02±1.08	5.159±1.52
>50	132	107.22 ± 32.82	108.70 ± 32.15	107.76±32.46	5.18±1.24	5.28±1.14	5.21±1.20
Total	440	103.16 ± 35.64	103.30±35.38	103.22±35.49	5.01±1.35	5.08±1.21	5.04±1.29
	F	*4.075*	*0.748*	*3.173*	*5.454*	*0.975*	*5.048*
Significance df		*2*	*2*	*2*	*2*	*2*	*2*
	p	*0.018*	*0.475*	*0.043*	*0.005*	*0.379*	*0.007*

The result of this study indicate that diabetes drugs users were 8.9%(n=39), females 4.5%(n=20), males 4.3%(n=19). The monotherapy 6.6%(n=29), bi-therapy 2%(n=9) and tri-therapy 0.2%(n=1), with the total diabetics (n=60), where (N=440). A proportion of 61.5% of all diabetic participants on treatment took metformin 850mg, which was the most, used diabetic drug and followed by 12.8% of the diabetic participant on treatment, who took glimepiride 3mg for the first indicated drug. Then 5.1% of the diabetic participant on treatment took daonil, 5mg as the second most prescribed drug combination. Around 6.6% of all participants, that is 74.3% of total diabetic participants on treatment. This was on monotherapy while 2% of all participants that is 23% of total diabetic participants on treatment was on bi-therapy and 0.2% of all participants, that is 2.5% of total diabetic participants on treatment, were on tri-therapy diabetic drugs. (See table 4.56).

Table 4.56: Distribution of diabetic drugs by gender in the study participants

Variable	N(440)	Male	Female
Current taking Diabetes medication			
Yes	39(8.9)	19(4.3)	20(4.5)
No	401(91.1)	172(39.1)	229(52.0)
Total	440(100)	249(56.6)	191(43.4)
Types of diabetes medication taken as 1st drug			
None	401(91.1)	229(52.0)	172(39.1)
Metiformine/Glycophage 850mg	24(5.5)	11(2.5)	13(3.0)
Amarel/Glimepiride 3mg	5(1.1)	3(0.7)	2(0.5)
Insulin Lente	1(0.2)	0(0.0)	1(0.2)
Insulin Mixte	1(0.2)	0(0.0)	1(0.2)
Galvus or Vildagliptin 50/Met 1000mg	2(0.5)	1(0.2)	1(0.2)
Galvus or Vildagliptin 50/Met 850mg	3(0.7)	1(0.2)	2(0.5)
Galvus or Vildagliptin 50/Met 500mg	1(0.2)	1(0.2)	0(0.0)
Gliclazide/Diamicron 60mg	2(0.5)	1(0.2)	1(0.2)
Total	440(100)	249(56.6)	191(43.4)
Type of diabetes medication taken as 2nd or 3rd drug			
None	34(87.1)	17(43.5)	17(43.5)
Daonil 5mg	2(5.1)	1(2.5)	1(2.5)
Amarel/Glimepirirde 3mg	1(2.5)	1(2.5)	0(0.0)
Insulin Mixte	1(2.5)	0(0.0)	1(2.5)
Gliclazide/Diamicron 60mg	1(2.5)	0(0.0)	1(2.5)
Total	39(100)	19(48.7)	20(51.2)
Monotherapy diabetes			
No	411(93.4)	234(53.2)	177(40.2)
Yes	29(6.6)	15(3.4)	14(3.2)
Total	440(100)	249(56.6)	191(43.4)
Bi-therapy diabetes			
No	431(98.0)	244(55.5)	187(42.5)
Yes	9(2.0)	5(1.1)	4(0.9)
Total	440(100)	249(56.6)	191(43.4)
Tri-therapy diabetes			
No	439(99.8)	249(56.6)	190(43.2)
yes	1(0.2)	0(0.0)	1(0.2)
Total	440(100)	249(56.6)	191(43.4)

4.6.3 Overweight, obesity, and central obesity for study participants in study area

The bivariate analysis results showed the association between body mass index (BMI) and gender. This study's findings presented a significant association between BMI and gender, p<0.001, where, female respondents (n=191) have more elevated BMI than male respondents (n=249). Levels of body fat accumulation by gender were expressed where only 27.7% were in normal weight class, 46.6% were in overweight class. Among 24.6% of obese participants, 15.5% were the female population with a gender-based BMI relationship significance of p<0.001. The findings are tabulated in Table 4.57 below.

Table 4.57: Bivariate analysis body fat accumulation (BMI category) of the study participants by gender

Variable	N(440)	Male	Female	Statistical Test X^2(df),*P*
BMI Group				
Underweight	5(1.1)	4(0.9)	1(0.2)	25.667(5);<.001
Normal Weight	122(27.7)	78(17.7)	44(10.0)	
Overweight	205(46.6)	127(28.9)	78(17.7)	
Obesity Class I	80(18.2)	33(7.5)	47(10.7)	
Obesity Class II	21(4.8)	6(1.4)	15(3.4)	
Obesity Class III	7(1.6)	1(0.2)	6(1.4)	
Total	440(100)	249(56.5)	191(43.5)	

The study findings displayed the bivariate analysis of the association between waist circumference levels and gender. The low-level Waist circumference (WC) (n=183), the high-level WC(n=94), and the very high-level WC(n=163) with p<0.001. The results are tabulated in Table 4.58 below.

Table 4.58: Bivariate analysis of waist circumference (Central fat accumulation) and gender of the study participants

Variable	N(440)	Male	Female	Statistical Test, X^2(df);*P*
Waist Circumference: Owolabi,2017				
Low: Men <94cm,Women<80cm	183(41.6)	150(34.1)	33(7.5)	1.089(2);<.001
High:Men94-102cm,Women 80-88cm	94(21.4)	56(12.7)	38(8.6)	
Very High: Men>102cm,Women>88cm	163(37.0)	43(9.8)	120(27.3)	
Total	440(100)	249(56.5)	191(43.5)	

The analysis results portrayed the comparison of mean value and standard deviation of the waist to hip ratio (WHR) in different age structures by location and gender. Men presented a slightly elevated mean value than women in different age structure: <40years (n=157), 40-50 years (n=151),>50 years (n=132), (N=440). Age differences were significant for Kicukiro male population <40years: 0.93±0.08, 40-50 years: 0.96±0.07, >50years: 0.97±0.06, p=0.04. The age differences were also significant for Rubavu Female population<40years: 0.80±0.08, 40-50 years: 0.86±0.09, >50years: 0.82±0.07, p=0.03. The results are tabulated in Table 4.59 below.

Table 4.59: Distribution of mean value and standard deviation of waist to hip ratio (WHR) of the study participants

Age	N=440	Kicukiro			Rubavu		
		Male	Female	Total	Male	Female	Total
<40	157	0.93±0.08	0.91±0.10	0.92±0.09	0.90±0.08	0.80±0.08	0.85±0.09
40-50	151	0.96±0.07	0.95±0.06	0.95±0.06	0.89±0.08	0.86±0.09	0.88±0.09
>50	132	0.97±0.06	0.94±0.06	0.96±0.06	0.91±0.08	0.82±0.07	0.88±0.09
Total	440(100)	0.95±0.07	0.93±0.07	0.94±0.07	0.90±0.08	0.83±0.09	0.87±0.09
	F	3.071	2.577	5.165	0.598	3.549	1.453
Significance df		2	2	2	2	2	2
	p	0.04	0.08	0.006	0.55	0.03	0.23

The analysis results presented the bivariate analysis for the association of the waist to hip ratio (WHR) WHO cut-off and gender. The association between WHR and gender was only significant for Kicukiro respondents. The central obesity for Kicukiro and Rubavu was 79.9% (n=207) and 44.2%(n=80) respectively. The results are tabulated in Table 4.60 below.

Table 4.60: Bivariate analysis of body fat distribution of WHO cut points of WHR by gender of the study participants

Variable	N(440)	Male	Female	Statistical test, X^2(df);*P*
Kicukiro				
Normal<0.85 F,<0.90M	52(20.1)	36(13.9)	16(6.2)	4.633(1);.022
Central Obesity >=0.85 F,>=0.90M	207(79.9)	109(42.1)	98(37.8)	
Total	259(100)	145(56.0)	114(44.0)	
Rubavu				
Normal<0.85 F,<0.90M	101(55.8)	54(29.8)	47(26.0)	1.491(1);.142
Central Obesity >=0.85 F,>=0.90M	80(44.2)	50(27.6)	30(16.6)	
Total	181(100)	104(57.5)	77(42.5)	

The analysis results portrayed the association of very high cut-off >=1 of WHR and gender. Rubavu WHR high cut-off was alone associated with gender and the proportion of high WHR levels was decreased. The very high level of WHR which is >=1, in Kicukiro was 17.0% and 8.8% for Rubavu with a significant gender relationship for only Rubavu, p=0.03. The findings are tabulated in Table 4.61 below.

Table 4.61: Bivariate analysis of body fat distribution of very high waist hip ratio (WHR) by gender of the study participants

Variable	**N(440)**	**Male**	**Female**	**Statistical test** X^2(df);*P*
Kicukiro				
Low WHR<1	215(83.0)	117(45.2)	78(37.8)	1.259(1);.170
Very High>=1	44(17.0)	28(10.8)	16(6.2)	
Total	259(100)	145(56.0)	114(44.0)	
Rubavu				
Low WHR<1	165(91.2)	91(50.3)	74(40.9)	4.064(1);.036
Very High>=1	16(8.8)	13(7.2)	3(1.7)	
Total	181(100)	104(57.5)	77(42.5)	

4.6.4. Dyslipidemia for all participants in the study area

Table 4.62 displayed the comparison of the mean value and standard deviation of total cholesterol, and triglyceride by age structure. The cholesterol and triglyceride were calculated in mg/dl and the F tests were also used to determine the age structure levels. The age difference was only significant for males <40years: 131.86±51.27, 40-50Years: 146.60±47.98 >50years: 162.26±47.3, p<0.001. The total mean value was for people <40years: 133.75±44.82, 40-50Years: 147.52±43.42, >50years: 157.37±49.03, p<0.001.

Table 4.62: Distribution of mean value of total cholesterol and triglycerides in mg/dl by age and gender

		Total Cholesterol (Mean ± SD)			Triglyceride (Mean ± SD)		
Age group	N=440						
		Male	Female	Total	Male	Female	Total
<40	157	155.97 ± 31.72	154.24 ±30.83	155.26 ± 31.27	131.86±51.27	136.42±33.88	133.75±44.82
40-50	151	167.48± 40.11	170.41 ± 37.51	169.0± 38.69	146.60±47.98	148.38±38.97	147.52±43.42
>50	132	170.99 ± 41.39	169.04 ± 32.90	170.28 ± 38.40	162.26±47.3	148.81±51.24	157.37±49.03
Total	440	164.41 ±38.12	164.57±34.82	164.48 ± 36.69	146.44±50.46	144.42±41.05	145.56±46.57
	F	*3.824*	*4.509*	*7.998*	*8.445*	*1.890*	*9.805*
Significance	*df*	*2*	*2*	*2*	*2*	*2*	*2*
	p	*0.023*	*0.01*	*<0.001*	*<0.001*	*<0.154*	*<0.001*

Table 4.63 displays the comparison of the mean value and standard deviation of high-density lipoprotein (HDL-c) and low-density lipoprotein (LDL-c) by age structure. The HDL and LDL were also calculated in mg/dl and the F tests were calculated for the age group's difference. There was a significant difference for males <40 years: 53.69±13.39, 40-50 years: 50.49 ±13.79, >50years: 46.47± 10.29, p=0.001.

Table 4.63: Distribution of mean value of high-density lipoprotein and low-density lipoprotein in mg/dl by age and gender

		High Density Lipoprotein (Mean ± SD)			Low Density Lipoprotein (Mean ± SD)		
Age group	N=440						
		Male	Female	Total	Male	Female	Total
<40	157	53.69±13.39	49.57 ± 8.82	51.98±11.86	76.53±28.70	78.97±30.27	77.54±29.29
40-50	151	50.49 ±13.79	47.64 ± 13.24	49.02±13.53	85.17±40.03	92.69±38.22	89.05±39.16
>50	132	46.47± 10.29	46.84 ± 10.61	46.60±10.37	91.64±38.42	90.41±35.68	91.19±37.31
Total	440	50.31 ± 12.87	48.10±11.24	49.35±12.23	84.16±36.09	87.4535.41	85.591±35.79
	F	*7.269*	*0.917*	*7.221*	*3.982*	*2.944*	*6.452*
Significance	*df*	*d2*	*2*	*2*	*2*	*2*	*1*
	p	*0.001*	*<0.402*	*0.001*	*0.02*	*0.05*	*0.002*

The bivariate analysis results showed that the association of lipid profile components was significantly associated with cardiovascular disease elevated risk (>10%) by the Framingham general risk score model, $p<0.001$. The high levels of lipid profile (total cholesterol, triglyceride, LDL-c, and low levels of HDL-c) are tabulated in Table 4.64.

Table 4.64: Distribution of relationship of lipid profile and cardiovascular diseases risk by Framingham general risk score

Variable	N(440)	FRGRS Model		Statistical test X^2(df);*P*
		Low CVD risk (<10%)	Elevated Risk (10-40%)	
Total Cholesterol				
Desirable :>200mg/dl	390(88.6)	384(87.3)	6(1.4)	68.409(2);.001
Borderline high :200-239mg/dl	30(6.8)	25(5.7)	5(1.1)	
High :>=240mg/dl	20(4.5)	11(2.5)	9(2.0)	
Total	440(100)	420(95.5)	20(4.5)	
Triglyceride				
Normal :<150mg/dl	285(64.8)	250(56.8)	35(8.0)	78.348(2);.001
Borderline high :150-199mg/dl	118(26.8)	66(15.0)	52(11.8)	
High 200-499mg/dl	37(8.4)	13(3.0)	24(5.5)	
Very high :>500mg/dl	0(0.0)	0(0.0)	0(0.0)	
	440(100)	329(74.8)	111(25.2)	
HDL-Cholesterol				
Low :High risk :<40mg/dl	89(20.2)	42(9.5)	47(10.7)	47.656(2);.001
Borderline low : 40-59mg/dl	284(64.5)	227(51.6)	57(13.0)	
High: No risk :>60mg/dl	67(15.2)	60(13.6)	7(1.6)	
Total	440(100)	329(74.8)	111(25.2)	
LDL-Cholesterol				
Optimal :<100mg/dl	313(71.1)	265(60.2)	48(10.9)	
Above optimal :100-129mg/dl	78(17.7)	47(10.7)	31(7.0)	68.479(3);.001
Borderline high:130-159mg/dl	42(9.5)	16(3.6)	26(5.9)	
High :160-189mg/dl	0(0.0)	0(0.0)	0(0.0)	
Very High : >=190mg/dl	7(1.6)	1(0.2)	6(1.4)	
Total	440(100)	329(74.8)	111(25.2)	

Table 4.65 expressed the analysis results for the association of lipid profile with cardiovascular risk diseases elevated risk (>10%) by the WHO/ISH model. These results also expressed a significant association of the lipid profile components to elevated cardiovascular diseases. The lipid profile high levels in elevated risk for total cholesterol were (n=9), borderline high (n=5); for triglyceride: borderline high (n=5), high (n=11); for HDL-C; borderline low (n=6) very low (n=13); for LDL-C: borderline high (n=10), high (n=0), very high (n=2) with the total sample of (N=440).

Table 4.65: Analysis of lipid profile and cardiovascular diseases risk by WHO/ISH score chart

Variable	N(440)	WHO/ISH Score model		Statistical test X^2(df);*P*
		Low CVD risk (<10%)	Elevated Risk (10-40%)	
Total Cholesterol				
Desirable :<200mg/dl	390(88.6)	384(87.3)	6(1.4)	93.724(2);.001
Borderline high :200-239mg/dl	30(6.8)	25(5.7)	5(1.1)	
High :>=240mg/dl	20(4.5)	11(2.5)	9(2.0)	
Total	440(100)	420(95.5)	20(4.5)	
Triglyceride				
Normal :<150mg/dl	285(64.8)	281(63.9)	4(0.9)	60.596(2);.001
Borderline high :150-199mg/dl	118(26.8)	113(25.7)	5(1.1)	
High : 200-499mg/dl	37(8.4)	26(5.9)	11(2.5)	
Very high :>500mg/dl	0(0.0)	0(0.0)	0(0.0)	
	440(100)	420(95.5)	20(4.5)	
HDL-Cholesterol				
Low :High risk :<40mg/dl	89(20.2)	76(13.3)	13(3.0)	26.078(2);.001
Borderline low : 40-59mg/dl	284(64.5)	278(63.2)	6(1.4)	
High: No risk :>60mg/dl	67(15.2)	66(15.0)	1(0.2)	
Total	440(100)	420(95.5)	20(4.5)	
LDL-Cholesterol				
Optimal :<100mg/dl	313(71.1)	309(70.2)	4(0.9)	
Above optimal :100-129mg/dl	78(17.7)	74(16.8)	4(0.9)	52.999(3);<.001
Borderline high:130-159mg/dl	42(9.5)	32(7.3)	10(2.3)	
High :160-189mg/dl	0(0.0)	0(0.0)	0(0.0)	
Very High : >=190mg/dl	7(1.6)	5(1.1)	2(0.5)	
Total	440(100)	420(95.5)	20(4.5)	

4.6.5 Metabolic syndrome for all participants in the study area

Table 4.66 displayed the analysis results of impaired and non-impaired components of the metabolic syndrome and its relationship to gender. Males presented impaired levels of fasting glucose, triglyceride, high level of blood pressure (HLBP), and high-density lipoprotein than females. Except for the waist circumference (WC). Three

metabolic syndrome components (HLBP, WC, HDL-C) were significantly associated with gender, p <0.05. Therefore, a total of Impaired Fasting glucose was 39.6%, and the male was 23.2%. Whereas the female was 16.4% with an insignificant gender relationship, p=0.487.

The Triglyceridemia for Metabolic Syndrome was 35.7%. The male population was 20.5% while the female was 15.2% with an insignificant relationship, p<0.817. The High Blood Pressure for Metabolic was 57.7%, the male was 36.6% while the female was 21.1% with a significant relationship, p=0.001. The HDL for abnormal Metabolic Syndrome was 39.3%. The male population was 10.9% while the female was 28.4% with a significant gender relationship p<0.001. Central obesity participants were 37.0%.

Table 4.66: Distribution of impaired and non-impaired components of metabolic syndrome by gender

Variables	N(440)	Gender		Statistical test
				X^2(df);*P*
Impaired Fasting Glycose for Metabolic Syndrome Classification FPG >= 100mg/dl(5.6mmol/l) or treatment(Rx)		Male	Female	
Normal<100mg/dl	266(60.4)	147(33.4)	119(27.0)	0.483(1);.487
IFG>=100MG/DL	174(39.6)	102(23.2)	72(16.4)	
Total	440(100%)	249(56.6))	191(43.4)	
Triglyceridemia for Metabolic Syndrom Classification(High level Triglyceride>=150mg/dl(1.7mmo/dl) or ttt for high TG				
High Triglycerides>=150mg/dl	157(35.7)	90(20.5)	67(15.2)	0.054(1);.817
Normal<150mg/dl	283(64.3)	159(36.1)	124(28.2)	
Total	440(100%)	316(71.8)	95(21.6)	
High Blood Pressure for Metabolic Syndrome Criteria HBP>= 130/85mmgh or Treatment of HBP+				
HLBP for MetS>=130/85mmgh	254(57.7)	161(36.6)	93(21.1)	11.293(1);.001
LLBP for MetS<130/85mmgh	186(67)	88(20.0)	98(22.3)	
Total	440(100%)	249(56.6)	191(43.4)	
HDL for Metabolic Syndrome Classification (low level men <40mg/dl(1mmol/l), low level women <50mg/dl(1.3mmol/l) or ttt for low HDL				
Normal	267(60.6)	201(45.7)	66(15.0)	96.563(1) ;<.001
Abnormal(Low levels)	173(39.3)	48(10.9)	125(28.4)	
Total	440(100%)	249(56.6)	191(43.4)	
Waist Circumference				
Central Obesity: F>88Cm,M>102Cm	163(37.0)	43(9.8)	120(27.3)	96.194(1) ;<.001
Normal: F<88Cm, M<102Cm	277(63.0)	206(46.8)	71(16.1)	
Total	440(100%)	249(56.6)	191(43.4)	

Table 4.67 presented the analysis results of the significant association of three components factor for metabolic syndrome (MetS) with elevated CVDs risk (>10%) by full model and reduced model of FGRS. The findings showed that high blood pressure (HBP), impaired fasting glucose (IFG), and triglyceride (TG) were significantly associated with an elevated cardiovascular diseases risk in Kicukiro plant. Whereas Rubavu showed two factors (HBP and TG) associated with cardiovascular diseases elevated risk (>10%), P<0.05. The triglyceride for Metabolic syndrome (TG MetS) was with the highest AOR: 12.879(4.48-36.99), P<0.001. This was followed by high blood pressure for Metabolic syndrome (HBP MetS) with AOR: 9.440(3.79-23.48), p<0.001.

Table 4.67: Association between metabolic syndrome components and level of CVDs Framingham general risk score

Variable	Full model (OR) CI95% (Risk>=10%)	p-value	Reduced model (AOR) CI 95% (Risk>=10%)	*P*-value
Kicukiro				
HBP MetS	10.564(4.79-23.29)	<0.001	9.440(3.79-23.48)	<0.001
IFG MetS	2.234(1.29-3.86)	0.004	2.202(1.15-4.20)	0.01
CO MetS	0.257(0.37-1.27)	0.2	-	-
TG MetS	2.637(1.47-4.70)	0.001	2.635(1.39-4.98)	0.003
HDL MetS	1.498(1.82-2.71)	0.1	-	-
Rubavu				
HBP MetS	8.636(2.75-27.11)	<0.001	8.720(4.399-26.99)	0.001
IFG MetS	1.036(0.41-2.58)	0.9	-	-
CO MetS	0.690(0.09-0.68)	0.006	0.345(0.11-1.00)	0.05
TG MetS	10.323(3.72-28.60)	<0.001	12.879(4.48-36.99)	<0.001
HDL MetS	1.855(0.67-8.09)	0.2	-	-

The full metabolic syndrome was calculated based on the fulfilled three or above of five metabolic syndrome factors. The respondents with the full metabolic syndrome were six times more likely to develop cardiovascular diseases at a risk> 10% than respondents who didn't have metabolic syndrome. However, results showed that

employees had an eight-fold likelihood to develop cardiovascular diseases while spouses had a five-fold likelihood to develop CVD at a risk >10%, p<0.001. The findings for full metabolic syndrome and Framingham general risk score elevated risk (>10%) are tabulated in Table 4.68 below.

Table 4.68: Association between full metabolic syndrome and level of risk of CVDs (FRSC)

Variable	**Full model (OR) CI95% (Risk>=10%)**	***P*-value**
Kicukiro		
Metabolic Syndrome	6.172(3.77-10.08)	<0.001
Rubavu		
Metabolic Syndrome	6.000(2.76-13.03)	<0.001
Employees		
Metabolic Syndrome	8.014(4.40-14.58)	<0.001
Spouses		
Metabolic Syndrome	5.284(2.31-12.07)	<0.001

Table 4.69 presented the analysis results of the components factor for metabolic syndrome (MetS) with elevated cardiovascular risk (>10%) by full model and reduced model of WHO/ISH model. All components were neither significant for the full model nor the reduced model.

Table 4.69: Association between metabolic syndrome components and CVDs risk (WHO/ISH)

Variable	Full model (OR) CI95% (Risk>=10%)	*P*-value
Kicukiro		
HBP MetS	7.479(-)	0.9
IFG MetS	1.887(0.47-7.47)	0.3
CO MetS	0.555(0.16-1.90)	0.3
TG MetS	3.126(0.76-12.82)	0.1
HDL MetS	0.500(0.14-1.77)	0.2
Rubavu		
HBP MetS	5.597(-)	0.9
IFG MetS	0.020(0.02-2.86)	0.2
CO MetS	0.663(0.10-4.35)	0.6
TG MetS	4.957(0.47-52.31)	0.1
HDL MetS	0.2(0.02-2.75)	0.2

Table 4.70 demonstrated the analysis results of the association of full metabolic syndrome toward cardiovascular diseases risk by the WHO/ISH model. The full model showed that the location (Kicukiro, Rubavu) and employees were significantly associated with cardiovascular diseases elevated risk, except for spouses, $p<0.05$. However, no significance was found in the reduced model.

Table 4.70: Association between full metabolic syndrome and CVDs risk (WHO/ISH)

Variable	Full model (OR) CI95% (Risk>=10%)	*P*-value
Kicukiro		
Metabolic Syndrome	25.203(3.31-191.49)	0.002
Rubavu		
Metabolic Syndrome	14.133(1.74-114.82)	0.01
Employees		
Metabolic Syndrome	16.707(3.70-75.31)	<0.001
Spouses		
Metabolic Syndrome	8.616(-)	0.9

The bivariate analysis results showed that there is an association between cardiovascular diseases elevated risk and metabolic syndrome. The findings are tabulated in Table 4.71 below.

Table 4.71: Distribution of metabolic syndrome by level of risk of cardiovascular diseases

Variable	N(440)	No MetS	MetS	Stastical test
				X^2(df); *P*
FGRS				
Low level risk (<10%)	329(74.8)	329(748)	112(25.5)	24.121(1);.02
Elevated level of risk (>10%)	111(25.2)	55(12.5)	56(12.7)	
Total	440(100%)	272(61.8)	168(38.2)	

4.7Cardiovascular diseases traditional risk factors and novel risk differentials among the study participants in the study area

4.7.1 Novel risk factors to cardiovascular diseases risk

Table 4.72 portrayed the analysis results of novel risk factors levels by location and gender. The proportions of the locations were at Kicukiro plant (n=259) and at Rubavu plant (n=181). Male employees presented elevated levels of C reactive protein and Serum uric acid than other groups, either for Kicukiro plant or Rubavu plant. The total high level >=7mg/dl of serum uric acid was 4.5% in Kicukiro. Thus, male employees were 4.1%, male spouses were 0.2%, and female spouses were 0.2%. whereas the total was 1.3% in Rubavu, 1.1% for male employees, and 0.2% for male spouses.

Table 4.72: Novel risk factors differentials by location, status, and gender of the study participants

Variable	n(259)	KICUKIRO Location			
		Employees		Spouses	
		Male	Female	Male	Female
C reactive protein					
Negative(hs-CRP<2mg/l)	232(52.7)	114(25.9)	25(5.7)	10(2.3)	83(18.9)
Positive(hs-CRP>2mg/l)	27(6.2)	21(4.8)	0(0.0)	0(0.0)	6(1.4)
Serum Uric Acid mg/dl					
Normal Uric Acid<7 mg/dl	239(54.3)	117(26.6)	25(5.7)	9(2.0)	88(20.0)
High Uric Acid>=7mg/dl	20(4.5)	18(4.1)	0(0.0)	1(0.2)	1(0.2)
Variable	n(181)	RUBAVU Location			
		Employees		Spouses	
		Male	Female	Male	Female
C reactive protein					
Negative(hs-CRP<2mg/l)	173(39.3)	93(21.1)	10(2.3)	5(1.1)	65(14.8)
Positive(hs-CRP>2mg/l)	8(1.8)	6(1.4)	1(0.2)	0(0.0)	1(0.2)
Serum Uric Acid mg/dl					
Normal Uric Acid<7 mg/dl	175(39.8)	94(21.4)	11(2.5))	4(0.9)	66(15.0)
High Uric Acid>=7mg/dl	6(1.3)	5(1.1)	0(0.0)	1(0.2)	0(0.0)

Table 4.73 portrayed the bivariate analysis results for novel risk factors associated with elevated cardiovascular diseases risk (>10%) by Framingham general risk score and WHO/ISH score chart model. The novel risk factors (C reactive protein and Serum uric acid) were significantly associated with cardiovascular diseases risk >10%, $p<0.05$. The proportion of 3.6% of study participants with high uric

acid>=7mg/dl had elevated CVD risk while 2.3% had low CVD risk, p<0.001 by Framingham general risk score model. The WHO/ISH model showed that 1.6% of study participants with positive CRP>2mg/l had elevated CVD risk while 6.6% had low CVDs risk, p<0.001. Around 0.9% of participants with High uric acid>=7mg/dl had thus elevated CVD risk while 5.0% had also low CVD risk, p=0.006.

Table 4.73: Bivariate analysis of novel risk factors by cardiovascular diseases risk based on FGRS and WHO/ISH score chart models

Variable	**N(440)**	**FGRS**		**Statistical test** X^2(df); *P*
		Low CVD risk (<10%)	Elevated CVD risk (10-40%)	
C reactive protein in mg/dl				
Negative(hs-CRP<2mg/l)	404(91.8)	317(72.0)	87(19.8)	35.693(1);<.001
Positive(hs-CRP>2mg/l)	36(8.2)	12(2.7)	24(5.5)	
Total	440(100)	329(74.8)	111(25.2)	
Serum Uric Acid mg/dl				
Normal Uric Acid<7 mg/dl	414(94.1)	319(72.5)	95(21.6)	9.315(1);<.001
High Uric Acid>=7mg/dl	26(5.9)	10(2.3)	16(3.6)	
Total	440(100)	329(74.8)	111(25.2)	
Variable	**n(440)**	**WHO/ISH Score Chart**		**Statistical test**
		Low CVD risk (<10%)	Elevated CVD risk (10-40%)	X^2(df);*P*
C reactive protein in mg/dl				
Negative(hs-CRP<2mg/l)	404(91.8)	391(88.9)	13(3.0)	20.059(1);<.001
Positive(hs-CRP>2mg/l)	36(8.2)	29(6.6)	7(1.6)	
Total	440(100)	420(95.5)	20(4.5)	
Serum Uric Acid mg/dl				
Normal Uric Acid<7 mg/dl	414(94.1)	398(90.5)	16(3.6)	7.482(1);.006
High Uric Acid>=7mg/dl	26(5.9)	22(5.0)	4(0.9)	
Total	440(100)	420(95.5)	20(4.5)	

Table 4.74 portrayed the multivariate analysis results of the association of novel risk factors (glycosylated hemoglobin (Hb1Ac), C reactive protein (CRP), and serum uric acid (SUA)) toward cardiovascular diseases risk. A part of the SUA, others were all significantly associated with cardiovascular diseases risk by Framingham general risk model>10%, p<0.05.

Table 4.74: Association between novel risk factors and CVDs risk (FRSC)

Variable	Full model (OR) CI95% (Risk>=10%)	p-value	Reduced model (AOR) CI 95% (Risk>=10%)	*P*-value
Kicukiro & Rubavu				
Normal HbA1c	Reference	-	Reference	-
Moderate HbA1c	3.029(0.93-9.79)	0.06	3.428(1.07-10.90)	0.03
High HbA1c	4.884(2.170-10.99)	<0.001	4.391(1.96-9.80)	<0.001
CRP	2.875(1.11-7.39)	0.02	4.482(2.03-9.88)	<0.001
Serum Uric acid	2.407(0.83-6.92)	0.1	-	-

Table 4.75. portrayed the multivariate analysis results of novels risk factors by WHO/ISH model. This result showed that one novel risk factor (CRP) was significantly associated with cardiovascular diseases risk only in the full model OR:5.53(1.33-22.95), p=0.01.

Table 4.75: Association between novel risk factors and CVDs risk (WHO/ISH)

Variable	Full model (OR) CI95% (Risk>=10%)	*P*-value
Kicukiro & Rubavu		
Normal HbA1c	Reference	
Moderate HbA1c	3.868(0.82-18.13)	0.08
High HbA1c	1.220(0.26-5.64)	0.7
CRP	5.535(1.33-22.95)	0.01
Serum Uric acid	1.043(0.21-5.03)	0.9

4.7.2 Traditional risk factors to cardiovascular diseases risk

Table 4.76 portrayed the analysis results of the traditional risk differentials for both Kicukiro plant (n=259) and in Rubavu plant (n=181) for the total sample of (N=440). Kicukiro male employees presented elevated levels for 11 traditional risk factors than other groups while female spouses dominated other groups for sedentary(n=41), obesity(n=40), central obesity(n=64), LDL-C(n=2). Rubavu male employees presented elevated levels of 13 traditional risk factors than other groups and followed by female spouses.

Kicukiro plant presented high levels of sedentarity >7hours than Rubavu plant where 9.3% of female spouses and 5.2% of male employees. Females dominated males for sedentarity >7hours risk factor while in Rubavu plant male employees dominated with 3.9% and followed by 3.4% of female spouses. Being with hypertension with >=140/90mmhg was dominated by male employees with 7.2% and followed by female spouses with 3.4%. Whereas Rubavu male employees dominated with 3.9% and followed by 1.6% of female spouses. Diabetes mellitus with >=125mg/dl was dominated by male employees with 4.7% and followed by female spouses with 2.7%, whereas in Rubavu plant male employees dominated with 3.9% and followed by female spouses with 3.4%. Smoking was dominated by male employees with 3.4% and followed by female spouses with 0.7% while Rubavu male employees was 1.3% and 0.7% of female spouses were smokers. Obesity>=30 BMI was dominated by female spouses with 9% and followed by 4.3% of male employees in Kicukiro while Rubavu male employees and female spouses were equally distributed at the same level of 3.6% of obesity. Central obesity>=94cm for females, >=102cm for Males, was dominated by 15.5% of female spouses and followed by male employees with 7% and female employees with 4% of central obesity while also in Rubavu 8% were female spouses and 2.5% of male employees.

Table 4.76: Traditional risk factors differentials by location, status, and gender of the study participants

Variable	n(440)	KICUKIRO PLANT LOCATION			
Traditional Modifiable risk factors to CVDs		Employees		Spouses	
		Male	Female	Male	Female
Sedentary>7hours	74	23(5.2)	9(2.0)	1(0.2)	41(9.3)
Hypertension 140/90mmgh and or on treated	57	32(7.2)	6(1.3)	4(0.9)	15(3.4)
Diabetes mellitus BS:125mg/dl or treated	36	21(4.7)	3(0.7)	0(0.0)	12(2.7)
Smoking	20	15(3.4)	0(0.0)	2(0.5)	3(0.7)
Obesity>=30 BMI	68	19(4.3)	7(1.6)	2(0.5)	40(9.0)
Central obesity>=94cm F, >=102cm M	113	30(7.0)	18(4.0)	1(0.2)	64(15.5)
Variable	**N(440)**	**KICUKIRO PLANT LOCATION**			
High stress for 270 workers only	44	39(9.0)	5(1.1)	-	-
Physical inactivity	186	88(20.0)	18(4.1)	10(2.3)	70(15.9)
Lack of fruits consumption	67	37(8.4)	10(2.2)	5(1.1)	15(3.4)
Lack of vegetable consumption	7	3(0.7)	0(0.0)	3(0.7)	1(0.2)
Alcohol consumption	204	118(27.0)	22(5.0)	9(2.0)	55(12.5)
Dyslipidemia					
Hypercholesterolemia>240mg/dl	13	7(1.6)	0(0.0)	0(0.0)	6(1.4)
Hypertriglyceridemia>150mg/dl	96	52(11.8)	7(1.6)	4(0.9)	33(8.5)
High LDL-C	3	1(0.2)	0(0.0)	0(0.0)	2(0.5)
Low HDL-C	46	24(5.5)	4(0.9)	2(0.50	16(3.6)
Traditional risk factors by status and gender					
Variable	**N(440)**	**RUBAVU PLANT LOCATION**			
		Employees		Spouses	
Traditional Modifiable risk factors to CVDs		Male	Female	Male	Female
Sedentarity>7hours	34	17(3.9)	2(0.5)	0(0.0)	15(3.4)
Hypertension 140/90mmgh and or on treated	25	17(3.9)	1(0.2)	0(0.0)	7(1.6)
Diabetes mellitus BS:125mg/dl or treated	34	17(3.9)	2(0.5)	0(0.0)	15(3.4)
Smoking	9	6(1.3)	0(0.0)	1(0.2)	3(0.7)
Obesity>=30 BMI	35	16(3.6)	3(0.7)	0(0.0)	16(3.6)
Central obesity>=94cm F, >=102cm M	50	11(2.5)	3(0.7)	1(0.2)	35(8.0)
High stress for 270 workers only	37	37(8.4)	0(0.0)	-	-
Physical inactivity	117	59(13.4)	4(0.9)	4(0.9)	50(11.4)
Lack of fruits consumption	44	27(6.1)	3(0.7)	0(0.0)	14(3.1)
Lack of vegetable consumption	3	1(0.2)	0(0.0)	0(0.0)	2(0.5)
Alcohol consumption	99	66(15.0)	2(0.5)	4(0.9)	27(6.1)
Dyslipidemia					
Hypercholesterolemia>240mg/dl	7	5(1.1)	0(0.0)	0(0.0)	2(0.5)
Hypertriglyceridemia>150mg/dl	59	32(7.3)	4(0.9)	2(0.5)	21(4.7)
High LDL-C	4	3(0.7)	0(0.0)	0(0.0)	1(0.2)
Low HDL-C	43	24(5.5)	3(0.7)	0(0.0)	16(3.6)

Table 4.77 summarized the comparative view of the cardiovascular diseases' absolute 10-year risk prediction and classification by location. The status was 44.5% of employees versus spouses with 30%. All were classified as having low cardiovascular diseases risk (<10%) by FGRS. Whereas WHO/ISH score chart classified 57.7% of employees versus 37.7% of spouses as having low cardiovascular diseases risk (<10%). The absolute cardiovascular risk levels by location showed that Kicukiro plant employees and spouses presented higher cardiovascular diseases absolute risk than Rubavu plant employees and spouses by the FGRS model. However, the WHO/ISH model showed that Rubavu and Kicukiro spouses had the same cardiovascular disease absolute risk level while Kicukiro plant employees remained at high risk than Rubavu plant employees.

Table 4.77: Cardiovascular diseases absolute risk level by study participants and location

Models	n(%)	Rubavu		Kicukiro	
FGRS	440	Employee	Spouse	Employee	Spouse
Low risk (<10%)	328(74.5)	88(20)	63(14.3)	108(24.5)	69(15.6)
2nd level risk (10-20%)	60(13.6)	16(3.6)	6(1.3)	21(4.7)	17(3.8)
3rd level risk (20-30%)	28(6.3)	2(0.4)	1(0.2)	17(3.8)	8(1.8)
4th level risk (30-40)	24(5.4)	4(0.9)	1(0.2)	14(3.1)	5(1.1)
5th level of risk (>40)	0(0.0)	0(0.0)	0(0.0)	0(0.0)	0(0.0)
Total	440(100)	110(25.0)	71(16.1)	160(36.3)	99(22.5)
WHO/ISH					
Low risk (<10%)	420(95.4)	106(24.0)	69(15.6)	148(33.6)	97(22.0)
2nd level risk (10-20%)	15(3.4)	2(0.4)	2(0.4)	9(2.0)	2(0.4)
3rd level risk (20-30%)	1(0.2)	1(0.2)	0(0.0)	0(0.0)	0(0.0)
4th level risk (30-40%)	3(0.6)	1(0.2)	0(0.0)	2(0.4)	0(0.0)
5th level of risk (>40%)	1(0.2)	0(0.0)	0(0.0)	1(100.0)	0(0.0)
Total	440(100)	110(25.0)	71(16.1)	160(36.3)	99(22.5)

The multivariate analysis results for traditional risk factors to cardiovascular diseases risk were divided into three levels: four unmodifiable risk factors, three workplace modifiable risk factors, and fifteen general modifiable risk factors. The findings showed that after the adjusted odds ratios (AOR) with the reduced model; three

unmodifiable factors, two modifiable workplace factors, and eight modifiable general factors were significantly associated with elevated CVDs risk >10%, 95%CI, p<0.05.

Three of the non-modifiable risk factors were statistically significant by the FGRS model. Gender factor was associated with CVDs risk with AOR= 0.541(0.32-0.90), p=0.01. Age structure from 40-50 years with AOR= 6.836(2.90-16.09), p<0.001. Age structure >50 years with AOR= 19.517(8.40-45.34), p<0.001. The family member who suffered the CVD with AOR=0.333(0.20-0.55), p<0.001. Only two workplace modifiable factors (Radiation and high stress) were statistically significant with AOR = 0.360(0.15-0.86), p=0.02, and AOR=21.398(2.65-172.59), p=0.004, respectively.

The eight modifiable factors (Physical inactivity, insufficiency of fruit and vegetable intake, smoking, exceeding four drinks for women and five for men, diabetes, and hypertension) were scientifically and statistically significant. The significance was observed for both full model and reduced model. Although exceeding four drinks for women and five drinks for men was statistically significant, alcohol consumption in 30 days was not significant alone. Its significance was observed in the full and reduced model on only the point of participants who have not taken alcohol in 30 days. Hence, not taking alcohol, slightly increased the cardiovascular risk with AOR=0.2, p=0.005. However, exceeding four and five drinks was associated with cardiovascular diseases risk with AOR=11.162, p<0.001. Diabetes and physical inactivity were highly associated with cardiovascular disease risk, AOR=29, p<0.001, AOR=13, p=0.001, respectively. The findings of the multivariate analysis using the Framingham general risk score model, are tabulated in Table 4.78 below.

Table 4.78: Association between traditional risk factors and cardiovascular diseases (FGRS)

Variable	Full model (OR) CI95% (Risk>=10%)	*P*-value	Reduced model (AOR) CI 95% (Risk>=10%)	*P*-value
Unmodifiable factors				
Gender	0.558(0.33-0.94)	0.02	0.541(0.32-0.90)	0.01
Age structure				
<40 years	Reference		Reference	
40-50years	6.823(2.89-16.07)	<0.001	6.836(2.90-16.09)	<0.001
>50years	19.733(8.48-45.91)	<0.001	19.517(8.40-45.34)	<0.001
Family member who suffer CVD	0.353(0.20-0.60)	<0.001	0.333(0.20-0.55)	<0.001
Family member who died from CVD	0.773(0.34-1.74)	0.5	-	-
Modifiable Workplace factors				
Radiation	0.392(0.15-1.00)	0.052	0.360(0.15-0.86)	0.02
Chemical handling	1.177(0.60-2.27)	0.6	-	-
Stress for only 270 employees				
Low level stress	Reference		Reference	
Moderate stress	2.465(0.30-19.68)	0.3	-	-
High stress	20.98(2.59-169.51)	0.004	21.398(2.65-172.59)	0.004
Modifiable general factors				
Location(Urban place)	1.395(0.65-2.95)	0.3	-	-
Status of participants(Employee)	1.578(0.74-3.34)	0.2	-	-
Sedentarity	1.040 (0.20-5.19)	0.9	-	-
Physical inactivity <600MET	13.150(1.54-111.6)	p0.01	13.496(1.63-111.29)	0.01
Low fruits intake	4.303(1.87-9.90	0.001	4.410(1.96-9.90)	<0.001
Low vegetable intake	6.729(1361-28.05)	0.009	7.839(1.90-32.27)	0.004
Tobacco smoke	3.754(1.01-13.95)	0.04	4.546(1.14-16.60)	0.02
Second hand smoke at home	1.146(0.43-3.04)	0.7	-	-
Second hand smoke at workplace	1.126(0.15-8.01)	0.9	-	-
Alcohol consumption in 30 days	0.223(0.09-0.55)	0.01	0.275(0.12-0.68)	0.005
Times exceeded 4 drinks for female	11.728(4.73-29.06)	<0.001	11.162(4.58-27.15)	<0.001
And 5 drinks for male in 30 days				
General obesity	1.047(0.20-5.33)	0.9	-	-
Central obesity(WHR)	1.462(0.63-3.37)	0.3	-	-
Diabetes	22.854(6.79-76.83)	<0.001	29.689(9.44-93.35)	<0.001
Previous HTN	4.105(1.86-9.06)	<0.001	6.440(3.36-12.32)	<0.001
Updated HTN	2.241(0.82—6.07)	0.1	-	-

Table 4.79 displays the multivariate analysis results of the traditional risk factors towards elevated cardiovascular diseases absolute risk >10% by the WHO/ISH model. The findings also presented four unmodifiable factors, three modifiable workplace factors, and fifteen modifiable general risk factors. After the factor's adjustment, the reduced model showed that only two unmodifiable risk factors and four modifiable general risk factors were significantly associated with cardiovascular diseases elevated risk >10%, 95% CI, $p<0.05$.

The reduced Model of the WHO/ISH score chart showed that two of the non-modifiable risk factors were statistically significant. Age structure from 40-50 years with AOR= 10.454(1.31-83.21), $p<0.02$, Age structure >50 years with AOR= 11.519(1.43-92.74), $p<0.02$. Having a family member who died from the CVDs was significant with AOR=0.233(0.08-0.65), $p<0.006$.

Of the eight modifiable factors only 3 factors (Alcohol intake, diabetes, and hypertension with previous blood pressure threshold) were statistically significant. This was observed on the full model and reduced model. However, exceeding four and five drinks was associated with cardiovascular diseases risk with AOR=5.243(1.08-25.31), $p=0.03$. Diabetes and hypertension as the prominent risk factors for cardiovascular diseases, were highly associated with cardiovascular diseases risk, AOR=3.620(1.29-10.12), $p=0.01$, AOR=15.992(3.50-72.98), $p<0.001$, respectively. All the factors' associations are tabulated in Table 4.79.

Table 4.79: Association between traditional risk factors and cardiovascular diseases (WHO/ISH)

Variable	Full model (OR) CI95% (Risk>=10%)	*P*-value	Reduced model (AOR) CI 95% (Risk>=10%)	*P*-value
Unmodifiable factors				
Gender	0.678(0.24-1.85)	p=0.4	-	-
Age structure				
<40 years	Reference		Reference	
40-50years	9.607(1.19-77.18)	0.03	10.454(1.31-83.21)	0.02
>50years	10.155(1.25-82.36)	0.03	11.519(1.43-92.74)	0.02
Family member who suffer CVD	0.502(0.17-1.42)	0.1	-	-
Family member who died from CVD	0.352(0.10-1.14)	0.08	0.233(0.08-0.65)	0.006
Modifiable Workplace factors				
Radiation	0.224(0.02-1.86)	0.1	-	-
Chemical handling	0.776(0.26-2.24)	0.6	-	-
Stress for only 270 employees				
Low level stress	Reference		Reference	
Moderate stress	8.077(-)	0.9	-	-
High stress	2.00(-)	0.9	-	-
Modifiable general factors				
Location(Urban place)	0.750(0.20-2.78)	0.6	-	-
Status of participants (Employee)	2.835(0.80-10.02)	0.1	-	-
Sedentary	0.635(0.07-5.22)	0.6	-	-
Physical inactivity <600MET	4.505(-)	0.9	-	-
Low fruits intake	1.617(0.36-7.27)	0.5	-	-
Low vegetable intake	1.034(0.10-16.9)	0.9	-	-
Tobacco smoke	4.141(1.01-16.9)	0.04	-	-
Second hand smoke at home	1.160(0.02-1.26)	0.08	-	-
Second hand smoke at workplace	2.196(0.27-17.61)	0.4	-	-
Alcohol consumption in 30 days	0.136(0.02-0.89)	0.03	0.192(0.03-0.99)	0.04
Times exceeded 4 drinks for female	4.901(0.81-29.48)	0.08	5.243(1.08-25.31)	0.03
And 5 drinks for male in 30 days				
General obesity	1.797(0.21-15.30)	0.5	-	-
Central obesity(WHR)	1.719(0.36-8.09)	0.4	-	-
Diabetes	5.132(1.31-20.00)	0.01	3.620(1.29-10.12)	0.01
Previous HTN	15.133(1.69-135.38)	0.01	15.992(3.50-72.98)	<0.001
Updated HTN	0.775(0.04-14.15)	0.8	-	-

CHAPTER FIVE

DISCUSSION, CONCLUSION AND RECOMMENDATION

5.1 Levels of the 10-year cardiovascular diseases risk predicted and models comparison among the study participants in the study area

Cardiovascular diseases risk predictors were the known cardiovascular disease risk factors, which have been used in this study to predict 10-year risk. All the used risk factors were with incontrovertible evidence of being associated with cardiovascular diseases (Yeates, 2015). Their selection showed how they were technically and economically crucial in the primary follow-up study, and accurately predicted the needed result. However, a choice of local predictors that are associated with cardiovascular diseases outcome could be included for future model creation. WHO/ISH Score chart applied six (Kaptoge et al., 2019), predictors' age, gender, smoking, TC, BP, diabetes, and five predictors, where there is no cholesterol capacity measurement (Savitharani, 2016; Arvind, 2015). Whereas FGRS applied eight predictors, which were age, gender, HDL, TC, Untreated and treated SBP, smoking, and diabetes (Yangfeng et al., 2006; D'Agostino et al., 2008; D'Agostino, 2011). The use of all these predictors was consistent with other studies that applied these two models. However, there are other multiple studies (Selvarajah et al., 2014) that used different predictors to predict the future conditions and diseases of their local population (Mansell, 2014).

The overall study results displayed a cardiovascular disease risk predicted by FGRS and WHO/ISH models. Males presented high CVDs Risk (16.1%), (2.7%) than females (9.3%), (1.5%) and The CVDs risk was elevated for Employees (16.8%), (3.6%) than for spouses (8.6%), (0.9%), respectively. Urban participants presented elevated CVDs risk (18.6%), (3.1%) than rural participants (6.8%), (1.3%), respectively. These findings were consistent with some studies. However, other studies exposed a cardiovascular disease transition such as stroke in the US, where rural and urban stroke trends crosscut in 2007 (Ambar, 2014). Employees' CVDs risk was high than the risk in spouses and high in men than in women (O'Neil,

2018). This difference was due to Gender-specific, workplace, and home Stress increases, (Regitz-Zagrosek, 2015) for workers. Additionally, the accumulation of most of the factors of the cardiovascular diseases for urban than rural populations (Miranda, 2011; Auley, 2013). Moreover, the current almost study's findings explain the rise of cardiovascular disease risk factors in rural communities (Prabhakaran, 2016).

This study's findings showed a minimal or fair level of agreement of 0.25 between the Framingham general risk score and the WHO/ISH score chart. This is not acceptable in the model prediction agreement by Cohen kappa due to the models' lack of discriminatory capacity for fulfilling the accuracy requirement of a test. A classifier or a model requires the interrater reliability coefficient to be perfect. It should be at least 0.8 for the accurate agreement between the used two models (FGRS and WHO/ISH Score chart) to be, interchangeably applied to the Rwandan population (Mary, 2012). The Framingham general risk score (FGRS) predicted 25.5% of the population to be at elevated cardiovascular disease absolute risk in the coming 10years (>=10%). Whereas the WHO/ISH score chart predicted only 4.6% of the population to be at elevated risk of (>=10%). This result showed a high predictable difference of 20.9% risk. This underlined a suspicion of underprediction of the WHO/ISH Score chart (WHO, 2007) and over prediction of FGRS (Buitrago et al., 2011). The correlated status of ROC curve performance with respectively, FGRS and WHO/ISH score chart, was 0.887 AUC, 0.847AUC all with a p-value <0.001, which showed a perfect performance. Although the ROC curve performance capacity was perfect, the interrater agreement of 0.25 Cohen kappa coefficient was low and consequently inadequate agreement (Mary, 2012). This could demote its ROC curve accuracy for underdiagnosis or overdiagnosis (Bantis, 2016).

Worldwide stroke and heart attack equated to 85% of all deaths caused by cardiovascular diseases (WHO, 2017). A recent multi-center study on the burden of stroke in Rwanda showed that 2.1% of all received patients had a stroke, and 61% of them died (Nkusi et al., 2017). Whereas these study findings showed that the 10-year predicted risk of fatal and non-fatal CVDs including stroke was between 4.6% and 25.5% of elevated Risk >=10% by WHO/ISH and FGRS models, respectively. This

may be a good future indicator if applied to the whole population for pro-actively planning preventive measures for lessening the burden of stroke and other CVDs in Rwanda. Reaching the preventive strategy goal could only be possible by setting practical barriers to changeable risk factors. Moreover, improving personal cardiovascular disease awareness risk level, and health-seeking behavior for heightening early health service utilization (Herbert & Moses, 2019) to reach the equipped stroke unit in due time (Aldo, 2018).

Cardiovascular prediction models including other disease prediction models are of great importance for current sound scientific health events and conditions forecasting and prevention (Mustafa, 2018). It is now the platform for creating strategic countermeasures for improving the population's quality of health. Additionally, reducing the physical, psychosocial, and economic burden of diseases on LMIC including Rwanda. Such diseases caused around 300 DALYs according to WHO, 2015 and 61% of death of all strokes received patients and 14% of worse disability (Nkusi et al., 2017; Herbert & Moses, 2019).

5.2 Proportion of behavioral factors associated with cardiovascular diseases among the study participants in the study area

The discussion of the second objective findings was organized regarding the seven modifiable behavioral risk factors. This discussion shows a better and deep clarification of behavioral factors' contribution to the cardiovascular diseases risk.

5.2.1 Level of smoking behavior for participants in the study area

The smoking prevalence as displayed in Table 4.5 showed a low smoking level of 6.8% of all smokers and 6.6% of daily smokers. The low second-hand smoking that occurred at home was 15.8% and 4% at work, while shisha was at 1.1%. However, a study done in SSA showed that Rwanda had a high smoking prevalence level from 2007 to 2014 of 20.9% for males and 12% for females (Brathwaite, 2015). In addition, in 2013, another study carried out in Rwanda showed a low prevalence of 8% (95%CI: 7.08–9.01) for a population between 15-34 years old (Habiyaremye, 2019). Shisha use in 2016/2017, among Kigali university students, was 26.1%

(Omotehinwa, 2018). However, the 2015 global health observatory repository showed 19.2% for males and 7.2% for females in Rwanda (WHO, 2015). The findings showed that smoking is high in males than in females (Table 4.6) where current smokers are 5.5% of males and 1.4 of females, p=0.005. The daily smoking for males was 5.2% and 1.4% for females, p=0.008. This is in agreement only with the significance of other studies carried out in Thailand where 67% of males smoked daily while the female was 41.9%, p=0.002 (Dujrudee, 2018). It is equally in agreement with another study done in Namibia on the prevalence of smoking where it was reported to be 8.8% (8.1–9.5) (Zhifei, 2019)).

5.2.2 Alcohol consumption behavior of the study participants

Alcohol consumption appeared to be high due to the age difference in this study with a mean age of 44.84(SD:8.2) years. Table 4.7 showed that 68.9% consumed alcohol in the last 30 days and 24.3% used to consume alcohol daily. Whereas WHO reported in 2016 that alcohol consumption in Rwanda for total consumption per capita of people of 15 years and above. The results were also 15% for beer and 3% for liquor and <1% for wine. However, the data was slightly low compared to the study done in Nigeria on a population with 39.10 (12.06) mean age. The study showed that 88.9% were current drinkers (Lasebikan, 2018). This is high compared to the study done in Namibia where the prevalence of alcohol take was 53.1% (51.5–54.6) (Zhifei, 2019).

Overall findings on alcoholic drink consumption showed that beer consumption for males was 44% with 2.8 mean standard drinks a day. Whereas the females' beer consumption was 23.3% with 1.6 mean standard drinks a day (Table 4.8). Wine consumption for males was 28.3% with 0.2 mean standard drink a day while the female's wine consumption was 12.4% with 0.2 mean standard drink a day (Table 4.9). The liquor consumption for males was 19.7% with 0.2 mean standard drink a day while the females' liquor consumption was 4.1% with 0.1 mean standard drink a day (Table 4.10). The findings of alcohol beverage average showed that males slightly took more units than females for beer and liquor but took an equal standard drink on wine. A high percentage of males took more standard drinks than females. This is in line with another study carried out in South Africa on alcohol use. This

showed that the prevalence of alcohol consumption was high for men than women with 41.5% versus 17%, respectively (Peltzer, 2011).

The findings presented the mean value of the largest intake for one occasion (Table 4.11) and taking 5drinks or more for males and taking 4 drinks or more for women (Table 4.12). This was highly and significantly varied by age for females. This showed that women took more drinks as long as their age increased, p=0.001, p=0.006 respectively. Therefore, these findings were consistent with Brazilian study results, where alcohol intake prevalence was high in men than in women in all age groups (Cynthia, et al., 2011). The study showed that a high percentage of men took more alcohol than women (71.5% versus 28.5%). However, the gender relationship was not significant in the times they exceeded the above drinks, p=0.207. However, the alcohol intake percentage significantly increased with age increase. This was shown in results, where participants <40years were 25.2%, 40-50 years were 35%, and>50years were 39.8% with p=0.02 (Table 4.13) (Lasebikan, 2018).

5.2.3 Level of fruits intake (weekly and servings intake) for participants in the study area

The overall findings on fruit consumption showed that 24.8% reported not eating fruits in a past typical week. The majority of participants of 53.3% ate fruit one day a week and 41.6% ate very insufficient fruits (under one serving). In addition, fruits intake insufficiency was low compared to South African fruit intake insufficiency of 68.5% (Peltzer, 2011). Around 38.7% of male employees reported eating fruits at worksite restaurants. However, a high proportion of male employees did not eat fruits with 14.3% of male employees versus 6.4% of female spouses. Although male employees dominated in eating more servings a day, female spouses dominated in eating fruits by two and more days a week with 12.5% than male employees with 7.5% (Table 4.14).

These findings were low in comparison with the WHO cut-off that recommended eating five servings of fruits a day. Therefore, none took fruits at the WHO recommended level. Furthermore, the results were in line, in terms of days of fruit consumption, with the steps study done in Rwanda in 2013. This study showed that

the mean day of fruit consumption was 1.6 days a week. In addition, many countries were found to not comply with the recommended threshold of a minimum level of consumption. This threshold is a daily 80-gram serving, or 400 grams of fruits and vegetables a day (FAO, 2017; Dhandevi, 2015).

5.2.4 Level of vegetable's intake (weekly and servings) for participants in the study area

The only percentage of no vegetable consumption was 2.8%, which was very low compared to fruit-eating by the study participants. However, the population style is to cook a few vegetables, mix them with water and beans, and serve them as vegetables (Imboga). This cooking way undermines the usual way of counting vegetable servings. Female spouses consumed vegetables for three and more days than male employees. However, male employees dominated in eating vegetables in two days and less. Although many people reported vegetable intake during five days of the week, the servings are insufficient. Almost 100% did not meet the recommended five portions of vegetable intake a day. Moreover, another study showed that 82% didn't meet the recommended daily intake.

It was 44.2% consumed vegetables daily in Tanzania (Beverly et al., 2018) while 87.8% didn't meet the recommended portions in Uganda (Kabwama, 2019). According to WHO, 2020 the availability of phytochemicals, polyphenol, potassium, dietary fibers, and flavonoids in fruits and vegetables are relevant elements in homocysteine and other free radicals' moderation. Hence, they provide the ability to prevent body parts' oxidative damage. Furthermore, they extend the prevention of coronary heart diseases and other cardiovascular diseases. Hence, their use will be important in strengthening the cardiovascular health of people.

5.2.5 Level of Oil intake for study participants in the study area

Three types of oil are the most used by the study participants. The sunseed oil dominated with 38.4% and followed by Mukwano oil at 17.7% and Golden oil at 12.5%. Then Palm 41.6%, sunflower 40.7%, and Soybean 9.8% were also the three prominent oil sources. A big proportion of oil components was unmarked on oil

bottles tag: total fat 43.4% unmarked, cholesterol content 51.8% unmarked, Mono-unsaturated fat 84.1% unmarked. Polyunsaturated 77% unmarked, saturated fat 78% unmarked, hydrogenated 98% unmarked, trans-fat 93.4% unmarked, and a Heating smoke point 74.1% unmarked (table 4.16).

The negative impact of Trans-fatty acids on cardiovascular life and mortality was demonstrated many years ago (Iqbal, 2014). The findings showed that trans-fat was unmarked (unlabeled) on the oil containers at 93.4% of all consumed oil. The trans-fatty acids must be traced and marked on the oil container. In addition, they must be reduced under the WHO recommended rate of 4% to save the life of the consumers. The 25% of cardiovascular risk increases for every 2% energy of hydrogenated fat (Nichols et al., 2012). Most of the used oil was processed from palm at a rate of 41.6%. Palm oil contains a part of mono-unsaturated fatty acids (Oleic, Linoleic acids). The last elements are subjected to be reduced in the manufacturing process and remain the saturated fatty acids parts (Palmitic, stearic, lauric, myristic). This has a strong harmful effect on cholesterol increase and on cardiovascular health (Iqbal, 2014).

It was observed that Palmitic acid was associated with myocardial infarction risk (MI) (OR: 2.76; 95%CI = 1.39–5.47 (Ismail, 2018; Sowmya, 2019). Around 74.1% of the oil used was not labeled regarding the heating smoking points, which may indicate to the consuming community not to overheat the oil. The fact that repeatedly heating oil use (Ng, 2014) increases lipid peroxidation and free radicals. These factors harm the heart and vessels through cholesterol and triglyceride accumulation. Hence, this leads to atherogenic plaque and aggravates cardiovascular diseases (Ganesan, 2018).

5.2.6 Physical activities for the study participants based on frequency, duration, and intensity of energy expenditure

Physical activity execution is the required lifestyle to strengthen the cardiovascular system (Saeid, 2012) and cognitive life (Leyland, 2019). In addition, it helps to prevent related diseases through endothelial function regeneration and mitochondrial adaptation (Matthew, 2018). However, the study findings showed in (Table 4.17) that

98.5% didn’t perform vigorous-intensity physical activity at work during the whole past week. And 87.5% couldn’t perform moderate-intensity physical activity at work. This showed a high level of inactivity at work concerning moderate and vigorous physical activity. Although there was a difference between exerting physical energy at work and walking to or from work, 87% couldn’t walk or bicycle to or from work.

The proportion of 13% of people who performed bicycling and walking to work used a mean time of 48.9min/person and 4.4 days/person. This is good and similar to other studies compared to 150min of moderate to vigorous physical activity (MVPA) required a week for a person (Table 4.18) (Pedro, 2018). (Table 4.19), (Table 4.20) showed that leisure, sport, and recreational vigorous-intensity physical activity were performed by 6.8% while moderate was 15% of the study participants. Moreover, more time was also significantly performed by men and 93.2% and 85% were inactive for vigorous and moderate-intensity sport, respectively.

At least 3.9% of moderate (600-1500MET) per week and 1.4% high energy expenditure (>1500MET) per week were inferior for 40 years while participants of 40-50 and > 50 years were 2.5% and 2.0% for moderate energy expenditure. At least 3.9% of moderate (600-1500MET) per week and 1.4% high energy expenditure (>1500MET) per week were inferior for 40 years while participants of 40-50 and > 50 years were 2.5% and 2.0% for moderate energy expenditure. The high-energy expenditure was 0% for both the age range of 40-50 and >50 years (Table 4.21).

Rubavu study participants presented an elevated percentage of 6.6% and 1.1% while Kicukiro presented 1.8% and 0.7% for moderate and high-energy expenditure, respectively. Employees spent more energy than spouses whereas the elevated percentage of employees are moderately and highly spend their weekly energy. This is obviously the same for males in comparison with females where 6.4% and 1.8% of males were more than 2.0% and 0.0% for females for moderate and high-energy expenditure, successively.

The obvious difference was marked between Kicukiro and Rubavu. The energy expenditure in those two locations was justified by the semi-urban region where people can walk to work and for other life-related requirements. This research

finding is slightly above the result of a study carried out in Rwanda for Government office employees in Kigali with 600 participants. The study showed that 61.1 % of the participant were not sufficiently active.

There was a concordance to the decrease of physical activity by age and observation of a slight increase in physical activity in men than in women (Mukaruzima, 2020). This study showed similar findings to the 2015 Rwanda non-communicable diseases report, where men were with high physical activity levels than women, as well as younger than older participants (WHO, 2015). In addition, the level of physical activities and low energy expenditure were found to be associated with cardiovascular diseases, (Ahad, 2016; Carl, 2019).

5.2.7 Sitting time /sedentarity for the participants in the study area

Sedentarity is a critical modern risk factor for metabolic diseases as well as cardiovascular diseases (Mohammad, 2018). Table 4.23 showed that 6.6% were required to sit for more than 10 hours and 21.6% were required to sit between 6 and 10 hours. Although workplace-sitting time was reported, it was never compensated by outside work, but rather increased (Clemes, 2014). Therefore, discussed strategies such as sit-stand desks, active workstations, walking during breaks, walking meetings, and counseling could be crucial if evidently applied. This can aid to reduce the amount of sitting time at work and outside work to prevent long sitting negative effects (Shrestha et al., 2018).

The association of sedentarity levels to metabolic syndrome was OR: 2.686 (1.42-5.06) p=0.002 for the moderate sitting period while the high sitting time period was 8-fold. This result association was slightly high in the regard to moderate sitting time and very higher in comparison to the study done in South Korea. The study showed a higher OR of 1.21-fold for participants who sat >7 hours than those who sat <7 hours (Jin et al., 2016). Although there was a big difference between the OR of the moderate and high sitting periods concerning Metabolic syndrome (MetS). The association of moderate and high sitting periods with cardiovascular disease risk was OR: 3.238(2.238-6.050) P<0.001, 3.772(1.718-8.285) p=0.001, 95% CI, respectively (Table 4.24). The findings showed high OR regarding the association of Metabolic

syndrome than CVD risk. The relationship of all components of metabolic syndrome (IFG, HBP, Triglyceride, Cholesterol, and Central obesity) with sedentarity levels was highly significant with p<0.0001. The relationships with other cardiovascular disease factors (Age, waist circumference, the status of participants: being employee or spouse) (Table 4.26) were all significant except for gender. These findings were in line with a Poland study, where the relationship between long sitting time was significant with central obesity. The findings indicated there was a statistically significant relationship between low physical activity and high triglyceridemia, low high-density lipoprotein, high cholesterol, and elevated blood pressure (Edyta, 2018).

5.3 Work condition factors to CVDs risk in the study area

5.3.1 Proportion of working condition factors for workers in the study area

5.3.1.1 Organizational hazard (Shift workers and Night workers)

The organizational hazards results showed that 46.3% regularly did shift work while 43.7% did night shift work. A percentage of 97.4% were males. 40.6% were under 40 years, 32.2% were between 40-50years while 27.1% were >50years (Table 4.27). The night shift and the occupation presented a substantial relationship with p<0.001. In the total of 43.7% of all regular night shift work, technical direction employees dominated other groups with 28.1%. Whereas 6.2% of logistic employees did regularly the night shift (Table 4.28). Around 9.3% of employees who regularly did night shifts were at elevated risk (10-40%) with x^2=3.9, p=0.03 (Table 4.29).

The relationship between the night shift and cardiovascular disease risk was significant and consistent with other studies. This showed that the duration of sleeping was linked to cardiovascular diseases at 40-50% of the risk increase of dying (Bridget, 2019). In addition, the Jordanian employee's study showed that monthly numbers of night shifts and their duration were significantly associated with CVDs with p = 0.012 and p<0.001, respectively (Rana, 2018).

The study findings showed that 43.7% reported doing the regular night shift and most of them were males at a level of 97.4% (Table 4.27). Moreover, technical direction employees were more subjected to serving the night shift at a level of

28.1%. This study showed that 9.3% of regular night shift employees were at elevated cardiovascular risk. Their respective significant relationships were for workplace direction, p<0.001(Table 4.29), and elevated cardiovascular risk, p=0.03. Another study done on Jordanian employees confirmed the link between the night shift and CVDs regarding the monthly numbers and duration time of the night shift. These factors were significantly associated with the high cardiovascular disease risk by 30 years Framingham risk model with p = 0.012 and 0.000, respectively (Rana, 2018). Thus, shift workers were more associated with coronary artery diseases than non-shift workers (Havakuk, 2018).

5.3.1.2 Physical hazards for industrial workers status participants

This study finding showed that 51.5%, 24.4%, 17.8%, and 11.5% were exposed to noise, vibration, Gamma and X rays, and cold chambers respectively (Table 4.29). These physical factors' relationships with gender were all significant with p<0.05(Table 4.30). However, the age and physical hazards relationships were not statistically significant (Table 4.31). The workplace departmental relationships with physical hazards were all statistically significant with 10.4% of technical workers exposed, p<0.001(Table.4.32). Another study showed that physical hazards caused effects on cardiovascular disease risk. This showed that the road-traffic noise raised the coronary heart disease risk by 8% per 10 dB(A) and rise when 50 dB(A), (Hahad, 2019; Thomas, 2014; Daniel, 2017; Thomas et al., 2018).

The vibration was associated with myocardial infarction with OR:1.6 (95% CI: 1.1–2.4) (Bodil, 2006; Dzhambov, 2016). The Gamma rays and X-rays caused ionization (John, 2011; Bjorn, 2016), and gamma rays radiation increased up to 7kGy affected fat composition, and increased trans-fatty acids (Ismail, 2007). Cold exposure was associated with cardiovascular diseases due to limitation of blood flow and myocardial damage (Bin, 2012; Tiina, 2018).

5.3.1.3 Psychological hazards for workers in the study area

5.3.1.3.1 Type of stress levels for workers in the study area

This This study's findings showed that only one item (Unclear duty) was with a high mean stress score of 4.1, which points to the role ambiguity (Iraj, 2013). The study done in Iran showed that role ambiguity and role conflict were considered to have a significant path relationship with job stress (Bhui, 2016).

Pressure or workload-based stress mean score rate was 3.6, which is very high stress. This is consistent with other study findings. The results explained that workload equated to the work demand was a key determinant of stress and fatigue in industrial repetitive work (Macdonald, 2003). The components of pressure-based stress such as working long hours displayed a higher OR:1.56 (95% CI: 1.344–1.824). This showed a positive relationship between occupational stress, cardiovascular disease effects, and work-life balance disturbance (Wong, 2019; Hsu et al., 2019).

The following three behavioral items (lack of opportunity to question the manager, unable to talk to the manager about an annoying thing, unable to get line manager encouragement) are very important to boost employee morale. They indeed improve the employee's engagement toward perfect management of workplace stress. The findings of this study as shown by the above table are consistent with other studies on factors such as lack of support (Bhui, 2016). As well as, poor worksite relationships, (Van, 2016), and difficulties to cope with change due to a lack of the opportunity to question managers (Wisse, 2016; Roy, 2015; Guy, 2019).

5.3.1.3.2 Stress relationship with department, gender, age, and CVD risk in the study area

The study's prevalence of workplace stress was 64.4% in moderate stress and 30% in high stress (Table 4.37). This is high compared to the industrial study done in Bangalore where the overall stress was 22.2% and 33.3% of managers versus 20.9% of supervisors (Basavakumar, 2017). This study showed that Stress increased with age and male-dominated females with $p=0.03$, and $p<0.001$, respectively. This is in

line with the study done in India where males presented more stress than females (Tandon, 2014).

Figure 4.3 showed that only two directions (Departments) dominated the other five directions. The technical department was with a high stress of 14% and moderate stress of 26.6%. The sales department followed with 6.6% of high stress and 12% of moderate stress. The relationship between stress levels and cardiovascular disease risk was significant with 17% of elevated stress in high cardiovascular disease risk, $p<0.001$(Figure 4.4). Table 4.38 showed a high difference of odd ratio where moderate stress was 2.5-fold versus high stress with 18-fold, $p<0.001$ for Framingham genera risk score. It was sevenfold for moderate stress, and 1-fold for high stress concerning WHO/ISH but not statistically significant (Table 4.39).

Although this study's level of stress and cardiovascular diseases associated with Framingham's general risk score was consistent with other studies. This study's results were far higher than those found in other studies for long working hours and associated cardiovascular disease risk. Other studies showed that the associated coronary heart disease and stroke risk increase was 1.12-fold (95% CI 1.03–1.21) and 1.21-fold (95% CI 1.01–1.45) respectively (Marianna, 2018). The exposed participants to stressors were with 10-40% excess risk compared to the unexposed group (Mika & Ichiro, 2015).

5.3.1.4 Chemical hazards for workers study participants

The findings of this study displayed 132 chemical hazards with 4126 of direct and indirect handling where technical employees have encountered more chemical substances than others. However, another study found that in 308 chemical hazards handling, 693 were direct exposure. This was increasingly a burden for technicians, operators, and agricultural workers as well as elementary workers (Montano, 2014).

Chemical hazards handling was neither associated with cardiovascular disease risk by Framingham general risk score with OR=1.1, p=0.6 nor by WHO/ISH with OR=0.7, p=0.6. This finding consists of the study about the solvents part where there was no association of organic solvents exposure to Hispanic Americans with

cardiovascular diseases at the workplace. However, metal and pesticide exposure were associated with cardiovascular diseases (Bulka et al., 2019). One of the presented chemical hazards was found to cause anthemia in another study but not in the current research (Joshi, 2019).

The study results showed that 57.8% of all the workers handled or encountered chemical hazards. Men were more exposed to chemical handling than women p=0.01(Table 4.41). Around 32.9% of technical direction (department) workers were more likely in chemical hazards handling, p<0.001(Figure 4.5). The association of whole chemical handling exposure, including all solvents for workplace employees, was OR 1.89(0.74-4.79), but not statistically significant (Table 4.42). On the other hand, other studies showed that some chemicals such as toluene cause hypokalemia and auricular-ventricular block (Zhou et al., 2011; Cruz, 2014). Moreover, xylene, hexane, lipophilic, heptane, ethyl ether trichloroethylene, trichlorotrifluoroethane chemicals were associated with cardiovascular diseases by causing metabolic change (Kim, 2012).

Furthermore, raising catecholamine to cause arrhythmia and death showed that the exposed group to solvent was significantly associated with electrocardiographic changes. Hence, the QRS complex change was high with an RR of 1.53(1.46-1.61), and the modified P wave was 1.02(1.01-2.28) and 1.15(1.08-1.49) for arrhythmia (Assadi, 2018). The solvents were found to increase cholesterol and affect the central nervous system (Zeliger, 2013). And as well as halogenated hydrocarbons such as chloroform (Sridhar et al., 2011; Butkiewicz et al., 2017).

5.3.2 Association of working conditions to cardiovascular diseases risk

This study finding showed all workplace hazards (working departments, shift workers, regular night Shifts, much sound, vibration, chemicals) that employees are exposed to, at different levels. Their association with cardiovascular disease risk expressed a significant Crude Odd ratio to workers. The findings showed that in human resources COR=3.52(1.08-11.48) 95% CI, p=0.037, night shift COR=4.257(1.03-17.44) 95% CI, p=0.044, much sound COR=0.209(0.071-0.61) pp=0.007. Whereas the AOR was only significant to one factor which was night shift

workers AOR= 2.41(1.27-4.58), p=0.007. Much noise(>=85Db) showed the insignificant result with AOR=0.54(0.28-1.04), p=0.06. These results were, hence, not different from other studies that showed an increase in cardiovascular risk. Additionally, they showed similar metabolic changes, and sugar spikes due to the night shift (Daniel, 2017; Rana, 2018; Keithellakpam et al., 2019; Li et al., 2019).

Some studies showed that Whole body vibration training executed at 30 Hz frequency with an amplitude of 3-mm peak-to-peak caused reactive hyperemia (RH). This exerts an acute effect on the endothelial system but does not show a direct significant effect on hypertension and heart rhythm (Aoyama et al., 2019). However, the risk increased among men and women Bulgarian industrial workers (European Agency for Safety and Health at Work, 2008; Dzhambov, 2016; Akinnuli, 2018).

5.4 Awareness on traditional Risk factors and Personal protective equipment usage among the study participants in the study area

5.4.1 Awareness on hypertension, diabetes, and dyslipidemia as prominent risk factors to CVD risk for all participants in the study area

The participant's awareness was related to three cardiovascular diseases' traditional risk factors, where people knew or were told previously about hypertension, diabetes, and dyslipidemia. This study showed that 30.7% were previously told to have hypertension. Around 49.7% of participants knew that hypertension control would help to curb cardiovascular diseases. This awareness level was low compared to the awareness level in a Cameroonian study, where the awareness level was 63.4% (Frank, 2018). Moreover, another study showed high results, where 75% were aware of, and recognized the need to improve their lifestyle to reduce cardiovascular risk factors (Table 4.44), (Ramirez, 2017).

Participants had more elevated awareness levels of hypertension with 49.7% than diabetes with 43.2% and dyslipidemia with 40.9%. They are aware that controlling them can help to fight cardiovascular diseases. These study findings are also low compared to the study carried out on immigrant Latinas. The results showed that 88% of non-Hispanic whites were aware that all components of Metabolic syndrome

were leading to death. In addition, 81% were aware of heart attack symptoms and the level of knowledge of risk factors for cardiovascular diseases. This has increased after the intervention among both Hispanics and non-Hispanic white (Deborah, 2015).

5.4.2 Workers using personal protective equipment (PPE) for prominent worksite hazards exposure to noise and chemical handling

Personal protective equipment was worn to protect employees from workplace hazards exposure. Such as 49.3% of employees were exposed to noise hazards and 43.3% of employees were exposed to chemical hazards. Although some workers wore PPE (Personal protective equipment) frequently, 15.9% and 4.8% of employees didn’t always wear PPE correctly for Chemical and noise exposure, respectively (Table 4.47). This study showed that most employees always wear PPE (Personal protective equipment) correctly. The Europian agency for safety and health at work demonstrated the exposure level and CVD development in 2005. This study showed that employees who don’t wear PPE correctly may develop cardiovascular diseases at 75dB of noise exposure and chemical exposure (Kim, 2012). The findings as shown in (Table 4.48) displayed a negatively correlated test, which explained a protective mark to whoever wore PPE. This revealed a significant negative correlated test, which also showed a protective mark to whoever wore PPE frequently for both WHO/ISH and FGRS models. This painted the importance of always wearing PPE when exposed to workplace hazards. Hence, it was emphasized by Kim's discussion in 2012 regarding the link between chemical exposure and cardiovascular disease development. He also discussed the negative impact of noise exposure on heart diseases. Consequently, as depicted in another study, the risk reduction mechanism (PPE wearing, sound moving, and blocking) below permissible exposure levels can prevent and control the incidence of cardiovascular diseases (Wu, 2017).

5.5 Proportion of people with biological factors among the study participants in the study area

5.5.1 Hypertension

The overall study findings on hypertension have shown a prevalence of 32.27% by previous blood pressure classification. This is not relatively high compared to other studies that showed a prevalence of 26% of workers participants in four African countries (Guwatudde et al., 2015). It was 46% and 35% in two different studies of the African region (Wamba, 2019). The prevalence was 52% in the South African nurse study (Monakali, 2018), 55.2% in the whole of Africa (Kaze et al., 2017), and 34.9% worldwide prevalence (Thomas et al., 2018). The prevalence has increased to 61.81%, (38% for males, and 23.8% for females) by the updated blood pressure classification with a difference increase of 29.54%. Employees have a relatively high hypertension prevalence from 35.92% to 65.18% compared to the spouses with 26.47% to 56.47% by previous and updated classification, respectively (Table 4.51).

Although health systems continually Increase the effort to tackle the issue of health coverage. This high prevalence of 65.18% found by the updated blood pressure classification would be covered if we only associate different theoretical culture-related theories and models. Such as health belief models, the theory of planned action and planned behavior, trans-theoretical theory, and clinical, workplace, and community models. Thus, coupled with structured non-pharmacological interventions (Kévin, 2010). This showed surprising results to fight the diseases in the community instead of waiting for the patients to come to the hospital through public health prevention strategies.

This second classifier showed an increase in hypertension prevalence which can astoundingly cause a low quality of health delivery. This is due to a low number of health professionals to support the high percentage of patients and an increase in drug use prescription and laboratory testing (Khera et al., 2018).

The combinants drug use findings were classified as dual therapy with 55.6%, monotherapy with 43.1%, and 1.4% tri-therapy (Table 4.52). Normally,

Pharmacological intervention was considered to be very crucial to sensibly reduce blood pressure. It was applied to patients with advanced levels of hypertension that could not be reduced with non-pharmacological intervention alone (Ipek, 2017). However, the current strategy of waiting for people at the health facility is rampantly implying the increase of hypertension beyond the normality of cut points due to low health-seeking behavior. It can also cause the failure of the application of non-pharmacological alone. This would be considered important in the early application (Hema, 2011), due to the cultural resistance of patients and halting hypertension consequences. Relying on the little time that a patient spends with a health professional in the consultation room. Therefore, this will not permit a health professional to convince the patient to adopt a new protective non-pharmacological intervention. Because it seems difficult for many people to change their modifiable behavior to fight hypertension. They rather believe in the pharmacological intervention, with which they are even not compliant if no planned structured community follow-up.

However, clinging to the previous blood pressure classification can hide the obvious rising burden of hypertension diseases. Its cardiovascular mean risk of 4% for normal blood pressure, 6% for prehypertension, 17% for hypertension stage 1, and 40% for hypertension stage 2 by the previous classification. The Framingham general risk score Prediction showed a high risk of stage 1 and stage 2 hypertension. Hence, this was caused by the retaining of a big number of people, who always and unknowingly develop high hypertension without the benefit of any health prevention strategy. On the other hand, the updated classification shows a minimized mean risk of 4% for elevated blood pressure. The risk increased at 9%, and 19% for hypertension stage 1 and 2, respectively (Figure 4.6). The second classification uncovers the reality of hypertension prevalence and its cardiovascular diseases associated risk. This really requires public health urgent interventions to maintain people in the normality. It will also help to prevent the progression of poorly controlled hypertension, and non-hypertensive drugs compliant toward hypertensive crisis (Ipek, 2017). This is and will remain the mark of the intersection of cardiology and health promotion.

We found that systolic and diastolic blood pressure increases by age (Table 4.48). Hypertension is more likely to develop in advanced age by 2-fold for 40-45 years. It is threefold for more than 50 years on two reduced models of the two-blood pressure classification (Table 4.53) (Thomas, 2016). In addition, HBP is high for men with a total systolic blood pressure mean of 135.06 ± 14.03 and 130.83±13.09 for women. The total diastolic blood pressure mean was 80.22±12.78 and 78.47±11.91 for males and females, respectively. With a single updated blood pressure classification significance of being likely to develop to males p-value <0.001(Table 4.48). However, the highest systolic blood pressure mean was found in males while the highest diastolic pressure mean levels were found in females (Table 4.48). The most prevalent hypertensive participants were found in employees where Kigali employees had high hypertension prevalence and followed by Rubavu employees. Kigali spouses were the third while Rubavu spouses come with a low prevalence (Table 4.50).

We observed that the association of unchangeable factors such as gender, Age (Thomas, 2016), Family history (Priyanga, 2015), and being employed or spouses was significant with a p-value <0.05. This was found for the reduced model of two blood pressure classifications except for gender, for a single previous classification with a p-value of 0.42 (Table 4.53). On the other hand, changeable factors such as central obesity were two to six-fold more associated with HBP in two reduced models. Alcohol intake in 30 days (Kazim, 2014), diabetes, stress level (Tanya, 2010), and smoking (Kaiye, 2017), were also significantly associated with hypertension except for alcohol intake (Luc, 2009). This was not significant on a single previous classification reduced model.

Although diabetes is a major risk factor for hypertension (Sowers & James, 1992), it was not significant in two reduced models of the previous and updated classification of blood pressure (Table 4.53). The high level of stress was missing in the reduced model. However, it was the highest significant factor in hypertension with seven-fold to be associated with hypertension in the crude model.

Overall, the findings on hypertension bring up the crucial need for early and primary prevention of hypertension using constructive, structured, and effective socio-

cultural strategies and psychological behavior change. Whereby some of which were consistent with other studies in their effective use. Such as the health belief model, where the improved result was significant with p=0.03). Planned behavior and planned action, social cognitive theory, stage of change theory, or trans-theoretical model are important theories. Hence, they highlight the consideration of workplace clinical and community approaches to improve the level of health promotion success (Chu-Hong, 2015; Eng, 2016; Azam, 2018; Jafaralilou, 2019; Mozhdeh, 2019).

5.5.2 Diabetes

The findings of this study showed that 13.6% of study participants were diabetics and 26.8% were prediabetics and 59.6% were normal participants. The gender and diabetes relationships were not significant (Table 4.54). These results painted a higher proportion compared to the findings of 2.8% stated by the world health organization in 2012 and Rwanda's non-communicable disease risk factor report of 2015. This showed that diabetes patients, were 3.06% and prediabetes patients were 1.59% in a survey conducted with the 6,662-study population (WHO, 2012).

Diabetes was statistically significant on either FGRS or WHO/ISH models. In addition, the study conducted at Kigali university teaching hospital demonstrated that diabetic microvascular complications preceded macrovascular complications. This was associated with 53% of neuropathy, 23% of retinopathy, and 20% of nephropathy versus 15% of peripheral vascular diseases. In addition, 4% of cerebrovascular diseases, and 3% of coronary artery diseases are also followed by diabetic microvascular complications (Rudasingwa, 2012).

The variation of mean blood sugar and glycosylated hemoglobin for age category difference was only significant for men and total mean value, respectively (Table 4.55). This expressed an increase in diabetes with aging (Rita, 2017). Out of 13.6% of total diabetics in all study participants, 8.9% are taking diabetics drugs. Metformin was the most used medication at a level of 61.5%. Around 74.3% of all diabetic participants were on monotherapy treatment. This is also consistent with other studies (Yi-Wei et al., 2017).

5.5.3 Overweight, Obesity and Central obesity for study participants in study area

This study's findings showed that 46.6% were overweight and were predominantly men. Around 24.6% were obese predominantly women with p<0.001(Table 4.57). This is high compared to the survey done in 2012 in Rwanda NCDs risk factor report of 2015, where overall obesity was 2.8%. Obesity was predominant in urban with 10.2%. However, it was slightly low in Kigali with 7.7%. The normal weight proportion was only 27.7% while 75% of all Rwandans were credited to be within normal weight level. However, another study showed that obesity in 2010 was 16.5% among women in Rwanda, 35% in Kigali, and 31.5% among women in other urban areas in Rwanda (Mukabutera, 2016).

Relying on Owolabi's study in 2017 waist circumference threshold for men and women. Around 21.4% of study participants were with high central obesity, where men were 12.7% versus 8.6% of women. The total number of participants with very high central obesity was 37%. Women dominated with 27.3% versus 9.8% of men (Table 4.58). Another study done in Dodoma Tanzania showed that women obese dominated men with 35.14% vs. 6.89%, p<0.001 (Munyogwa, 2018).

The prevalence of general obesity and central obesity was low compared to the study done in South Africa. The study results showed that central obesity was 66.6% while general obesity by BMI was 46% and central obesity by WHR was 57.8% (Owolabi, 2017).

This study’s findings showed that regarding the World health organization cut point, the WHR results were divided into two different district plants. Kicukiro had high central obesity of 79.9%, dominated by men with 42.1% versus 37.8% of women. whereas Rubavu had 44.2% of central obesity and was dominated by men with 27.6% versus 16.6% (Table 4.60). The age-related variation of WHR mean values was significant for only Kicukiro males and Rubavu females and kept a continuous rise for men. This explained the rise of abdominal obesity in men by age (Table 4.59).

These findings displayed a higher level of central obesity based on WHR with the World health organization threshold than measurements obtained using tape for waist circumference based on NCEP ATP-III cut points (Table 4.60). However, the application of the very high threshold (>=1), showed a reduction of central obesity in Kicukiro with 17%. This central obesity level was dominated by males with 10.8% versus 6.2% whereas Rubavu had 8.8% also dominated by males with 7.2% versus 1.7% of females (Table 4.61). However, the waist to hip ratio displayed a slightly elevated level of central obesity than a mere waist circumference.

We have observed, in another study carried out by de Koning in 2007, that these two measures correlated well for cardiovascular disease prediction. This showed that every 1 cm and 0.01 U increase of waist circumference and WHR, respectively (deKoning, 2007). There was also an increase of 2% and 5% in the relative risk of cardiovascular diseases event. However, another study showed a slight increase in central obesity by WC than WHR in Malaysian adults' study and concluded to be a good indicator for Malaysian adults (Norfazilah, 2016).

5.5.4 Dyslipidemia for all participants in the study area

Total cholesterol and triglyceride age-related incremental variation were significant, except for females on triglyceride with insignificant incremental variation $p<0.1$ (Table 4.62). Low-density lipoprotein's mean value increased with the age while High-density lipoprotein decreased dominantly for men than women with the age. Even though, it was not significant for women (Table 4.63). However, the Rancho Bernado study carried out by Assiamira in 1997, painted discrepant results. The study explained that total cholesterol and Low-density lipoprotein decreased for men with age for a very older community from 50-93 years. This may explain a sound age difference with the current community (Assiamira, 1997). In addition, another study showed a tendency to reduction of Total cholesterol, Triglyceride, and Low-density lipoprotein in the elderly (+65 years). It has also shown the stabilization of HDL for people inferior and superior to 65 years (Zhao, 2018).

The relationship of the lipid profile to cardiovascular disease risk, as depicted in this study's findings (Table.4.64), showed elevated numbers of the lipid profiles. The

studied lipids were total cholesterol (TC), triglyceride (TG), Low-density lipoprotein (LDL-C), and High-density lipoprotein (HDL-C). They were all at elevated cardiovascular disease risk (>10%) by Framingham general risk score model. Where among 25.2% of elevated risk, were in high and very high lipid profiles. There were all scientifically significant ($p<0.001$). It was also found that the second model (WHO/ISH), displayed 4.5% of elevated cardiovascular disease risk (>10%), where lipid profile slightly increased cardiovascular disease risk with $p<0.001$ (Table 4.65). These findings were also consistent with another study, where an increase in one of the lipid profiles (Total cholesterol) was associated with myocardial infarction and stroke. Hence, its reduction was inversely associated with CVD (Mee et al., 2017).

5.5.5 Metabolic syndrome for all participants in the study area

No component of the metabolic syndrome was associated with cardiovascular disease risk by the WHO/ISH model. However, three separate components of the metabolic syndrome (hypertension, Impaired fasting glycemia, and Hypertriglyceridemia) were significant in the reduced model by FGRS. A low level of high-density lipoprotein (HDL) and central obesity was not significant. However, other studies showed an association between cardiovascular disease with an increase in myocardial infarction (MI) risk by central obesity and an increase in stroke risk by HDL (Kazlauskienė, 2015).

The full metabolic syndrome was also statistically significant on the full model with cardiovascular diseases risk by FGRS whether for location or participant's status. This finding is surely consistent with another study result, which clearly demonstrated the association of the metabolic syndrome with cardiovascular diseases (Barbara, 2002; Wilson, 2005).

The findings of this study, regarding metabolic syndrome components relationship with gender. This revealed that only two components were only dominated by females with a significantly high level of abnormalities than the males. The High-density lipoprotein was 28.4% for females versus 10.9% for males. Whereas Central obesity, was 27.3% for females versus 9.8% for males, $p<0.001$. Other three components among others, impaired blood sugar, Triglyceride, and High blood

pressure were all dominated by men and only significant for High blood pressure. In addition, a high level of blood pressure for metabolic syndrome was 36.6% for males versus 21.1% for females, p<0.001(Table 4.66). Moreover, males were dominated by females with 46.3% versus 29.3% of males, among farmer's participants in South Africa (Maritza, 2017).

The total metabolic syndrome prevalence was 38.2%. Its relationship with cardiovascular diseases elevated risk was slightly significant with 12.7% in elevated risk (>10%) versus 12.5% in low risk (<10%), (Table 4.71). However, a systematic review study carried out in SSA, 2019, showed that according to NCEP-ATP III the prevalence of metabolic syndrome was 17.1% (95%CI: 12.8-22.0) (Maritza, 2017). This result is far low than the current prevalence study of 38.2%, (Faijer-Westerink, 2019). The estimates of this African metabolic syndrome prevalence from 17% to 27% in 2012 are nearly approaching the current finding figures (Okafor, 2012).

A meta-analysis study carried out by Mottillo concluded that metabolic syndrome was associated with a 2-fold increase in cardiovascular diseases and a 1.5-fold, increase in all-cause of mortality (Salvatore et al., 2010).

5.6 Cardiovascular diseases traditional risk factors and novel risk differentials among the study participants in the study area

5.6.1 Novel risk factors to cardiovascular diseases risk

These study findings are consistent with another study done in 2010, where several studies confirmed the relationship of serum uric acid with cardiovascular disease morbidity and mortality. It stated that an increase of roughly1mg/dl was associated with a roughly 1-hazard ratio of total mortality and cardiovascular mortality. It was also associated with an increase of about 13% for metabolic syndrome. Again, a rough increase of one odd ratio for hypertension and a 48% increase for women's stroke events (Adriana, 2010). C reactive protein was also found to contribute to cardiovascular events and mortality. Moreover, high sensitivity C reactive protein (hs-CRP) with its inflammation-causing capacity was the impetus for complications associated with atherosclerotic plaques (Audrey, 2015; Francisco, 2016).

This study finding displayed CRP to be either associated with cardiovascular disease risk by FGRS with AOR=4.482(2.03-9.88), P=0.001or by WHO/ISH model OR=5.535(1.33-22.95), p=0.01. This result showed that CRP had an obvious association with cardiovascular disease risk. This is consistent with other studies that demonstrated CRP to be both a marker and mediator of atherosclerosis. Moreover, it is an independent predictor of abrupt heart death, ischemic stroke, myocardial infarction, and finally cardiovascular events (Amit, 2015; Zhuang, 2019; Koosha et al., 2020). Serum uric acid was neither associated with cardiovascular disease risk by FGRS nor the WHO/ISH model in this study. In contrary to other studies, which showed the association of Serum Uric Acid (SUA) with cardiovascular diseases (Muiesan, 2016; Rahimi-Sakak, 2019).

Moderate and high Glycosylated hemoglobin (HB1Ac) were associated with cardiovascular disease risk by only the FGRS model. Other studies demonstrated that HB1AC could be a predictive biomarker for coronary artery disease in non-diabetic (Ewid et al., 2019) and diabetic patients. Hence, the increase of HB1Ac was positively associated with cardiovascular diseases and fatal coronary heart diseases (Zhang, 2012; Jennifer et al., 2013; Danesh et al., 2014).

5.6.2 Traditional risk factors to cardiovascular diseases risk

The findings of this study showed that male employees dominated other groups with a high prevalence of people with traditional risk factors. Except for levels of general obesity, central obesity, inactivity, and high triglyceridemia for Kicukiro female spouses. Obviously, Rubavu female spouses presented a low level of cardiovascular disease traditional risk factors than all other groups (Table 4.75). It was clearly depicted in another study, comparing traditional risk factors for south Asian countries and European countries. This showed that the TC/HDL ratio and diabetes explained the excess of risk factors in south Asia more than in Europe (Rabanal et al., 2017).

Kicukiro study participants (Employees and spouses) dominated Rubavu study participants (Employees and spouses) with a high prevalence of cardiovascular risk (>= 10%) (Table 4.76). Where, 18.6% versus 6.8% of elevated cardiovascular risk

(>=10%) by FGRS model, for Kicukiro and Rubavu study participants, respectively. Even the second model (WHO/ISH score chart) showed that the prevalence of elevated cardiovascular risk (>=10%) dominated with 3.1% versus 1.3% of Kicukiro and Rubavu study participants, respectively. Therefore, traditional risk factors were practical guidance that heralded the development of the prediction and treatment of cardiovascular diseases. Risk factors don't immediately mean causality. However, they can in one way or another, explain the increase in disease and mortality events. They can be the basic targets for treatment, and modification to delay, and or prevent cardiovascular diseases (Emil, 2012).

These study findings showed that three unmodifiable factors were significant by the FGRS reduced model (Gender/Male, age, and family member who suffer CVDs). On the other hand, relationships with family members who died from CVDs and age were significant by the WHO/ISH reduced model. This result is similar to other studies where age was demonstrated as an independent risk factor for cardiovascular diseases (Dhingra, 2012).

Males were found to have a higher incidence of cardiovascular diseases than females. Even though women were more prone to high hospitalization rate and mortality due to cardiovascular diseases (stroke, heart failure, heart failure and coronary heart disease, stroke) (Zujie, 2019). The two models showed a segregated significance concerning the two factors (The family member who suffers CVDs and the Family member who died from CVDs). However, other studies demonstrated that there was an increased risk of sudden cardiac death (SCD) four-fold and two-fold for first-degree and second-degree relatives, respectively (Ranthe, 2013). Another study showed that 12.2% of CVD patients reported having a parent or sibling with a heart attack or angina before age 50 years (Emelia, 2017).

Even though the WHO/ISH did not show significance to workplace factors, the FGRS model demonstrated the industrial exposure to two factors. The models showed that radiation and high stress were associated with cardiovascular disease risk. In fact, this is similar to other studies that revealed the link between occupational stress with cardiovascular diseases (Jian, 2016), (Luigi et al., 2018). Concerning occupational radiation exposure, the International Commission for

radiological protection (ICRP) elaborated on this issue. The commission showed that after 10 years exposure to a dose of 0.5 Sievert (Sv) may cause the development of cardiovascular diseases in 1% of exposed people to radiation. Moreover, the undetectable radiation dose-risk level was below 0.5 Grey (Gy) (Baselet, 2016). Another study done in England did not reveal any effect of radiation cumulative exposure below 0.1 Sv. However, heart disease was only detected in people exposed around 40 years on more than 0.4 Sv. Yet, the mortality was low in this cohort (Zhang, 2019). Moreover, each Gamma-ray is approximately ten times more energetic than an X-ray and 100 rad=1 sievert=1 Gray.

Eight modifiable general factors (urban place, being employee, sedentarity, second-hand smoking, general obesity, central obesity, and updated hypertension) were studied. These factors were not statistically significant in the reduced model by FGRS. On the other hand, twelve general modifiable factors were also studied. Those factors are: (location, participant status, sedentarity, physical inactivity, low fruit intake, low vegetable intake, tobacco smoke, second-hand smoking, general obesity, central obesity, and updated hypertension). They were not statistically significant in the reduced model by WHO/ISH model. However, other studies have demonstrated the link between cardiovascular diseases with urbanization (Smith, 2012), sedentarity, general obesity and central obesity (Carl, 2019), and second-hand smoking (Olasky, 2012). Being either employee or spouse was not significantly linked to cardiovascular diseases in the full model of all general risk factors, probably due to the crossover effect of employees to spouses or vice versa or due to lifestyle sharing (Kang, 2017).

Eight general modifiable risk factors were statistically and significantly linked to cardiovascular diseases. However, one of them taking alcohol in the last 30 days was not alone significant. It was only significant in association with the time of exceeding four drinks by women and five drinks by women. The more times the person exceeds the standards, the more the association becomes high AOR=11.162(4.58-27.15). This result is consistent with other studies where the link to cardiovascular diseases was established for alcohol (Larsson, 2020), inactivity (Carl, 2019; Ahad, 2016), diabetes (Alessandra et al., 2013; Leon, 2015), and hypertension (Flávio, 2019). The fruit and vegetable intake was inversely associated with cardiovascular diseases, cancer, and

premature death. An estimate of 5.6 to 7.8 million worldwide premature death was attributed to Low fruit (<500g/day) and vegetable (<800g/day) intake in 2013 (Karl, 2012; Dagfinn, 2017). Another study showed that cardiovascular diseases and mortality were reduced by fruits and vegetables eating at a level of three to four servings per day (equivalent to 375–500 g/day), (Victoria et al., 2017). In addition, a reduction of peripheral vascular diseases (Heffron, 2017).

5.7 Conclusions

1. This study showed that cardiovascular disease risk is high in employees than in spouses for both two models (Framingham general risk score and WHO/ISH model). Although the ROC Curve of the Framingham risk score model and WHO/ISH is perfect for a correlated rater, their level of agreement is minimal. This brings an issue of overestimation in Framingham general risk score and underestimation in WHO/ISH score chart. The use of the above well-performing prediction model in clinical and preventative medicine practice is important until the development of a local model.
2. This study showed that amongst the 7 behavioral factors, three (alcohol, oil, and sedentarity) were consumed a lot more by the society than other factors (Smoking, Fruits, and vegetables, and Physical activities). It was also found that the weekly quantity and servings consumptions for fruits and vegetables were low. Only smoking, low fruit and vegetable intake, sedentarity, and excessive alcohol intake were significantly associated with cardiovascular disease risk.
3. It was found that among the 8 working conditions (Shift work, night shift, occupational stress, much sound, vibration, cold, chemical substance, and radiation), shift work, night shift and chemical handling/ or encountering were with an elevated proportion of the study participants. Only 4 working conditions (Nightshift, occupational stress, much sound>85db, and radiation) were statistically associated with cardiovascular disease risk.
4. The study determined that the awareness level of three variables (if controlling hypertension, diabetes, and dyslipidemia) can help to prevent cardiovascular diseases was below fifty percent for all study participants. It

was also found that the chemical handling and sound exposure negative effect can be controlled. The higher the frequency of wearing personal protective equipment, the lower the risk of developing cardiovascular diseases for employees.

5. The study found that among the 6 biological factors (Hypertension, diabetes, general obesity, central obesity, dyslipidemia, and metabolic syndrome), only three variables (hypertension, central obesity, and metabolic syndrome) have the elevated levels than the three remaining factors. Although the HBP prevalence was around one-third of the study population by the measurement of the old threshold, it was almost doubled with the new threshold measurement. This could increase the queue at the hospital and reduce the treatment quality. It was equally shown that hypertension and diabetes are the prominent risk factors associated with cardiovascular diseases elevated risk.
6. This study found that among the novel risk factors, Glycosylated hemoglobin (HB1AC) and C reactive protein (CRP) presented more levels than serum uric acid (SUA). It was also found that elevated levels of glycosylated hemoglobin and C reactive protein were significantly associated with cardiovascular disease risk. There is no significant difference found across all groups regarding glycosylated hemoglobin. However, Kicukiro male employees and Rubavu male spouses have high levels of SUA while it is Kicukiro male employees only who have elevated CRP. It was also found that the difference between groups was significant regarding the traditional factors. Kicukiro male employees and Rubavu male employees dominated other groups on larger alcohol intake, physical inactivity, stress, and low fruits intake. Whereas Kicukiro female Spouses dominated other remaining groups on, central obesity and sedentarity.

5.8 Recommendations

i. This study's findings on the cardiovascular diseases risk prediction showed that a third of the respondents had an elevated risk of developing cardiovascular diseases in ten years. It is recommended to ensure the

proactive prevention of cardiovascular diseases by using the Framingham general risk score.

ii. This study showed the effect of behavioral factors on cardiovascular diseases factors. It is recommended to ensure the moderation of these behavioral predisposing factors and increment of behavioral protecting factors. It is also recommended to the ministry of health and the agricultural ministry for a joint Agri-health effort program. To ensure the availability and preparation of indigenous and improved fruits and vegetables for daily consumption.

iii. This study's findings on workplace conditions, are the first findings for elucidating the manufacturing industry factors toward cardiovascular disease risk in Rwanda. It is recommended to the ministry of health, the Ministry of labor, and industry administrations to ensure the safety of employees. Hence, observing the safety requirement (PELs, TWA, TLV), creating workplace health policies to protect employees, minimize the risk, and increase the employee's longevity and production.

iv. This study's results determined that the awareness of hypertension, diabetes, and metabolic syndrome diseases was low in the study area. Wearing personal protective equipment was associated with a protective capacity against the chemical and sound harm to employees. It is, hence, recommended to create a cultural-based occupational and community awareness. It is also recommended to ensure the use of tangible cues to action, instead of using force in the unknown or vacuum in the industrial safety.

v. The findings of this study on biological factors are the prominent risk factors for cardiovascular diseases. It is recommended to the ministry of health, adopt, and practice the approach of hypertension level-based prevention strategies. To ensure the early non-pharmacological interventions are applied and monitored. To ensure the awareness of personal responsibility, choice of oil by label check to reduce unnecessary saturated fat and trans-fat.

vi. This study's results highlighted the difference between groups and showed that employees are having high cardiovascular disease risk than spouses. It is recommended to track the Workplace exposure effect on workers by

monitoring the levels of traditional and Novel risk factors. Ensuring the people accompaniment by cultural theories to change their lifestyle.

5.9 Suggestion for further studies

- A follow-up study of the local population to create our own national model to predict cardiovascular diseases.
- Study the pure African culture-related theories to fight cardiovascular diseases with a highlight of the difference of African American population due to the slavery and colonization epigenetic and long-term stress on hypertension.
- Study of African obesity versus African values of being obese (fat) as taken to be healthy and rich.

5.10 Contribution of the current study to learning

- The first and foremost contribution is the comparison of the WHO/ISH model and the Framingham general cardiovascular risk prediction model. Additionally, in the Rwandan population of industrial brewery employees and their spouses' community. Hence, it has a direct implication in:
    - Early quantification of cardiovascular disease burden at workplace and community.
    - It implies also proactive management of the burden and quality of health improvement.
    - Informing the future follow-up study to create our own model due to poor agreement of the two models, which was not yet used by the local population.
- Early highlight of Professional implication of new cutoff of Hypertension-on-hypertension prevalence increment, consultation long lines, which will consequently, increase poor service, High utilization of antihypertensive drugs. Hence, underscores the capital importance of health promotion to curb the issue and balance the middle ground scale before treatment and after the treatment of the Rwandan population.

- Highlight the beverage industry's chemical presence with departmental differential use and risk. It contributed also to the minimization of chemical impact on the employees with safety measures toward a standardized Rwandan workplace safety without cardiovascular diseases and other diseases due to chemical manipulation.
- The implication goes beyond the risk reduction and minimization with the creation of a properly secured workplace and community environment safe from cardiovascular diseases.
- Highlight the impact of workplace stress, night shift, sedentary, and inactivity. In addition, indicate their great and growing negative effect on the development of metabolic diseases and cardiovascular disease risk. Thus, a reduction in production in the Rwandan workplace.
- Inform the policymakers on the industrial working conditions to facilitate the development of standardized country workplace policy. Moreover, serve as a relevant model to heighten worksite health and minimize the negative effect of hazardous activities.
- Due to the accumulation of many harmful behaviors, which increases the development of non-communicable diseases among other cardiovascular diseases. It has been shown by the high prevalence of hypertension before and after the new cutoff despite the treatment guidelines.

    - This study has informed the development of a culture behavior change theory concept (Culture treason theory). This theory was fostered after realizing the growing tension of cardiovascular diseases and difficulties of behavior change.
    - Its development will positively influence future preventive and rehabilitative approaches. In addition, it will support instilling the inner behavioral power change and alternative proposition. Moreover, this theory concept was clearly and deeply developed elsewhere out of this thesis.

- This study has also contributed to the highlight of novel risks, and their association with cardiovascular diseases. In addition, explained novel risk, which could be used in model development prior to a cohort study.

REFERENCES

Abu, F. R.(2018). Shift Work and the Risk of Cardiovascular Diseases and Metabolic Syndrome Among Jordanian Employees. *Oman Med J. 33*(3), 235-242.

Adriana, I. A. (2010, Jul). Serum uric acid and cardiovascular disease,. *Maedica (Buchar)/A journal of clinical medecine, 5*(3), 186–192.

Ahad, W. N. (2016). Quantifying the Association Between Physical Activity and Cardiovascular Disease and Diabetes: A Systematic Review and Meta Analysis. *J Am Heart Assoc, 5*, e002495.

Akinnuli, B., Dahunsi, O., Ayodeji, S., & Bodunde, O. (2018). Wholebody vibration exposure on earthmoving equipment operators in construction industries, Cogent. *Engineering, 5*(1), 1507266..

Alberti, K. Z. (2005). Themetabolic syndrome, new worldwide definition. *The Lancet, 366*(9491), 1059–1062.

Aldo, A. M.-G. (2018). Update in the Early Management and Reperfusion Strategies of Patients with Acute Ischemic Stroke. *Critical Care Research and Practice,*, 15.

Alessandra , S., Lucianne , R., Roberta , A., Catia, C., Carlos, A., & Marilia , d. (2013). Impact of Diabetes on Cardiovascular Disease: An Update. *International Journal of Hypertension,10*(1155), 653789.

Ambar, K. A. (2014). Urban-Rural Differences in Coronary Heart Disease Mortality in the United States:1999–2009. *Public Health Rep, 129*(1), 19–29.

Ambrose, J. B. (2004). The pathophysiology of cigarette smoking and cardiovascular disease: an Update. *J Am Coll Cardiol,43*(10), 1731-7.

American Heart Association. (2005). *Heart Disease and Stroke Statistics: 2005 Update.* . Dallas, TX: AHA.

Amit, K. S. (2015). C-reactive protein, inflammation and coronary heart disease. *The Egyptian Heart Journal*, *67*, 89–97.

Anne, E. P. (2004). heart disease and work,heartjournal, 2004;. *Heart Journal, 90,*1077–1084.

Anokye, M. A. (2020). Sample size determination in survey research. *Journal of scientific research & resports*, *26*(5), 90-97.

Ansell, B. N. (2004). Anti-inflamatory properties of HDL. *RevEndocr Metab Disord,* 51-358.

Aoyama, A., Yamaoka-Tojo, M., Obara, S., Shimizu, E., Fujiyoshi, K., Noda, C., . . . Ako, J. (2019). Acute Effects of Whole-Body Vibration Training on Endothelial Function and Cardiovascular Response in Elderly Patients with Cardiovascular Disease. *Int Heart J, 60*(4), 854-861.

Apovian, C. B. (2008). Adipose macrophage infiltration is associated with insulin resistance and vascular endothelial function in obese subjects. *Arterioscler Thromb Vasc Biol*, *28*, 1654–1659.

Apovian, C. N. (2012, March 6). Obesity and cardiovascular disease. *Circulation, 125*(9), 1178-82.

Arend, M. A. (2007, Sept). Clinical epidemiology of heart failure. *Heart, 93*(9), 1137-46.

Arranz, S. G.-B.-M.-R.-R. (2012). Wine, Beer, Alcohol and Polyphenols on Cardiovascular Disease and Cancer. *Nutrients*, *4*(7), 759-781.

Arvind, R. D. (2015). Implications of Cardiovascular Disease Risk Assessment Using the WHO/ISH Risk Prediction Charts in Rural India. *PLOS ONE*; *10*(8), e0133618.

Ashley, E. N. (2004). *Cardiology Explained*. London: Remedica.

Assadi, S. N. (2018). Electrocardiographic changes and exposure to solvents. *Journal of Arythmia, 34*(1), 65–70.

Assiamira, F. E.-C. (1997). Total, LDL, and HDL Cholesterol Decrease with Age in Older Men and Women, the Rancho Bernardo Study 1984–1994,. *Circulation., 96*, 37–43.

Audrey, H. W. (2015). Relation of serum uric acid to cardiovascular disease. *international journal of cardiology*. *213*, 4-7.

Auley, D. G. (2013). Comparative study of risk factors of cardiac diseases amongurban and rural population, . *Int J Hum Genet.* , 15-9.

Azam, L. R. (2018). Factors Predicting Self-Care Behaviors among Low Health Literacy Hypertensive Patients Based on Health Belief Model in Bushehr District, South of Iran. *Int J Hypertension*. *14*(1), 4-12.

Babisch, W. (2003). Stress hormones in the research on cardiovascular effects of noise. *Noise Health, 5*(18), 1–11.

Balder, J. W. (2017). Lipid and lipoprotein reference values from 133,450 Dutch Lifelines participants. *Journal of Clinical Lipidology*, *1, 11*(4), 1055-1064.e6.

Bantis, L. F. (2016). Comparison of two correlated ROC curves at a given specificity or sensitivity level. *Stat Med., 35*(24), 4352–67.

Bantle, J. R. (2000). Effects of dietary fructose on plasma lipids in healthy subjects. *Am J Clin Nutr , 72,* 1128–1134.

Barbara, E. R. (2002, Oct). Components of the Metabolic Syndrome and Risk of Cardiovascular Disease and Diabetes in Beaver Dam. *DIABETES CARE, 25*(10), 94-112..

Basavakumar, S. A. (2017). assessment of work-related stress and its associated factors among managerial staff in an industry. *National Journal of Research in Community Medicine, 6*(1), 009-014.

Baselet, B. C. (2016). Cardiovascular diseases related to ionizing radiation: The risk of low-dose exposure (Review. *International journal of molecular Medicine*, 1623-1641.

Bengt, S. (2015). Cardiovascular Disease. In S. C. Bengt, & B. A. Gunnar F. Nordberg (Ed.), *Handbook on the Toxicology of Metals (Fourth Edition)* (Vol. I, pp. 313-331). New York: Elsevier academic

Bergman, R. K. (2006). Why visceral fat is bad: mechanisms of themetabolic syndrome. Obesity. *Silver Spring*, *14*(suppl1), 16S–19S.

Berrington, d. G.-C. (2010). Body mass index and mortality among 1.46 million white adults. *N Engl JMed*, *363*, 2211–2219.

Beverly, M., Ikenna, C. E., Ramadhani, A., Salim, A., Paul, K., Marcel, T., . . . Nicole, P.-H. (2018). Insufficient Fruit and Vegetable Intake in a Low- and Middle-Income Setting: A Population-Based Survey in Semi-Urban Tanzania. *Nutrients, 10*, 222.

Bhui, K. S.-M. (2016). Perceptions of work stress causes and effective interventions in employees working in public private and non-governmental organisations: a qualitative study. *BJPsych Bulletin, 40*, 318-325,.

Bin, L. S. (2012). Effects of Cold Air on Cardiovascular Disease Risk Factors in Rat. *Int. J. Environ. Res. Public Health, 9*, 2312-2325.

Bitsika, V. S. (2014). HPA and SAM axis responses as correlates of self- vs. parental ratings of anxiety in boys with an Autistic Disorder. *Physiol. Behav.*, *127*, 1–7.

Bitton, A. G. (2010). The Framingham Heart Study's impact on global risk assessment. *Progress in Cardiovascular Diseases*, *53*(1), 68-78.

Bjorn, b. c. (2016). cardiovascular diseases related to ionizing radiation: the risk of low-dose exposure (review. *international journal of molecular medicine, 38*, 1623-1641.

Blackburn, H., & Darwin, l. (2012, Nov). Stories From the Evolution of Guidelines for Causal influence in epidemiologic association:1953-1965. *AJE. American journal of epidemiology, 176*(12), 1071-1077.

Bodil, B. L. (2006). Vibration exposure and myocardial infarction incidence:the VHEEP case–control study,. *Occupational Medicine, 56*, 338–344. doi:doi:10.1093/occmed/kql024

Bolger, N. D. (1989). The contagion of stress across multiple roles. *Journal of Marriage and Family,*, 51, 175 – 183.

Bozkurt, B. (2021). Universal Definition and Classification of Heart Failure. *Journal of cardiac failure. 27*(4), 387-413.

Bradburn, M. C. (2003). Survival Analysis Part II: Multivariate data analysis – an introduction to concepts and methods. *British Journal of Cancer, 89*, 431 – 436.

Brainerd, E. C. (2004). Autopsy on an Empire: Understanding Mortality in Russia and the Former Soviet Union.NBER working paper 10868, National Bureau of Economic Research. *Cambridge, MA*. Retrieved from http://www.nber.org/papers/w1086

Brathwaite, R. A. (2015). A Systematic Review of Tobacco Smoking Prevalence and Description of Tobacco Control Strategies in Sub-Saharan African Countries;2007 to 2014. *PLoS ONE, 10*(7), e0132401.

Bray, G. A. (2004). Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *Am J Clin Nutr, 79*(4), 537-43.

Bridget, M. K. (2019). Sleep Duration Linked to Cardiovascular Disease,. *Circulation., 139*, 2483–2484.

Brown, C. D.-P. (2008). Sugary drinks in the pathogenesis of obesity and cardiovascular diseases. *International Journal of Obesity, 32*, S28–S34.

Buitrago, F., Juan, I., Lourdes, C., Gerónimo, P., Luis, M., Manuel, E., & al., e. (2011). Original and REGICOR Framingham Functions in a Nondiabetic Population of a Spanish Health Care Center:A Validation Study. *Ann Fam Med, 9*(5), 431-8.

Bulka, C. M. (2019). Association of occupational exposures with cardiovascular disease among US Hispanics/Latinos. *heart, 105*, 439–448.

Butkiewicz, M., Wang, Y., Bryant, S., Lowe , E., Weaver, C., & Meiler, J. (2017). "High-throughput screening assay datasets from the PubChem database,". *Chemical Informatics, 3*(1), 1.

Carl, J. L. (2019). Sedentary Behavior, Exercise, and Cardiovascular Health. *Circ Res., 124*, 799-815.

Carmoa, G. M. (2008). Can we measure the ankle–brachial index using only a stethoscope, A pilot study,. *Oxford journal fp,*, 22-26.

Carnethon, M. W. (2009). Worksite wellness programs for cardiovascular disease prevention: a policy statement from the American Heart Association. *Circulation, 120*, 1725–41.

Carnevale, F., & Iavicoli, S. (2014). Bernardino Ramazzini (1633–1714): a visionary physician, scientist and communicator. *Occupational and Environmental Medicine*, *72*(1), 2-3.

Casey, S.(2019). The reliability of the ankle brachial index: a systematic review. *J Foot Ankle Res 12,* 39.

Cassani, R. S. (2009). Prevalence of Cardiovascular Risk Factors in a Population of Brazilian Industry Workers, . *Arq Bras Cardiol*, *92*(1), 15-21.

Corey, C. G., King, B. A., Coleman, B. N., Delnevo, C. D., Husten, C. G., Ambrose, B. K., & Apelberg, B. J. (2014). Little filtered cigar, cigarillo, and premium cigar smoking among adults—United States, 2012–2013. *MMWR. Morbidity and mortality weekly report*, *63*(30), 650.

Cavagioni, L., & Pierin, AMG (2012). Cardiovascular risk among health professionals working in pre-hospital care services. *Journal of the USP School of Nursing*, *46*, 395-403.

CDC. (2004). *Preventing Heart Disease and Stroke: Addressing the Nation's Leading Killers.* National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Department of Health and Human Services. Atlanta: CDC At-A-Glance Report.).

Chan, W. P. (2015). Ankle-Brachial Index Measurement. *Medline*, *2*(6), e000257.

Chen, X. Z. (2015). Regulation of Coronary Endothelial Function by Interactions between TNF-α,LOX-1 and Adiponectin in Apolipoprotein E Knockout Mice. *J Vasc Res, 52*, 372-382.

Chia, Y. G. (2015). Validation of the Framingham general cardiovascular risk score in a multiethnic Asian population: a retrospective cohort study. *BMJ , 5*, e007324.

Chong, A. (2016). *Home and work stress spillover: the roles of social support and positive reappraisals.* Unpublished PhD desertation, Ohio: Kent State University.

Christopher, C. I. (2014). Family history of cardiovascular disease (CVD), perceived CVD risk, and health-related behavior: A review of the literature. *J Cardiovasc Nurs*, *29*(2), 108–129.

Chu-Hong, L. S.-T.-X.-Q.-Q.-H. (2015). Community-based interventions in hypertensive patients: a comparison of three health education strategies. *BMC Public Health., 15*, 33.

Chung, H.(2017). Carbon disulfide exposure estimate and prevalence of chronic diseases after carbon disulfide poisoning-related occupational diseases. *Ann of Occup and Environ Med. 29*, 52.

Clark, T., & Bradburn, M. L. (2003). Survival analysis. Part I: basic concepts and first analyses. . *Br J Cancer , 89*, 232–238.

Clemes, S. P. (2014). Sitting time and step counts in office workers. *Occup Med, 64*(3), 188-92.

Cohen, S. J.-D. (2007). Psychological stress and disease. . *JAMA*, *298*(14), 1685–7.

Colby, S. P., & Karen, H. (2014). Validation of the Use of the Effort-Reward Imbalance Scale in Human Services Using Confirmatory Factor Analysis,. *Journal of the Society for Social Work and Research, 5*(4), 565-587.

Cole, TB. (2019). Smoking Cessation and Reduction of Cardiovascular Disease Risk. *JAMA. 322*(7), 651.

Cooper, C. M. (2013). *Occupational Sources of Stress: A Review of the Literature Relating to Coronary Heart Disease and Mental Ill Health In: Cooper C.L. (eds) From Stress to Wellbeing Volume 1* (Vol. 1). London, UK: Palgrave Macmillan.

Corner, M. (2002). *Health Behaviors*, UK: University of Leeds.

Cornner, M., & Norman, P. (1996). *Predicting Health Behaviour*. UK: Buckingham.

Coronel, R. d. (2001). Defining heart failure, On behalf of the editorial team of Cardiovascular Research. *Cardiovascular Research*, 419–422.

Coupland, A. P. (2017). The definition of stroke. *Journal of the Royal Society of Medicine, 110*(1), 9–12.

Covas, M. (2004). *The Mediterranean Diet and the Contribution and Role of Alcohol. In Comprehensive Handbook of Alcohol Related Pathology* (Vol. 1). San Diego,, USA: ElsevierAcademic Press.

Cox, D. (1972). Regression models and life tables (with discussion),. *Journal of the Royal Statistical Society, ,Series B 34*, 187–220.

Cruz, S. L. (2014). Review of toluene action: clinical evidence, animal studies and molecular targets. *Journal of drug and alcohol research, 3*(10), 4303.

Cui, Y., Blumenthal, R. S., Flaws, J. A., Whiteman, M. K., Langenberg, P., Bachorik, P. S., & Bush, T. L. (2001). Non–high-density lipoprotein cholesterol level as a predictor of cardiovascular disease mortality. *Archives of internal medicine*, *161*(11), 1413-1419.

Cynthia, C. W., Marcos, S., Monica, L. Z., Raul, C., Marcos, Z., Ronaldo, R. L., & Ilana, P. (2011, Dec). Differences in drinking patterns between men and women in Brazil,. *Brazilian Journal of Psychiatry, 33*(4). S1516.

D'Agostino, R. B. (2011). Validation of the Framingham Coronary Heart Disease Prediction Scores. *Jama, 286*(2), 180-187.

D'Agostino, R., Ramachandran, S., Vasan, M., Pencina, P., Wolf, M., Joseph, M., & al, e. (2008). General Cardiovascular Risk Profile for Use in Primary Care, The Framingham Heart Study. *Circulation., 117*, 743-53.

D'Angelo, G. E. (2005). Fructose feeding increases insulin resistance but not blood pressure in Sprague-Dawley rats. *Hypertension;, 46,* 806–811.

Dagfinn, A. (2012). Soft drinks, aspartame, and the risk of cancer and cardiovascular disease. *Am J Clin Nutr, 96,* 1249–51.

Dagfinn, A. E. (2017). Fruit and vegetable intake and the risk of Cardiovascular disease, total cancer and all cause mortality—a systematic review and dose response meta-analysis of prospective studies. *International Journal of Epidemiology,* 1029–1056.

Dagfinn, A. N. (2016). Whole grain consumption and risk of cardiovascular disease, cancer, and all cause and causes pecific mortality systematic review and dose-response meta-analysis of prospective studies, . *BMJ*, 353, i2716.

D'Agostino RB., ,. G. (2001). Validation of the Framingham coronary heart disease prediction scores: results of a multiple ethnic groups investigation. *JAMA, 286*(2), 180-7.

Danesh , J., Emanuele , D., Pei , G., Hassan , K., Adam , S., David, W., & Stephen , K. (2014). Glycated Hemoglobin Measurement and Prediction of Cardiovascular Disease. *JAMA, 311*(12), 1225-1233.

Daniel, A. L., & G. F. (2009). Behavioural biologists don't agree on what constitutes behavior. *Animal behaviour, 78*(1), 103-110.

Daniel, I. F.-H. (2008). Uric Acid and Cardiovascular Risk,. *Engl J Med, 359*(17), 1811–1821.

Daniel, J. (2017, Jan). What Is a Safe Noise Level for the Public?,. *AJPH, 107*(1).

David, M. N. (2007). Impaired Fasting Glucose and Impaired Glucose Tolerance,. *Diabetes Carer, 30*(3), 753-759.

David, P., Mark , A., Sidney, C., Richard, C., Jeffrey, W., Michael , A., . . . & Joseph, L. (2004). , Atherosclerotic Vascular Disease Conference, Executive Summary: Atherosclerotic Vascular Disease Conference Proceeding for Healthcare Professionals From a Special Writing of the American Heart Association. *Circulation, 109*(21), 2595-2604.

Davood , K., Farzad , H., Hamid, S., Ewout , W., Mohammadreza , B., & Fereidoun, A. (2012). Clinical Usefulness of the Framingham Cardiovascular Risk Profile Beyond Its Statistical Performance,The Tehran Lipidand Glucose Study. *AJC,, 76*(3).

Deborah, K.-G. A. (2015). Awareness of Cardiovascular Disease and Preventive Behaviors among Overweight Immigrant Latinas. *J Cardiovasc Nurs., 30*(5), 447–455.

de Groot, R. (2019). Urban-rural differences in the association between blood lipids and characteristics of the built environment: a systematic review and meta-analysis. BMJ global health. 4(1), e001017.

deKoning, L. M. (2007, Apr). Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart J, 28*(7), 850-6.

DeKoning, L. M. (2012). Sweetened beverage Consumption, incident coronary heart disease, and biomarkers of risk in men. *Circulation, 126*, 1301-1313.

DeMarco, M., Josef, C., Mark , W., Kenneth , R., W., H., Thomas , H., . . . Cheryl , A. (2011). Hypertension Status, Treatment and Control among Spousal Pairs in a Middle-aged Adult Cohort. *Am J Epidemiol*. *174*(7), 790-796.

Despres, J. (2012). Body Fat Distribution and Risk of Cardiovascular Disease an Update. *Circulation., 126*, 1301-1313.

Dhandevi, P. R. (2015, Oct). Fruit and Vegetable Intake: Benefits and Progress of Nutrition Education Interventions- Narrative Review Article. *Iran J Public Health, 44*(10), 1309–1321.

Dhingra, R. (2012). Age as a Cardiovascular Risk Factor. *Med Clin North Am, 96*(1), 87–91.

Dokken, B. B. (2008, JUL). The Pathophysiology of Cardiovascular Disease and Diabetes: Beyond Blood Pressure and Lipids. *Diabetes Spectrum, 21*(3), 160-165.

Donadini, G. S. (2008). Arsenic, Cadmium and Lead in Beers from the Italian Market. *J. Inst. Brew*, *114*(4), 283–288.

Douglas, S. L.(2009). Cardiovascular Outcomes Research Team. Trends in risk factors for cardiovascular disease in Canada: temporal, socio-demographic and geographic factors. *cmaj*. *4*(3-4), E55-66.

Dujrudee, C. N. (2018). Comparison of Gender Differences in Smoking Behaviors, Intention to Quit, and Nicotine Dependence among Thai University Students,. *Journal of Addiction, 2018*, 1155.

Dzhambov, A. D. (2016). Heart disease attributed to occupational noise, vibration and other co-exposure: Self-reported population-based survey among Bulgarian workers. *Med Pr., 67*(4), 435-45.

Eberly, L. A., Rusingiza, E., Park, P. H., Ngoga, G., Dusabeyezu, S., Mutabazi, F., ... & Bukhman, G. (2019). 10-Year heart failure outcomes from nurse-driven clinics in rural sub-Saharan Africa. *Journal of the American College of Cardiology*, *73*(8), 977-980.

Eckardstein, A. H. (2005). Current understanding of the metabolism and biological actions of HDL. *Curr Opin Clin Nutr Metab Care, 8*, 147-152.

Edyta, S. E. (2018). Relationship Between Sitting Time, Physical Activity, and Metabolic Syndrome Among Adults Depending on Body Mass Index (BMI),. *Med Sci Monit, 24*, 7633–7645.

Elisa, U. (2010). *Occupation health and safety report.* Australia: Mc grow.

Elizabeth, B. (2018). Moran Trends in Healthcare Expenditures Among US Adults With Hypertension: National Estimates, 2003–2014. Journal of the American Heart Association. 7, e008731.

Elliott, S. K. (2002). Fructose, weight gain, and the insulin resistanc syndrome. *Am J Clin Nutr*, *76*, 911–922.

Emelia, J. B. (2017). Heart Disease and Stroke Statistics-2017 Update: A Report From the American Heart Association. *Circulation, 135*(10), e146–e603.

Emil, M. d. (2012). The Evolution and Refinement of Traditional Risk Factors for Cardiovascular Disease. *Cardiol Rev, 20*(3), 118–129.

Eng, J. M. (2016). Impact of a Workplace Health Promotion Program on Employees Blood Pressure in a Public University. *PLoS One., 1*(2), e0148307.

Eric, J. S. (2010, August 17). Physical Physica Activity and Cardiovascular Health, Lessons Learned From Epidemiological Studies Across Age, Gender, and Race/Ethnicity,. *Circulation, Volume 122*,(Issue 7).

European Agency for Safety and Health at Work. (2008). *Workplace exposure to vibration in Europe: an expert review.* UK: Office for Official Publications of the European Communities.

Ewid , M., Hossam , S., Syed , M., Nazmus , S., Wael, A., Omer , R., . . . & Rami, A. (2019). Glycated hemoglobin predicts coronary artery disease in non-diabetic adults. *BMC Cardiovascular Disorders, 19,* 309.

Faijer-Westerink, H. J., Kengne, A. P., Meeks, K. A., & Agyemang, C. (2020). Prevalence of metabolic syndrome in sub-Saharan Africa: A systematic review and meta-analysis. *Nutrition, Metabolism and Cardiovascular Diseases, 30*(4), 547-565.

FAO. (2017). *fruit and vegetables for health initiative,WHO/FAO Joint report.* Geneva: FAO.

Farah, V. E. (2006). Nocturnal hypertension in mice consuming a high fructose diet. *Auton Neurosci, 130,* 41–50.

Flávio, D. F. (2019). High Blood Pressure and Cardiovascular Disease. *Hypertension, 75,* 285–292.

Florin, M. M. (2011). Exposure to cold environments at working places and cardiovasculare disease. *rcis, 33,* 197 – 208.

Francesco, P. C. (2016). Cardiovascular disease and hypertension in sub-Saharan Africa: burden, risk and interventions. *Intern Emerg Med., 11*, 299–305.

Francisco, A. H. (2016). High-Sensitivity C-Reactive Protein and Cardiovascular Disease across Countries and Ethnicities,. *Clinics (Sao Paulo), 71*(4), 235–242.

Frank, L. T. (2018). Prevalence, awareness, treatment, and control of hypertension in Cameroonians aged 50 years and older: A community- based study. *Health Sci Rep, 1*, 44.

Frary, C. J. (2005). Food sources and intakes of caffeine in the diets of persons in the United States. *J Am Diet Assoc, 105*, 110-113.

Epstein, F. H. (1996). Cardiovascular disease epidemiology: a journey from the past into the future. *Circulation, 93*(9), 1755-1764.

Fujii, Y., Kishimoto, S., & Higashi, Y. (2020). Finger blood flow after the cold challenge with primary Raynaud's syndrome: a case report. *European heart journal. Case reports, 4*(6), 1–5.

Fung, T. M. (2009). Sweetened beverage Consumption and risk of coronary heart disease in women. *Am J Clin Nutr, 89*, 1037–42.

Fuster, V. (2014). Global Burden of Cardiovascular Disease, Time to Implement Feasible Strategies and to Monitor Results. *Journal of the American college of Cardiology, 64*.

Fuster, V., Kelly, B. B., & Vedanthan, R. (2011). Promoting global cardiovascular health: moving forward. *Circulation, 123*(15), 1671-1678.

Ganesan, K. K. (2018). Impact of consumption and cooking manners of vegetable oils on cardiovascular diseases- A critical review,. *Trends in Food Science & Technology, 71*, 132–154.

Genuth, S. A. (2003). Follow-up report on the diagnosis of diabetes mellitus. *Diabetes Care, 26*(11), 3160-3167.

Gersh, B. S. (2010). Novel therapeutic concepts: the epidemic of cardiovascular disease in the developing world: global implications. *Eur Heart J., 31*(16), 642–648.

Gessner, B.D.(2019). A Systematic Review of Systemic Cobaltism After Wear or Corrosion of Chrome-Cobalt Hip Implants.Journal of patient safety.15(2), 97–104.

Giuseppe, M. R. (2013). 2013 ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). *European Heart Journal, 34*(28), 2159–2219,.

Gonullu, H. K. (2011). ST Elevation Myocardial Infarction Due to Carbon Monoxide Poisoning. *The Eurasian journal of medicine, 43*(2), 125–128.

Grundy , S. (2006). Metabolic syndrome: connecting and reconciling cardiovascular and diabetesworlds,". *Journal of theAmerican College of Cardiology, 47*(6), 1093–1100.

Grundy, S. C. (2005). Diagnosis and management of the metabolic Syndrome: an American Heart Association/NationalHeart, Lung, and Blood Institute Scientific statement. *Circulation, 112*(17), 2735–2752.

Gul, F.(2022). *Peripheral Vascular Disease. In: StatPearls*.Treasure Island (FL): StatPearls.

Guwatudde, D., Nankya-Mutyoba, J., Kalyesubula, R., Laurence, C., Adebamowo, C., Ajayi, I., & al., e. (2015). The burden of hypertension in sub-Saharan Africa: a four-country cross sectional study. *BMC Public Health, 15*, 1211.

Guy, N, Törnroos, M., & Salin, D. (2019). Effort-reward imbalance: A risk factor for exposure to workplace bullying. *Frontiers in psychology*, *10*, 386.

Habiyaremye, F. R. (2019). Tobacco use and associated factors among Rwandan youth aged 15-34 years: Findings from a nationwide survey, 2013. *PLoS ONE, 14*(10), e0212601.

Hahad, O. K.-S. (2019). The cardiovascular effects of noise. *Dtsch Arztebl Int, 116*, 245–50.

Haitham, M. A. (2012). Effects of Physical Activity on Cardiovascular Disease,. *Amercan journal of cardiology, 102*(2), 288–295.

Havakuk, O. Z. (2018). Shift Work and the Risk of Coronary Artery Disease: A Cardiac Computed Tomography Angiography Study. *Cardiology 2018, 139*, 11-16.

Heather, L. G. (2005). Peripheral arterial Disease. *Circulation, 111*, e169-e172.

Heffron, S. P. (2017). Greater Frequency of Fruit and Vegetable Consumption Is Associated With Lower Prevalence of Peripheral Artery Disease,. *Arterioscler Thromb Vasc Biol, 37*, 1234-1240.

Hema, S. S. (2011). Non-pharmacological Interventions in Hypertension: A Community-based Cross-over Randomized Controlled Trial. *Indian J Community Med, 36*(3), 191–6.

Herbert, C., & Moses, J. (2019). Cardiovascular Disease Healthcare Utilization in Sub-Saharan Africa: A Scoping Review. *Int J Environ Res Public Health, 16*, 419.

Hoang, A. Assessment of Methylene Chloride–Related Fatalities in the United States, 1980-2018. *JAMA Intern Med. 181*(6), 797–805.

Hong, Y. M. (2008). Impact of plaque rupture and elevated C-reactive protein on clinical outcome in patients with acute myocardial infarction: an intravascular ultrasound study. *J Invasive Cardiol, 20*(9), 428-435.

Hong-Kyu, k. C.-H.-Y.-W.-J. (2013). Impaired Fasting Glucose and Risk of Cardiovascular Disease in Korean Men and Women. *Diabetes Care Feb, 36*(2), 328-335.

Houston, M. (2002). *he Role of Vascular Biology, The Role of Vascular Biology*. Retrieved from https://www.scripps.org/assets/documents/houston_gr_jan_20. pdf

Houston, M. (2018). The role of noninvasive cardiovascular testing, applied clinical nutrition and nutritional supplements in the prevention and treatment of coronary heart disease. *Therapetic advances in cardiovascular disease, 13*(3), 85-108.

HSE. (2005). Whole-body vibration, The Control of Vibration at Work Regulations 2005 Guidance on Regulations. *crown*. Retrieved from https://www.hse.gov.uk/pUbns/priced/l141.pdf

Hsu , Y.-Y., Chyi-Huey , B., Chien-Ming, Y., Ya-Chuan , H., Tzu-Ting, L., & Chih-Hung , L. (2019). Long Hours' Effects on Work-Life Balance and Satisfaction, *BioMed Research International, 2019,* 8.

Huang, J.J. (2013 , jun). Effects of a workplace multiple cardiovascular disease risks reduction program. *Asian Nurs Res (Korean Soc Nurs Sci), 7*(2), 74-82.

Hussain, M. M. (2014, JUN). Intestinal Lipid Absorption and Lipoprotein Formation. *Curr Opin Lipidol, 25*(3), 200–206.

Ikäheimo T. M. (2018). Cardiovascular diseases, cold exposure and exercise. *Temperature, 5*(2), 123–146.

ILO. (2014). *safety and health in the use of chemicals Report at work, World Day for safety and health at work.* Geneva: International Labor Organization.

Imamura, F. L. (2015). Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type ,diabetes:

systematic reviewmeta-analysis, and estimation of population attributable fraction. *BMJ* , 351-357.

Iozzo, P. (2011). Myocardial, perivascular, and epicardial fat. *Diabetes Care, 34*, 371–S379.

Ipek, E. O. (2017). Hypertensive crisis: an update on clinical approach and management. *Curr Opin Cardiol, 32*(4), 397-406.

Iqbal, M. (2014). Trans fatty acids – a risk factor for cardiovascular disease. *Pak J Med Sci, 1*(30), 194-197.

Iraj, S. S. (2013). Investigating the effect of role conflict and role ambiguity on employees' job stress: Articulating the role of work-family conflict. *Management Science Letters*, 1927–1936.

Ising, H. K. (2004). Health effects caused by noise: evidence in the literature from the past 25 years. *Noise Health. 2004, 6*(22), 5–13.

Ismail, S. M. (2018). Systematic review of palm oil consumption and the risk of cardiovascular disease. *PLoS ONE, 13*(2), e0193533.

Ismail, Y. U. (2007). Effects of gamma irradiation on trans fatty acid composition in ground beef. *Food Control, 18*, 635–638.

Swain, J. D., Pugliese, D. N., Mucumbitsi, J., Rusingiza, E. K., Ruhamya, N., Kagame, A., ... & Morton Bolman, R. (2014). Partnership for sustainability in cardiac surgery to address critical rheumatic heart disease in sub-Saharan Africa: the experience from Rwanda. *World journal of surgery*, *38*(9), 2205-2211.

Jafaralilou, H. I. (2019). The impact of theory-based educational intervention on improving helmet use behavior among workers of cement factory iran. *J Egypt Public Health Assoc., 94*, 1.

James, B. M. (2002, Oct). Fasting and Postchallenge Glycemia and Cardiovascular Disease Risk. *Diabetes Care, 25*(10), 1845-1850.

James, N. R. (2014). Risk Factors for Cardiovascular Diseases in Youth. *Exerc Sport Sci Rev., 42*(4), 145-152.

Jamison, D. T. (2006). Disease and Mortality in Sub-Saharan Africa. In D. T. Jamison, *2nd edition,Cardiovascular diseases,*. Geniva: The International Bank for Reconstruction and Development/The World Bank.

Janet, M. T. (2009). Coronary Heart Disease Risk Factors. *JAMA, 302*(21), 2388.

Jaspinder, K. (2014). A Comprehensive Review on Metabolic Syndrome. *Cardio Res Pract.*

Jemaa, R.(2020). Prevalence of cardiovascular risk factors in the Tunisian population: The ATERA-survey, *Archives of Cardiovascular Diseases Supplements.12*(1), 1878-6480.

Jennifer, L.(2019). Cardiovascular Risks Associated with Gender and Aging. J. Cardiovasc. *Dev. Dis. 6*(19), 3390.

Jennifer, P. K., Cahill, L. E., Hu, F. B., Rexrode, K. M., Manson, J. E., & Rimm, E. B. (2013). Hemoglobin a1c is associated with increased risk of incident coronary heart disease among apparently healthy, nondiabetic men and women. *Journal of the American Heart Association*, *2*(2), e000077.

Jian, L. A. (2016). Work stress and cardiovascular disease: a life course perspective. *J Occup Health, 58*, 216-219.

Jin, Y. N., Juyoung, K., Kyung, H. C., Young, C., Jaewoo, C., Jaeyong, S., & Eun-Cheol, P. (2016). Associations of sitting time and occupation with metabolic syndrome in South Korean adults: a cross-sectional study,. *BMC Public Health, 16*(1), 943.

John, E. B. (2011). Radiation as a Risk Factor for Cardiovascular Disease. *ANTIOXIDANTS & REDOX SIGNALING, 15*(7).

Jones, J. H. (2002). *Self-reported work related illness in Great Britain. HSE Epidemiology and Medical Statistics Unit.*

Joshi, D. R. (2019). An Overview on Common Organic Solvents and Their Toxicity. *JPRI, 28*(3), 1-18.

Joshua, A. (2002). Diseases of the veins. *Circulation, 106*, 2170-2172 .

Kabwama, S. N. (2019). Low consumption of fruits and vegetables among adults in Uganda: findings from a countrywide cross-sectional survey,. *Archives of Public Health, 77*(4), 0332-6

Kaiye, G. X. (2017). The life-course impact of smoking on hypertension, myocardial infarction and respiratory diseases. *Sci Rep, 7*, 4330.

Kamarudeen, S. (2014). *Constraints and Opportunities in Rwanda's Industrial Sector: Kamarudeen-Soederbom-2013-Working-Paper.pdf.* London: International Growth Centre. Retrieved from https://www.theigc.org/wp-content/uploads/2014/09/Kamarudeen-Soederbom-2013-Working-Paper.pdf

Kang, M.-Y. H.-C. (2017). Crossover effect of spouse weekly working hours on estimated 10-years risk of cardiovascular disease. *PLoS ONE, 12*(8). e0182010.

Kaplan, N. G. (2010). *Kaplan's Clinical Hypertension.* Philadelphia: Lippincot Williams & Wilkins.

Kaplan, N. L. (2002). *Kaplan's Clinical Hypertension.* Philadelphia: Lippincot Williams & Wilkins.

Kaptoge, S., Pennells, L., DeBacquer, D., Cooney, M., Kavousi, M., Stevens, G., & al., e. (2019). The WHO CVD Risk Chart Working Group, World Health

Organization cardiovascular disease risk charts:revised models to estimate risk in 21 global regions. *Lancet Glob Health, 7*, 1332–45.

Karl Peltzer & Nancy Phaswana-Mafuya (2012) Fruit and vegetable intake and associated factors in older adults in South Africa, *Global Health Action, 5*(1), 18668,

Kaze, A., Schutte, A., Erqou, S., Kengne, A., Echouffo-Tcheugui, J., & al, e. (2017). Prevalence of hypertension in older people in Africa: a systematic review and meta-analysis. *J Hypertens, 35*(7), 1345-52.

Kazim, H. R. (2014). Alcohol-induced hypertension: Mechanism and prevention. *World J Cardiol, 5*, 245–52.

Kazlauskienė, L. B. (2015). Metabolic syndrome related to cardiovascular events in a 10-year prospective study. *Diabetol Metab Syndr, 7*, 102.

Keithellakpam, K., Mohammad , A., Brijesh , K., Mishra, R., Jhamb , S., & Venkata , M. (2019). *Association of postprandial triglyceride responses with insulin resistance among rotational night shift healthcare workers*. New York: wiley.

Kévin , D. (2010). Social Cognitive Theory in IS Research – Literature Review, Criticism, and Research Agenda. *ICISTM, 54*, 3-642.

Khera, R., Lu, Y., Lu, J., Saxena, A., Nasir, K., & Jiang, L. e. (2018). Impact of 2017 ACC/AHA guidelines on prevalence of hypertension and eligibility for antihypertensive treatment in United States and China: nationally representative cross-sectional study. *BMJ., 362*, k2357.

Kim, K.-W. Y.-H. (2012). The Effects of Hazardous Chemical Exposure on Cardiovascular Disease in Chemical Products Manufacturing Workers,. *Toxicol. Res, 28*(4), 269-277.

Kiran J. Biddinger. (2020). Association of Habitual Alcohol Intake With Risk of Cardiovascular Disease, *JAMA.5*(3), e223849.

Kirsten, F. L.-C. (2014). Work Status and Return to the Workforce after Coronary Artery Bypass Grafting and/or Heart Valve Surgery: A One-Year-Follow Up Study. *Rehabil Res Pract*. *10*, 1155

Ki-Woong, K. Y.-H. (2012). The Effects of Hazardous Chemical Exposure on Cardiovascular Diseasein Chemical Products Manufacturing Workers. *Toxicol. Res., 28*(4), 269-277.

Koosha, P., Hamidreza, R., Nizal, S., Mehrbod, V., Mohammad, T., Erfan, S., & Masoumeh, S. (2020). High Sensitivity C-Reactive Protein Predictive Value for Cardiovascular Disease: A Nested Case Control from Isfahan Cohort Study (ICS). *Global Heart, 15*(1), 3.

Krishnamurthi, R. V. (2013). Global and regional burden of first-ever ischaemic and haemorrhagic stroke during 1990-2010: findings from the Global Burden of Disease Study 2010. *Lancet Glob Health, 1*(5), e259-81.

Kuklina, E. V. (2010). Assessing and Managing Risk for Cardiovascular Disease: A Worldwide Perspective. *North American Journal of Medicine and Science, 3*(2), 1246-1258.

Lange, S. T. (2007). Profound influence of different methods for determination of the ankle brachial index on the prevalence estimate of peripheral arterial disease. *BMC Public Health, 7*, 147.

Lantin, A. V. (2013). Occupational exposure to cobalt is not associated with incipient signs of dilated cardiomyopathy in a Belgian refinery. *Occup Environ Med, 70*(6), 386-392.

Larsson, S. C., Burgess, S., Mason, A. M., & Michaëlsson, K. (2020). Alcohol consumption and cardiovascular disease: a Mendelian randomization study. *Circulation: Genomic and Precision Medicine, 13*(3), e002814.

Lasebikan, V. A. (2018). Prevalence of alcohol consumption and alcohol use disorders among outdoor drinkers in public open places in Nigeria. *BMC Public Health, 18*, 400.

Lee, B. J. (2014). Air pollution exposure and cardiovascular disease. *Toxicological research, 30*(2), 71–75.

Leeder, S. (2014). *A race against time: the challenge of cardiovascular disease in developing Economies.* Colombia: The Earth Institut. Retrieved from https://www.researchgate.net/publication/277279425

Leon, B. M. (2015, Oct). Diabetes and cardiovascular disease: Epidemiology biological mechanisms, treatment recommendations and future research. *World J Diabetes, 6*(13), 1246-1258.

Leon, D. M. (2010). Alcohol increases circulatory disease mortality in Russia: acute and chronic effects or misattribution of cause. *Int J Epidemiol, 39*, 1279–90.

Leyland, L. S. (2019). The effect of cycling on cognitive function and well-being in older adults. *PLoS ONE, 14*(2), e0211779.

Li, Q., Yang, Z., Gu, H., Lu, S., Shi, Q., Xing, Y., ... & Su, Q. (2014). Association between serum uric acid levels and cardiovascular disease in middle-aged and elderly Chinese individuals. *BMC cardiovascular disorders*, *14*(1), 1-8.

Li, X., Dong, Q., Wang, B., Song, H., Wang, S., & Zhu, B. (2019). The influence of occupational noise exposure on cardiovascular and hearing conditions among industrial workers. *Scientific Reports*, *9*(1), 1-7.

Lim, S. V.-R. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions1990–2010: a systematic analysis for the Global Burden. *Lancet., 380*(9859), 2224–60.

Linna, A. (2020). Effects of occupational cobalt exposure on the heart in the production of cobalt and cobalt compounds: a 6-year follow-up. *International archives of occupational and environmental health. 93*(3), 365–374.

Linton, M. Y. (2019). The Role of Lipids and Lipoproteins in Atherosclerosis. *NCBI*. Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK343489/

Liu, G. L. (2015, Sept). Cardiovascular System Response to Carbon Dioxide and Exercise in Oxygen-Enriched Environment at 3800 m. *International journal of environmental research and public health, 19*(9), 11781–11796.

Longo, M.(2019). Adipose Tissue Dysfunction as Determinant of Obesity-Associated Metabolic Complications. International journal of molecular sciences. 20(9),2358.

Lorene, G. C.-C. (2015). Food habits and risk of cardiovascular disease in schoolchildren from Ouro Preto, Minas Gerais,. *Rev. Nutr., Campinas, 28*(2), 133-142.

Luc, D. M. (2009). Alcohol Consumption and Risk of Hypertension: Does the Type of Beverage or Drinking Pattern Matter? *Rev Esp Cardiol, 62*(6), 603-5.

Luckhaupt, S. E., & Calvert, G. M. (2014). Prevalence of coronary heart disease or stroke among workers aged< 55 years—United States, 2008–2012. *MMWR. Morbidity and Mortality Weekly Report*, *63*(30), 645.

Lugasi, A. B. (2015). CAFFEINE INTAKE IN HUNGARY – A POPULATION BASED ESTIMATION. *Acta Alimentaria, 42*(2), 242–250.

Luigi, I., Marcello , C., Igor , P., Maura , G., Nicola , M., Michele, M., & Pierluigi, C. (2018). Work Related Stress, Well-Being and Cardiovascular Risk among Flight Logistic Workers: An Observational Study. *Int. J. Environ Res. Public Health, 15*, 1952.

Luis , R., & César , C. (2012, Mars 02). Uric acid and cardiovascular risk considered. *an update e-journal of the ESC Council for cardiology Practice, 10*.

Lukwiya, O. C. (2013 , Mar). Epidemiology of ischaemic heart disease in sub-Saharan Africa. *Cardiovasc J Afr, 24*(2), 34-42.

Maas, A., & Appelman , Y. (2010). Gender differences in coronary heart disease, Netherlands. *Heart Journal, 18*(12), 598-603.

Macdonald, W. (2003, July). The Impact of Job Demands and Workload on Stress and Fatigue. *Australian psychologist, 38*(2), 102-117.

Mahmood, S. L. (2013, Sep). The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective. *Lancet*, 999-1008.

Malik, V. P. (2010). Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes a meta-analysis. *Diabetes Care, 33*, 2477–83.

Mann, J. (2007). Dietary carbohydrate: relationship to cardiovascular disease and disorders of carbohydrate metabolism. *European Journal of Clinical Nutrition*, *61*(Suppl 1), S100–S111 .

Mansell, H. S. (2014). Validity of cardiovascular risk prediction models in kidney transplant recipients. *Scientific World J.* 2014.

Manyema, M. L. (2012). modelling the potential impact of a sugar-sweetened beverage tax on stroke mortality, costs and health-adjusted life years in South Africa. *BMC Public Health*. *16*(1), 1-10.

Marianna, V. M. (2018). Long Working Hours and Risk of Cardiovascular Disease,. *Current cardiology reports, 20*(11), 1-7.

Maritza, J. K. & Nell, T. A. (2017). The prevalence of the metabolic syndrome in a farm worker community in the Boland district, South Africa. *BMC Public Health*, *17*(1), 1-10.

Mark, A. C., Christopher, J. W., William, R. H., Michael, H. C., Shellie, C. J. (2008). Atherosclerotic Peripheral Vascular Disease Symposium II Executive Summary. *Circulation, 118*, 2811-2825.

Martín-Timón, I.(2014). Type 2 diabetes and cardiovascular disease: Have all risk factors the same strength?. World journal of diabetes, 5(4), 444–470.

Mary, E. C. (2016, June). Dietary Sodium and Cardiovascular Disease Risk - Measurement Matters,. *the new England journal of medicine*, *375*(6), 580.

Mary, L. (2012). Interrater reliability: the kappa statistic. *Biochem Med(Zagreb), 22*(3), 276-82.

Matthew, A. N. (2018). Cardiovascular Effects and Benefits of Exercise. *Front Cardiovasc Med, 5*, 135.

McEachan, R. C., Lawton, R. J., Jackson, C., Conner, M., & Lunt, J. (2008). Evidence, theory and context: Using intervention mapping to develop a worksite physical activity intervention. *BioMedical Central Public Health, 8*, 326.

Mee, K. K., Kyungdo, H., Hun-Sung, K., Yong-Moon, P., Hyuk-Sang, K., Kun-Ho, Y., & Seung-Hwan, L. (2017, Oct). Cholesterol variability and the risk of mortality, myocardial infarction, and stroke: a nationwide population-based study,. *Eur Heart J., 38*(48), 3560–3566.

Mehan, M. S. (2006). Profile of non-communicable diseases risk factors in an industrial setting. *j postgrad med, 52*(3), 276-82.

MGI. (2012). *Global labor full report, the world at work.* Seoul: McKinsey Global Institute. Retrieved from https://www.mckinsey.com/~/media/McKinsey/Featured%20Insights/Employment%20and%20Growth/The%20world%20at%20work/MGI%20Global_labor_Full_Report_June_2012.ashx

Michael, R. K., & Cathy, S. (2014, Nov). Family history of cardiovascular disease,. *Can Fam Physician, 60*(11), 1016.

Michelle, L. K. (2015). The Application of High-Sensitivity C-Reactive Protein in Clinical Practice: A 2015 Update. *US Pharm., 40*(2), 50-53.

Mika, K., & Ichiro, K. (2015). Work Stress as a Risk Factor for Cardiovascular Disease. *Curr Cardiol Rep, 17*(9), 74.

Mika, K., Päivi, L., Ritva, L., Hilkka, R., Jussi, V., & Juhani, K. (2002). Work stress and risk of cardiovascular mortality:prospective cohort study of industrial employees. *bmj, 325*, 857.

Ming, W., Larry, W. G., Tedd, L. M., James, B. K., Michael, P. S., & Steven, N. (2000). Low Fasting Plasma Glucose Level as a Predictor of Cardiovascular Disease and All-Cause Mortality,. *Circulation, 101*, 2047-2052.

Mion Júnior, D., Junior, O. K., & Gomes, M. A. M. (2006). V Diretrizes brasileiras de hipertensão arterial. *Sociedade Brasileira de Cardiologia. Sociedade Brasileira de Hipertensão. Sociedade Brasileira de Nefrologia*.

Miranda, J. R. (2011). Differences in cardiovascular risk factors in rural, urban and rural-to-urban migrants in Peru. . *Heart, 97*(10), 787–96.

Mocumbi, A. O. (2012). Lack of focus on cardiovascular disease in sub-Saharan Africa. *cdt.org, 2*.

Mohammad, G. S. (2018). The Global Epidemic of the Metabolic Syndrome. *Curr Hypertens Rep, 20*(2), 12.

Monakali, S. G. (2018). Prevalence, awareness, control and determinants of hypertension among primary health care professional nurses in Eastern Cape,South Africa. *African J Primary Health Care Family Med, 10*(1), a1758.

Montano, D. (2014). Chemical and biological work-related risks across occupations in Europe: a review. *Journal of Occupational Medicine and Toxicology, 9*, 28.

Moon, K. (2012). Arsenic exposure and cardiovascular disease: an updated systematic review. Current atherosclerosis reports. 14(6), 542–555.

Mozaffarian, D. W. (2005). Beyond Established and Novel Risk Factors Lifestyle Risk Factors for Cardiovascular Disease. *CIRCULATION, 117*, 3031-3038.

Mozaffarian, D., Emelia, J. B., Alan, S. G., Donna, K. A., Michael, J. B., Mary, C., . . . Heather, J. F. (2015). Heart Disease and Stroke Statistics, Update. *Circulation, 131*(4), e29-e322.

Mozhdeh, H. A.-F.-N. (2019). Transtheoretical Model of Health Behavioral Change: A Systematic Review. *Iran J Nurs Midwifery Res, 24*(2), 83–90.

Muiesan, M. L.-R. (2016). Uric Acid and Cardiovascular Disease: An Update. *European Cardiology Review, 11*(1), 54–9.

Mukabutera, A. N. (2016). Overweight or Obesity prevalence, trends and risk factors among women in Rwanda: A cross-sectional study using the Rwanda Demographic and Health Surveys 2000–2010. *Rwanda Journal Series F: Medicine and Health Sciences, 3*(1), 235 - 242.

Mukaruzima, L. A. (2020). Leisure-time physical activity practices and the influencing factors among government office employees in Kigali, Rwanda. *Occupational Health Southern Africa*, *26*(1), 3 – 7.

Munyogwa, M. J. (2018). The Prevalence of Abdominal Obesity and Its Correlates among the Adults in Dodoma Region, Tanzania: A Community-Based Cross-Sectional Study. *Hindawi Advances in Medicine, 2018*.

Münzel, T.(2011). Andreas Daiber and Tommaso Gori, Nitrate Therapy New Aspects Concerning Molecular Action and Tolerance.*Circulation, 123*, 2132–2144.

Mustafa, J. A., & ., M. S. (2018). Ensemble approach for developing a smart heart disease prediction system using classification algorithms. *Res Rep Clin Cardiol, 1*, 33-45.

Nahimana, MR.(2018). A population-based national estimate of the prevalence and risk factors associated with hypertension in Rwanda: implications for prevention and control. *BMC Public Health. 18*, 2 (2018).

Nanchahal, K. D. (2000). Alcohol consumption, metabolic cardiovascular risk factors and hypertension in women,. *International Journal of Epidemiology, 29*, 57–64.

National Research Council. (1977). Biological effects of arsenic on man. In: Arsenic. Washington DC:. *National Academy of Sciences*, 173–91.

Navas-Acien, A. G. (2007). Lead exposure and cardiovascular disease--a systematic review. *Environmental health perspectives, 115*(3), 472–482.

NCEP. (2001). National Institutes of Health, Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Executive Summary of the Third Report of theNational Cholesterol Education Program (NCEP) Expert Panel. *JAMA, 285*, 2486-97.

Nehal, H., & Kanwal, K. (2011). impact of karasek job demand control model on the job satisfaction of the employees of nadra. *ijcrb*. *Interdisciplinary journal of contemporary research in business*, *3*(5), 566-594.

Ng, C. L. (2014). Heated vegetable oils and cardiovascular disease risk factors. *Vascul Pharmacol, 61*(1), 1-9.

Ngoungoua, E. B., Victor, A., Philomène, K., Roger, M., Ecke, N. J., Carine Ndong, A., . . . Philippe, L. (2012). Prevalence of cardiovascular disease in Gabon: A population study. *Elsevier,Archives of Cardiovascular Diseas, 105*, 77—83.

Nia, A. F. (2010). Torsades de pointes tachycardia induced by common cold compound medication containing chlorpheniramine. *Eur J Clin Pharmacol, 66*(11), 1173-1175.

Nichols, M., Townsend, N., Scarborough, P., Luengo-Fernandez, R., Real, J., Gray, A., & Rayner, M. (2012). European Cardiovascular Disease Statistics. *European Heart Network*. Retrieved from http://www.ehnheart.org/cvd-statistics.html

Nihal, A. M., & Tekin, S. (2007). cox regression models with nonproportional hazards applied to lung cancer survival data,. *Hacettepe Journal of Mathematics and Statistics, 36*(2), 157 – 167.

Nkusi, A., Muneza, S., Nshuti, S., Hakizimana, D., Munyemana, P., Nkeshimana, M., & al, e. (2017). Stroke Burden in Rwanda:A Multicenter Study of Stroke Management and Outcome. *World Neurosurg., 106*, 462-9.

Norfazilah, A. S. (2016). Abdominal Obesity Indicators: Waist Circumference or Waist-to-hip Ratio in Malaysian Adults Population. *Int J Prev Med, 7*, 82.

Norhayati, M. S. (2013). Cardiovascular risk: associated factors, assessment and agreement between WHO/ISH risk prediction chart and Framingham Scoring System among Primary Care Patients in Kelantan, Malaysia. *ijcrimph. International Journal of Collaborative Research on Internal Medicine & Public Health, 5*(12), 0-0.

Noubiap, J. J., Bigna, J. J., & Nansseu, J. R. (2015). Low Sodium and High Potassium Intake for Cardiovascular Prevention:Evidence Revisited With Emphasis on Challenges in Sub-Saharan Africa. *The Journal of Clinical Hypertension, 17*(1), 81-83.

O'Neil, A. S. (2018). Gender/Sex as a Social Determinant of Cardiovascular Risk. *Circulation., 137*, 854–64.

Ogeng'o, J. A., Gatonga, P., & Olabu, B. O. (2011). Cardiovascular causes of death in an east African country: an autopsy study. *Cardiology journal*, *18*(1), 67-72.

Oguoma, V., Ezekiel, U., Timothy, C., Kester , A., Innocent, C., & Ross, S. (2015). Prevalence of cardiovascular disease risk factors among a Nigerian adult population:elationship with income level and accessibility to CVD risks screening,. *BMC Public Health., 15*, 397.

Okafor, C. I. (2012). The metabolic syndrome in Africa: Current trends. *Indian J Endocrinol Metab, 16*(1), 56–66.

Olasky, S. J. (2012). Second hand smoke and cardiovascular disease in Low and Middle Income Countries: a case for action. *Glob Heart, 7*(2), 151–160.

Olijhoek , J., Van Der Graaf, y., Banga, J.-D., Algra, A., Rabelink, T., & Visseren, F. (2004). The Metabolic Syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease or abdominal aortic aneurysm. *European Heart Journal*, *25*(4), 342–348.

Oluseyi, A. O. (2019). Evaluation of Health Risk Level of Hand-Arm and Whole-Body Vibrations on the Technical Operators and Equipment in a Tobacco-Producing Company in Nigeria. *Journal of Healthcare Engineering, 12,* 2019.

Olvera, L. E. (2020, jan). *Cardiovascular Disease*. (StatPearls, Editor) Retrieved from https://www.ncbi.nlm.nih.gov/books/NBK535419/.

Omair, Y., Bibhu, D. M., Seth, S., Parag , H., Michael , J., Khurram, N., . . . & Matthew, J. (2013). High-Sensitivity C-Reactive Protein and Cardiovascular Disease. *J Am Coll Cardiol., 62*, 397-408.

Omotehinwa, O. J. (2018). Shisha use among students in a private university in Kigali city, Rwanda: prevalence and associated factors,. *BMC Public Health, 18*, 713.

OSHA,. (2020). *protecting yourself from noise in construction, OSHA pocket guide.* Occupational safety and health administration, workers safety series. OSHA. Retrieved from https://www.osha.gov/Publications/3498noise-in-construction-pocket-guide.pdf

Owolabi, E. O. (2017). Central obesity and normal-weight central obesity among adults attending healthcare facilities in Buffalo City Metropolitan

Municipality, South Africa: a cross-sectional study. *Journal of Health, Population and Nutrition. 36*(1), 1-10.

Packer, M. (2016). Cobalt cardiomyopathy: a critical reappraisal in light of a recent resurgence. *Circulation: Heart Failure, 9*(12), e003604.

Palikaras, K. (2017). Ectopic fat deposition contributes to age-associated pathology in Caenorhabditis elegans. *Journal of lipid research. 58*(1),72–80.

Pearson, T. M. (2003). Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. *Circulation, 107*(3), 499-511.

Pedro, F. S.- M. (2018). Moderate- to- Vigorous Physical Activity and All- Cause Mortality: Do Bouts Matter?,. *Journal of the American Heart Association, 7*, e007678.

Peltzer, K. D. (2011). Alcohol use and problem drinking in South Africa: findings from a national population-based survey. *Afr J Psychiatry, 14*, 30-37.

Perelman, R. (2011). *Perelman's pocket cyclopedia of cigars*. Glendale, CA:. Chromatic Lithographers.

Permentier, K. V. (2017). Carbon dioxide poisoning: a literature review of an often forgotten cause of intoxication in the emergency department. *International journal of emergency medicine,, 10*(1), 14.

Peter, L., & Pierre, T. (2005). Pathophysiology of Coronary Artery Disease,. *Circulation, 111*, 3481-3488.

Peter, W. (2016). Estimation of cardiovascular risk in an individual patient without known cardiovascular diseases. *Journal of the American College of Cardiology, 66*(24), 2812.

Petrukhin, I., & Lunina, E. (2012). Cardiovascular disease risk factors and mortality in Russia: challenges and barriers. *Public Health Reviews, 33,* 436-49.

Phillips, J., & Banyangiriki, J. (2015). Modifiable Risk Factors for Cardiovascular Disease: The Case of an African University. *African Journal for Physical, Health Education, Recreation and Dance*, *66*(24), 2812..

Plissonneau, P. P. (2015, Oct). Cold climate could be an etiologic factor involved in Raynaud's phenomenon physiopathology. Epidemiological investigation from 954 consultations in general practic. *Int Angiol, 34*(5), 467-74.

Poirier, P., Cornier, M., Mazzone, T., Stiles, S., Cummings, S., Klein, S., . . . & Franklin, B. (2011). Bariatric surgery and cardiovascular risk factors: a scientific statement from the American Heart Association. *Circulation, 123,* 1683–1701.

Poirier, P., Giles, T. D., Bray, G. A., Hong, Y., Stern, J. S., Pi-Sunyer, F. X., & Eckel, R. H. (2006). Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation, 113*(6), 898-918.

Prabhakaran, D. P. (2016). Cardiovascular Diseases in India, Current Epidemiology and Future Directions. *Circulation, 133,* 1605–20.

Priyanga, R. D. (2015). The influence of family history of Hypertension on disease prevalence and associated metabolic risk factors among Sri Lankan adults. *BMC Public Health., 15,* 576.

Pyakurel, P. P. (2016). Cardiovascular risk factors among industrial workers: a cross–sectional study from eastern Nepal. *J Occup Med Toxicol, 11*(25), 016-019.

Qiang, Z. S.-Y.-N. (2012, jul). Percent body fat is a better predictor of cardiovascular risk factors than body mass index. *Braz J Med Biol Res, 45*(7), 591-600.

Rabanal, K. S., Meyer, H. E., Tell, G. S., Igland, J., Pylypchuk, R., Mehta, S., ... & Jackson, R. (2017). Can traditional risk factors explain the higher risk of cardiovascular disease in South Asians compared to Europeans in Norway and New Zealand? Two cohort studies. *BMJ open*, *7*(12), e016819.

Rahimi-Sakak, F. M. (2019). Serum uric acid and risk of cardiovascular mortality: a systematic review and doseresponse meta-analysis of cohort studies of over a million participants. *BMC Cardiovascular Disorders*, *19*(1), 1-7.

Rahman, M., Nakamura, K., Seino, K., & Kizuki, M. (2015). Socio-demographic factors and the risk of developing cardiovascular disease in Bangladesh,. *Am J Prev Med, 48*(4), 456-61.

Rafieian-Kopaei, M. (2014). Atherosclerosis: process, indicators, risk factors and new hopes. *International journal of preventive medicine*, *5*(8), 927–946.

Rajagopal, G. S. (2012). High-density lipoprotein cholesterol: How High. *Indian journal of endocrinology and metabolism*, *16*(2), S236–S238.

Ramirez, F. C. (2017). Association Between Self- Reported Potentially Modifiable Cardiac Risk Factors and Perceived Need to Improve Physical Health: A Population- Based Study. *J Am Heart Assoc*, *6*(5), 117 – 129.

Rana, A. F. (2018). Shift Work and the Risk of Cardiovascular Diseases and Metabolic Syndrome among Jordanian Employees. *Oman Medical Journal, 33*(3), 235-242.

Ranthe, M. F. (2013). Risk of cardiovascular disease in family members of young sudden cardiac death victims. *European Heart Journal, 34*, 503-511.

Regitz-Zagrosek, V. S. (2015). Gender in cardiovascular diseases: impact on clinical manifestations, management, and outcomes. *Europian heart journal, 37*(1), 24-34.

Reibis, Rona. (2019) The importance of return to work: How to achieve optimal reintegration in ACS patients. *European journal of preventive cardiology.* 1(12), 2047-4881.

Rhonda, B. T. (2009). An overview of cardiovascular risk factor burden in sub-Saharan African countries: a socio-cultural perspective;. *Globalization and Health, 5*(10), 1186 -9.

Rice, M. B. (2014). Climate change. A global threat to cardiopulmonary health. *American journal of respiratory and critical care medicine,, 189*(5), 512–519.

Ridker, P. M. (2003). Clinical Application of C-Reactive Protein for Cardiovascular Disease Detection and Prevention. *Circulation, 107*, 363–369.

Rita, R. K. (2017). Diabetes and Aging: Unique Considerations and Goals of Care. *Diabetes Care, 40*(4), 440-443.

Robert, C. K., Avilés-Santa, M. L., Christina, M. P., David, B. H., Molly, J., Sheila, F. C., . . . Neil, W. (2014). Body Mass Index, Sex, and Cardiovascular Disease Risk Factors Among Hispanic/Latino Adults: Hispanic Community Health Study/Study of Latinos. *J Am Heart Assoc., 3*(4), e000923.

Rohan, Khera.(2020). Financial Toxicity in Atherosclerotic Cardiovascular Disease in the United States: Current State and Future Directions, *Journal of the American Heart Association, 9*, e017793.

Roger, V. L., Lloyd-Jones, D. M., Adams, R. J., Berry, J. D., & Brown, T. M. (2011). Heart disease and stroke statisticsd2011 update: a report from the American heart association. *Circulation, 123*(4), e18e209.

Romina, G.-M. (2020). Soft drink intake is associated with weight gain, regardless of physical activity levels: the health workers cohort study. *International Journal of Behavioral Nutrition and Physical Activity*. *17*(1), 1-10

Roy, A., Prabhakaran, D., Jeemon, P., Thankappan, K. R., Mohan, V., Ramakrishnan, L., ... & Sentinel Surveillance in Industrial Populations Study Group. (2010). Impact of alcohol on coronary heart disease in Indian men. *Atherosclerosis*, *210*(2), 531-535.

Roy , K. (2015). Causes of stress before, during and after organizational change: a qualitative study. *Journal of Organizational Change Management*, 301 - 314.

Rudasingwa, G. A. (2012, Nov). Clinical patterns and complications of African diabetic patients: preliminary data from Kigali University Teaching Hospital, Rwanda,. *African Journal of Diabetes Medicine*, *20*(2).

Rugge, B., Balshem, H., Sehgal, R., Relevo, R., Gorman, P., & Helfand, M. (2011). Screening and Treatment of Subclinical Hypothyroidism or Hyperthyroidism. *NCBI,AHRQ*. Retrieved from https://europepmc.org/article/NBK/nbk 83496.

Sacco, R. L. (2013). An Updated Definition of Stroke for the 21st Century: A Statement for Healthcare Professionals From the American Heart Association/American Stroke Association. *Stroke., 44*, 2064–2089.

Saeid, G. I. (2012). Exercise and the Cardiovascular System. *Cardiology Research and Practice, 10*, 1155.

Sakaguchi, K. (2015). Glucose area under the curve during oral glucose tolerance test as an index of glucose intolerance. *Diabetology international, 7*(1),53–58.

Salvatore, M., Kristian, B., Jacques, G., Lawrence, J., Louise, P., Paul, P., . . & Mark, J. (2010). The Metabolic Syndrome and Cardiovascular Risk: A Systematic Review and Meta-Analysis. *Journal of the American College of Cardiology*, *56*(14), 1113-1132.

Santosh, K. S. (2020). Cardiovascular Disease Risk Factors among Group C Employees in a Tertiary Health Care Centre in Puducherry: A Cross-sectional Study. *nternational Journal of Medicine and Public Health, 10*(4), 147-151.

Sara, E. L. (2014). Prevalence of Coronary Heart Disease or Stroke Among Workers Aged <55 Years-United States, 2008–2012,. *United States,, 63*(30).

Sarma, P. R. (2019). Prevalence of risk factors of non-communicable diseases in Kerala, India: results of a cross-sectional study. *BMJ journals*. *BMJ open, 9*(11), e027880.

Savitharani, B. M. (2016). Utilization of who-ish 10-year cvd risk prediction chart as a screening tool among supporting staff of a tertiary care hospital, Mysuru,India. *Heart India, 4*, 13-6.

Schnall, P. B. (2000). The Workplace and Cardiovascular Disease. *Occup Med, 15*(1), 1-5.

Schramm, A. (2016). Increased intima-media thickness in rayon workers after long-term exposure to carbon disulfide. *Int Arch Occup Environ Health. 89*, 513–9.

Sean, j. J. (2012). Acute and chronic effects of glyceryl trinitrate therapy on insulin and glycose regulation in human, *Journal of Cardiovascular Pharmacology and Therapeutics, 18*(3), 211-216.

Selvarajah, S., Kaur, G., Haniff, J., Cheong, K., Hiong, T., van, Y., & al., e. (2014). Comparison of the Framingham Risk Score, SCORE and WHO/ISH cardiovascular risk prediction models in an Asian population. *Int J Cardiol., 176*(1), 211-8.

Shlomo, S. S. (2003). Aging and Diseases of the Heart,. *Circulation, 108*(14).

Shrestha, N., Kukkonen-Harjula, K., Verbeek, J., Ijaz, S., Hermans, V., & Pedisic, Z. (2018). Workplace interventions (methods) for reducing time spent sitting at work. *Cochrane*. doi:doi/10.1002/14651858.CD010912.pub5/epdf/abstract

Silva, J. (2003). Colesterol, lípidos e doença vascular. Lidel, Edições Técnicas Lda.Shlomo Stern, Solomon Behar and Shmuel Gottlieb, Aging and Diseases of the Heart, *Circulation. 108*(14), 3264 - 3275.

Simao, M. M. (2002). Cardiovascular diseases: profile of male workers from a distillery in the countryside of são paulo state. *Revista Eletrônica de Enfermagem, 4*(2), 27 – 35.

Sivan, R. J. (2016, May). Inter-relation between autonomic and HPA axis activity in children and adolescents,. *Biol Psychol., 117*, 16–25.

Smallegange, R. C. (2010). Sugar-fermenting yeast as an organic source of carbon dioxide to attract the malaria mosquito Anopheles gambiae. *Malar J, 25*(9), 292.

Smith, , S. (2012). *Urbanization and cardiovascular disease: Raising heart-healthy children in today's cities.* GENEVA: The World Heart Federation. Retrieved from http://www.worldheart.org/urbanization

Sowmya, K. R. (2019). The palm oil industry and noncommunicable diseases. *Bull World Health Organ, 97*, 118–128.

Sridhar , N., Krishnakishore , C., Sandeep , Y., Sriramnaveen , P., Manjusha , Y., & Sivakumar , V. (2011). Chloroform poisoning-a case report. *Ren Fail., 33*, 1037–9.

Stephan, G. G. (2015). The Esc Textbook of Preventive Cardiology: Clinical Practice. *Oxford University Press*, 43-53.

Stephen , J., David , M., Murray , D., Ian , B., David , H., V Michael, E., . . . & Andrew, M. (2003). Stress" and coronary heart disease. *psychosocial risk factors, Medical Journal of Australia, 178*(6), 272-276.

Stewart, S. E. (2010). Prevalence and correlates of hypertension: a cross-sectional study among rural populations in sub-Saharan Africa. *J Hum Hypertens, 12*, 786-95.

Stuart, W., Grant, SC., & Samer, A. (2018). Statistical premer: developing and validating a risk prediction model. *European Journal of Cardio-Thoracic Surgery, 52*(2), 203-208.

Suresh, K.(2012) . Sample size estimation and power analysis for clinical research studies. *J Hum Reprod Sci*. *5*(1), 7-13.

Suzuki, J. S. (2015, December). Current therapies and investigational drugs for peripheral arterial disease. *Hypertens Res, 39*(4), 183-191.

Tandon, J. C. (2014). effect of age and gender on occupational stress:a study on teaching fraternity. *ijetmas, 2*(2), 2349-4476.

Tanya, M. (2010). Chronic Psychosocial Stress and Hypertension. *Curr Hypertens Rep, 12*, 10-16.

Taqueti, V. R. (2018). Coronary Microvascular Disease Pathogenic Mechanisms and Therapeutic Options: JACC State-of-the-Art Review. *Journal of the American College of Cardiology, 72*(21), 2625–2641.

Taravatmanesh, S. K. (2017). Determining the Factors Associated with Cardiovascular Disease Recurrence: Tehran Lipid and Glucose Study. *J Teh Univ Heart Ctr, 12*(3), 107-113.

Tatiana, C. F. (2015). Noise exposure and hypertension: investigation of a silent relationship. *BMC Public Health, 15*, 328.

Telmo, P. (2012). Dyslipidemia and Cardiovascular Risk: Lipid Ratios as Risk Factors for Cardiovascular Disease. *Dyslipidemia-From Prevention to Treatment, 14*, 279-302.

Thiriet, M. (2018). Cardiovascular Disease: An Introduction. *Vasculopathies, 8*(1), 1-90.

Thomas , J., Alsabeeh , N., Apovian, C., Megan, C., Coffman , G., Diana , C., . . . & Howard, C. (2008). Weight, Blood Pressure and Dietary Benefits After 12 Months of a Web-based Nutrition Education Program (DASH for Health): Longitudinal Observational Study. *J Med Internet Res*. *37*(1), 24-34.

Thomas, B., Aletta, E., Maciej, T., Ariti, C., Burrell, L., Castillo, R., & al., e. (2018). May Measurement Month 2017: an analysis of blood pressure screening results worldwide. *Lancet Global Health, 6,* e736–43.

Thomas, M. T. (2014). Cardiovascular effects of environmental noise exposure. *European Heart Journal, 35,* 829–836. doi:doi:10.1093/eurheartj/ehu030

Thomas, M., Frank, P., Sebastian, S., Johannes, H., Andreas, D., & MetteS. (2018). Environmental Noise and the Cardiovascular System,. *Journal of the American College of Cardiology, 71*(6), 688-697.

Thomas, W. (2016). Hypertension and Aging. *Ageing Res Rev, 26,* 96–111.

Thukkani , A., & Kinlay , S. (2015). Endovascular intervention for peripheral artery disease. *Circ Res., 116*(9), 1599-613.

Tiina , M. (2018). Cardiovascular diseases, cold exposure and exercise. *TEMPERATURE, 5*(2), 123–146.

Tora, G. W. (2016, jun). The role of diseases, risk factors and symptoms in the definition of multimorbidity – a systematic review. *Scandinavian Journal of Primary Health Care, 34*(2), 112-121.

Tsutsumi, A. (2015). Prevention and management of work-related cardiovascular disorders. *Int J Occup Med Environ Health, 28*(1), 4-7.

Tzoulaki, I. P. (2016). Worldwide Exposures to Cardiovascular Risk Factors and Associated Health Effects Current Knowledge and Data Gaps. *Circulation, 133*(23).

UK HDL-Consensus Group. (2004). Role of fibrates in reducing coronary risk:a UK consensus. *Curr Med Res Opin,, 20,* 241-247.

Van , T. (2016). Relationship at work as a cause of occupational stress: the case of academic women in Vietnam. *Int J Ment Health Syst, 10,* 42.

Vera, R.-Z. S.-P. (2015). Gender in cardiovascular diseases: impact on clinical manifestations, management, and outcomes. *Europian heart journal*. *37*(1), 24-34.

Victoria , M., Andrew , M., Mahshid , D., Sumathy , R., Xiaohe , Z., & Sumathi , S. (2017). Fruit, vegetable, and legume intake, and cardiovascular disease and deaths in 18 countries (PURE): a prospective cohort study. *Lancet, 390*, 2037–49.

Wallach, J. S. (2020). Evaluation of confounding in epidemiologic studies assessing alcohol consumption on the risk of ischemic heart disease. *BMC Med Res Methodol, 20*(64). doi:https://doi.org/10.1186/s12874-020-0914-6

Wamba, A. T. (2019). The impact of interventions for the primary prevention of hypertension in Sub-Saharan Africa: A systematic review and meta-analysis. *PLoS ONE, 14*(7), e0219623.

Wayne, W. L. (2016). *Confounding and Effect Measure Modification.* Retrieved from https://sphweb.bumc.bu.edu/otlt/MPH-Modules/BS/BS704-EP713_Confounding-EM/BS704-EP713_Confounding-EM_print.html

WHO. (2004). *Assessing the burden of disease from work-related hearing impairment at national and local levels, Occupational noise,Environmental Burden of Disease Series, No. 9,*. Geneva: WHO. Retrieved from https://www.who.int/quantifying_ehimpacts/publications/en/ebd9.pdf

WHO. (2008). *2008 STEPwise Approach to Chronic Disease Risk Factor Survey Report.* Geneva: World Health Organization. Retrieved from https://www.who.int/ncds/surveillance/steps/2007_Report_StKitts.pdf

WHO. (2012). *Introduction to health settings.* Geneva: World Health Organization.

WHO. (2015). *Global Health Observatory data repository, Prevalence - most recent adult survey Data by country, tobacco use, Rwanda Steps.* Geneva: WHO. Retrieved from http://apps.who.int/gho/data/node.main.TOB1249?lang=en

WHO. (2015). *Rwanda Non-communicable Diseases Risk Factors Report.* Geneva: World Health Organization.

WHO. (2016). *alcohol consumption: levels and patterns,2016, Recorded alcohol per capita (15+), consumption (in litres of pure alcohol) by type of alcoholic beverage, 2016 Recorded alcohol per capita(15+) consumption, 1961–2016.* Geneva: WHO.

WHO. (2017). *Fact sheets: Cardiovascular diseases,2017.* Geneva: Fact sheets: Cardiovascular diseases, 2017. Retrieved from https://www.who.int/news-room/fact-sheets/detail/ cardiovascular-diseases-(cvds).

WHO. (2018). *Rwanda Country statistics and global health estimates and UN partners, Global Health.* Geneva: WHO.

WHO. (2020). *Fruits and vegetables antioxidants content to prevent cardiovascular diseases.* Geneva: World Health Orgnaization.

WHO. (2021). *Types of cardiovascular disease*. Retrieved from www.who.int: https://www.who.int/cardiovascular_diseases/en/cvd_atlas_01_types.pdf

WHO. (2007). *Prevention of cardiovascular disease: Pocket guidelines for assessment and management of cardiovascular risk.* Geneva: WHO.

WHO/WEF. (2008). *Preventing Noncommunicable Diseases in the Workplace through Diet and Physical Activity,WHO/World Economic Forum Report of a Joint Event.* Geneva: World Health organization.

William, R., Jerry , G., Sidney , C., Jr, Mary , M., Gregory , M., . . . & William, H. (2008). Atherosclerotic Peripheral Vascular Disease Symposium II Nomenclature for Vascular Diseases. *Circulation, 118*, 2826-2829.

Wilson, P. F. (2005). Metabolic syndrome as a precursor of cardiovascular disease and type 2 diabetes mellitus. *Circulation, 112*(20), 3066–307.

Winkelmayer, W. C. (2005). Habitual Caffeine Intake and the Risk of Hypertension in Women. *JAMA, 294*(18), 2330-5.

Wisse, B. S. (2016). When Change Causes Stress: Effects of Self-construal and Change Consequences. *J Bus Psychol, 31*, 249–264.

Wolfgang , B., & Heike , K. (2007). *Hearts and Minds at Work in Europe A European work-related public health report on Cardiovascular Diseases and Mental Ill Health*, Europe: Essen.

Wong, K., Chan, A. H., & Ngan, S. C. (2019). The effect of long working hours and overtime on occupational health: a meta-analysis of evidence from 1998 to 2018. *International journal of environmental research and public health, 16*(12), 2102.

World Health Organization. (2013.). *Country cooperation strategy brief: India.May*. Geneva: World Health Organization.

World Health Orgnaization. (2007). *Prevention of cardiovascular disease: Pocket guidelines for assessment and management of cardiovascular risk.* World Health Orgnaization. Retrieved from https://www.who.int/cardiovascular diseases/guidelines/PocketGL.ENGLISH.AFR-D-E.rev1.pdf

Wu, X. D. (2017). Cardiovascular Risk Factors in Noise-Exposed Workers in China: Small Area Study. *Noise Health*, *19*, 245–253.

Xia, E., Yu, Y., Xu, E., Mei, J., & Sun, W. (2019). From Risk Prediction Models to Risk Assessment Service: A Formulation of Development Paradigm. *arXiv preprint arXiv:1903.07551*.

Yang, L., Wu, H., Jin, X., & al, e. (2020). Study of cardiovascular diseases prediction model based on random forest in eastern China. *Sci Rep., 10*, 5245.

Yangfeng, W., Xiaoqing, L., Xian, L., Ying, L., Liancheng, Z., Zuo, C., & al, e. (2006). Estimation of 10-Year Risk of Fatal and Nonfatal Ischemic Cardiovascular Diseases in Chinese Adults. *Circulation., 14*, 2217-25.

Yeates, k. L. (2015). A global perspective on cardiovascular disease in vulnerable Populations. *Can J Cardiol., 31*(9), 1081–93.

Yi-Wei, W., Si-Jia, H., Xiao, F., Jin, C., Yun-Tao, L., Ling, T., & Qian, H. (2017). Metformin: a review of its potential indications. *Drug Des Devel Ther, 11*, 2421–2429.

Yuling, H. (2009). The Burden of Cardiovascular Disease in Asia: Big challenges and ample opportunities for Action and Making a Difference. *Clinical Chemistry.*, 1450-1452.

Zabina, H. S. (2001). Monitoring behavioral risk factors for cardiovascular disease in Russia. *Am J Public Health, 91*(10), 1613-4.

Zahran, S. M. (2017). Association of beverage consumption with obesity in healthy adults. *Arab Journal of Nutrition and Exercise (AJNE)*, 37.

Zamanian, Z. R. (2013). Investigation of the effect of occupational noise exposure on blood pressure and heart rate of steel industry workers. *Journal of environmental and public health, 2013*.

Zeliger, H. I. (2013). Lipophilic chemical exposure as a cause of cardiovascular disease. *Interdiscip Toxicol, 6*, 55–62.

Zhang, N.(2020). Association between metal cobalt exposure and the risk of congenital heart defect occurrence in offspring: a multi-hospital case-control study. *Environ Health Prev Med. 25*, 38.

Zhang, W. R. (2019). Mortality from heart diseases following occupational radiation exposure: analysis of the National Registry for Radiation Workers (NRRW) in the United Kingdom. *J. Radiol. Prot, 39*, 327–353.

Zhang, Y. H. (2012). Glycosylated Hemoglobin in Relationship to Cardiovascular Outcomes and Death in Patients with Type 2 Diabetes: A Systematic Review and Meta-Analysis. *PLoS ONE, 7*(8), e42551.

Zhang, Z. (2018). Time-varying covariates and coefficients in Cox regression models. *Annals of translational medicine, 6*(7), 121.

Zhao, P. L. (2018). Age- and sex-related difference of lipid profile in patients with ischemic stroke in China. *Medicine, 97*(23), 10930.

Zhifei, H. B. (2019). Prevalence of Alcohol and Tobacco Use among Men and Women in Namibia. *Int. J. Environ. Res. Public Health, 16*, 59.

Zhou, Y., Wu, H., Zhang, Y., Sun, H., Wong, T., & Li, G. (2011). Ionic mechanisms underlying cardiac toxicity of the organochloride solvent dichloromethane. *Toxicology*. *290*(2-3), 295-304.

Zhuang, Q. C. (2019). Association of high sensitive C-reactive protein with coronary heart disease: a Mendelian randomization study. *BMC Medical Genetics, 20*(170), 1186.

Zujie, G. Z. (2019). Gender differences in cardiovascular disease. *Medicine in Novel Technology and Devices*. *4*, 100025.

APPENDICES

Appendix I: Participant consent form

Protocol Nr……..

Protocol Title: Risk prediction and factors associated with cardiovascular diseases among workers and their spouses in two drink processing industries in Rwanda.

Dear Participant

You have been invited to take part in a research thesis with NSANZABERA Charles Ph.D. student at Jomo Kenyatta University of Agricuture and Technology (JKUAT) titled: **Risk prediction and factors associated with cardiovascular diseases among workers and their spouses in two drink processing industries in Rwanda.**

Before joining the project in questions, you need to read this information form, since it contains important information to assist you in deciding whether or not signing up to participate, is in your best interests. We request that you ask as many questions as you wish in order to make sure that you understand the procedure of the study, the risks and benefits. If you have a question about this documents that has not been sufficiently answered or explained, don't hesitate to ask one of the research team members for more information.

The study has been approved by the National Research Committee (NHRC) in the Ministry of Health and Institutional Review Board of College of medicine and Health Science of University of Rwanda since it complies with medical ethics standards. Additionally, the study will be conducted according the Helsinki Declaration and Guide on Best Clinical Practices.

You may choose not to participate in the study or to leave it at any time simply by informing the investigator. If you decide not to participate in the study or to retract your consent, you will not lose any advantage that you would be due.

If client refused to participate, please check this box 

I have been told about the study and I have understood its main aim that it is Voluntary and Confidential, and the results will be used to improve community health.

Therefore, I willingly accept to participate in this study.

Respondent’s Signature/Thumb print…….Date……

Appendix II: Questionnaires

Participant's questionnaires

Instructions:

i. These questionnaires will be filled by the interviewer
ii. Read thoroughly the questions below for the participants
iii. Fill in the number below regarding the asked information

Q/N	Location and Date	Responses	Code
1	Location ID	☐☐☐	L1
2	Interviewer ID	☐☐☐	L2
3	Date of completion of the instruments	☐☐☐☐☐☐☐☐ dd mm year	L3
4	Consent has been read and obtained	Yes 01 No 02 if no END	L4
5	Interview language	English 01 Kinyarwanda 02 03 Other :……	L5
7	Time of the interview	☐☐☐☐ Hour Min	L6

SOCIO-DEMOGRAPHIC CHARACTERISTIC OF RESPONDENTS			
Q/N	**Question**	**Responses**	**Code**
8	Sex	male 01 Female 02	C1
9	Date of birth	DOB: ……/……../……	C2
10	How old are you?	Age:……	C3
11	Place of birth	Western province 01 Northern Province 02 03	C4

		Southern Province Eastern Province Kigali town 05 Foreign country: 06	
12	Marital status	Single 01 Married 02 Divorced 03 Living together 04 Widow 05	C5
13	Religion	Roman Catholic 01 Protestant 02 Muslim 03 Others 04	C6
14	Occupation and working department for workers	Commerce and marketing 01 Technical 02 Human Resources 03 Logistic 04 Finance 05 Indicate the Position:…… 06 Go to C	C7
15	Occupation spouses	Indiacte what is currently doing …….. Go to C9	C8
16	Tenure	0-4years 01 5-9years 02 03	C9

		10-14years 15-19years 04 20-24years 05 25-29years 06 30 and more 07	
17	Level of education	Primary 01 A3 02 A2 03 A1 04 A0 05 Masters 06 Ph.D. 07 **If is a spouse go to T1**	C9

Working conditions for employees			
N/Q	**QUESTIONS**	**ANSWERS AND CODES**	**Code**
18	Are you in your current position doing shift works	Yes 01 No 02	W1
19	Are you regularly doing a night shift work	Yes 01 No 02	W2
20	Are you regularly working in	Yes 01	W3

	cold chambers, refrigeration?	No 02	
21	How many days a week are you affected in cold chambers?	A day a week 01 2days a week 02 3days a week 03 4days a week 04 5 days or more a week 05	W4
22	Are you regularly experiencing much vibration in your area of work?	Yes 01 No 02	W5
23	Are you regularly experiencing much sounds in your area of work?	Yes 01 No 02	W6
24	Are you wearing PPE when working in area with much noises?	Yes 01 No 02	W7
25	How often are you wearing PPE (Ear Plugs)?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	W8

26	Are you handling or breathing chemicals, gas, fumes and dust or reagents in your area of work?	Yes 01 No 02 If no go to W11	W9
27	Which ones are you handling in this list	CO 01 CO2 02 Nitrites 03 Cobalt 04 Other 05 If other go toW10a	W10
28	If other chemical, Gas, fumes, dust	Specify……. …… ……..	W10a
29	Are you wearing PPE at work when handling Chemicals, Gas, Dust and Fumes?	Yes 01 No 02 If no got to S1	W11
30	How often are you wearing PPE?	A 01	W12

Occupational stress

Now I am going to ask you about stress related to the working conditions, It is recognized that working conditions affect worker well-being, it is important that your responses reflect your work in the last six months			
31	Are you clear of what is expected of you at work?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S1
32	Are you clear of what are your duties and responsibilities?	Never 01 Seldom 02 Sometimes 03 Often 04 05 Always	S2
33	Do you know how to go about getting your job done?	Never 01 Seldom 02	S3

		Sometimes 03 Often 04 Always 05	
34	Do you have unachievable deadlines?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S4
35	Do you have to work very intensively?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S5
36	Do you have to neglect some tasks because you have too much to do?	Never 01	S6

		Seldom 02 Sometimes 03 Often 04 Always 05	
37	Are you pressured to work long hours?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S7
38	Are you unable to take sufficient breaks?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S8

		Always	
39	Are you subject to bullying at work?	Never 01 Seldom 02 Sometimes 03 Often 04 Always 05	S9
40	Do you have sufficient opportunities to question managers about change at work?	Strongly disagree 01 Disagree 02 Neutral 03 Agree 04 Strongly agree 05	S10
41	Do you receive the respect at work you deserve from your colleagues?	Strongly disagree 01 Disagree 02 Neutral 03	S11

		Agree 04 Strongly agree 05	
42	Can you talk to your line manager about something that has upset or annoyed you about work?	Strongly disagree 01 Disagree 02 Neutral 03 Agree 04 Strongly agree 05	S12
43	Is your line manager encourages you at work?	Strongly disagree 01 Disagree 02 Neutral 03 Agree 04 Strongly agree 05	S13

Behavioral and lifestyle history of the participants			
Tobacco Use			
Now I am going to ask you some questions about various health behaviors. Things like smoking, drinking alcohol, eating fruits and vegetables and physical activity. Let start with Tobacco			
44	Do you currently smoke any **tobacco products**, such as cigarettes, cigars or pipes? *(use showcard)*	Yes 01 No 02 if no Go to T6	**T1**
45	Do you currently smoke tobacco products daily?	Yes 01 No 02	**T2**
46	How old were you when you first started smoking daily?	Age in years ☐☐	**T3**
49	On average, how many of the following do you smoke each day? *(record for each type, use showcard*	Manufactured cigarettes ☐☐	**T4a**
		Hand-rolled cigarettes ☐☐	**T4b**
		Pipes full of tobacco ☐☐	**T4c**
		Cigars, cheroots, cigarillos ☐☐	**T4d**
		Others ☐☐	**T4e**

		If other go to T4others	
50		If other specify…….. ……	**T4other**
51	In the past, did you ever smoke daily?	Yes 01 No 02	**T5**
52	How old were you when you stopped smoking daily?	Yes 01 No 02	**T6**
53	How long ago did you stop smoking daily? *(record only 1, not all 3)*	Years ago ☐☐ If Known, go to T8	**T7a**
		OR Months ago ☐☐ If Known, go to T8	**T7b**
		OR Weeks ago ☐☐	**T7c**
54	Do you currently use any smokeless tobacco such as [snuff, chewing tobacco, betel]? *(use showcard)*	Yes 01 No 02	**T8**
55	Do you currently use smokeless tobacco products daily?	Yes 01	**T9**

		No 0	
56	On average, how many times a day do you use …. *(record for each type, use showcard)*	Snuff, by mouth	**T10a**
		Snuff, by nose	**T10b**
		Chewing tobacco	**T10c**
		Betel, quid	**T10d**
		Other	**T10e**
		Other (specify)	**T10 other**
57	In the past, did you ever use smokeless tobacco such as [snuff, chewing tobacco, or betel] daily?	Yes 01 No 02	**T11**
58	During the past 7 days, on how many days did someone in your home smoke when you were present?	Number of days a week ☐☐	**T12**
59	During the past 7 days, on how many days did someone smoke in closed areas in your workplace (in the building, in a work area or a specific office) when you were present?	Number of days a week ☐☐	**T13**
Alcohol Consumption			
Next questions ask about the consumption of alcohol			
60	Have you ever consumed an alcoholic drink such as beer, wine, spirits, fermented cider or (add other local examples)?	Yes 01 No 02	**A1a**
61	Have you consumed an alcoholic drink within the past 12 months?	Yes 01 No 02	**A1b**
62	During the past 12 months, how frequently have you had at least one alcoholic drink?	Daily 01 02 5-6 days per week	**A2**

	(read responses, use showcard)	03 1-4 days per week 04 1-3 days per month 05 Less than once a month	
63	Have you consumed an alcoholic drink within the past 30 days?	Yes 01 02 No	**A3**
64	During the past 30 days, on how many occasions did you have at least one alcoholic drink?	Number of days ☐☐	**A4**
65	During the past 30 days, when you drank alcohol, on average, how many standard alcoholic drinks did you have during one drinking occasion?	Number of days ☐☐	**A5**
66	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 ☐☐	**A6**
67	During the past 30 days, how many times did you have **For Men**: Five or more **For Women**: Four or more Standard alcoholic drinks in a single drinking occasion?	Number of times ☐☐	**A7**
68	Are you taking alcoholic drinks while at work?	Never 01	**A8**

		Seldom 02 Sometimes 03 Often 04 Always 05	

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.

Q/N	Questions	Responses	Code
69	In a typical week, on how many days do you eat fruit? *(use showcard)*	Number of days If zero days go toD3	**D1**
70	How many servings of fruit do you eat on one of those days? (USE SHOWCARD)	Number of servings	**D2**
71	In a typical week, on how many days do you eat vegetables? (USE SHOWCARD)	Number of days If zero days, go to D5	**D3**
72	How many servings of vegetables do you eat on one of those days? (USE SHOWCARD)	Number of servings	**D4**

73	What type of oil or fat is most often used for meal preparation in your household?	Vegetable oil 01 Lard or suet 02 Butter or ghee 03 Margarine 04 None in particular 05 None used 06 other If other go to D5 others	**D5**
		Other Specify…….. ……	**D5 Other**

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			
74	Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like [carrying or lifting heavy loads, digging or construction work] for at least 10 minutes continuously? [INSERT EXAMPLES] (USE SHOWCARD)	**Yes** 01 **No** 02 **If No, go to P4**	**P1**
75	In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days ☐☐	**P2**
76	How much time do you spend doing vigorous-intensity activities at work on a typical day?	Hours: minutes ☐☐:☐☐	**P3** **(a-b)**
77	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 01 No 02 if no go to P7	**P4**
78	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days ☐☐	**P5**
79	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours: Minutes ☐☐ ☐☐ :	**P6** **(a-b)**

Travel to and from places

The next questions exclude the physical activities at work that you have already mentioned.

Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship. (Insert other examples if needed)

80	Do you walk or use a bicycle (pedal cycle) for at least 10 minutes continuously to get to and from places?	Yes 01 No 02 if no go to P10	**P7**
81	In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?	Numbers of days ☐☐	**P8**
82	How much time do you spend walking or bicycling for travel on a typical day?	☐☐ ☐☐ Hours: minutes :	**P9** **(a-b)**

Recreational activities

The next questions exclude the work and transport activities that you have already mentioned.

Now I would like to ask you about sports, fitness and recreational activities (leisure), [Insert relevant terms].

83	Do you do any vigorous-intensity sports, fitness or recreational (leisure) activities that cause large increases in breathing or heart rate like [running or football] for at least 10 minutes continuously?	Yes 01 No 02 if no go to P13	**P10**
84	In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (leisure) activities?	Number of days ☐☐	**P11**

85	How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours : minutes ☐☐ : ☐☐	**P12** **(a-b)**
86	Do you do any moderate-intensity sports, fitness or recreational (leisure) activities that cause a small increase in breathing or heart rate such as brisk walking, (cycling, swimming, and volley-ball) for at least 10 minutes continuously?	Yes 01 No 02 if no go to P16	**P13**
87	In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (leisure) activities?	Number of days ☐☐	**P14**
88	How much time do you spend doing moderate-intensity sports, fitness or recreational (leisure) activities on a typical day?	Hours : minutes ☐☐ : ☐☐	**P15** **(a-b)**
Sedentary behavior			
The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent sitting at a desk, sitting with friends, traveling in car, bus, train, reading, playing cards or watching television, but do not include time spent sleeping. *[insert examples] (use showcard)*			
89	How much time do you usually spend sitting or reclining on typical day?	**Hours : minutes**	**P16** **(a-b)**
History of Cardiovascular diseases			
90	Have you ever had your blood pressure measured by a doctor or other health worker?	Yes 01 No 02 If no go to H	**H1**

91	Have you ever been told by a doctor or other health worker that you have raised blood pressure or hypertension?	Yes 01 02 No	**H2a**
92	Are you currently taking any drug or medication for raised blood pressure?	Yes 01 No 02	**H2b**
93	Indicate the medications you are taking	Specify……. …….. ……..	**H2c**
94	Have you ever been told by a doctor or other health worker that you have diabetes?	Yes 01 No 02 if no go to H4a	**H3a**
95	Are you currently taking any drug or medication for your Diabetes condition?	Yes 01 No 02	**H3b**
96	Indicate the medication you are taking	Specify……. …….. ……..	**H3c**
97	Have you ever been told by a doctor or other health worker that you have dyslipidemia?	Yes 01 No 02 If no go toH5	**H4a**
98	Are you currently taking any drug or medication for your dyslipidemia condition?	Yes 01 No 02	**H4b**

99	Indicate the medication you are taking	Specify…… …….. ……..	**H4c**
100	Is any person in your family suffer CVDs?	Yes 01 No 02 if no go to H7	**H5**
101	Which CVDs did or does he suffer?	Type:…….	**H6**
102	Which relationship do you have	Father 01 Mother 02 Brother 03 Sister 04 Uncle 05 Aunt 06 Grandfather 07 Grand mother 08	**H6a**

103	Is any person died in Your family due to CVDs?	Yes 01 No 02 if no go to M1	**H7**
104	Indicate relationship with this person	Relationship:	**H7a**

ANTROPOMETRIC AND BIOCHEISTRY REPORT FORM				
Q/N	**MEASUREMENT**	**Type of measurement**	**Responses**	**Code**
Anthropometric measurement				
105	Interviewer ID			M1
106	Device ID			M2a
107	Device ID			M2b
108	Height	In centimeters (Cm)		M3
109	Weight If too large for scale 777	In Kilograms(Kg)		M4
110	For **women**: are you pregnant?	Yes 01 No 02	If yes Go to	M5
111	Waist Circumference	In centimeters(Cm)		M6
112	Hip Circumference	Cm		M6a

Blood pressure measurement				
113	Cuff size use	Small	01	M7
		Medium	02	
		Large	03	
114	Reading 1	Systolic (mmHg)		M8a
		Diastolic(mmHg)		M8b
115	Reading 2	Systolic(mmHg)		M9a
		Diastolic(mmHg)		M9b
116	Reading 3	Systolic(mmHg)		M10a
		Diastolic(mmHg)		M10b
117	During the past two weeks, have you been treated for raised blood pressure with drugs (Medication) prescribed by a doctor or other health worker?	Yes	01	M11
		No	02	
Biochemical measurements				
118	During the past 12 hours have you had anything to eat or drink, other than water?	Yes	01	B1
		No	02	
119	Technician ID			B2
120	Device ID			B3
121	Time of day blood	Hours: Minutes	:	B4

	specimen taken(24 hour clock)			
122	Fasting blood glucose *Choose accordingly mg/dl or mmol/l*	mg/dl	.	B5
		mmol/l	.	
123	Total Cholesterol *Choose accordingly mg/dl or mmol/l*	Mg/dl	.	B6
		mmol/l	.	
124	HDL Cholesterol *Choose accordingly mg/dl or mmol/l*	Mg/dl	.	B7
		mmol/l	.	
125	Triglyceride *Choose accordingly mg/dl or mmol/l*	Mg/dl		B8
		Mmol/l		
126	LDL Cholesterol *Choose accordingly mg/dl or mmol/l*	Mg/dl		B9
		Mmol/dl		
127	CRP(C Reactive Protein *Choose accordingly mg*	mg		B10
128	Glycated Hemoglobin(HB1AC) Percentage	%		B11
129	Uric Acid	Mg/dl		B12
130	During the past two weeks, have you been treated for raised	Yes	01	B13

02

	cholesterol with drugs (medication) prescribed by a doctor or other health worker?	No		

Amasezeran yo kubazwa ku bushakashatsi ku bushake

Numero y'ubushakashatsi……

Izina ry'ubushakashatsi: iteganya ry'ibyago byo gufatwa n'indwara z'umutima n'imitsi ndetse n'ibindi byatuma zifata bamwe mubakozi n'abafasha babo munganda ebyiri zenga ibyo kunywa mu Rwanda.

Bwana/madamu mukozi/ mufasha w'umukozi,

Mwatumiwe kugira uruhare mu bushakashatsi bwitwa: **iteganya ry'ibyago byo gufatwa n'indwara z'umutima n'imitsi ndetse n'ibindi byatuma zifata bamwe mubakozi n'abafasha babo munganda ebyiri zenga ibyo kunywa mu Rwanda** burimo gukorwa na NSANZABERA Charles, umunyeshuri muri Kaminuza ya Jomo Kenyatta.

Mbere yo kugira uruhare muri ubu bushakashatsi, ukeneye gusoma, Kumva no gusobanukirwa amakuru yagufasha gufata umwanzuro wo kugira uruhare mu bushakashatsi ku bushake kandi inyungu zawe zitabangamiwe. Turagusaba kubaza ibibazo byinshi uko ubishoboye, kugirango usobanukirwe uko ubushakashatsi buzagenda, ingaruka ndetse n'inyungu zizabuvamo. Niba ufite ibibazo cyangwa icyo utasobanikiwe kuri ubu bushakashatsi, ntugire impungenge zo kubaza abagize itsinda ry'ubushakashatsi kugirango usobanukirwe.

Ubushakashatsi bwemewe na komite ngengamahame ya ministere y'ubuzima mu Rwanda kuko yujuje amahame mpuzamahanga mu by'ubushakashatsi. Na none

kandi ubu bushakashatsi buzubahiriza amahame n'amabwiriza y'imigendekere myiza y'ubushakashatsi nkuko biteganywa na Helsinki.

Ushobora guhitamo kujya mu bushakashatsi cyangwa kutabujyamo ubimenyesheje umushakashatsi. Niba ufashe icyemezo cyo kutitabira ubushakashatsi cyangwa cyo guhagarika amasezerano yo kubazwa ku bushakashatsi ku bushake ntuzatakaza uburenganzira kubyo wemerewe.

Niba Umukozi Cyangwa umufasha w'umukozi yanze kubazwa, bigaragaze ☐

Nasobanuriwe ibijyanye n'ubushakashatsi. Numvise neza impanvu nyamukuru y'ubu bushakashatsi kandi ko kubazwa nta gahato kandi ko ibisubizo bizagirwa ibanga ibizava mu bushakashatsi bizazamura ubuzima bw'abaturage. Kubera iyo mpanvu, nemeye kubazwa nta gahato

Umukono cyangwa igikumwe cy'ubazwa……..…Itariki…….....

IBIBAZO

Ibibazo bignewe ubazwa

Amabwiriza y'ibaza:

i. Uru rutonde rw'ibibazo byuzuzwa nubaza

ii. Somera neza ibibazo byose ubazwa

iv. Uzuza ukurikije uruhererekane rw'ibibazo

Q/N	**Igihe abakora ubushakashatsi naho buri kubera**	**IBIZUBIZO**	**Kode**
1	Numero iranga aho ubushakashatsi buri kubera	☐☐☐	L1

2	Numero y'ubaza	☐☐☐	L2
3	Itariki wasorejeho uru rurutonde rw'ibibazo	☐☐☐☐☐☐☐☐ itariki ukwezi Umwaka	L3
4	Amasezerano yo kwemera kubazwa yabonetse aranasomwa	Yego 01 Oya 02 niba ari Oya Soza ubushakashatsi	L4
5	Ururimi ibazwa ryakorewemo	English 01 Kinyarwanda 02 Izindi :……. 03	L5
7	Igihe ibazwa ryatangiriye	☐☐☐☐ Hour Min	L6

Ibirango rusange by'Imiterere n'imiturire y'ababazwa			
Q/N	**Ibibazo**	**Ibisubizo**	**Kode**
8	Igitsina	Umuga 01 Umugore 02	C1

9	Igihe yavukuye	Igihe yavukiye: ……/……../……	C2
10	Ufite imyaka ingahe?	Imyaka:……	C3
11	Aho yavukiye	Intara y'uburengera zuba 01 Intara Y'amajyaruguru 02 Intara y'Amajyepfo 03 Intara y'iburasirazuba 04 Umujyi Wa Kigali 05 Mumahanga: 06 Niba ari 06 vuga igihugu:……	C4
12	Iranga mimerere	Ingaragu 01 Afite Umufasha/Gushyingirw 02 Baratandukanye 03 Baribanira gusa 04 Umupfakazi 05	C5
13	Idini	Roman Catholic/Gatolika 01 Protestantanti 02 Umusilamu 03 Abandi 04 Niba ari 04 havuge……. Niba uri umufasha w'umukozi jya kuri C8	C6
14	Umurimo ukora	Ubucuruzi No kwamamaza 01	C7

	ndetse nigice ukoreramo	Tekiniki 02 Urwego Rwabakozi 03 Abahahira Uruganda 04 Abashinzwe ibigega n'imari 05 Ukora akahe kazi:…… Jya kuri C9	
15	Akazi k'umufasha(umugore cg umugabo) w'umukozi	ukora iki ubu …….. Jya kuri C9	C8
16	imyaka umaze mukazi	0-4years(Imyaka) 01 5-9years(Imyaka) 02 10-14years(Imyaka) 03 15-19years(Imyaka) 04 20-24years(Imyaka) 05 25-29years(Imyaka) 06 Imyaka 30 nirenga 07	C9
17	Amashuri nimpamya bumenyi ufite	Primary(Amashuri abanza) 01 A3 (Amashuri atatu y'isumbuye) A2 (Amashuri 6 yisumbuye) 03 A1(Icyiciro cya mbere cya kaminuza) 04	C9

05

02

		A0(Icyiciro cya 2 cya Kaminuza) Masters(Icyiciro cya 3 cya Kaminuza) 06 Ph.D.(Icyiciro cya Kane cya Kaminuza 07 **Niba uri umufasha w'umukozi jya kuri T1**	

Imiterere y'akazi kuba kozi b'uruganda			
N/ Q	**Ibibazo**	**Ibizubizo na kode**	**kode**
18	Waba ukora izamu (nijoro) ndetse no kumanwa mukazi ukora ubu?	Yego 01 Oya 02	W1
19	Waba ukora izamu (Nijoro) buri gihe?	Yego 01 Oya 02	W2
20	Waba ukorera mu cyumba gikonjesha burigihe?	Yego 01 Oya 02	W3
21	Niminsi ingahe mucyuimweru ukorera mucyumba gikonje cg gikonjesha?	Umunsi 1 mucyumweru 01 Iminsi 2 mucyumweru 02	W4

		Iminsi itatu Mucyumweru 03 Iminsi 4 mucyumweru 04 05 Iminsi 5 cg irenga mucyumweru	
22	Waba ukoresha ibyma bitigisa umubiri aho ukorera buri gihe?	Yego 01 Oya 02	W5
23	Waba ukorera ahantu hari urusaku rwinshi burigishe?	Yego 01 Oya 02	W6
24	Ese waba wambara uturinda rusaku iyo uri gukorera ahari urusaku?	Yego 01 Oya 02	W7
25	Wambara uturinda rusaku kangahe?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 Kenshi 04 05	W8

		Burigihe	
26	Waba ukora cg uhumeka Ibinyabutabire (Uburozi),Umwuka, imyotsi, umukungugu cg ibindi byangiza aho ukorera cg byahungabanya ubuzima bwawe?	Yego 01 Oya 02 Niba ari oya jya kuri W11	W9
27	Ese muribi nibihe uhura nabyo?	CO 01 CO2 02 Nitrites 03 Cobalt 04 Other 05 Niba hari ibindi jya W10a	W10
28	Niba hari ibindi Ibinyabutabire,umwuka wangiza,imyotsi,umukungugu, bivuge	Bivuge ……. …… …….	W10a
29	Ese wambara	Yego 01	W11

	agahumekerwamo mugihe uri gukorera ahari ibinyabutabire,imyotsi,Umwuka wangiza, n'umukungugu?	Oya 02 Niba ari oya jya S1	
30	Nikangahe wambara agahumekerwamo?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 Kenshi 04 Burigihe 05	W12
Ibibazo byo kunanirwa no guta umutwe bituruka kukazi(Stressi)			
Ubu ngiye kukubaza ibirebana nibibazo ndetse no guta umutwe bituruka kumiterere yakazi kawe, birazwiko imiterere y'akazi yagira icyo yangiza kubuzima bw'umukozi, ningenzi ko usubiza uko byakugendekeye kuva mumezi atandatu ashize.			
31	Ese waba wumva cg ujya wumva neza ibigukeneweho kukazi?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03	S1

		Kenshi 04 Burigihe 05	
32	Ese waba usobanukiwe neza nibyo usabwa ndetse ninshingano zawe?	Ntanarimwe 01 Gake cyane 02 Rimwe Narimwe 03 Kenshi 04 Burigihe 05	S2
33	Ese wabuzi ibigomba gukorwa kugirango akazi kawe gatungane neza?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 Kenshi 04 05	S3

		Burigihe	
34	Ese waba utarangiriza akazi kugihe gitegetswe?	Ntanarimwe 01 Gakee cyane 02 Rimwe narimwe 03 Kenshi 04 Burigihe 05	S4
35	Ese ugomba gukora cyane kuburyo utindamukazi kugirango urangize inshingano zawe??	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 Kenshi 04 Burigihe 05	S5
36	Ese hari imirimo imwe nimwe wirengagiza kuberako ufite	Nta narimwe 01	S6

	byinshi byo gukora?	Gake cyane 02 Rimwe narimwe 03 Kenshi 04 Burigihe 05	
37	Ese waba uhatirwa nakazi gukora amasaha menshi?	Ntanarimwe 01 Gake cyane 02 Rimwe Narimwe 03 Kenshi 04 Burigihe	S7
38	Ese waba udashobora kubona ikiruhuko gihagije?	Ntanarimwe 01 Gake Cyane 02	S8

		Rimwe narimwe 03 Kenshi 04 Burigihe 05	
39	Ese waba uhita bakagukoba ndetse bakakumwaza basakuza cyane kukazi?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 kenshi 04 Burigihe 05	S9
40	Ese ujya ugira igihe gihagije cyo kubaza umukoresha wawe impamvu ibintu byahindutse kukazi?	simbyemeye nagato 01 Simbyemera 02 Ndifashe 03 Ndabyemera 04 Ndabyemera Cyane 05	S10
41	Ese waba ubona icyubahiro	Simbyemeye Nagato 01	S11

	ukwiriye mubo mukorana byahafi?	Simbyemera 02 Ndifashe 03 ndabyemera 04 Ndabyemera Cyane 05	
42	Ushobora kubwira ugukuriye byahafi mukazi ikitagushimishije cg kigutesha umutwe mukazi?	Simbyemeye Nagato 01 Simbyemeye 02 Ndifashe 03 Ndabyemeye 04 Ndabyemeye Cyane 05	S12
43	Ese umuyobozi wawe byahafi agutera ingabo mubitugu mukazi kawe?	Simbyemeye nagato 01 Simbyemeye 02	S13

		Ndifashe 03 Ndabyemeye 04 Ndabyemeye Cyane 05	
Imibereho nimyitwarire by'ubazwa			
Gukoresha cg kunywa Itabi			
Ubu noneho Ngiye kukubaza ibibazo bitandukanye birebana nimyitwarire mubuzima. Mbese nko kunywa itabi, Kunywa inzoga, Kurya imbuto n'Imboga no gukora (Siporo)imyitozo ngorora mubiri. Reka dutangirire Kw'itabi.			
44	*Ese ubu unywa itabi iryariryo ryose nk' isigara,Ikigoma,Inkono yitabi?(mwereke aho bishushanyije)*	Yego 01 Oya 02 if no Go to T5	**T1**
45	Ese unywa itabi ry'umwotsi buri munsi?	Yego 01 Oya 02	**T2**
46	Warufite imyaka ingahe igihe watangiraga kunywa itabi?	Imyaka	**T3**
49	Muri rusange unywa itabi ringana iki	Manufactured cigarettes/Itabiri ryomuruganda	**T4a**

	(erekana ikarita ishushanyijeho ubwoko bwitabi)	Hand-rolled cigarettes/Itabi bazinga mugipapuro cg mw'ikoma	**T4b**
		Pipes full of tobacco/Inkono y'itabi	**T4c**
		Cigars, cheroots, cigarillos/Ikigoma cg ibitabi binini cyane	**T4d**
		Ubundi bwoko bw'itabi Niba harubundi bwoko bwitabi jya T4others	**T4e**
50		Vuga ubundi bwoko …….. ……	**T4other**
51	Ese cyera wigeze unywa itabi?	Yego 01 Oya 02 Niba ari oya jya	**T5**
52	Warufite imyaka ingahe igihe warekaga itabi?	Imyaka	**T6**

53	Hashize imyaka ingahe uretse kunywa itabi? *Andika aho yibuka gusa singombwa hatatu*	Imyaka ishize Niba izwi, jya kuri T8	**T7a**
		Cg amezi ashize Niba igihe kizwi, jya kuri T8	**T7b**
		cg ibyumweru bishize	**T7c**
54	*Ese waba unywa irindi tabi ritagira imyotsi nka(Itabi bashyira mumazuru,Ubugoro,cg betel cg irindi tabi bahekenya) erekana ikarita rishushanyijeho*	Yego 01 Oya 02	**T8**
55	Ese waba unywa itabi ritagira umwotsi burigihe?	Yego 01 Oya 0	**T9**
56	Ducishirije waba urifata inshuro zingahe kumunsi? *(andika inshuro imbere yitabi ukurikije iriri ku ishusho)*	Snuff, by mouth/Itabi ryo mukanwa cg ubugoro nibindi bias nkabwo	**T10a**
		Snuff, by nose/Itabi ryo mumazuru cg ubugoro bwo muazuru nibindi bias nkabwo	**T10b**
		Chewing tobacco/Itabi bahekenya	**T10c**
		Betel, quid	**T10d**

		Ubundi bwoko bwitabi	**T10e**
		Irindi (rivuge)	**T10 other**
57	Ese waba warigeze ukoresha itabi ritagira umwotsi nka(ivu ryo mumazuru, ubugoro cg betel ? mwereke ikarita)	Yego 01 Oya 02	**T11**
58	Muminsi irindwi ishize haba hari umuntu wanywereye itabi murugo muhari?	Iminsi yarinyweye mucyumweru	**T12**
59	Muminsi 7 ishize nikangahe umuntu yanywereye itabi ahegereye aho mukorera (Munzu imbere cg mubiro nahandi mukorera?)	Iminsi yarinyweye mucyumweru	**T13**
Kunywa inzoga/Ibyo kunywa bisembuye			
Ibibazo bikurikiyeho bibaza ibirebana no kunywa inzoga			
60	Wigeze unywa ikinyobwa gisembuye nka byeri, divayi, Kanyanga cg urwagwa nizindi zisembye?	Yego 01 Oya 02	**A1a**
61	Waba waranyoye kubinyobwa bisembuye mumezi cumi nabiri ashize?	Yego 01 Oya 02	**A1b**
62	Mumezi cumi nabiri ashize ninshuro zingahe waba waranyoye byibuze kuri kimwe muribi binyobwa?	Buri munsi 01 iminsi5-6 mucyumweru 02	**A2**

	mwereke ikarita	1-4 mucyumweru 03 1-3 Mucyumweru 04 Munsi ya Rimwe mukwezi 05	
63	Waba waranyoye ibinyobwa bisembuye muminsi 30 ishize?	Yego 01 Oya 02	**A3**
64	Muminsi 30 ishize ni kangahe waba waranyoye byibura icupa rimwe?	umubare wiminsi | | |	**A4**
65	Muminsi mirongo itatu ishize, ducishirij cg se tugereranije ninkamacupa (Ibirahuri Bingahe) angahe wanyoye buri uko wajyaga kunywa?	Umubare wamacupa cg w'ibirahuri (erekana ikigero) | | |	**A5**
66	Muminsi 30 ishize, nikihe kigero kinini kikinyobwa gisembuye wafataga buri uko wajyaga kunywa, Ubariye hamwe buri bwoko bwibinyobwa bisembuye byose?	Largest number/Umubare wamacupa cg Wibirahuri | | |	**A6**
67	Muminsi 30 ishize, n'inshuro zingahe wafashe Umugabo: 5 cg	Umubare winshuro | |	**A7**

	birenga(Arenga) Umugorre:4 cg birenga(Arenga) Ikigero mpuzamahanga kikinyobwa gisembuye buri uko wajyaga kunywa? 1Standard drink=375ml of beer (5%)=100ml of wine(12%)=30ml of Spirit(40%)		
68	Waba unywa ibinyobwa bisembuye uri kukazi?	Ntanarimwe 01 Gake cyane 02 Rimwe narimwe 03 Kenshi 04 Burigihe 05	**A8**
Ibyo kurya ukunda kurya			
Ibibazo bikurikiraho birabaza imbuto nimboga ukunda kurya. Hano Mfite ikarita ishushanyijeho ibyo kurya, yerekana ingero z'imbuto n'imboga ziboneka mukarere kanyu. Buri foto iri kw'ikarita yerekana imbuto zikwiriye kw'igaburo. Mugihe uri gusubiza ibi bibazo tekereza izo waryaga buri cyumweru mu mwaka ushize.			

Q/N	Ibibazo	Ibisubizo	Kode
69	Mu cyumweru cyose, n'iminsi ingahe urya imbuto? *(use showcard/Erekana ikarita)*	Umubare wiminsi Niba ari zero jya D3	**D1**
70	Nikigero kingana iki cy'igaburo ry'imbuto ufungura buri munsi umwe muyo wavuze haruguru? (*Erekana ikarita)*	umubare w'amagaburo	**D2**
71	Mucyumweru cyose n'iminsi ingahe urya imboga? (*Erekana ikarita)*	Umubare w'iminsi Niba ari zero jya D5	**D3**
72	Namagaburo angahe y'imboga urya buri munsi muyo wavuze haruguru *(erekana ikarita)*	Umubare w'amagaburo	**D4**
73	Nubuhe bwoko bw'amavuta cg ibinure ukoresha keshi utunganya igaburo murugo iwawe?	amavuta y'imboga 01 Lard or suet/ 02 Amavuta y'inka 03 Margarine/ 04 05	**D5**

		abonetse yose Ntayo 06 Niba arayandi jya D5 ayandi	
		Ayandi Yavuge…… ……	**D5** **Other**
Imyitozo ngororamubiri/Siporo			
Noneho ngiye kukubaza igihe umara ukora uburyo butandukanye bw'imyitozo ngororamubiri mu cyumweru cyose.Gerageza gusubiza nubwo waba utiyiziho kwitabira cyane siporo. Banza utekereze igihe umara ukora akazi. Tekereza akazi nkikintu ukora udahembwe cg kugirango uhembwe, nko gukora,kwiga,amahugurwa,imirimo yo murugo,gusarura,kuroba,guhiga ibyo kurya,gushaka akazi,gukata ibyatsi mubusitani,koza imodoka. Mugusubiza ibi bibazo:'Imirimo isaba imbaraga nyinshi'' nimirimo isaba ingufu nyinshi, ituma umuntu ahumeka cyane cg ituma umutima utera bwangu,''Imirimo isaba imbarag zigereranyije''nimirimo isaba imbaraga zigereranyije kandi ituma umuntu adahumeka cyane kani umutima udatera cyane.			
Ibibazo		**Ibisubizo**	**Kode**
Uko ingufu ukoresha kukazi zingana			
74	Ese akazi ukora kaba kagusaba ingufu nyinshi bigatuma uhumeka cyane ndetse numutima ugatera cyane (guterura ibintu	**Yego** 01 **Oya** 02	**P1**

	biremereye,gucukura,kubaka guhera kuminota icumi kuzamura urimo gukoresha ingufu? [*Shyiramo izindi ngero] Erekana ikarita*)	**Niba ari oya jya kuri P4**	
75	Mucyumweru cyose n'iminsi ingahe ukora imirimo isaba ingufu nyinshi mukazi kawe?	Umubare w'iminsi ☐☐	**P2**
76	Nigihe kingana iki umara ukora akazi gasaba ingufu nyinshi mumunsi wose?	Isaha: Iminota ☐☐:☐☐	**P3** **(a-b)**
77	Ese akazi ukora kaba kagusaba ingufu zigereraninyije zituma guhumeka byiyongera numutima ugatera ariko bitari cyane nko guterura ibintu bitaremereye cyane, kugenda genda mukazi wihuta ariko utarengeje intambwe ijana mumunota? *[shyira izindi ngero] (Erekana ikarita)*	Yego 01 Oya 02 Niba ari oya jya kuri P7	**P4**
78	Mucyumweru cyose n'iminsi ingahe ukora imirimo isaba ingufu ziri murugero mukazi kawe ka buri munsi?	Umubare wiminsi ☐☐	**P5**
79	Nigihe kingana iki ukora imirimo isaba imbaraga zigereranije kumunsi wose?	Isaha: Iminota ☐☐ ☐☐	**P6** **(a-b)**

Urugendo rwo kuva ahantu cg ujyayo			
Ibibazo bikurikiyeho ntibirebana nimirimo isaba ingufu yo kukazi umaze kuvuga haruguru. Noneho ndasha kukubaza uburyo ukoresha ujya cg Uva aho uba wagiye. Urugero kukazi,mumurima, Guhaha, kwisoko, ujya gusenga.(Cg ahandi)			
80	Ese ugendesha amaguru cg ukoresha igare iminota irenze icumi iyo ujya cg uva iyo wagiye?	Yego 01 Oya 02 Niba ari oya jya P10	**P7**
81	Mu cyumweru cyose, niminsi ingahe ugenda namaguru cg nigare birenze iminota icumi gukomeza, iyo ujya cg uva aho uba wagiye?	Umubare w'iminsi ☐☐	**P8**
82	Nigihe kingana iki umara ugenda?	Isaha: iminota ☐☐ : ☐☐	**P9** **(a-b)**
Imyidagaduro			
Ibibazo bikurikiyeho bitandukanye nibirebana nakazi ndetse ningendo wavuze haruguru. Noneho Ndashaka kukubaza ibirebana na sporo, Imyidagaduro, kubyina, gukina umupira wamaguru cg uwintoki?			
83	Waba ukora siporo igusaba ingufu nyinshi, guterura ibyuma, kubyina bituma umutima utera cyane ndetse uhumeka cyane (Gukina ruhago cg kwirukanka)?	Yego 01 Oya 02 Niba ari oya P13	**P10**

84	Mucyumweru cyose, niminsi ingahe wakoraga siporo isaba ingufu nyinshi, guterura ibyuma cg gukina umupira wamaguru cg indi myidagaduro?	Umubare w'iminsi ☐☐	**P11**
85	Nigihe kingana (Amasaha) iki Umara ukora siporor isaba imbaraga nyinshi guterura ibyuma gukina umupira mumunsi wose?	Isaha : Iminota ☐☐ : ☐☐	**P12** **(a-b)**
86	Waba ukora sporo cg se imyidagaduro isaba imbaraga ziri murugero zituma guhumeka no gutera kumutima byiyongera gahoro nko kugenda wihuta ukora nkintambwe ijana mumunota, Koga cg gukina umupira wamaboko (nka volleyball) uhereye kuminota icumi kuzamura?	Yego 01 Oya 02 Niba ari Oya jya P16	**P13**
87	Mucyumweru cyose, niminsi ingahe ukora siporo isaba ingufu ziri murugero (Kubyina cg indi myidagaduro)?	Umubare wiminsi ☐☐☐	**P14**
88	Nigihe cyingana iki umara ukora siporo cg indi myidagaduro isaba imbaraga zoroheje mumunsi wose?	amasaha : Iminota ☐☐ : ☐	**P15** **(a-b)**
Imyitwarire yo kumara igihe kirekire wicaye udakora siporo cg utagenda			
Ibibazo bikurikiyeho birebana no kwicara umwanya munini kukazi cg murugo cg kugenda ugaruka wicaye, urikumwe ninshuti zawe harimo nigihe umara mubiro, wicaranye ninshuti,ugenda mumodoka, mumodoka zitwara abagenzi(Bus),Igare			

ry'umwotsi(Gariyamoshi),usoma, ukina amakarita uraba televisiyo na filime, ariko ntushyiremo igihe umara uryamye(Usinziriye). *Erekana ikarita iriho ingero*			
89	Nigihe kingana iki umara wicaye cg ugenda wicaye kuntebe zifite amapine umunsi wose?	**Amasaha : Iminota**	**P16** **(a-b)**
Amateka kundwara z'umutima n'imitsi			
90	Ese waba warigeze wipimisha Umuvuduko w'amaraso upimwe na muganga cg undi muvuzi?	Yego 01 Oya 02 Niba ari oya jya kuri H3a	**H1**
91	Waba warigeze ubwirwa numuganga cg umuvuzi ko ufite indwara y'umuvuduko w'amaraso cg Iperitansiyo?	Yego 01 Oya 02	**H2a**
92	Hari umuti waba uri kunywa ubungubu uvura Indwara y'umuvuduko wamaraso?	Yego 01 Oya 02	**H2b**
93	Vuga amazina y'imiti waba ufata ubu ngubu.	Specify/amazina y'imiti……. …….. ……..	**H2c**
94	Waba warigeze ubwirwa numuganga cg umuvuzi ko	Yego 01 Oya 02	**H3a**

	urwaye indwara yisukari nyinshi(Diyabete)	Niba ari oya jya kuri H4a	
95	Waba hari imiti ufata ya diyabete?	Yego 01 Oya 02	**H3b**
96	Vuga imiti ufata ubu ya diyabete	Amazina yimiti ufata……. …….. ……..	**H3c**
97	Ese waba warigeze ubwirwa numuganga cg umuvuzi ko waba ufite ibinure byinshi mumubiri?	Yego 01 Oya 02 Niba ari oya jya kuri H5	**H4a**
98	Ese waba hari imiti ufata ivura igabanya ibinure byinshi mumubiri?	Yego 01 Oya 02	**H4b**
99	Indicate the medication you are taking/Vuga imiti igabanya ibinure mumubiri ufata ubu.	Amazina y'imiti……. …….. ……..	**H4c**
100	Ese haba hari umuntu mumuryango wiwanyu urwaye indwara y'umutima cg imitsi?	Yes 01 Oya 02 Niba ari oya jya kuri H7	**H5**
101	Niyihe ndwara y'umutima cg se imitsi Arwaye?	Ubwoko bw'indwara:…….	**H6**
102	Muphana iki nuyumuntu?	Papa 01 02	**H6a**

		Mama Umuvandimwe 03 Mushiki wanjye 04 Data wacu/marume 05 Mansenge 06 Sogokuru 07 Nyogokuru 08	
103	Haba harumuntu mumuryango wanyu wapfuye azize indwara y'umutima cg imitsi?	Yego 01 Oya 02 Niba ari oya jya kuri M1	**H7**
104	Mupfana iki nuyumuntu?	Isano	**H7a**

IFISHI Y'IBIPIMO BY'INGANO NIMIKORERE Y'UMUBIRI W'UMUNTU				
Q/N	**Ibipimo**	**ubwoko bw'ibipimo**	**Ibisubizo**	**kode**
Anthropometric measurement/Ibipimo by'ingano y'umubiri w'umuntu				
105	No y'ubaza			M1
106	No y'igikoresho cya1			M2a
107	No y'igikoresho cya 2			M2b
108	Uburebure	In centimeters (Cm)		M3
109	Ibiro	In		M4

	(niba ibiro biruta umunzani andika) 777	Kilograms(Kg)		
110	For **women**:are you pregnant? Kuba gore: ese waba utwite?	Yego 01 Oya 02	Niba ari yego jya M7	M5
111	Umuzenguruko winda	In centimeters(Cm)		M6
112	Umuzenguruko w'amatako	In Cm		M6a
Ibipimo b'umuvuduko w'amaraso				
113	Igano yigitambaro gifata kukuboko	Nigito Kiraringaniye Nikinini	01 02 03	M7
114	Gupima Inshuro ya 1	Systolic (mmHg)		M8a
		Diastolic(mmHg)		M8b
115	Gupima Inshuro ya 2	Systolic(mmHg)		M9a
		Diastolic(mmHg)		M9b
116	Gupima Inshuro ya 3	Systolic(mmHg)		M10a
		Diastolic(mmHg)		M10 b
117	Mubyumweru bibiri bishize waba warivuje umuvuduko	Yego	01	M11

	wamaraso bakaguha imiti yanditswe na muganga cg numuvuzi?	Oya	02	
Ibipimo by'imikorere y'umubiri				
118	Mumasaha cumi nabiri ashize haba harikindi kintu wanyoye uretse amazi?	Yego Oya	01 02	B1
119	Technician ID/No y'ufata amaraso			B2
120	Device ID/No y'imashini			B3
121	Time of day blood specimen taken(24 hour clock)/Igihe amaraso yafatiwe(Kukigero cy'Amasaha 24)	Isaha: Iminota	:	
122	Fasting blood glucose/Isukari yo mumaraso utararya *Choose accordingly mg/dl or mmol/l/(Hitamo ukurikije niba ari mg/dl cg mmol/l)*	mg/dl mmol/l		B5
123	Total Cholesterol/Ibinure bya kolesterol yose hamwe *Choose accordingly mg/dl or mmol/l(Hitamo ukurikije niba ari mg/dl cg mmol/l)*	Mg/dl mmol/l		B6
124	HDL Cholesterol(Ibinure byo mubwoko bwa kolesterol bifite ireme ryinshi) *Choose accordingly mg/dl or mmol/l(Hitamo ukurikije*	Mg/dl mmol/l		B7

	niba ari mg/dl cg niba ari mmol/l)			
125	Triglyceride/Ibinure byo mubwoko bwa tiligiliseride) *Choose accordingly mg/dl or mmol/l(Hitamo ulurikije niba ari mg/dl cg ari mmol/l*	Mg/dl		B8
		Mmol/l		
126	LDL Cholesterol/Ibinure byo mbwoko bwa kolesterol ifite ireme rike *Choose accordingly mg/dl or mmol/l(Hitamo ukurikije niba ari mg/dl cg ari mmol/l)*	Mg/dl		B9
		Mmol/dl		
127	CRP(C Reactive Protein/Poroteyine C *Choose accordingly mg/Hitamo ukurikije niba ari mg/dl*	mg		B10
128	Glycated Hemoglobin(HB1AC)/Ikigero cya HB1AC Percentage/Ijanisha	%		B11
129	Uric Acid/Acide irike	Mg/dl		B12
130	Mubyumweru bibiri bishize waba warigeze wivuza ibinure byinshi byo murwego rwa kolesterol, ukanywa imiti wandikiwe namuganga?	Yego Oya	01 02	B13

Appendix III: School Data collection authorization

JOMO KENYATTA UNIVERSITY OF AGRICULTURE AND TECHNOLOGY

OFFICE OF THE DIRECTOR

KIGALI CAMPUS

P.O.BOX 3373, KIGALI, RWANDA. Email: director .kigalicampus@jkuat.ac.ke

REF: JKU/13/05/460 20th November, 2017.

RECOMMENDATION LETTER FOR ETHICAL CLEARANCE

Dear Sir/Madam,

RECOMMENDATION LETTER FOR, NSANZABERA Charles Reg N0.**PHD/TM410-C010-0013/2015.**

This is to confirm that **NSANZABERA Charles** Reg N0.**PHD/TM410-C010-0013/2015.**is a bona fide student of JKUAT, Kigali Campus pursuing PhD in Public Health with the topic "Risk prediction and factors associated with cardiovascular diseases among workers and their spouses in two beverage processing industries in Rwanda" this was presented to the Institution research review panel with a pass at our Institution JKUAT. He has completed his course work and now working on His research.

Any assistance accorded to him shall be highly appreciated.

Yours Faithfully,

PROF. CHERUIYOT W.K, PhD

DIRECTOR

JKUAT is ISO 9001:2008 Certified

Setting trends in higher education, research and innovation

Appendix IV: BPS Approval

(3)

JOMO KENYATTA UNIVERSITY OF AGRICULTURE AND TECHNOLOGY

DIRECTOR, BOARD OF POSTGRADUATE STUDIES

P.O. BOX 62000
NAIROBI - 00200
KENYA
Email: director@bps.jkuat.ac.ke

TEL: 254-067-52711/52181-4
FAX: 254-067-52164/52030

REF: JKU/2/11/ TM410-C010-0013/2015

21ST JUNE, 2017

NSANZABERA CHARLES
C/o SPH
JKUAT

Dear Mr. Nsanzabera,

RE: APPROVAL OF PHD RESEARCH PROPOSAL AND SUPERVISORS

Kindly note that your research proposal entitled **"Risk prediction and factors associated with cardiovascular diseases among workers and their spouses in two beverage processing industries in Rwanda"** has been approved. The following are your approved supervisors:-

1. Dr. Daniel Nyamongo
2. Dr. Marcel Ndengu

Yours sincerely

PROF. MATHEW KINYANJUI
DIRECTOR, BOARD OF POSTGRADUATE STUDIES

Copy to: - Dean, SPH

Appendix V: Field data collection authorization

Bralirwa
PART OF THE HEINEKEN COMPANY

BRALIRWA Ltd

HUMAN RESOURCES DIRECTION/COSH DEPARTMENT

B.P 131 Kigali

N° contact: Tél. 0788 520 297

21/03/2018

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RE: ACCEPTANCY FOR RESEACH DATA COLLECTION.

I, *Dr. Jean Pierre KABAREGA,* Company Occupational health and safety Manager of Bralirwa Ltd, Certify that Mr. *NSANZABERA CHARLES,* is authorized to carry out his study on cardiovascular diseases risk factors in our workplace organization and this with the aim of improving and promoting the health of employees in Bralirwa Workplace.

Sincerely

Dr Jean Pierre KABAREGA

BRALIRWA Ltd SERVICE MEDICAL B.P. 131 KIGALI-RWANDA KABAREGA Jean Pierre

Bralirwa ltd trade Register G 001 | VAT No. 100004348 | www.bralirwa.com | bralirwa@heineken.com
Kigali (Head Office) | P.O. Box 131 | Tel: +250 252 582993 | Fax: +250 252 585693
Gisenyi | P.O. Box 180 | Tel: +250 252 540372 | Fax +250 252 540356

Appendix VI: Introduction letter from Rwanda Ministry of Education

REPUBLIC OF RWANDA

Kigali, 30/11/2017
N° 2735 /12.00/2017

MINISTRY OF EDUCATION
P.O.BOX 622 KIGALI

The Head of Rwanda National Ethics Committee
Kigali.

Dear Sir/Madam,

RE: Research Project Proposal for Review

I wish to introduce **Mr. Nsanzabera Charles,** PhD student in Public Health, Jomo Kenyatta University of Agriculture and Technology to you. He is seeking Research Clearance Certificate to carry out research in Rwanda.

The Title of her research project is "**Risk Prediction and Factors Associated with Cardiovascular Disease among Workers and their Spouses in two Beverage Processing Industries in Rwanda".** As it is required by the research regulations, the project proposal should be reviewed by the Rwanda National Ethics Committee.

It is in this regard that I am requesting that this project be considered on your review schedule.

I take this opportunity to thank you for your continued collaboration.

Yours sincerely,

25/11/2017

Marie-Christine GASINGIRWA, Ph.D
Director General of Science, Technology and Research

Cc.

- Hon. Minister of Education
- Hon. Minister of State in Charge of TVET
- Hon Minister of State in charge of Primary and Secondary Education
- Permanent Secretary, Ministry of Education

Appendix VII: Rwanda Biomedical Center Collaboration note

RWANDA BIOMEDICAL CENTER

A Healthy People. A Wealthy Nation

Kigali, 07/02/2018

Ref: No 247/25 /RBC/2018

Office of Director General

Chairman of the Rwanda National Health Research Committee (NHRC)
Ministry of Health

Re: COLLABORATION APPROVAL NOTE

Dear Chairman of NHRC,

I, Dr. Jeanine U. Condo, Director General of Rwanda Biomedical Centre confirm that I am aware of the study entitled ***"Risk prediction and factors associated with cardiovascular diseases among workers and their spouses in two beverage processing industries in Rwanda"***, A study whose specific objectives include the prediction of the 10 year cardiovascular diseases risk, determining the behavioural, biological and work condition factors associated with cardiovascular diseases as well as determining the proportion of people with Hypertension, Diabetes and Dyslipidemia, overweight and obesity and difference between two by two groups in risk factors, novel risk development and the level of cardiovascular disease risk among workers of Kicukiro soft drink plant and Rubavu brewery plant and their spouses

This cross-sectional quantitative study will use both stratified and simple Random sampling techniques to select participants (employees) from their departments and spouses or retirees at each site, respectively. The study population totalizes 822 participants aged 30 – 75 composed by 299 workers and 187 spouses in Kicukiro and 204 workers and 132 spouses in Rubavu plant. The study tools include standardized questionnaire with Clinical and anthropometric measures form, laboratory form for biochemical samples; and cardiovascular risk assessment based on WHO/ISH risk prediction chart and FGRS. Data analysis will be performed in SPSS featuring inbuilt cox hazard regression model (Framingham general cardiovascular risk score) and WHO/ISH risk prediction chart to determine the 10-year CVD risk in the population of the study, Cohen Kappa analysis to determine the level of risk prediction agreement between models, Bivariate and multivariate analysis to determine the factors associated with high risk categories and underlying risk factors correlated to predictor variables and finally Anova model to compare the group participants in two different area regarding cardiovascular risk factors differences. Findings from this study will provide a comprehensive understanding of the linkage of workplace, community and cardiovascular diseases, showing the riskier and unsafe working conditions and the strategy to address them, protecting and advising the workforce, employers, community and the policy makers to develop a safe environment as well as helping to reduce the cost spent to cardiovascular diseases and increase awareness and behaviour change toward CVDs Risk Factors in Rwanda.

The study PI is Mr NSANZABERA Charles, PhD Student at in Public Health programme at Jomo Kenyatta University of Agriculture and Technology and Co-PI is Dr Evariste NTAGANDA from Rwanda Biomedical Center , Non Communicable Disease Division /Cardiovascular Diseases Unit , supervised by Dr Daniel NYAMONGO SAGWE and Dr Marcel NDENGO . DG's office, through Medical Research Center and NCD Division is ready to collaborate with the study team to ensure effective implementation.

Sincerely,

RWANDA BIOMEDICAL CENTER

Jeanine U. Condo, MD, PhD
Associate Professor of Public Health
Director General /RBC

Appendix VIII: Ethical Clearance

REPUBLIC OF RWANDA/REPUBLIQUE DU RWANDA

NATIONAL ETHICS COMMITTEE / COMITE NATIONAL D'ETHIQUE

Telephone: (250) 2 55 10 78 84
E-mail: info@rnecrwanda.org
Web site: www.rnecrwanda.org

Ministry of Health
P.O. Box. 84
Kigali, Rwanda.

FWA Assurance No. 00001973
IRB 00001497 of IORG0001100

May 11, 2018
No.121/RNEC/2018

NSANZABERA CHARLES
Primary Investigator
(A student at Jomo Kenyatta University)

Your research project: "**RISK PREDICTION AND FACTORS ASSOCIATED WITH CARDIOVASCULAR DISEASES AMONG WORKERS AND THEIR SPOUSES IN TWO BEVERAGE PROCESSING INDUSTRIES IN RWANDA** " has been evaluated by the Rwanda National Ethics committee.

		Involved in the decision		
			No (Reason)	
Name	Institute	Yes	Absent	Withdrawn from the proceeding
Dr.Jean-Baptiste MAZARATI	Biomedical Services (BIOS)	X		
Prof. Eugène RUTEMBESA	University of Rwanda	X		
Dr.Laetitia NYIRAZINYOYE	University of Rwanda	X		
Dr. Egide KAYITARE	University of Rwanda	X		
Sr.Domitilla MUKANTABANA	Kabgayi Nursing and Midwife school	X		
Dr. David K. TUMUSIIME	University of Rwanda	X		
Dr. Lisine TUYISENGE	Kigali Teaching Hospital	X		
Dr. Claude MUVUNYI	Biomedical Services (BIOS)	X		

After reviewing your protocol during the RNEC meeting of March 10, 2018 where quorum was met, and revisions made on the advice of the RNEC submitted on 08 May 2018, we **hereby provide approval for the above mentioned protocol.**

Please note that approval of the protocol and consent form is valid for **12 months.**
You are responsible for fulfilling the following requirements:

1. Changes, amendments, and addenda to the protocol or consent form must be submitted to the committee for review and approval, prior to activation of the changes.
2. Only approved consent forms are to be used in the enrollment of participants
3. All consent forms signed by subjects should be retained on file. The RNEC may conduct audits of all study records, and consent documentation may be part of such audits.
4. A continuing review application must be submitted to the RNEC in a timely fashion and before expiry of this approval.
5. Failure to submit a continuing review application will result in termination of the study.
6. Notify the Rwanda National Ethics committee once the study is finished.

Sincerely,

Date of Approval: May 11, 2018
Expiration date: May 10, 2019

Dr. Jean- Baptiste MAZARATI
Chairperson, Rwanda National Ethics Committee.
C.C.
- Hon. Minister of Health.
- The Permanent Secretary, Ministry of Heath