THE STUDY ON OCCUPATIONAL RISK FACTORS IN THE MANIFESTATION OF CARDIOMETABOLIC SYNDROME IN AND AROUND BIJAPUR

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Certificate

This is to certify that this thesis entitled **"The study on occupational risk factors in the manifestation of cardiometabolic syndrome in and around Bijapur"**is a bonafide work of **Mr. Sanjeev Srinivas Walvekar** and was carried out under our supervision and guidance in the Department of Biochemistry, Shri B.M.Patil Medical College, Hospital & Research Centre, Bijapur, Karnataka, India.

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I declare that the thesis entitled **"The study on occupational risk factors in the manifestation of cardiometabolic syndrome in and around Bijapur"** has been prepared by me under the guidance of Professor Jeevan G. Ambekar, Department of Biochemistry, BLDE University's Shri B.M.Patil Medical College, Hospital & Research Centre Bijapur, Karnataka, India. No part of this thesis has formed the basis for the award of any degree or fellowship previously.

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I dedicate this thesis to

My Parents who have groomed me to higher level

Late Shri Srinivas Walvekar & Late Smt Sudha Walvekar.

ABSTRACT

Background

The stress at the work place (Occupational Stress) can be inferred from the existence of problems in the employee/environment interaction or measured in terms of health problems. The prevalence of cardiovascular disease (CVD) and its risk factors among occupational groups have been reported. Cardiometabolic syndrome is identified as a risk factor and the pre-state for CVD and type-2 diabetes. Metabolic syndrome is a cluster of risk factors including obesity, hyperglycemia, hypertension and dyslipidemia. The association between the level of occupational stress and development of metabolic syndrome remains unclear.

Objectives

We aimed to determine the impact of occupational risk factors including stress on metabolism in people with different working environment and to estimate the differences in prevalence and risk of metabolic syndrome with relation to level of stress among individuals with different occupations.

Methods

A cross sectional study was conducted on subjects (n=405) with three different occupations with age ranging from 30-60 years: Bank employees (n=97), Bus drivers (n=90) and police constables (n=108).The subjects not belonging to these three occupational groups served as the control group (n=110). The level of stress was assessed using a validated perceived stress scale (PSS), including 14-items. All the participants were further divided into 2 subgroups on the basis of PSS scale: (a) Stressed and (b) Non-stressed. Fasting blood glucose, Glycosylated HbA1c, lipid profile, cortisol, insulin, Lipoprotein(a), Homocysteine, C-reactive protein (CRP) in serum and microalbumin in urine were measured.

Results

We found a significant rise in the serum cortisol level in all the participants those identified as under stress (stressed subgroups) based on Perceived stress scale scoring. There was a statistically significant difference in the levels of Cortisol between stressed and non-stressed subgroups of all the three occupations. While no significant difference was noticed in serum cholesterol and HDL cholesterol between stressed and non-stressed subgroups.

A significant positive correlation was observed between serum cortisol and fasting blood glucose ($p< 0.001$), Glycosylated HbA1c ($p<0.05$) in the stressed subgroup of bank employees and police constables. On the basis of NCEP ATP III guidelines, participants were further classified into metabolic and non-metabolic syndrome subgroups. The prevalence of metabolic syndrome was 38 % among all the study group participants (n=295). Waist circumference level ($p<0.001$) was significantly raised in subjects with metabolic syndrome subgroups of bus drivers, bank employees and police constables.

The CVD risk factors were quite high among the study group participants. We found that 10.9% subjects had BMI > 30 Kg / m^2; 8.8% increased waist circumference; 15.6% elevated LDL cholesterol: 13.7% raised triglyceride; 14.7% hypertension, diabetes 16.9% and smoking habit 26.2 %.

Conclusion

The above findings demonstrate a significant association between level of stress and risk factors of metabolic syndrome in the participants of three study groups. Risk factors of metabolic syndrome were significantly higher in stressed subgroups when compared with non-stressed subgroups. The study indicates that many employees from the studied occupational groups were at the risk of developing CVD, type-2 diabetes and Cardiometabolic syndrome. The study emphasizes about addressing the health complications arising from the job stress and a more aggressive working health policy has to be implemented at all work places.

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ABBREVATIONS

AACE	American Association Of Clinical Endocrinologists.
ACR	Albumin creatinine ratio.
ANOVA	Analysis of Variance.
ATP III	Adult Treatment Panel III.
BMI	Body mass index.
bpm	Beat per minute.
CHE	Cholesterol esterase.
CHO	Cholesterol oxidase.
CMS	Cardiometabolic syndrome.
Conc	Concentration
Creat	Creatinine
CRP	C-Reactive protein.
CVD	Cardiovascular Disease.
DBP	Diastolic blood pressure.
DM	Diabetes mellitus.
ESRD	End stage renal disease.
FBS	Fasting blood glucose.
H0	Null hypothesis.
H1	Alternate hypothesis.
HBA1c	Hemoglobin A1c.
HDL	High density lipoprotein.

HOMA-IR	Homeostasis model assessment- Insulin resistance.
HPA	Hypothalamic pituitary adrenal.
HRV	Heart rate variability.
IDF	International Diabetes Federation.
IR	Insulin resistance.
LDL	Low-density lipoprotein.
NAFID	Nonalcoholic fatty liver disease.
NCD	Non communicable disease.
NCEP	National Cholesterol Education Program.
OGTT	Oral glucose tolerance test.
PCOS	Polycystic ovarian syndrome.
PSS	Perceived stress scale.
SBP	Systolic blood pressure.
SD	Standard deviation.
SNS	Sympathetic nervous system.
SPSS	Statistical package for social science.
T2DM	Type 2- diabetes mellitus.
TC	Total cholesterol.
TG	Triglyceride.
VLDL	Very low density lipoprotein.
WC	Waist circumference.
WHO	World Health Organization.
WHR	Waist to hip ratio.

CHAPTER-I

INTRODUCTION

CHAPTER I

INTRODUCTON

1.1. PURPOSE OF THE STUDY

The WHO has described the health as a state of complete physical, mental and social well-being and not merely an absence of disease or infirmity (WHO-1948). In the present life, most of the individuals are facing a variety of problems to maintain a good health. The problem includes unhealthy environment, changes in food habits, sedentary life style, and excessive consumption of alcoholic beverages, smoking and lack of health awareness, leading to various metabolic disorders like Diabetes, Obesity, Hypertension and Dyslipidemia. Good health is man's most precious possession.

India is a diverse country and many states in India are passing through an epidemiological health transition with high rates of urbanization. Urbanization has led to economic improvement. The consequences of which are increased food consumption and decreased physical activity. One of the effects of this economic transition is a shift in the disease spectrum from communicable to non-communicable diseases (NCDs) (Mohan V et al., 2008). NCDs, especially cardiovascular disease, diabetes mellitus have emerged as a major public-health problem in India. The morbidity and mortality, the most productive phase of life is posing serious challenges to Indian society and economy (Reddy KS., 2006). The huge burden of cardiovascular diseases (CVD) in the Indian Subcontinent is the consequence of the large population and the high prevalence of CVD risk factors (Goyal A et al., 2006).

Cardiometabolic syndrome also referred as 'Metabolic syndrome' has been devised to include a cluster of cardiovascular disease (CVD), diabetes mellitus risk factors or risk markers that tended to occur together. People identified with metabolic syndrome are associated with a 2-fold increase in risk of CVD and four to five fold risk of developing type 2-diabetes compared to people without the syndrome (Alberti KGMM, et al., 2005). However, with stringent dietary restrictions and physical exercise, the metabolic syndrome is preventable and treatable. Such aggressive action will significantly reduce the risk of developing further complications like type 2-diabetes and cardiovascular disease. Reaven in 1988, first time termed the syndrome as 'Syndrome X'. Later it was named as Reaven's Syndrome, Insulin resistance syndrome, etc. It is identified clinically by the presence of

abnormalities like abdominal obesity, elevated triglycerides, low levels of high-density lipoprotein cholesterol (HDL-C), elevated blood pressure and high blood glucose and/or insulin resistance. As per the National Cholesterol Education program- Adult treatment panel III (NCEP-ATP III), cardiovascular disease is the major outcome of the metabolic syndrome. But many people with this syndrome have insulin resistance, which confers increased risk for type 2 diabetes. When diabetes becomes clinically apparent, CVD risk rises sharply (Grund SM et.al, 2004).

The work place environment has become focus for interventions aimed at the reduction of risk for the chronic diseases (Violanti JM et al 2009). Most of the people employed, spend a substantial amount of their time at the premises of their work site. They may face a variety of hazards owing to chemicals, biological agents, physical factors, etc. The interventions of affecting risk factors in the workplace can be a fruitful approach for chronic diseases in the total population. As per the World Health Organization (WHO), occupational health risk is the tenth leading cause of morbidity and mortality (Mandal MK, 2009). The risk factors in the workplace may lead to cancers, accidents, respiratory diseases, hearing loss, stress related disorders and communicable diseases and others. Occupational stress has become almost globalized.

The stress at work place is explained in terms of the interaction between a person and their (work) environment and it is the awareness of not being able to cope with the demands of one's environment. The presence of stress can be inferred from the existence of problems in the employee/environment interaction or measured in terms of health problems. The specific nature of each working environment poses unique risk factors such as shift work, night shift, job strain, job dissatisfaction, sedentary working style all of which may contribute to illness including diabetes and CVD. Moreover these risk factors are not usually found isolated but in combination. Most of the studies from the developed countries have provided a large body of evidence that job strain and different CHD indicators, such as the incidence of myocardial infarction. Recent studies have reported that the presence of metabolic syndrome which is composed of five CHD risk factors: obesity, high triglyceride (TG) and low high-density lipoprotein cholesterol (HDL) concentration, elevated blood pressure and impaired glucose tolerance is a risk factor for cardiovascular disease (Grund SM et al., 2004).

The risk of CVD & DM is attributed to metabolic syndrome, whether the prevalence of the risk is high or normal is unclear. We have no evidence that metabolic syndrome directly

influence the CVD risk profile. To the best of our knowledge this issue has never been studied in a specific population of different occupational subjects.

There is a growing concern worldwide about the metabolic syndrome. The association between the level of occupational stress and metabolic syndrome remains unclear. Moreover, the relationship between job and metabolic syndrome is least reported. Few studies have shown an association between metabolic syndrome and risk of development of cardiovascular disease and diabetes among the selected occupations (Saberi HR et al., 2011; Thayyil J et al., 2012). The role of factors such as occupational stresses, physical inactivity, prolonged working hours, night shifts, job strain, inappropriate dietary habits and sedentary working style in the development of metabolic syndrome remains to be established. As per our knowledge, there are no studies that have examined impact of occupational stress on metabolism; and the prevalence of metabolic syndrome among bank employees, bus drivers and police constables. There is the absence of such data about the influence of work stress on the risk of development of metabolic syndrome. Also it remains unclear about the level of occupational stress and its impact on cardiovascular risk factors. This study will bridge the research gap. We assume that the outcome of the study will benefit these employees for averting metabolic syndrome; lead a healthy life to serve the society in a better way.

1.2 HYPOTHESIS

The levels of stress (long working hours, shift work, irregular duty schedules, sedentary working style and irregular dietary habits) in different occupations are associated with the prevalence of metabolic syndrome.

H0: Null hypothesis

- There may not be any significant difference in level of stress in different occupations.
- There may not be any significant impact of occupational stress on metabolism of glucose, lipid and hemodynamic factors.
- There may not be any significant difference in glucose metabolism between individuals with different occupations.
- There may not be any significant difference in lipid metabolism between individuals with different occupations.
- There may not be any significant difference in hemodynamic factors between individuals with different occupations.
- There may not any significant association between occupational stress and metabolic syndrome.

H1: Alternate hypothesis

- There will be significant difference in level of stress in different occupations.
- There will be significant impact of occupational stress on metabolism of glucose, lipid and hemodynamic factors.
- There will be significant difference in glucose metabolism between individuals with different occupations.
- There will be significant difference in lipid metabolism between individuals with different occupations.
- There will be significant difference in hemodynamic factors between individuals with different occupations.
- There will be significant association between occupational stress and metabolic syndrome.

1.3 AIMS AND OBJECTIVES

Aim

We aimed to determine the impact of occupational risk factors including level of stress on metabolism in people with different working environment; and to estimate the differences in prevalence and risk of metabolic syndrome with relation to level of stress among individuals with different occupations.

Objectives:

1. To estimate level of stress among different occupations.
2. To find any significant difference in level of stress between different occupational groups.
3. To determine the impact of occupational stress on metabolism of glucose & lipid
4. To determine the impact of occupational stress on insulin function & hemodynamic factors.
5. To assess the correlation between level of stress and metabolic changes among different occupational groups.
6. To assess the correlation between level of stress and blood pressure among different occupational groups.
7. To determine the prevalence of metabolic syndrome on the basis of various standard guidelines among different occupational groups.

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CHAPTER-II

REVIEW OF LITERATURE

CHAPTER II

REVIEW OF LITERATURE

2.1. INTRODUCTION

Non communicable diseases which include Diabetes mellitus and cardiovascular disease are estimated to cause 3.5 million deaths around the world each year. The WHO has developed an action plan for implementation of global strategies in prevention and control of non-communicable diseases (WHO. 2008). The term "metabolic syndrome" has been advocated as a simple clinical tool to describe those individuals at increased risk of type 2- diabetes and cardiovascular diseases due to the metabolic dysfunction. It also forms a conceptual basis for understanding some of the pathophysiological links between metabolic risks, diabetes and CVD.

Metabolic syndrome as a greater health risk is receiving significant attention worldwide. Metabolic syndrome is not a new condition. The clustering of metabolic syndrome risks with cardiovascular disease and DM has been recognized very long back. But the modern concept of metabolic syndrome began when Reaven proposed a conceptual framework which links between biological events in a single path physiological construct (Reaven, 2005). He suggested that insulin resistance provides common mechanisms underlying the associated abnormalities such as blood pressure, lipid abnormalities and glucose intolerance. Several attempts have been made to develop a set of components that constitute metabolic syndrome. There has been a lot of debate about whether metabolic syndrome should be considered a syndrome at all.

A syndrome defines a group of signs and symptoms of a common underlying pathology, but the real pathology of metabolic syndrome is still not yet fully known. Some authors argue that although the symptoms of the metabolic syndrome often appear together, they may reflect diverse disease processes. It was proposed that using metabolic risk or Cardio metabolic risk could be the better way to describe the syndrome. It was also remarked that it is useful as a concept rather than using the word "syndrome" (Kahn R et al., 2005).

2.1.1 Definition and history

Cardio metabolic syndrome, an another name for 'Metabolic syndrome' is a cluster of interconnected biochemical, physiological clinical, and metabolic factors that increases the risk of type 2- diabetes mellitus and cardiovascular disease over next few years (Alberti M et al.,2009). Abdominal obesity, elevated blood pressure, elevated fasting glucose, high serum triglycerides and lowered high density cholesterol are the medical conditions considered to diagnose the metabolic syndrome. The risk factors related to diabetes were explained in 1920 only (Joslin. 1921). The physician Dr. Jean Vague observed that the upper body obesity was related to development of diabetes, atherosclerosis and gout (Vague. 1956). In 1927, Maranon, the founder of modern endocrinology in Spain, described the fact that the arterial hypertension is a pre-diabetical stage and this concept was similarly applied to obesity (Milici. 2010). In the early 1950s, Jean Vague presented a series of reports on the sexual differentiation of obesity and its consequences. Vague later described the relationship between abdominal obesity, fat distribution and their association with diabetes and other chronic disorders (Vague, 1956). In a study carried over on the Finnish population, a strong link between glucose intolerance, hyperinsulinaemic, and coronary heart disease was observed. (Pyorala et al. , 1979).

The term "Metabolic syndrome" was used by Haller for the association of obesity, diabetes mellitus, hyperlipoproteinemia and hyperuricemia (Haller. 1977). In 1988, MrReaven proposed the name 'Syndrome X' to the group of associated conditions which were important in the development of coronary artery disease (Reaven., 1988). In the proposal, the association between adipose tissue and insulin resistance was acknowledged by him though he did not mention about obesity. Later this syndrome was renamed as 'The Insulin resistance Syndrome' (Haffner et al., 1996). In 2001, the Centers for Disease Control and Prevention approved the request by the American Association of Clinical Endocrinologists (AACE) for a new diagnostic code, ICD-9-CM 277.7 for "Dysmetabolic Syndrome X" and thus, created a new disease (Peters. 2007).

2.1.2 Criteria to diagnose metabolic syndrome

For the first time, World Health Organization proposed the definition for metabolic syndrome in 1999. It was the first definition accepted with specific thresholds which included dyslipidemia, insulin resistance, obesity, hypertension and microalbuminuria (Alberti et al., 1998).

European Study of Insulin resistance incorporated the Insulin resistance as one of the components to define the metabolic syndrome. Post 2-hour glucose tolerance test was deemed to define the Insulin resistance. However, it was unsuitable for clinical practice (Balkau et al., 1999). In 2001, The National Cholesterol Education Program Adult Treatment Panel (NCEP/ATP) released its definition (Cleeman ., 2001). Later the committees like joint expert group of the National Heart, Lung and Blood Institute and the American Heart Association (Lorenzo et al., 2006) and the International Diabetes Federation (IDF) proposed the definition for the metabolic syndrome (Alberti et al., 2006). These definitions excluded the insulin resistance and given prominence to fasting blood glucose. They have given due importance to abdominal obesity. All these groups agreed on the core components of metabolic syndrome but have different clinical criteria that identify individuals with the syndrome.

Presently, the two most widely used definitions are those of the NCEP: ATP III and IDF. Of these, 2001 Third Report of the NCEP's Adult Treatment Panel has emerged as the most commonly used definition, primarily because it provides a relatively simple approach for diagnosing the metabolic syndrome by employing easily measurable risk factors (Moebus., 2006). Specifically, the NCEP defines the metabolic syndrome as having 3 or more of the following 5 cardiovascular risk factors: 1) central obesity (waist circumference: men ≥102 cm; women ≥88 cm); 2) elevated triglycerides (≥150 mg/dl); 3) diminished high density lipoprotein (HDL) cholesterol (men < 40 mg/dl; women <50 mg/dl); 4) systemic hypertension (≥130/85 mm Hg); and 5) elevated fasting glucose (≥110 mg/dl). In 2004, this NCEP definition was revised (rNCEP) by lowering the threshold for fasting glucose to ≥100 mg/dl in concordance with American Diabetes Association criteria for impaired fasting glucose (Grundy SM., 2004). Also, thresholds for central obesity were lowered from strictly ≥102 cm in men and 88cm in women to greater than or equal to these values.

Finally, the NCEP definition includes patients being treated for dyslipidemia, hyperglycemia, or systemic hypertension. Regardless of which definition is used, insulin resistance and central obesity are postulated to be the key components of the metabolic syndrome and both lead to glucose intolerance and dysglycemia. Consequently, even a small change in the fasting glucose threshold may have an important impact on the associated cardiovascular risk. For this reason, there has been considerable debate over the impact of lowering the fasting glucose threshold from ≥110 to ≥100 mg/dl (Salvatore M., 2010). Though the metabolic syndrome does not encompass all cardiovascular risk factors, it

provides a picture of a predominantly increasing risk for a growing portion of the population. The ATP III guidelines were intended to be more useful with simple measures that were applicable in both clinical and research settings (Zimmet P, 2005).

In 2005, the International Diabetes Federation (IDF) proposed their own definition of metabolic syndrome intended for global application in clinical practice and represents modifications to the WHO definition and ATP III criteria. The IDF places more emphasis on abdominal obesity as the core feature of the syndrome as it is independently associated with each of the other metabolic syndrome factors including insulin resistance. The IDF took an additional step to develop ethnic specific values for waist circumference cut off points based on various sources of epidemiologic data. IDF recognizes dyslipidemia or treatment for dyslipidemia, and hypertension or previous diagnosis for hypertension as risk factors.

However, IDF does not exclude diabetics from the definition (Alberti et al., 2006). The prevalence of metabolic syndrome was highest with modified NCEP ATP III and least with NCEP ATP III. This difference in prevalence may be attributed to waist circumference cut-offs of 102 and 88 cm for men and women respectively in NCEP ATP III, while 90 and 80 cm for men and women respectively in modified NCEP ATP III (Dhanaraj E et al., 2009). The use of waist circumference to assess abdominal adiposity is greater than BMI. The cut-off value for waist circumference is likely to be population specific as there are clear differences across ethnic populations in the relationship between overall adiposity, abdominal obesity and visceral fat accumulation (Chee-Eng T et al., 2004).

The WHO definition includes those persons with high risk of developing diabetes as well as people diagnosed with type 2- diabetes. The obesity component can be calculated by the waist to hip ratio or BMI and includes microalbuminuria, which links the syndrome with risk for developing chronic kidney disease. The WHO definition has been criticized for including type 2 diabetics in the definition and not reserving the diagnosis of metabolic syndrome for those who are in danger for developing diabetes.

Additionally, the WHO definition includes impaired glucose tolerance measured by oral glucose tolerance test (OGTT) or 2-hour post glucose challenge as part of its criteria, tests considered by some to be less practical and an added cost with a small added value of predicting cardiovascular risk (Grundy et al., 2004). They focus specifically on waist circumference, which is a measure of central obesity. It was agreed that there should not be an obligatory component. Three abnormal findings out of five would qualify a person for the

metabolic syndrome. For the waist circumference, national or regional cut points may be applicable. Metabolic syndrome has been approved worldwide as a diagnostic tool. However it could do not provide conclusive evidence to support the clinical utility of the metabolic syndrome as a diagnostic category. As there is variation in the definition for metabolic syndrome, clinicians find it difficult to interpret the syndrome in the clinical practice.

Table 1: Definition of metabolic syndrome by different organizations

	WHO	R-NCEP-ATP III	IDF
Obesity	WHR >0.90 (male) >0.85 (female) Or BMI >30 kg/m2	WC ≥90 cm (male) ≥80 cm (female	WC ≥90 cm (male) ≥80 cm (female
Serum Triglyceride	≥150 mg/dl	≥150 mg/dl	≥150 mg/dl
Serum HDL Cholesterol	< 35 mg/dl (male) < 39 mg/dl (female	<40 mg/dl (male) <50 mg/dl (female)	<40 mg/dl (male) <50 mg/dl (female
Blood Pressure	≥140/90 mmHg. Or on medication.	≥130/85 mmHg. Or medical treatment of previously diagnosed hypertension	≥130/85 mmHg Or medical treatment of previously diagnosed hypertension
Fasting Plasma Glucose	FPG≥110 mg/dl	≥100 mg/dl	≥100.0mg/dl. Or previously diagnosed T2DM
Other risk Factors	Urinary albumin excretion rate≥20 µg/min Or albumin/ creatinine ratio≥30 mg/g		
Diagnosis	Impaired FPG + any 2 criteria	Any 3 criteria	WC + any 2 criteria

R-NCEP ATP III: Revised National Cholesterol Education Program. Adult Treatment Panel III; WHO: World Health Organization; IDF: International Diabetes Federation. HDL-C: High-density lipoprotein cholesterol.

2.2 PREVALENCE OF METABOLIC SYNDROME

Studies have the accuracy of the metabolic syndrome by applying these each set of criteria but the difference lies in the prevalence of metabolic syndrome due to the differences in the screening criteria (Alberti K. G et al., 2006). One study compared the prevalence of metabolic syndrome using WHO and ATP-III definitions among 8608 subjects. About 86% people were classified the same by both the definitions 23.9% by WHO and 25.1% by ATP III definitions (Ford ES, Giles WH., 2003). The WHO criteria have been applied mainly in European cohorts (Ilanne et al., 2004).

In a research study conducted by DECODE (Diabetes Epidemiology: Collaborative Analysis of Diagnostic Criteria in Europe) Study Group, variations were found in the prevalence of the metabolic syndrome based on the three definitions utilized. It was a cross-sectional study consisting of 4190 men and 4950 women non-diabetics from seven DECODE study centers. In this study, the prevalence of the syndrome using the WHO, ATP-III and the EGIR criteria ranged from 16.5-24.7% in men and 15.2-20.9% in women aged 30-77 years (DECODE Study Group ., 2005). A study was undertaken to find the capacity of metabolic Syndrome to identify impaired glucose tolerance. It was informed that metabolic Syndrome could be used to screen for individuals likely to fail an oral glucose tolerance test. 61 to 93% with the three components of metabolic syndrome (impaired fasting glucose, large waist circumference and raised triglyceride levels) failed glucose tolerance test (Meigs J et al., 2005). A 12-year follow-up data was examined from a Finnish study of 2682 middle-aged men who did not have cardiovascular disease or diabetes at baseline (Lakka et al 2002).

The prevalence of CHD was 19% in people with both the metabolic syndrome and diabetes versus 9% in those with neither and 7.5% in the small percentage of the study population that had diabetes but not the metabolic syndrome. These results suggest that for most diabetic patients, cardiovascular risk is related not to diabetes itself but to the concomitant presence of the metabolic syndrome (de Simone et al., 2005). A study was undertaken in the Qatari population to examine the differences in gender and age prevalence of metabolic syndrome. According to the revised criteria of NCEP ATP III and IDF, the prevalence of metabolic Syndrome in studied subjects was 26.7% and 33.9% respectively. The prevalence of metabolic Syndrome by both definitions was more in the 30-39 years age group among males, and the 40-49 years age group among females (Bener A et al., 2010). As per another study done with the application of WHO criteria for metabolic syndrome, it

was found that the prevalence of metabolic syndrome varied between 7 and 36% for men of age group 40–55 years and between 5 and 22% for women of the same age group (Aguilar Salinas CA et al., 2003). A wide range of prevalence of metabolic syndrome in Asian population is reported. (Misra A et al., 2005; Misra A et al., 2007). In many countries there is very little difference between rates of metabolic syndrome among women and men, there are some countries that have noticeably greater numbers of women than men that meet the metabolic syndrome criteria (Lorenzo C et al. 2007; Lilu J et al 2006). Because sex-related differences in metabolic syndrome prevalence are universal, differences between women and men within specific countries may be due to the inclusion criteria for each of the definitions was based upon epidemiological data. Although the definitions may still be used to estimate prevalence in any population, it remains unclear how each of the individual criteria may impact sex-specific prevalence rates within certain countries.

2.2.1 Prevalence of metabolic syndrome in India

In the future 70% of new incident cases of diabetes will be located in the developing countries (Yach D ., 2004). Among the ten leading countries with diabetes, five are in Asia (Roglik et al 2000). In 2025, India will rank first with 57 million diabetics. The recent data are shown that the prevalence of metabolic syndrome in Asian Indians is dependent on the region, lifestyle pattern and extent of urbanization (Misra A et al., 2009). The prevalence of metabolic syndrome was reported to be 20% to 25% in south Asians in one study (Nestel R et al., 2007). In a community based cross-sectional study from Haryana India, (43.6%) of metabolic syndrome cases were found in the age group of 65 years and above and the minimum (6.4%) in the age group of 20-34 years. And also an increasing trend of the prevalence rate was observed with increase in the age (Pathania D et al., 2013). In India, the studies had been done to find the prevalence of metabolic syndrome and most of the studies had used ATP-III. But in a study about the prevalence of metabolic syndrome in south Indian population by various definitions, it was found that by IDF 25.8% of individuals > 20 years were having metabolic syndrome as compared to 18.3% for ATP-III (Deepa et al. 2006). Another study from Bangalore (Kanjlal et al 2008), concluded that prevalence of metabolic syndrome (by ATP-III) was 40.3% as compared to 34.9% by IDF definition. High prevalence of metabolic syndrome in the study could be attributed to modern life style adopted by residents of a highly urbanized population of Chandigarh, an Union Territory of India (Mangat C et al 2010). Metabolic syndrome was found in 9.3 per cent rural population.

Traditional societies and population residing in and around rural areas were expected to have low prevalence as these were not exposed to modernization (Sarkar S et al., 2006).

In view of the increasing prevalence of type 2- diabetes and hypertension in India, it is reasonable to assume that there is an increasing epidemic of Cardiometabolic syndrome going on in India at the moment. Since the Cardiometabolic syndrome is a long-term process that starts early in life and is involved in the patho- physiology of type 2- diabetes and atherosclerosis, vigorous early management of the syndrome will have a significant impact on the prevention of both diabetes and CVD (Zimmet et al., 2005). In one study, it was concluded that metabolic syndrome is a highly prevalent but under recognized and under treated condition. The concurrence of abdominal obesity, borderline hyperglycemia, atherogenic lipoprotein phenotype, and/or hypertension in a patient constitutes a system of linked pathogenesis and high atherogenicity (Miranda et al., 2005). The huge burden of cardiovascular diseases in the Indian Subcontinent is the consequence of the large population and the high prevalence of CVD risk factors (Goyal et al., 2006).

2.3 THE COMPONENTS OF METABOLIC SYNDROME

The clustering of insulin resistance and other components of metabolic syndrome is often observed in south Asians and they may lead to type 2 DM and cardiovascular diseases and make South Asians more prone to develop these diseases (Ferrannini E et al., 2007). Studies have indicated the association between essential hypertension and metabolic syndrome (Bjorntorp p et al., 2000: Kelishadi R et al., 2005). As per a study done in Japanese subjects who had obesity, dyslipidemia or impaired glucose tolerances were at higher risk for the development of hypertension(Hiroyuki Takase et al ., 2008).

2.3.1 Obesity

Obesity, a chronic imbalance between energy intake and energy expenditure is becoming a worldwide epidemic. Obesity increased significantly from 23 to 30.5% between 1988 and 2000, and the increase occurred for both men and women and in all age groups (Flegal KM., 2002). The prevalence of obesity and metabolic syndrome is rapidly increasing among children and adolescents, especially in groups with the lowest level of education. In the United States, the prevalence of overweight children reached approximately 14%, which is 3 times higher than the prevalence observed 40 years ago. A vicious circle of higher birth weight, childhood obesity, metabolic syndrome, and type 2- diabetes mellitus has evolved. Obesity is well documented as a contributing factor in diabetes, cardiovascular disease (CVD), hypertension, stroke, cancer, osteoarthritis, asthma, and sleep apnea (Stein CJ et al.,2004). Overweight, obesity, and the metabolic syndrome have recently emerged as strong independent risk factors for chronic kidney disease (CKD) and ESRD (Ihab M et al., 2007).

Many cohort studies have documented the association of obesity with metabolic syndrome, resulting in to myocardial infarction, cerebrovascular disease and may be the sudden death (Halpern A et al., 2003).Central adiposity is a key feature of the metabolic syndrome, as reflected by the strong relationship between the prevalence of the syndrome and waist circumference and increasing adiposity. The study done in South Africa in 2009 reported that central obesity measured by waist circumference was the most prevalent component of the metabolic syndrome, (58.6% vs 49.5%) for rural and urban women respectively (Motala A et al., 2009).Another hospital based study in Botswana also showed that obesity (28.7%) and overweight (27.3%) were the major high risk factors for the metabolic syndrome among hospital workers (Kelliny C et al., 2007). The concept of metabolic syndrome in children and adolescents is still under debate because ofless number

of studies being undertaken. The presence of obesity in children and adolescents in relation to metabolic syndrome is of a concern (Weiss R et al., 2004). In the Symposium on metabolic syndrome, Dyslipidemia, Hypertension and type 2- diabetes in Children and Adolescents, organized by the Brazilian Society of Diabetes, it was concluded that the action should be aimed at the prevention of obesity in children and adolescents (Halpern A et al., 2010). Studies have established that the presence of anyone component of metabolic syndrome increases the possible risk of having one or more other components (Blumenthol RS .,2005).In a cross sectional study conducted by NHANES III (National health and Nutrition survey III), it was found that metabolic syndrome was significantly associated with self-reported myocardial infarction or stroke. Also insulin resistance, hypertriglyceridemia and hypertension were independently associated with greater risk of myocardial infarction (Ninomiya JK et al., 2004).

2.3.2 Dyslipidemia

The changes in the levels of serum lipoproteins are the one of the earliest manifestations of metabolic syndrome and can contribute to the development of cardiovascular disease. Dyslipidemia is one of the component of metabolic syndrome since the definitions include hypertriglyceridemia (defined as serum triglycerides $\geq$ 150 mg/dl) and a low HDL cholesterol concentration (defined as HDL-cholesterol < 40 mg/dl for men and < 50 mg/dl for women by NCEP ATP III, or HDL-cholesterol < 35 mg/dl for men and < 40 mg/dl for women by WHO) as components. Individuals with metabolic syndrome in addition to obesity, present high level for cardiovascular disease. The abnormalities like, elevated triglyceride level, decreased HDL-Cholesterol are the most likely to occur together than separately and are key components of the metabolic syndrome. The new IDF definition differs from the ATP III definition in that it requires evidence of central obesity in the diagnosis of metabolic syndrome. The rationale for this requirement is that central obesity is more strongly correlated with the other metabolic syndrome features than is any other parameter (Carr DB et al., 2004). Recommended cut-points for waist circumference vary for other ethnic groups. Cut-points for South Asians and Chinese are 90cm and 80cm for men and women, respectively. These cut-points have been recommended by the WHO (Alberti KGMM et al., 2006). Data from Asian Indians showed that the risks of having diabetes increased significantly at a waist circumference of 85cm in men and 80cm in women (Snehalatha C., 2003). While the methods of calculating cut-points and the recommended cut-points have

varied between studies, the current approach with ethnic group-specific cut-points is consistent with the WHO recommendations (Alberti KGM et al., 2006).

Patients with type 2- diabetes have an increased prevalence of lipid abnormalities, which contribute to higher rates of CHD. High triglyceride and low HDL cholesterol levels were significantly related to all coronary heart disease events and to coronary mortality in patients with type 2- diabetes (Lehto S, et al., 1997). Moreover, the prevalence of CHD in diabetic patients increases significantly with the addition of metabolic syndrome components (Alexander CM et al., 2003). According to Third National Health and Nutrition Examination Survey (NHANES III) data, people who did not have metabolic syndrome, had the lowest risk for cardiovascular disease (CVD) events, those with metabolic syndrome had an intermediate level of risk, and those with diabetes had the highest level of risk (Park YW et al., 2003).

2.3.3 Metabolic syndrome and diabetes

Diabetes mellitus is a metabolic disorder marked by the presence of hyperglycemia due to defective insulin secretion, defective insulin action or both. The chronic hyperglycemia of diabetes is associated with relatively specific long-term microvascular complications affecting the eyes, kidneys and nerves, as well as an increased risk for cardiovascular disease (CVD) (Goldenberg R., 2013). The characteristic signs and symptoms in diabetes are polydipsia, polyphagia, polyuria accompanied by fatigue, weight loss and weakness. The understanding of diabetes mellitus goes back to centuries before Christ. The diabetes was described to be associated with the passage of much urine. The ancient Indian physicians have left a great heritage, especially regarding the diagnosis and treatment of diabetes mellitus. Charak and Sushruta of ancient India (400- 600 B.C.) were well versed with many of the currently well known facts of the disease and named it ‘madhumeh’ (rain of honey) as they found that the urine tasted sweet and attracted ants. It is notable that even in that ancient period; diabetes insipidus was described as a separate entity.

The origin of current understanding of some aspects of diabetes can be traced to discoveries made in Europe between sixteenth and eighteenth centuries. A Swiss physician better known as Paracelsus (1494–1541), allowed the urine of patients with diabetes to evaporate and observed a white residue. He incorrectly thought that this residue consisted of salt and proceeded to attribute excessive thirst and urination in these patients to salt deposition in the kidneys (Medvei VC.,1993). The islet cells of the pancreas were discovered by Paul Langerhans, a young German medical student. In 1916, Sharpey-Shafer of Edinburgh

suggested that a single chemical was missing from the pancreas and proposed its name as "insulin." The term insulin originates from the word Insel, which means in German on an islet or island. The key breakthrough came from the Toronto University with the discovery of insulin in 1921. Banting FG and J.J.R. Macleod were awarded the Nobel Prize in 1923(Das AK, Shah S., 2011).

Ironically, although scientific advances have led to effective strategies for preventing diabetes, the pathway to cure has remained elusive. In fact, if one views diabetes from a public health and overall societal standpoint, little progress has been achieved toward conquering the disease during the past 200 years, and we are arguably worse off now than we were in 1812. Despite the fact that it is possible that some people had milder forms of hyperglycemia at that time, they largely escaped clinical detection. In 2012, the commonly encountered spectrum of diabetes is very different. Regardless of the fact that severe insulin deficiency still occurs, it now accounts for only about 10% of cases overall and can be readily treated with insulin. The vast majority of patients with diabetes is overweight and has a combination of insulin resistance and impaired insulin secretion. The prevalence of this form of diabetes has been increasing dramatically, particularly in the past three to four decades, resulting in a worldwide epidemic that has made diabetes one of the most common and most serious medical conditions humankind has had to face (Polonsky KS., 2012).

Diabetes and metabolic syndrome have been increasing in prevalence worldwide since at least the middle of the twentieth century. It seems that this trend is continuing apace into the new millennium. Diabetes is currently one of the most common non-communicable diseases globally. The core components of this condition include elevated plasma glucose as well as atherogenic dyslipidemia, vascular dysfunction and inflammation, a pro-thrombotic state and a pro-inflammatory state (Cameron AJ., 2007).

The studies provide considerable evidence about the link of metabolic syndrome and diabetes with an increased risk of cardiovascular diseases. Various estimates suggest that the number of deaths among people with diabetes is due to cardiovascular causes such as ischaemic heart disease and stroke (Dunstan, et al., 2002). Comparing with those without diabetes, the risk of coronary artery disease, stroke and peripheral arterial disease was two to four times higher in the diabetic population, more particularly among women. Similarly, risk for cardiovascular diseases was elevated in those with the metabolic syndrome (Brown CD. Et al.,2000). Many now see diabetes as having an equivalent risk for future cardiovascular disease events as that associated with previously diagnosed coronary heart disease (Sicree, RA et al., 2008). Several

epidemiological studies have shown that presence of the metabolic syndrome increases the probability of developing type 2-diabetes three to four fold, and that the risk increases with the number of elements of the syndrome present. This has been shown using several different definitions of the metabolic syndrome (Ford ES., 2005). Some very strong risk factors for development of type 2- diabetes do not appear in the definition, the most important being age and family history. These factors are among the strongest predictors of diabetes in diabetes risk (Lindstrom J et al., 2003). In 2002, David E published an analysis and reported that men who met the WHO definition of the metabolic syndrome in which adiposity was defined as waist-hip ratio > 0.90 or body mass index ≥ 30 kg/m^2 had a nearly nine fold greater likelihood of developing diabetes than men without the metabolic syndrome. Use of the NCEP definition of the metabolic syndrome detected only 61 percent of prevalent and 41 percent of incident diabetes, although specificity was quite high (David E.L et al., 2002).

2.3.4 Insulin resistance and metabolic syndrome

Insulin is a peptide hormone secreted by the β cells of the pancreatic islets of Langerhans and maintains normal blood glucose levels by facilitating cellular glucose uptake, regulating carbohydrate, lipid and protein metabolism and promoting cell division and growth. Insulin resistance is defined as the body's inability to respond to and use the insulin it produces. Insulin resistance may be linked to obesity, hypertension, and abnormal levels of fat in the blood. Classically this refers to impaired sensitivity to insulin mediated glucose disposal (Reaven G., 2004).

The first international definition for metabolic syndrome was given by the World Health Organization (WHO) in 1999.This was focusing on the glucose intolerance and insulin resistance. The European Group for the Study of Insulin Resistance (EGIR) in 1999 proposed a definition to be employed in non-diabetic patients only and put the insulin resistance in the centre, as well. In 2001 the National Cholesterol Education Program – Third Adult Treatment Panel (ATP III) set a new definition concentrating rather on the obesity and dyslipidemia. In 2002 the American Association of Clinical Endocrinology (AACE) presented a position statement, again stressing the role of insulin resistance in the syndrome. In 2005 the International Diabetes Federation (IDF) offered a consensus worldwide definition of the metabolic syndrome. This document shows the complexity of the problem of metabolic syndrome and divides the definition in four parts: part one contains definition for use of clinical practice, part two gives the additional metabolic criteria for research, part three gives details about the recommendations for treatment, and part four suggests some future works.

The definition for use in clinical practice is similar to the ATP III criteria focusing on the dominant obesity, but still keeping type 2- diabetes in the definition of the syndrome and strongly recommends the oral glucose tolerance test (OGTT). On the other hand, central obesity and insulin resistance were acknowledged equally significant causative factors. Thus, IDF definition could be characterized as a "mixture" of WHO and ATP III criteria. In the Indian context, the increasing tendency of its inhabitants to develop metabolic syndrome due to genetic predispositions and change of life-style due to the impact of westernization and rapid urbanization, has led the people to become more vulnerable to developing IR (Misra A et al., 2003).

According to Reaven, IR is the central pathophysiological feature of the cluster of metabolic abnormalities, which are associated with metabolic syndrome (Reaven., 1988). Globally, several studies have suggested that subjects with metabolic syndrome are more insulin resistant and are at greater risk for CVD than those without metabolic syndrome(Hsu CH et al., 2013). A study conducted in a representative sample of the PCOS (Polycystic ovary syndrome) population, the authors found significant differences between women with PCOS and non PCOS (control) (Jamil AV et al., 2015). Insulin resistance, the associated hyperinsulinemia and hyperglycemia, and adipocyte cytokines (adipokines) may also lead to vascular endothelial dysfunction, an abnormal lipid profile, hypertension, and vascular inflammation, all of which promote the development of atherosclerotic cardiovascular disease (ASCVD) (Lindsay et al., 2004; Koh KK ,et al 2005). A study from japan concluded that the BMI, Triglyceride, SBP were significantly correlated with insulin resistance (Chizumi Yamada, et al.2012). The path physiological perspective of insulin resistance demonstrates how this single defect, leads to a variety of pathological changes, resulting in increased risk for a constellation of clinical conditions like type 2- diabetes, cardiovascular disease, essential hypertension, polycystic ovarian syndrome, non-alcoholic fatty liver disease, gallstone disease, cancer (i.e., breast cancer) and sleep apnea (Reaven., 2004). The metabolic syndrome is the clinical epidemiological perspective which assembles a group of related metabolic risk factors and uses this grouping for the prediction of future cardiovascular events. In a Korean follow up study, it was reported that elevated fasting insulin emerged as an independent predictive factor for the development of subsequent metabolic syndrome over a 5 year period in a well characterized cohort of apparently healthy adults (Ki-Chul C Sung et al., 2011).

2.3.5 Insulin resistance and HOMA-IR

The concept of insulin resistance was the one that of the components to diagnose metabolic syndrome as per World Health Organization. NCEP ATP III and IDF have not chosen insulin measurement as the required component to diagnose the metabolic syndrome. But insulin resistance plays an important part in the development of metabolic syndrome (Ferrannini E et al., 1991).In recent years, there is a widespread scientific approach in this aspect as it has become apparent that insulin resistance occurs well in advance and develops early in the pathological process leading to diabetes (Boral A et al., 2011).The measurement of insulin resistance is helpful for detecting its presence, severity in those persons who has not yet developed abnormal glucose tolerance or diabetes. However, currently it is not the common practice to measure insulin resistance for clinical utility. It is to a great extent confined to research studies. The measurement of insulin resistance by hyperinsulinaemic euglycaemic clamp is considered as the reference technique and a gold standard method which is costly, cumbersome and invasive (Defronza RA t al., 1979). Hence many studies have adopted simple noninvasive alternative methods. They are HOMA (Homeostasis model assessment), which was reached by a mathematical calculation. (Matthews et al.,1985). Another one is QUICKI method. It is derived by using the inverse of the sum of the logarithms of the fasting insulin and fasting glucose. This is useful for measuring insulin sensitivity (IS), which is the inverse of insulin resistance (IR) (Katz A et al., 2000).

HOMA-IR is a mathematical equation used to estimate the insulin resistance from fasting glucose and insulin levels. The HOMA model is used to yield an estimate of insulin sensitivity and β-cell function from fasting plasma insulin and glucose concentrations (Matthews et al., 1985).It was postulated that elevated fasting glucose levels reflected a compensatory mechanism that maintained fasting insulin levels when there was a reduced insulin secretory capacity, and that fasting insulin levels were elevated in direct proportion to diminished insulin sensitivity. A mathematical feedback model based on these hypotheses was constructed to estimate the degrees of beta cell function and insulin sensitivity that would equate to the steady state plasma glucose and insulin levels observed in an individual (Turner RC et al., 1979) .The relationship between glucose and insulin in the basal state reflects the balance between hepatic glucose output and insulin secretion, which is maintained by a feedback loop between the liver and β-cells (Wallace TM et al., 2004). Hepatic IR can be estimated based on the effects of reduced insulin secretion capacity, leading to increased

hepatic glucose efflux. This increase in basal plasma glucose stimulates increased secretion of insulin within the portal vein, until glucose levels return to normal: thus the "feed-back" loop between the liver and β-cells. The basal plasma insulin levels necessary to maintain normal glucose levels are directly proportional to the grade of IR(Turner RC et al.,1979).

In a study from Mexico City, β-cell function and IR were assessed cross-sectionally using HOMA in 1,449 Mexicans with normal or impaired glucose tolerance (IGT). Subjects were followed up for 3.5 years in order to ascertain the incidence of diabetes and to examine any possible relationship with baseline β-cell function and IR. By 3.5 years, 4.4% of subjects with normal glucose tolerance (NGT) and 23.4% with IGT had progressed to diabetes. The development of diabetes was associated with higher HOMA-IR at baseline (Haffner SM et al.,1996) HOMA-IR has been utilized as a measure of IR in over 500 published articles (Wallace T.M. et al., 2004). Many cross-sectional studies have utilized HOMA-IR as a measurement of IR (Ausk KJ,et al., 2010; Healy GN et al., 2011) .Ausk et al. revealed a significant relationship between all-cause and CVD mortality across quartiles of HOMA-IR in subjects without diabetes. Healy et al. investigated the associations between increasing levels of sedentary time and cardio-metabolic risk in a sample of 4,757 adults (≥20 years) from the 2003-2006 NHANES. Results revealed a significant positive trend across quartiles of total sedentary time and HOMA-IR ($p < 0.001$).

2.3.6 Stress and Metabolic syndrome

Stress is defined as the process in which environmental demands exceed the adaptive capacity of an organism; resulting in psychological and biological changes that may place individuals at risk for disease (Cohen et al., 1995).Modern society has led to profound changes in lifestyle. Body weights are on the rise, diets are becoming less healthy and people are becoming increasingly sedentary, resulting in elevations of blood pressure and metabolic alterations that increase atherothrombotic risk.

In fact, obesity, insulin resistance, and diabetes are becoming public health problems of epidemic proportions (Seidell JC., 2000; Grundy SM., 2002). Also the modern society imposes demands on many that lead to difficulties in coping with their situations and more chronic stress. Stress activates the sympatho adrenal system and the hypothalamic-pituitary-adrenocortical (HPA) axis. Defense reactions involve catecholamine release, vagal withdrawal, cortisol secretion, and activation of the renin-angiotensin system (Folkow B., 1997). The functions of these mediators help the individual during short-term stress.

When stress is frequent, adaptation (coping) is lacking, the ability to shut off the stress response is deficient, or the responses to stress are inadequate and compensatory mechanisms are activated, the allostatic load may become overwhelming and the adaptive processes become maladaptive (McEwen BS., 1998). Brunner and coworkers performed a nested case-control study among working men aged 45 to 63 in the Whitehall II cohort to investigate associations between markers of neuro hormonal and inflammatory activity and presence (or previous presence) of the metabolic syndrome. The authors applied two definitions of the metabolic syndrome to their material, and obtained similar results for the two sets of criteria. Main findings are that cases had elevated urinary excretion of cortisol metabolites and normetanephrine (a marker for sympathetic activity), lower heart rate variability (HRV), and elevated levels of interleukin-6 (IL-6) and C-reactive protein (Brunner EJ et al., 2002).

Previous studies in the Whitehall II cohort have demonstrated an association between lower social position, which is linked to increased stress, and the metabolic syndrome. The authors conclude that neuroendocrine stress axes are activated in the metabolic syndrome and that chronic stress may be a causal factor in its development (Hjemdahl P., 2002). Stress plays a major role in the pathogenesis of metabolic syndrome (Chrousos GP., 2000). The probable explanation given to the link between Cortisol and metabolic syndrome was that patients with the metabolic syndrome present neuroendocrine abnormalities compatible with chronic stress. Psychosocial stress may contribute to the etiology of the metabolic syndrome through mechanisms involving both the sympathetic nervous system (SNS) and the hypothalamic-pituitary-adrenal (HPA) axis, which mediate the neuroendocrine response to stress. Stimulation of the HPA axis, with increased cortisol production, causes accumulation of intra-abdominal adipose tissue.

Catecholamines, released during sympathetic nervous system activation, and cortisol, resulting from hypothalamic-pituitary-adrenal axis activation, have the effects on glucose and lipid metabolism leading to impaired glucose tolerance and insulin resistance (Chrousos GP., 2000). The Perceived Stress Scale is the one among tools assessing the perceived stress. Perceived Stress Scale (PSS) was prepared by Sheldon Cohen. 1983 based on a theoretical perspective. The Perceived Stress Scale (PSS) items were intended to find the extent to which respondents find their lives unpredictable, uncontrollable, and overloaded. (Cohen S., 1983). PSS is not a diagnostic instrument, but it is proposed to make comparisons between individuals' perceived stress related to current, objective events. It assesses the degree to which situations in one's life are considered as stressful. The perceived stress is the one

which measures the level of stress based on stressful incidents, capability to face them at an individual level.

Earlier studies have indicated the relationship between stress and

- Hypertension and cardiovascular disease (Franke W et al., 2002).
- Low birth weight (when pregnant mothers experience stress) (Rondo et al., 2003).
- Respiratory illness (Sandberg et al., 2000).
- Immune response, including progression from HIV to AIDS (Burns V et al., 2002).
- Visceral obesity and metabolic syndrome (Peeke PM et al., 1995) as well as many other diseases.

Despite the fact that the definition of stress, the temporal characteristics of the stressors, and the scales used to assess the stressors varied greatly for the studies cited above, findings consistently showed a relationship between stress and illness. However, studies that focus more on chronic stress may capture a more complete picture of the relationship between stress and illness. Chronic stress, whether it is periodic or constant, is persistent over time. This persistence, perhaps through its cumulative effects, may produce more adverse health outcomes than acute stress events, which are discrete and time-limited in nature (Baum A, et al., 1999).

2.4 OTHER MANIFESTATIONS

Occupational health and metabolic syndrome

The prevalence of the Cardiometabolic syndrome (CMS) has gradually increased in all populations worldwide over the last 20 years making this one of the major global public health challenges (Alberti et al., 2006). The WHO reports 35% of cardiac disease related deaths in India by 2030 will be attributable to occupational stress. According to the WHO, 30% of suicide deaths in India are due to occupational stress. A recent study in South India indicated the prevalence of over 25% of job related stress in industrial workers, owing to severe working conditions (Mohan GM, et al., 2008).

Metabolic Syndrome and Related Disorders

Metabolic Syndrome and related Disorders provides an interdisciplinary forum to explore the path physiology, recognition, and treatment of the conditions associated with the evolving entity of metabolic syndrome. These include central obesity, endothelial dysfunction, insulin resistance, dyslipidemia, glucose intolerance, type 2- diabetes, prothrombotic and pro-inflammatory states, hypertension and cardiovascular disease. For a majority of those affected, poor nutritional status and insufficient physical activity are the root causes in the disease process.

The disorders like obstructive sleep apnea and polycystic ovary syndrome found to have the association of metabolic syndrome. It is pointed out that the subjects with obstructive sleep apnea along with metabolic syndrome have a higher incidence of cardiovascular morbidity and mortality. (Hamilton S et al ., 2004).It is reported that insulin resistance plays a pathogenic role in the development of polycystic ovary syndrome. (Apridonidze T, et al., 2005). Gluek et al. found that about 46.4% of women with polycystic ovary syndrome in the NHANES population had the metabolic syndrome. (Glueck et al., 2003).

Polycystic ovarian syndrome is clinically defined as oligomenorrhea associated with hyperandrogenism. Dialectologists are familiar with PCOS because of its frequent occurrence as a precursor to diabetes (Julie L, et al., 2003). Women with metabolic Syndrome (Syndrome X) are more prone to developing Polycystic Ovarian Syndrome (PCOS)**.** A root cause of PCOS is **i**nsulin resistance**.** This hormonal imbalance can cause a wide variety of symptoms, ranging from menstrual irregularity, infertility and excessive facial and body hair to acne, fatigue, mood swings and male pattern baldness. In PCOS, Insulin resistance is more than a biomarker of the disease, but is rather an active contributor

to its pathogenesis. Obesity aggravates the clinical presentation of PCOS (Kiddy DS et al., 1990). However, even in lean women, PCOS is often accompanied by abnormalities of insulin secretion and higher basal blood glucose than weight-matched controls (Kulshreshtha B et al., 2008).Elevated alanine aminotransferase (ALT) serum levels are a common finding in PCOS.(Vassilatou E et al., 2010). Moreover, in PCOS women with abnormal ALT, insulin sensitivity is markedly decreased ($P < 0.001$). One study found that 55% (48 / 88) of PCOS women had both hepatic steatosis and high HOMA-IR scores ($P = 0.033$) (Gambarin M et al., 2007)

The PCOS subjects with metabolic Syndrome are also at greater risk of developing Gestational Diabetes and have higher rates of liver, breast and colon cancer (Dey R et al., 2011).The riseof obesity. Sedentary lifestyle, food habits, cultural influences and also a genetic predisposition can cause dyslipidemia, hypertension, abdominal obesity and insulin resistance which are the main features of metabolic syndrome. Polycystic ovary syndrome (PCOS) is a condition directly associated with obesity, insulin resistance and metabolic syndrome, and the PCOS has its relationship and overlap with the metabolic syndrome. The relationship between the two syndromes is mutual: PCOS women have a higher prevalence of metabolic syndrome and also women with metabolic syndrome commonly present the reproductive/endocrine trait of PCOS. Prevention and treatment of metabolic syndrome and PCOS are similar for various aspects. It is necessary to treat excess adiposity and insulin resistance, with the overall goals of preventing cardiovascular disease and type 2- diabetes and improving reproductive failure in young women with PCOS. First of all, lifestyle changes, then pharmacological therapy, bariatric surgery and laparoscopic ovarian surgery represent the pillars for PCOS treatment. (Caserta D et al., 2014).

The development of nonalcoholic fatty liver disease (NAFLD) is strongly associated with the metabolic syndrome as reflected by the fact that approximately 90% of the patients with NAFLD have more than one feature of metabolic syndrome and about 33% have three or more criteria (Almeda-Valdes, P et al., 2009). (NAFLD) refers to a histological spectrum of liver damage from simple steatosis to advanced fibrosis and cirrhosis in individuals without relevant alcohol consumption (< 20 g/day) (Angulo P. 2002). In a cross sectional study it was observed that 51.4% of patients of NAFLD had metabolic syndrome and statistical significance was found in Aspartate amino transferase, diabetes mellitus and lipid profile (Gaharwar R., 2015).

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CHAPTER-III

MATERIAL AND METHODS

CHAPTER III
MATERIALS AND METHODS

3.1 STUDY DESIGN

A cross sectional study was conducted on three occupational groups, i.e. banking service, police service and public transport.

3.1.1 Sample size

The distribution of participants who participated in this study was as follow.

Sample size in the studied groups

Group	Subjects	Number of participants
I	Control subjects	110
II	Bank employees	97
III	Bus drivers	90
IV	Police constables	108
	Total	405

3.1.2 Sample size calculation

The estimated minimum sample size for this study according to the equation was 87, rounded to 90 per group. The prevalence rate of metabolic syndrome considered was 35 % [Reported 33.17%] (Thiruvagounder M et al., 2010).

The following sample size calculation formula was used (Daniel, 1999).

$$\boldsymbol{n = Z^2 \frac{P(1-P)}{d^2}}$$

Where *n* = sample size,

Z = Z statistic for a level of confidence

P= expected prevalence or proportion.

d = precision

Z statistic (*Z*): For the level of confidence of 95%, Z value is 1.96. In this study, the results are presented with 95% confidence intervals (CI).

P = Prevalence rate 35 %.

D = Precision, 0.1

$n = (1.96)^2 * 0.35 (1- 0.35) / (0.1)^2$

$= 3.8416* 0.35* 0.65 / 0.01$

n = 87.4 (Rounded to 90 in each group)

3.2 INCLUSION AND EXCLUSION CRITERIA

3.2.1 Inclusion criteria

- The employees working as the bus drivers, bank employees and police constables served as the study group.
- The subjects not belonging to these three selected occupations have served as the control group.
- Only male subjects of the age group 30-60 years were randomly included in the study. As the ladies staff was very less in transport service (bus drivers) and police department, only male participants were included in the overall study. As the retirement age is at 60 years, age restriction was up to 60 years only.

3.2.2 Exclusion criteria

- Subjects below 30 and above 60 years of age were excluded from the study.
- Female subjects were excluded from the study.
- Subjects with infective diseases, cancer (any type), tuberculosis and psychiatric complications were excluded from the study.

3.3 ETHICS

3.3.1Informed consent

Informed written consent was obtained from the participants in the study (Appendix I)

3.3.2 Institutional approval

An Institutional Ethical Clearance Certificate was obtained to conduct this study from Institutional Ethical committee of BLDE U Sri B.M. Patil medical college, hospital and research center. An informed consent was also obtained from all the subjects before their participation in the study.

3.3.3 Declaration of Helsinki

We followed the declaration of Helsinki during the entire study.

3.4 STUDY PROTOCOL

All the participants were interviewed using a structured questionnaire. Some questions were specific to the respective occupations. The information like age, history of diabetes, hypertension and Presence of symptoms related to diabetes if any, were collected. Further we collected information about family history of cardiovascular diseases and diabetes in first degree relatives: father, mother, sister, brother.

The details about smoking, alcohol consumption and tobacco chewing were also collected. All relevant information was compiled.

3.4.1 Anthropometry measurements

All the participants standing body height was measured. A digital scale with accuracy of ± 100 gm was used to measure body weight and was recorded in Kg. Waist circumference was measured in a horizontal plane, using a standard inelastic tape. Hip circumference was measured at the widest circumference around the buttocks. Body mass Index (BMI) was calculated by dividing weight in Kg by the square of height (in meter). Waist-to-hip-ratio (WHR) was calculated by dividing waist circumference by hip circumference. Blood pressure was measured on the right arm after 10 minutes rest in the sitting position by using sphygmomanometer. Systolic and diastolic were read to the nearest 5 mmHg.

Physical activity: In this study, independent self-report of activities of the individual were identified and recorded. The respondent was asked to remember the physical activities (PA) which he had done during the last one month, how many days in a week and time spent on each activity per week. The physical activities were categorized into low, average and regular physical activity respectively.

Habits: The information about smoking, alcohol consumption and tobacco chewing were also the independent self-reporting. The participants presently having these habits were only recorded.

3.4.2 Measurement of perceived stress and the scoring

To evaluate the occupational stress, the Perceived Stress Scale (PSS) was used which contains 14 items or questionnaires. Perceived Stress Scale is the most validated psychological tool for measuring the perception of stress. It measures the degree to which situations in one's life are considered as stressful. The perceived stress is the one which measures the level of stress based on stressful incidents, capability to face them at an individual level. The questions in the PSS-14 items were asked about the feelings and thoughts of the subjects during the past month. The PSS consisted of 14 items (Cohen S, et al. 1983), seven positive and seven negative. The negative element was intended to assess the lack of control and the negative affective reactions, while the positive element measured the degree of the ability to cope with the existing stressors. Each item was rated on a five-point scale from 0 = 'never' to 4 = 'very often', covering the previous month. The PSS scores were obtained by reversing the responses (e.g., 0 = 4, 1 = 3, 2 = 2, 3 = 1 and 4 = 0) to the four positively stated items (items 4, 5, 7, and 8) and then summing across all the scale items. The scores ranged from 0 to 56, with higher scores indicating higher levels of perceived stress and the lower scores indicating lower levels of stress (Cohen S, et al. 1983). The PSS-Questionnaire-14 item translated into the local language (Kannada) was provided in case if it was required. The PSS score was divided into two sections. The score, 28 being the operational cutoff value of the upper bound and were labeled as 'stressed' and the score less than 28 as 'non stressed' respectively. This cut off value was selected in accordance with a similar study from Pakistan (Shah M, et al 2010) and India (Brahmbhatt KR et al. 2013).

3.4.3 Sample collection

Venous blood and urine samples were collected from the participants with overnight fasting. The blood was collected in the test tube containing EDTA as an anticoagulant for the estimation of glycosylated HbA1c, the test tube containing sodium fluoride as an anticoagulant for blood glucose estimation and the plain test tube for the rest of the investigations. The urine was collected in a sterile container for the estimation of Microalbumin.

3.5 BIOCHEMICAL ANALYSIS

All the biochemical estimations were done in the clinical biochemistry laboratory of BLDE U Sri B.M. Patil medical college, hospital and research center, using Agappe auto analyzer, and Roche C 311 fully auto analyzer.

3.5.1 Estimation of glucose

Fasting blood glucose was estimated by using GOD-PAP method (Trinder P. 1969). (Agappe diagnostics).

Principle

Glucose is oxidized to gluconic acid and hydrogen peroxide in the presence of glucose oxidase. Hydrogen peroxide further reacts with phenol and 4-aminoantipyrine by the catalytic action of peroxidase to form a red coloured quinoneimine dye complex. Intensity of the colour formed is directly proportional to the amount of glucose present in the sample.

$$\text{Glucose} + O_2 + H_2O \xrightarrow{Glucose\ Oxidase} \text{Gluconic acid} + H_2O_2$$

$$2H_2O_2 + 4\text{Aminoantipyrine} + \text{Phenol} \xrightarrow{Peroxidase} \text{Red Quinoneimine dye} + 4\ H_2O$$

Reagents

Reagents	Concentration
Phosphate buffer (pH 7.40)	100 mmol/l
Phenol	10 mmol/l
Glucose oxidase	>1000 U/L
Peroxidase	>600 U/L
4-Aminoantipyrine	270 mmol/l

Procedure

	Blank	**Standard**	**Sample**
Working reagent	1000 µl	1000 µl	1000 µl
Glucose. Standard (100.0 (mg/ dl)	-	10 µl	-
Sample	-	-	10 µl

After mixing, the standard and sample with the reagent respectively, incubated at 37^0 C for ten minutes. The change in absorbance was measured at 505/ 630 nm against reagent blank.

The program was set in the instrument as follow

Mode of Reaction	End point
Slope of reaction	Increasing
Wavelength	505 / 630
Temperature	37^0 C
Standard concentration	100.0 mg / dl
Blank	Reagent blank
Linearity	500.0 mg / dl
Incubation Time	10 min
Sample volume	10 µl
Reagent volume	1000 µl
Cuvette	1 cm light path.

The calculation

Glucose conc. (mg/dl) = Absorbance of sample / Absorbance of Standard * 100

3.5.2 Estimation of Triglyceride

Serum triglyceride was estimated by glycerol phosphate – oxidase (GPO-PAP) method. (Bucolog , 1973).

Principle:

Triglycerides were enzymatically hydrolyzed by lipase to glycerol and free fatty acids. The glycerol was subsequently measured by a coupled enzymatic reaction system. The glycerol released was phosphorylated to glycerol-3-phosphate by glycerol kinase. The glycerol-3-phosphate was oxidized by glycerol phosphate oxidase to produce dihydroxyacetone phosphate and hydrogen peroxide. Peroxidase catalyze the reaction of hydrogen peroxide with 4-aminoantipyrine and 3,5-dichloro-2-hydroxybenzene sulfonate. The intensity of the chromogen (quinoneimine) formed was proportional to the triglycerides concentration in the sample.

$$\text{Triglyceride} + \ H_2O \xrightarrow{Lipase} \text{Glycerol} + \text{Fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{Glycerol\ Kinase} \text{Glycerol} - 3 - \text{p} + \text{ADP}$$

$$\text{Glycerol} - 3 - \text{p} + O_2 \xrightarrow{G-3-p-oxidase} \text{Dihydroxyacetone} - \text{p} + H_2O_2$$

$$2H_2O_2 + 4 - \text{Aminoantipyrine} + 4 - \text{Chlorophenol peroxidase Quinoneimine} + \ 4H_2O_2$$

Reagents

Reagents	Concentration
Glycerol kinase	> 1.5U/ml
4-chlorophenol	6 mmol/l
Magnesium chloride	5 mmol/l
Glycerol-3-phosphate Oxidase	>4 U/ml
Peroxidase	>0.8 U/ml
4-aminoantipyrine	0.75 mmol/l
ATP	0.9 mmol/l pH 7.0

Procedure

	Blank	**Standard**	**Sample**
Working reagent	1000 µl	1000 µl	1000 µl
Triglyceride Standard. (200.0 mg /dl)	-	10 µl	-
Sample	-	-	10 µl

After mixing, the standard and sample with the reagent respectively, incubated at 37^0 C for ten minutes. The change in absorbance was measured at 505/ 630 nm against reagent blank

The program was set in the instrument as follow

Mode of Reaction	End point
Slope of reaction	Increasing
Wavelength	505 / 630
Temperature	37^0 C
Standard concentration	200.0 mg / dl
Blank	Reagent blank
Linearity	1000.0 mg / dl
Incubation Time	10 min
Sample volume	10 µl

The calculation

Triglyceride conc (mg/dl) = Absorbance of sample / Absorbance of Standard * 200

3.5.3 Estimation of Total Cholesterol

Serum Cholesterol was measured by cholesterol oxidase- peroxidase (CHOD-PAP) enzymatic method (Allain GC et al., 1974).

Principle

Cholesterol esters were hydrolyzed by cholesterol esterase to cholesterol and free fatty acids. Free cholesterol was oxidized by cholesterol oxidase to cholest-4-en-3-one and hydrogen peroxidase. This hydrogen peroxidase combined with 4-aminoantipyrine to form chromophore (quinoneimine dye) which was measured at 505/ 630 nm.

$$\text{Cholesterol ester} + \text{H}_2\text{O} \xrightarrow{CHE} \text{Cholesterol} + \text{fattyacids}$$

$$\text{Cholesterol} + \text{O}_2 \xrightarrow{CHO} 4\,\text{cholesten} - 3 - \text{one} + \text{H}_2\text{O}_2$$

$$2\text{H}_2\text{O}_2 + \text{Phenol} + 4\text{Aminoantipyrine} \xrightarrow{POD} \text{Red quinone} + 4\text{H}_2\text{O}$$

CHE: Cholesterol esterase; CHO: Cholestrol Oxidase: POD: Peroxidase.

Reagents

Reagents	Concentration
Pipes buffer	50 mmol/l
Phenol	24 mmol/l
Sodium cholate	0.5 mmol/l
Cholesterol Esterase	≥ 180 U/L
Cholesterol Oxidase	≥ 200 U/L
Peroxidase	≥1000 U/L

Procedure

	Blank	**Standard**	**Sample**
Working reagent	1000 µl	1000 µl	1000 µl
Cholesterol Standard (200.0 mg /dl)	-	10 µl	-
Sample	-	-	10 µl

After mixing, the standard and sample with the reagent respectively, incubated at 37^0 C for ten minutes. The change in absorbance was measured at 505/ 630 nm against reagent blank.

The program was set in the instrument as follow.

Mode of Reaction	End point
Slope of reaction	Increasing
Wavelength	505 / 630
Temperature	37^0 C
Standard concentration	200.0 mg / dl
Blank	Reagent blank
Linearity	1000.0 mg / dl
Incubation Time	10 min
Sample volume	10 µl
Reagent volume	1000 µl
Cuvette	1 cm light path.

3.5.4 Estimation of HDL (High density lipoprotein). Cholesterol

Method

Enzymatic method (Direct method) (Castelli GT, et al 1977)

Principle

The reaction between cholesterol other than HDL and enzyme for cholesterol assay is suppressed by the electrostatic interaction between polyanions andcationic substances. Hydrogen peroxide is formed by the free cholesterol in HDL by cholesterol oxidase. Oxidative condensation of EMSE and 4-AA is caused by hydrogen peroxide in the presence of peroxidase. The absorbance of the resulting red-purple quinine is measured to obtain the cholesterol value in HDL.

$$\text{Lipoprotein other than HDL} \xrightarrow{Polyanions} \text{supress reaction with enzyme}$$

$$\text{Lipoprotein other than HDL} \xrightarrow[\text{Cationic substances}]{\text{Polyanions}} \text{Suppress reaction with enzyme.}$$

$$\text{HDL (Cholestrol esters)} + H_2O \xrightarrow{Chloesterol\ esterase} \text{(Free cholesterol)} + \text{Free fatty}$$

$$\text{HDL (Free cholesterol)} + O_2 \xrightarrow{Cholesterol\ oxidase} \text{Cholestenone} + H_2O_2$$

$$2H_2O_2 + 4 - \text{aminoantipyrine} + \text{EMSE} + H_3 + O \xrightarrow{Peroxidase} \text{Red purple quinine} + 5H_2O$$

Reagent

Reagent 1 (R1)	Concentration
Cholesterol oxidase	< 1500 U/L
Peroxidase	< 4 KU/L
N,N-bis(4-sulphobutyl)-m-toludine-disodium(DSBmT)	<1 mM
Ascorbic oxidase	<3000U/L
Reagent 2 (R2)	
Buffer, Cholesterol estarase	<2000 U/L
4-Aminoantipyrine (4-AAP)	<mM
Preservative	0.1%

After mixing, the standard and sample with the reagent respectively, incubated at 37^0 C for five minutes. The change in absorbance was measured at 578 nm against reagent blank.

Procedure

	Blank	**Calibrator**	**Sample**
Working reagent (R1)	450 µl	450 µl	450 µl
HDL-Cholesterol Calibrator	-	10 µl	-
Sample	-	-	10 µl
Mix and incubate for five minutes at 37^0 C			
Working reagent (R2)	150 µl	150 µl	150 µl

The program was set in the instrument as follow

Mode of Reaction	End point
Slope of reaction	Increasing
Wavelength	578 nm
Temperature	37^0 C
Calibrator	67.0 mg / dl
Blank	Reagent blank
Linearity	1000.0 mg / dl
Incubation Time	(5+5) min
Sample volume	10 µl
Reagent volume	450 µl + 150 µl
Cuvette	1 cm light path.

3.5.5 Estimation of Homocysteine (HCY)

Method

(Enzymatic method) (Sheshadri S et al. 2002)

Principle

Homocysteine test is based on a series of enzymatic reactions causing a decrease in absorbance value due to NADH oxidation to NAD. Homocysteine concentration in the sample is directly proportional to the quantity of NADH converted to NAD.

$$T(Hcy) \rightarrow (Hcy)free$$

$$\text{(Hcy) free} + \text{SAM} \xrightarrow{S-Methyltransferase} \text{Methionine} + \text{SAH}$$

$$SAH + NADH\ \ SAM \xrightarrow{Hydrolase+Adenosine\ deaminase} Inosine + NAD$$

$$SAH + NADH \quad SAM \xrightarrow[Glutamatedehydrogenase]{Hydrolase + Adenosine\ deaminase} Inosine + NAD$$

SAM: S-Adenosyl- methionine: SAH: S-Adenosylhomocysteine

Reagents

Reagent R1	Concentration
S- adenosylmethionine	0.1 mmol/l
NADH	0.2 mmol/l
TCEP	0.5 mmol/L
2- Oxoglutarate	5.0 mmol/l
Reagent R2	
Glutamate dehydrogenase	10 KU/l
SAH Hydrolase	3.0 KU/l
Adenosynedeaminase	5.0KU/l
HCY methyltransferase	5.0 KU/l

Procedure

	Blank	**Calibrator**	**Sample**
Working reagent (R1)	450 µl	450 µl	450 µl
Homocysteine Calibrator	-	25 µl	-
Sample	-	-	25 µl
Mix and incubate for five minutes at 37^0 C			
Working reagent (R2)	125 µl	125 µl	125 µl

After mixing, the standard and sample with the reagent respectively, incubated at 37^0 C for Five minutes. Read the absorbance (A1) at 340 nm and then read again after 2.5 minutes (A2) against reagent blank.

Calculation

$$\boldsymbol{HCY\ Concentration(\mu mol/l)} \frac{\boldsymbol{Absorbance of sample(A2-A1)}}{\boldsymbol{Absorbance of Calibrator(A2-A1)}} \text{x}\ \boldsymbol{Calibrator concn}$$

3.5.6 Estimation of Lipoprotein (a) [Lp (a)]

Method

Turbidimetric Immunoassay (Poulik M.D. et al)

Reagent

Reagent 1 (R1). Buffer solution	Glycine buffer. pH: 8.3
Reagent 2. (R2). Latex reagent	Latex particles covalently bound rabbit antibody

Principle

Latex particles coated with anti-human Lp (a) are agglutinated when mixed with samples containing Lp (a). The agglutination causes an absorbance change dependent upon Lp (a) content of the sample

Procedure

	Blank	**Calibrator**	**Sample**
Glycine Buffer (R1)	375 µl	375 µl	375 µl
Lp(a) Calibrator. (80.0 mg/dl)	-	10 µl	-
Sample	-	-	10 µl
Mix and incubate for five minutes at 37^0 C			
Lp(a) Latex reagent (R2)	65 µl	65 µl	65 µl

After mixing, the Calibrator and sample with the reagent respectively, incubated at 37 ^{0} C for Five minutes. Read the absorbance (A1) at 600 nm and then read again after 2.5 minutes (A2) against reagent blank.

Calculation

$$\boldsymbol{Lp(a)Concentration(mg/dl)} = \frac{\boldsymbol{Absorbanceofsample(A2-A1)}}{\boldsymbol{AbsorbanceofCalibrator(A2-A1)}} \text{x Calibrator Concn}$$

3.5.7 Estimation of Microalbumin (M.A)

Method

Turbidimetric Immunoassay (Winocour PH., 1992)

Principle

This Microalbumin test is based upon the reactions between albumin and latex-covalently bound antibodies against human albumin. The latex particles are agglutinated when mixed with samples containing Microalbumin. The agglutination causes an absorbance change. This is directly proportional to the amount of Microalbumin present in the sample.

Reagents

Diluent. (R1); Glycine buffer	100 mmol/l, pH: 10.0
Latex reagent. (R2)	Particles coated goat IgG with anti-human albumin, pH : 8.2

Procedure

	Blank	Calibrator	Sample
Glycine Buffer (R1)	375 µl	375 µl	375 µl
Calibrator. (41.0 mg/L)	-	10 µl	-
Sample	-	-	10 µl
Mix and incubate for five minutes at 37^0 C			
Latex reagent(R2)	40 µl	40 µl	40 µl

After mixing, the Calibrator and sample with the reagent respectively,incubated at 37^0 C for Five minutes. Read the absorbance (A1) at 600 nm and then read again after five minutes (A2) against reagent blank.

Calculation

$$\boldsymbol{M.A.\ Concentration\ (mg/L)} = \frac{\boldsymbol{Absorbance\ of\ sample\ (A2-A1)}}{\boldsymbol{Absorbance\ of\ Calibrator\ (A2-A1)}} \times \boldsymbol{calibrator\ concentration}$$

Microalbumin value is expressed as albumin to creatinine ratio. (Mg/gm of creatinine.)

Calculation

Albumin:creatinine ratio (ACR): It is ratio of urinary albumin (microalbumin) to urinary creatinine; it is expressed as milligram of microalbumin excreted per gram of urinary creatinine(ChavanVU.,et al).

$$\boldsymbol{ACR\ (mg/g)} = \frac{\boldsymbol{microalbumin\ (mg/dl)}}{\boldsymbol{Creatinine\ (mg/dl)}} \boldsymbol{x\ 1000}$$

3.5.8 Estimation of Glycosylated.HbA1C(Processed on Roche C311 Auto analyzer)

Method: "Tina-quant" (turbidimetric inhibition immunoassay***). (***Chang J ., et al 1998***)***

The concentration A1c (HbA1c) is measured as a percentage of a total hemoglobin in human whole blood (%HbA1c). Consequently, the hemoglobin contained in red blood cells is released by hemolysis of the sample. This method uses the Hemolyzing Reagent containing a detergent (tetradecyl trimethyl ammonium bromide - TTAB) to specifically lysate red blood cells. HbA1c and Hemoglobin levels in the sample are determined from the obtained hemolysate by two independent reactions. HbA1c During the first stage of the reaction: the HbA1c in the sample reacts with the anti-HbA1c specific antibody (Reagent A1) to form soluble antigen-antibody complexes. Then the polyhapten is added (Reagent A2). The polyhapten reacts with the specific antibody excess from the first reaction, producing insoluble immune complexes which can be measured turbidimetrically at 340 nm.

Reagents

Reagent A1: Monospecific antibodies anti-HbA1c in pH 6.2 buffer.

Reagent A2: Polyhapten-HbA1c in pH 6.2 buffer.

The program was set in the instrument as follow

Reaction type	End point
Wave length	340 nm
Temperature	37^0 C
Sample volume	10 µl
Reagent A1 volume	250 µl
Reagent A2 volume	50 µl
Reagent A1 incubation	300 seconds
Reagent A2 incubation	300 seconds

Calculation

%HbA1c = 91.5 x HbA1c (g/dl) / Hb(gm/dl) + 2.15

3.5.9 Estimation of C - reactive protein (CRP)

Method: Immuno turbidimetric Method (Price CP., et al 1987)

Principle

CRP test is based upon the reactions between C- reactive protein (CRP) and latex covalently bound antibodies against human CRP. CRP values are determined photometrically.

Reagents

Buffer (Reagent. 1) R1t	Phosphate buffer. 7.3
Latex reagent (Reagent) R2	Latent particles covalently bound with goat IgG

Procedure

	Blank	**Standard**	**Sample**
Working reagent	500 µl	500 µl	500 µl
CRP Standard(1.5 mg /dl)		10 µl	
Sample	-	-	10 µl

Mix well. Read absorbance A1 after 5 seconds, incubate for 120 seconds and read absorbance A2 at 550nm.

Calculations

CRP Conc in mg/dl = ΔA for Test / ΔA for Calibrator * Calibrator Conc.

3.5.10 Estimation of Serum Cortisol

Cortisol was estimated by Elisa method. (Burtis CA et al., 1994)

Principle

The principle of enzyme immunoassay test follows the competitive binding procedure. The competition occurs between an unlabeled antigen (present in standards, controls and samples) and an enzyme labeled antigen (Conjugate) for a limited number of antibody binding sites on microwell plate. The washing and decanting procedure remove unbound materials. After the washing step, the enzyme substrate is added. The enzymatic reaction is terminated by addition of stop solution. The absorbance is measured at 450/630 nm. The intensity of colour formed is inversely proportional to the concentration of cortisol in the sample.

Reagent

1) Cortisol Enzyme reagent.
2) Steroid conjugate buffer.
3) Cortisol biotin reagent.
4) Substrate A. TMB Substrate. (Tetramethylbenzidine)
5) Substrate B. H_2O_2 substrate. (Hydrogen peroxide)
6) Stop solution. 1 N HCL

Procedure

The ELISA for the quantitative analysis of Cortisol levels in serum. This test kit operates on the basis of competition between the hormone conjugate and the Cortisol in the sample for a limited number of binding sites on the antibody coated plate.

The sample or standard solution (25 µl) was first added to the microplate. Next, working cortisol enzyme reagent (50 µl) and Cortisol biotin reagent (50 µl) were added. The mixture was shaken and incubated at room temperature for one hour. During the incubation,

competition for binding sites was taken place. The plate was then washed removing all the unbound material. The bound hormone conjugate was detected by the addition of 100 µl of working substrate solution (TMB + H_2O_2).The mixture was incubated at room temperature for fifteen minutes. The Reaction was stopped by adding 50 µl stopping solution (1N HCL). Quantitative test results were obtained by measuring and comparing the absorbance reading of the wells of the samples against the standards with a micro plate reader at 450/630nm. The extent of color development was inversely proportional to the amount of Cortisol in the sample or standard.

3.5.11 Estimation of Serum Insulin

Method

Serum insulin level was measured by ELISA method. (David CR et al., 1996)

Principle

This ELISA is a solid phase two-site enzyme immunoassay. It is based on the direct sandwich technique in which two monoclonal antibodies are directed against separate antigenic determinants on the insulin molecule. During incubation insulin in the sample reacts with anti-insulin antibodies bound to the microtitration well and with peroxidase-conjugated anti-insulin antibodies. A washing step removes unbound enzyme labeled antibody. The bound conjugate is detected by reaction with 3, 3', 5, 5'-tetramethylbenzidine (TMB substrate). The reaction is stopped by adding acid to give a colorimetric endpoint that is read at a wavelength of 450 nm using a microplate reader.

Reagents

1) Microplate: microplate coated with a monoclonal anti-insulin antibody.
2) Enzyme Conjugate 11X: Peroxidase conjugated mouse monoclonal anti-insulin
3) Enzyme Conjugate Buffer.
4) Substrate TMB: 3, 3', 5, 5'-tetramethylbenzidine (TMB) colorless solution.
5) Stop Solution: 0.5M H_2SO_4.

Procedure

This ELISA is a solid phase two-site enzyme immunoassay. It is based on the direct sandwich technique in which two monoclonal antibodies were directed against separate antigenic

determinants on the insulin molecule. During incubation insulin in the sample reacts with anti-insulin antibodies bound to the micro titration well and with peroxidase-conjugated anti-insulin antibodies. A washing step removes unbound enzyme labeled antibody. The bound conjugate was detected by reaction with 3, 3', 5, 5'-tetramethylbenzidine (TMB, a frequently used chromogenic in ELISAs). The reaction was stopped by adding acid to give a colorimetric endpoint that was read spectrophotometrically at a wavelength of 450 nm using a microplate reader.

3.5.12 Estimation of Insulin resistance: By HOMA.

In each participant, the degree of insulin resistance was estimated by HOMA (Homeostasis model assessment), according to the formula, Fasting insulin (µmol/l) * Fasting blood glucose (mg/dl) / 405 described by Matthews. (Matthews, et al. 1985)

Diagnosis of the Cardiometabolic syndrome (Metabolic syndrome):

Diagnosis of the metabolic syndrome was based on Revised National Cholesterol Education Program, Adult training program III (NCEP ATP III). Out of these following criteria, the presence of any three components, qualified the subject as having metabolic syndrome.

1 Serum Triglyceride level ≥ 150.0 mg/dl.

2 Serum High density level Cholesterol < 40.0 mg/dl.

3 Fasting blood glucose level ≥ 100.0 mg/dl.

4 Waist circumference ≥ 90.0 cm.

5 Blood pressure 130/ 85 mmHg.

3.6 DATA ANALYSIS

All characteristics were summarized descriptively. For continuous variables, the summary statistics of number, arithmetic mean (referred to as mean), and standard deviation about the arithmetic mean (SD) were used. For categorical data, the number and percentage were used in the data summaries.

A one way ANOVA between subjects of four groups was conducted to compare the difference of anthropometric and Biochemical parameters between the study group and control group. Also we used the post hoc comparison using Tukey test to test indicated significant difference between the groups. Means and standard deviations were calculated for both controls and cases in each group i.e. (bus drivers, bank employees, and police constables).

We employed bivariate correlation analysis using Pearson's correlation coefficient (r) and intraclass correlation coefficient (which ever applicable) to test the strength and direction of relationships between the interval levels of variables.

A Chi-square (χ^2) test was employed to determine the significance of differences between groups for categorical data. For continuous data, the differences of the analysis variables were tested with the t-test. If the p-value is > 0.05, then the results will be considered to be not significant and if p-value is < 0.05 then results would be considered to be statistically significant. Data were analyzed using SPSS software version 16. Descriptive statistics were used to describe the sample and scale characteristics.

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CHAPTER-IV

RESULTS AND DISCUSSION

Chapter IV

RESULTS AND DISCUSSION

4.1.LEVEL OF STRESS IN DIFFERENT OCCUPATIONS

This chapter outlines the results of basic characteristics like Age, weight, height, waist circumference and hip circumference and biochemical analysis like fasting blood glucose, lipid profile, serum cortisol and serum insulin. Also data analyses of all the participated subjects (four groups) have been recorded in this chapter.

4.1.1. Cortisol and PSS Scorein different occupational groups

Table No 2: Basic Characteristics of individuals of different occupational groups

Characteristics	Group I (N=110)	Group II (N=97)	Group III (N=90)	Group IV (N=108)	Total (N=405)	p value
Age (Years)	46.52±0.73	47.28±0.71[b,c]	44.60±0.71[d,e]	42.17±1.00[f]	45.11±0.41	0.000
Height (Cm)	162.68±0.91	166.0±1.02[b,c]	165.32±0.95[d]	170.82±0.51[f]	166.23±0.45	0.000
Weight (Cm)	60.13±0.35	69.82±1.06[b,c]	67.56±1.08[d,e]	75.61±0.62[f]	68.23±0.49	0.000
BMI (Kg / m^2)	22.99±0.32	25.51±0.45[c]	24.79±0.39[e]	25.94±0.21[f]	24.78±0.18	0.000
Waist circumference(Cm)	74.83±0.69	91.71±0.99[b,c]	91.19±1.56[d,e]	81.33±1.00[f]	84.24±0.64	0.000
Hip circumference(Cm)	89.18±0.68	97.77±0.79[b,c]	96.36±1.41[d,e]	81.59±1.38[f]	90.81±0.64	0.000
W/H Ratio	0.84±0.01	0.94±0.01[b,c]	0.95±0.01d,[e]	1.01±0.01[f]	0.93±0.01	0.000
Systolic Blood Pressure (mmHg)	120.52±0.78	125.28±1.11[c]	124.56±1.06[e]	123.06±1.18	123.23±0.53	0.006
Diastolic BloodPressure (mmHg)	78.17±0.62	82.04±1.02[c]	80.91±0.86	79.96±0.92	80.19±0.43	0.011
Pulse Rate (bpm)	80.43±0.66	79.32±0.71[a,b]	84.10±0.73[e]	82.40±0.51	81.50±0.33	0.000

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]. Values are expressed as Mean±SEM; ANOVA followed by Post Hoc Tukey's multiple comparison tests was applied, superscript 'a, b, c, d, e, f' shows the significant difference between (1,2), (1,3), (1,4), (2,3), (2,4), (3,4) groups respectively.

Table 2 shows the descriptive statistics pertaining to baseline characteristics of control subjects, bank employees, bus drivers and police constables. The BMI shows the higher mean value in bank employees and police constables. As per the guidelines given for Asians the BMI above 25 is considered as overweight.

ANOVA result shows significant mean differences between/among four studied groups in the anthropometric parameters. Waist circumference of bank employees and bus drivers were significantly raised above the cut off value given for Asians.

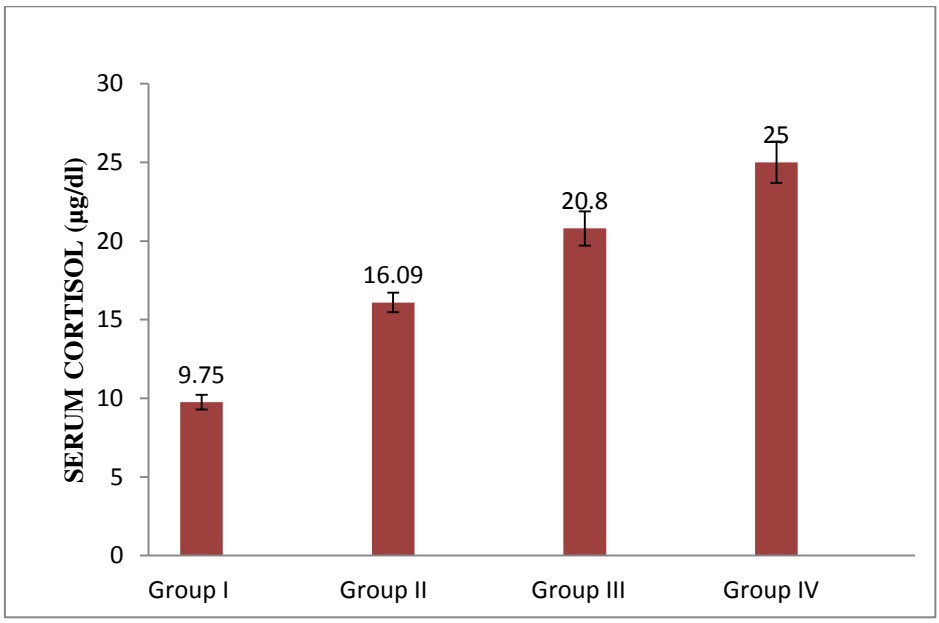

Fig No: 1 The Serum Cortisol level in different occupational groups

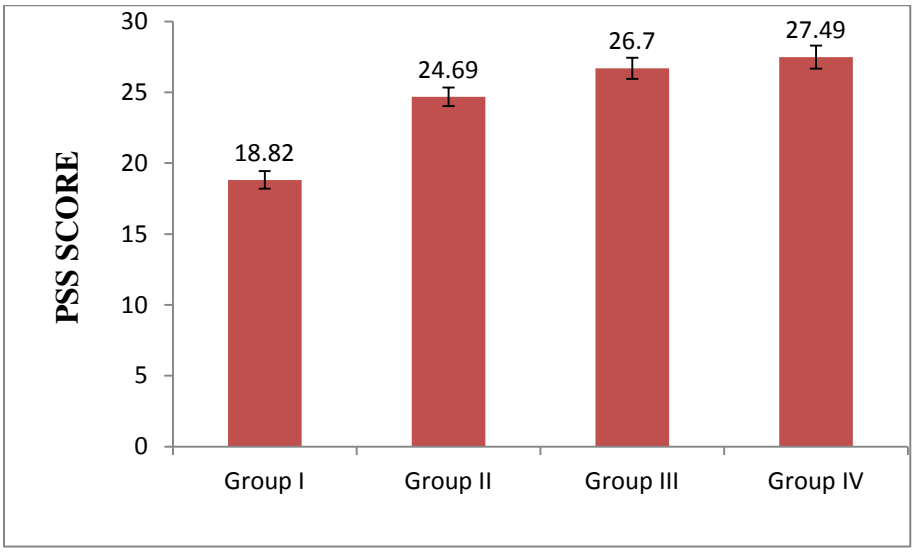

Fig No: 2 The PSS Score in different occupational groups

Figure 1 and 2 show the levels of serum cortisol and PSS in all the four studied groups respectively. Comparing to controls, the Cortisol and PSS levels were significantly raised in the three occupational groups. The figures show raised levels of serum cortisol and PSS in group IV subjects comparing to other three groups.

Table No 3: Details about habits and physical activities of participants of all four groups

Participants	Total no of individual group	Smoking	Alcohol consumption	Tobacco chewing	Physical activity		
					Low	Average	Regular
Group I	110	26.8	26.5	17.9	36.2	41.6	22.2
Group II	97	17.7	31.9	16.8	46.9	38.1	15
Group III	90	23.8	47.4	32.2	52.5	35.6	11.9
Group IV	108	47.9	65.8	31.1	46.6	30.1	23.3

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police Constables]

Table 3 shows the details about the habits like smoking, Alcohol consumption and tobacco chewing. Also the physical activity in different stages of all the four groups studied. The values are given in terms of percentage.

Table No 4: PSS and Cortisol level in stressed and non-stressed subgroups of Group I (Control group) participants

Characteristics	Stressed PSS > 29 (no=22)		Non Stressed PSS <29 (no=88)		p Value
	Mean	SD	Mean	SD	
Cortisol (µg/dl)	8.81	3.41	9.99	5.24	0.315
PSS	29.14	1.78	16.64	5.28	0.000***

* p<0.05, **p<0.01, ***p<0.001

Table No 5: PSS and Cortisol level in stressed and non-stressed subgroups of Group II (Bank employees) participants

Characteristics	**Stressed PSS > 29 (no=30)**		**Non Stressed PSS <29 (no=67)**		**p Value**
	Mean	SD	Mean	SD	
Cortisol (µg/dl)	17.91	4.86	15.27	6.43	0.048*
PSS	31.73	2.39	21.54	5.22	0.001**

* p<0.05, **p<0.01, ***p<0.001

TableNo 6: PSS and Cortisol level in stressed and non-stressed subgroups of Group III (bus drivers) participants

Characteristics	**Stressed PSS > 29 (no=36)**		**Non Stressed PSS <29 (no=54)**		**p Value**
	Mean	SD	Mean	SD	
Cortisol (µg/dl)	29.38	10.00	15.09	5.66	0.000***
PSS	33.42	5.31	22.22	3.92	0.000***

* p<0.05, **p<0.01, ***p<0.001

Table No 7: PSS and Cortisol level in stressed and non-stressed subgroups of Group IV (Police constables) participants

Characteristics	**Stressed PSS > 29 (no=41)**		**Non Stressed PSS <29 (no=67)**		**p Value**
	Mean	SD	Mean	SD	
Cortisol (µg/dl)	40.86	11.01	14.09	7.67	0.000***
PSS	35.78	6.00	22.42	5.13	0.000***

*p<0.05, **p<0.01, ***p<0.001

Table 4 to 7 show the stress level in the subjects of all the four groups studied. We found that there was a statistical significant difference in the levels of Cortisol and PSS between stressed and non-stressed subjects of II, III, and IV groups. In group I subjects the PSS level was significantly increased. The PSS value of 29 was used as the cut off value to identify the subjects as stressed and non-stressed.

Table No 8: Correlation between Cortisol and basic characteristics in stressed and non-Stressed subgroups of Group II (bank employees) subjects.

Variables	Stressed (n=30) r-value	p-value	Non stressed (n=67) r-value	p-value
Age	0.052	0.786	0.071	0.566
BMI	0.117	0.540	-0.159	0.199
Waist circumference	0.181	0.340	-0.071	0.566
Waist to hip ratio	-0.005	0.981	0.207	0.093
Systolic blood pressure	0.064	0.981	0.039	0.754
Diastolic blood pressure	0.167	0.379	-0.021	0.868

The Pearson correlation coefficient (r) between cortisol and basic characteristics in bank employees is presented in table 8. There was no significant correlation between serum cortisol and the basic characteristics in stressed and non-stressed groups.

Table No 9: Correlation between serum Cortisol and basic characteristics after adjusting for the habits (Overall) in Stressed and non-stressed subgroups of Group II (bank employees) subjects.

Variables	Overall (n=56) r-value	p-value	Stressed (n=18) r-value	p-value	Non-stressed (n=38) r-value	p-value
Age	0.024	0.860	-0.456	0.057	0.101	0.545
BMI	-0.076	0.577	-0.277	0.265	0.081	0.627
Waist circumference	-0.079	0.561	-0.401	0.099	0.093	0.577
Waist to hip ratio	0.035	0.796	-0.460	0.298	0.099	0.552
Systolic blood pressure	0.217	0.108	0.230	0.358	0.280	0.089
Diastolic blood pressure	0.087	0.525	0.112	0.659	0.160	0.337

Table 9 illustrates Correlation between serum Cortisol and basic parameters, after adjusting for the habits in stressed and Non-stressed bank employees. There was no significant correlation between serum cortisol and the basic characteristics in stressed and Non-stressed groups of bank employees.

Table No 10: Correlation between Cortisol and basic characteristics in stressed and non-Stressed groups of Group III (bus drivers) subjects

Variables	Stressed (n=36) r-value	p-value	Nonstressed (n=54) r-value	p-value
Age	-0.088	0.611	-0.388	0.004**
BMI	0.070	0.683	-0.009	0.947
Waist circumference	0.121	0.482	-0.204	0.139
Waist to hip ratio	0.240	0.159	-0.191	0.167
Systolic blood pressure	-0.051	0.767	-0.088	0.525
Diastolic blood pressure	0.046	0.788	-0.336	0.013*

* p<0.05, **p<0.01, ***p<0.001

Table 10 shows correlation coefficient (r) between serum cortisol and basic characteristics in stressed and Non-stressed group of bus drivers. It was observed that age, diastolic blood pressure was negatively correlated with serum cortisol in non-stressed group of bus drivers. The values were statistically significant.

Table No 11: Correlation between serum Cortisol and basic characteristics after adjusting for the habits (Overall) in Stressed and non-stressed subgroups of Group III (bus drivers) participants

Variables	Overall (n=51) r-value	p-value	Stressed (n= 21) 'r' 'value	p-value	Non stressed (n=30) r-value	p-value
Age	-0.305	0.030*	-0.354	0.115	-0.396	0.030*
BMI	-0.072	0.616	-0.040	0.864	-0.025	0.896
Waist circumference	0.086	0.549	0.181	0.433	-0.353	0.056
Waist to hip ratio	0.152	0.288	0.295	0.194	-0.377	0.040*
Systolic blood pressure	-0.260	0.066	-0.269	0.238	-0.132	0.488
Diastolic blood pressure	-0.261	0.065	-0.143	0.535	-0.324	0.080

* p<0.05, **p<0.01, ***p<0.001

Table No 12: Correlation between Cortisol and basic characteristics in Stressed and non-Stressed subgroupsof Group IV (police constables) participants

Variables	Stressed (n=41) 'r 'value	p-value	Non-stressed (n=67) r-value	p-value
Age (Years)	0.038	0.406	0.180	0.073
BMI (Kg / m^2)	-0.210	0.188	0.055	0.658
Waist circumference	-0.104	0.519	-0.147	0.235
Waist to hip ratio	0.408	0.004	0.090	0.233
Systolic blood pressure	-0.005	0.488	0.152	0.109
Diastolic blood pressure	0.068	0.335	0.085	0.246

Table 11 depicts about Correlation between serum Cortisol and basic characteristics, after adjusting for the habits. It is observed that serum cortisol was negatively correlated with age (r=-305, p< 0.05, r= - 0.396, p < 0.05) in overall and Non-stressed group of bus drivers respectively. Also it was observed that serum cortisol was negatively correlated with waist to hip ratio (r= - 0.377, p= 0.040) in non-stressed group.

Table 12 shows correlation coefficient (r) between serum cortisol and basic characteristics in stressed and non-stressed group of police constables. No significant association between serum cortisol and basic characteristics was found between stressed and non-stressed groups.

Table No 13: Correlation between Cortisol and other variables excluding the habits in (Overall) in Stressed and non-stressed subgroups of Group IV (police constables) subjects.

Variables	Overall (n=76) r-value	p-value	Stressed (n= 29) 'r 'value	p-value	Non-stressed (n=47) r-value	p-value
Age (Years)	-0.016	0.455	0.206	0.142	0.095	0.262
BMI (Kg / m^2)	-0.192	0.096	-0.173	0.371	-0.142	0.343
Waist circumference	-0.165	0.156	-0.089	0.647	0.154	0.301
Waist to hip ratio	0.395	0.000***	0.478	0.004**	-0.075	0.307
Systolic blood pressure	-0.082	0.240	-0.136	0.241	0.075	0.308
Diastolic blood pressure	-0.051	0.331	-0.003	0.493	0.038	0.401

* p<0.05, **p<0.01, ***p<0.001

Table 13 indicates about the Correlation between serum cortisol and variables in the groups 'stressed' and 'Non-stressed' after excluding the subjects with the habits of smoking, tobacco chewing and alcohol consumption. It is observed that serum cortisol was significantly correlated with waist to hip ratio (r= 0.478, p<0.01) in the stressed group as well as in overall group (r = 0.395, p< 0.001).

4.1.2. Glucose and insulin indices: its concentration and relationship with stress in different occupational groups

Table No 14: Glucose and Insulin level in differentoccupationalGroups

Characteristics	Group I (N=110)	Group II (N=97)	Group III (N=90)	Group IV (N=108)	Total (N=405)	p value
Fasting Blood glucose (mg/dl)	97.96±1.08	117.90±3.95^{c}	107.36±5.37	115.09±4.42^{f}	109.39±1.98	0.001
Glycosylated HbA1c(%)	4.89±0.06	6.04±0.13^{c}	5.74±0.12^{e}	5.88±0.12^{f}	5.62±0.06	0.000
Serum Insulin (µIU/ml)	5.87±0.58	16.07±1.16^{a,c}	11.59±0.91^{e}	14.62±1.19^{f}	11.92±0.53	0.000
HOMA-IR-IR	1.52±0.19	4.79±0.39^{c}	3.65±0.40^{e}	4.72±0.48^{f}	3.63±0.20	0.000
Microalbumin mg/ gmof Creat.	15.78±0.59	21.01±2.04^{b,c}	22.17±1.42^{d,e}	28.87±1.25^{f}	21.94±0.73	0.000
Cortisol (µg/dl)	9.75±0.47	16.09±0.62^{a,b,c}	20.80±1.09^{d,e}	25.00±1.31^{f}	17.79±0.55	0.000
PSS	18.82±0.62	24.69±0.66^{b,c}	26.70±0.75^{e}	27.49±0.82^{f}	24.29±0.40	0.000

[Values are expressed as mean±SEM. ANOVA followed by Post Hoc Tukey's multiple comparison test. Superscript 'a, b, c, d, e, f' express the significant difference between (1,2), (1,3), (1,4), (2,3), (2,4), (3,4) groups respectively. [Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

Table 14 shows the values of fasting blood glucose, Glycosylated HbA1c, Insulin, Cortisol and other biochemical parameters. There was a significant rise in fasting blood glucose level in the bank employees and police constables comparing to controls.

A one way between subjects of four groups, ANOVA was conducted to compare the Biochemical parameters. Almost all the mean indicators were significantly different among all the groups. Also with the post hoc comparison using Tukey, the test indicated significant difference between the groups with the 'p'value is less than 0.001.

Table No 15: Fasting blood glucose and Insulin indices in stressed and Non-stressed subgroups of Group I (Control group) participants

Variables	Stressed PSS > 29 (no=22)		Non -Stressed PSS <29 (no=88)		p Value
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	97.73	10.8	98.02	11.46	0.913
Glycosylated HbA1c (%)	4.79	0.62	4.92	0.695	0.447
Insulin (µIU/ml)	6.07	7.04	6.10	6.63	0.985
HOMA-IR IR	1.59	2.42	1.55	1.95	0.944

* p<0.05, **p<0.01, ***p<0.001

Table 15 shows the levels of fasting blood glucose and other variables in stressed and Non-stressed groups of control subjects. No significant difference of levels was noticed between stressed and Non-stressed group of control subjects.

Table No 16: Fasting blood glucose and Insulin indices in stressed and Non-stressedsubgroups of Group II (Bank employees) participants

Variables	Stressed PSS > 29 (no=30)		Non Stressed PSS <29 (no=67)		p Value
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	116.80	32.80	118.39	41.58	0.854
Glycosylated HbA1c (%)	5.87	0.92	6.11	1.37	0.375
Insulin (µIU/ml)	20.51	15.14	14.74	10.09	0.029*
HOMA-IR IR	5.91	4.22	4.45	3.75	0.091

Table 16 shows a significant difference in the serum insulin levels between stressed and Non-stressed group II subjects (p<0.05).

Table No 17: Fasting blood glucose and Insulin indices in stressed and non-stressed subgroups of in Group III (Bus drivers) participants

Variables	Stressed PSS > 29 (no=36)		Non Stressed PSS <29 (no=54)		p Value
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	121.25	66.66	98.09	34.71	0.034*
Glycosylated HbA1c (%)	6.01	1.28	5.57	0.94	0.060
Insulin (µIU/ml)	13.95	9.81	10.02	7.36	0.033*
HOMA-IR IR	4.84	4.38	2.85	3.06	0.013*

* p<0.05, **p<0.01, ***p<0.001

The table 17 shows a significant rise in the fasting blood glucose from mean 98.09 to 121.25 mg/dl (p<0.05), serum insulin from 10.02 to 13.95 µIU/ml (p<0.05) and HOMA-IR 2.85 to 4.84 in the stressed group III subjects.

Table No 18: Fasting blood glucose and other variables in subjects with stressed and Non-stressed in Group IV (Police constables)

Variables	Stressed PSS > 29 (no=41)		Non Stressed PSS <29 (no=67)		p Value
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	125.12	45.98	105.94	28.52	0.000**
Glycosylated HbA1c (%)	6.02	1.17	5.48	0.60	0.000***
Insulin (µIU/ml)	16.23	13.44	13.63	11.60	0.288
HOMA-IR-IR	5.92	5.78	3.98	4.30	0.049*

* p<0.05, **p<0.01, ***p<0.001

Table 18 indicates the fasting blood glucose and other variables in stressed and Non-stressed group IV subjects. There was a significant rise in fasting blood glucose (p< 0.001),GlycosylatedHbA1C (p<0.001) and HOMA-IR (p< 0.05) levels of stressed subjects.

Table No 19: Correlation between Cortisol and glucose indices in stressed and non-Stressedsubgroups of (Group II) bank employees.

Variables	Stressed (n=30) r-value	p-value	Non-stressed (n=67) r-value	p-value
Fasting blood glucose	0.470	0.009**	0.033	0.788
Glycosylated. HbA1c	0.410	0.024*	-0.058	0.641
Serum Insulin	0.034	0.857	-0.192	0.120
HOMA-IR-IR	0.130	0.493	-0.162	0.190
PSS	0.478	0.008**	0.041	0.740

* p<0.05, **p<0.01, ***p<0.001

The Correlation between cortisol and other studied biochemical parameters in bank employees is presented in table 19. In the stressed group serum cortisol level was significantly correlated with fasting blood glucose (r= 0.470, p<0.01), Glycosylated HbA1c (r=0.410, p<0.05), PSS (r= 0.478, p<0.01). There was no significant correlation between serum cortisol and other variables in subjects grouped as non-stressed.

Table No 20: Correlation between serum Cortisol and glucose indices after adjusting for the habits; (Overall) in Stressed and non-stressed subgroups of Group II (bank employee) participants.

Variables	Overall (n=56) r-value	p-value	Stressed (n=18) r-value	p-value	Non stressed (n=38) r-value	p-value
Fasting blood glucose	0.248	0.065	0.143	0.570	0.295	0.072
Glycosylated HbA1c	0.154	0.257	0.187	0.456	0.185	0.265
Serum Insulin	0.459	0.000***	0.218	0.385	0.595	0.000***
HOMA-IR	0.544	0.000***	0.263	0.293	0.660	0.000***
PSS	0.188	0.166	-0.085	0.785	0.091	0.585

* p<0.05, **p<0.01, ***p<0.001

Table no 20 Illustrates the correlation coefficient (r) between serum cortisol and biochemical parameters in the groups ‘stressed’ and ‘Non-stressed’ after excluding the subjects with the habits of smoking, tobacco chewing and alcohol consumption. Overall it was found that there was a statistically significant positive correlation between serum cortisol and serum

insulin, (r=0.459, p<0.000)HOMA-IR (r= 0.544, p < 0.000). The same positive correlation between serum cortisol and insulin (r= 0.595, p < 0.000), HOMA-IR(r= 0.660, p< 0.001) was observed in Non-stressed subjects once the subjects with habits were excluded. It suggests the effect of habits on the positive association between serum cortisol and fasting blood glucose, Glycosylated HbA1c and PSS.

Table No 21: Correlation between serum Cortisol and glucose indices after adjusting for the habits; (Overall) in Stressed and non-stressed subgroups of Group III (Bus drivers) participants.

Variables	Stressed (n=36) r-value	p-value	Non-stressed (n=54) r-value	p-value
Fasting blood glucose	0.034	0.845	0.259	0.059
Glycosylated HbA1c	0.040	0.816	0.230	0.094
Serum Insulin	0.030	0.864	-0.024	0.861
HOMA-IR	0.048	0.781	0.112	0.422
PSS	0.381	0.022*	0.180	0.194

* p<0.05, **p<0.01, ***p<0.001

The table no 21 shows the Correlation between cortisol and biochemicalparameters in bus drivers. It was observed that serum cortisol was positively correlated with PSS (r=0.381, p< 0.05) in stressed group of bus drivers.

Table No 22: Correlation between serum Cortisol and glucose indices, after adjusting for the habits; (Overall), Stressed and non-stressed subgroups of Group III (bus drivers)

Variables	Overall (n=51) r-value	p-value	Stressed (n= 21) r-value	p-value	Non-stressed (n=30) r-value	p-value
Fasting blood glucose	0.134	0.347	-0.075	0.747	-0.121	0.525
Glycosylated HbA1c	0.215	0.131	-0.058	0.802	0.121	0.524
Serum Insulin	-0.081	0.572	-0.184	0.424	-0.224	0.282
HOMA-IR	0.041	0.777	-0.143	0.536	-0.179	0.344
PSS	0.579	0.000***	0.060	0.798	0.127	0.505

Table 22 shows the correlation coefficient between serum cortisol and biochemical parameters in the groups ‘stressed’ and ‘Non-stressed’ of bus drivers after excluding the subjects with the habits of smoking, tobacco chewing and alcohol consumption.

We have noticed a positive correlation of PSS with cortisol which were statistically significant (r=0.579, p< 0.001) in overall subjects. However after excluding the subjects with habits, the same variables have not shown statistical significant correlation with cortisol in both stressed and Non-stressed groups.

Table No 23: Correlation between Cortisol and glucose indices in Stressed and non-Stressed subgroups of Group IV (police constables) participants

Variables	Stressed (n=41) 'r' value	p-value	Non-stressed (n=67) 'r' value	p-value
Fasting blood glucose	0.357	0.011*	0.013	0.459
Glycosylated HbA1c	0.424	0.003**	0.048	0.349
Serum Insulin	-0.183	0.253	-0.098	0.429
HOMA-IR	-0.350	0.025	-0.075	0.546
PSS	0.479	0.001**	0.191	0.061

* p<0.05, **p<0.01, ***p<0.001

The table 23 shows the Pearson correlation coefficient (r) between cortisol and other studied biochemical parameters in police constables. It was observed that there was a positive correlation between Cortisol and fasting blood glucose (r= 0.357, p< 0.05), Glycosylated HbA1c (r=0.424, p< 0.01) and PSS (r=0.479, p< 0.01) in the stressed group. No any such correlation was found in Non-stressed group of police constables.

Table No 24: Correlation between serum Cortisol and glucose indices, after adjusting for the habits; (Overall), Stressed and non-stressed subgroups of Group IV (police constables).

Variables	Overall (n=76) r-value	p-value	Stressed (n= 29) r-value	p-value	Non stressed (n=47) r-value	p-value
Fasting blood glucose	0.383	0.000***	0.343	0.034*	-0.021	0.443
Glycosylated HbA1c	0.491	0.000***	0.407	0.014*	-0.033	0.414
Serum Insulin	-0.095	0.413	-0.243	0.204	-0.031	0.836
HOMA-IR	-0.080	0.492	-0.346	0.066	-0.004	0.981
PSS	0.711	0.000***	0.500	0.003**	0.110	0.232

* p<0.05, **p<0.01, ***p<0.001

Table 24 indicates about the Pearson correlation coefficient (r) between serum cortisol and biochemical parameters, PSS in the groups, overall, 'stressed' and 'Non-stressed' after excluding the subjects with the habits of smoking, tobacco chewing and alcohol consumption. Overall we found that there was a positive correlation between Cortisol and, fasting blood glucose (r =0.383, p<0.001), Glycosylated HbA1c (r= 0.491 p< 0.001) , and PSS (r = 0.711, p< 0.001). The correlation between serum cortisol and PSS (r= 0.500, p< 0.01) was statistically significant even after the subjects with habits were excluded.

4.1.3. Lipid profile: its concentration and relationship with stress in different occupational groups

Table No 25: Lipid profile in different occupational Groups

Characteristics	Group I (N=110)	Group II (N=97)	Group III (N=90)	Group IV (N=108)	Total (N=405)	p value
Triglyceride (mg/dl)	117.94±2.68	137.52±9.24	148.43±8.81[e]	151.58±9.31[f]	138.38±3.97	0.009
Total Cholesterol (mg/dl)	178.40±2.62	182.05±3.10[b]	180.38±3.30[d]	200.74±5.41[f]	185.67±1.96	0.000
HDL. Cholesterol (mg/dl)	47.38±0.60	39.78±0.89[b,c]	42.22±1.05[d,e]	36.31±0.62[f]	41.46±0.44	0.000
LDL Cholesterol (mg/dl)	107.43±2.68	114.76±3.13[b]	108.47±3.65[d]	134.11±4.99[f]	116.53±1.95	0.000
VLDL Cholesterol (mg/dl)	23.59±0.54	27.50±1.85	29.69±1.76[e]	30.32±1.86[f]	27.68±0.79	0.009
Lipoprotein (a) (mg/dl)	6.40±0.22	14.69±1.23[c]	15.10±0.72[e]	15.59±1.00[f]	12.77±0.47	0.000

[Values are expressed as mean±SEM. ANOVA followed by Post Hoc Tukey's multiple comparison tests. Superscript 'a, b, c, d, e, f' express the significant difference between (1,2), (1,3), (1,4), (2,3), (2,4), (3,4) groups respectively.[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

Table 25 shows the values of Lipid profile and LP (a). The patients presented higher values of all Biochemical parameters in cases except HDL Cholesterol which was lowered in cases compared to controls.

Table No 26: Lipid profile in stressed and non-stressed subgroups of Group I (Control group) participants

Characteristics	**Stress PSS > 29 (n=22)**		**Non Stressed PSS <29 (no=88)**		**p-Value**
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	122.10	25.38	116.90	28.83	0.441
Total Cholesterol (mg/dl)	174.73	25.85	179.32	27.93	0.485
HDL Cholesterol (mg/dl)	46.27	4.9	47,66	6.55	0.355
LDL Cholesterol (mg/dl)	104.04	26.89	108.28	28.47	0.528
VLDL Cholesterol (mg/dl)	24.42	5.07	23.38	5.76	0.441

Table No 27: Lipid profile in subjects with stressed and Non-stressed in Group II (Bank employees)

Characteristics	**Stress PSS > 29 (n=30)**		**Non Stressed PSS <29 (no=67)**		**p-Value**
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	120.67	91.23	145.06	90.59	0.224
Total Cholesterol (mg/dl)	179.63	26.47	183.13	32.34	0.605
HDL Cholesterol(mg/dl)	39.53	8.35	39.90	8.94	0.851
LDL Cholesterol (mg/dl)	115.97	28.42	114.23	32.05	0.799
VLDLCholesterol(mg/dl)	24.11	18.25	29.01	18.12	0.224

Table No 28: Lipid profile in subjects with stressed and Non-stressed in Group III (Bus drivers)

Characteristics	Stress PSS > 29 (n=36)		Non Stressed PSS <29 (no=54)		p-Value
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	164.58	85.35	137.67	81.36	0.135
Total Cholesterol (mg/dl)	183.31	37.01	178.43	27.08	0.472
HDL Cholesterol (mg/dl)	42.97	11.65	41.72	8.68	0.562
LDL Cholesterol (mg/dl)	107.42	39.23	109.17	31.65	0.816
VLDL Cholesterol (mg/dl)	32.92	17.07	27.03	16.27	0.135

Table No 29: Lipid profile in subjects with stressed and non-stressed in Group IV(Police constables)

Characteristics	Stress PSS > 29 (n=41)		Non Stressed PSS <29 (no=67)		p-Value
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	163.73	138.90	141.06	61.30	0.246
Total Cholesterol (mg/dl)	209.66	67.03	193.16	52.94	0.159
HDL Cholesterol (mg/dl)	36.54	8.04	36.18	5.21	0.779
LDL Cholesterol (mg/dl)	140.38	57.69	128.77	52.13	0.283
VLDL Cholesterol (mg/dl)	32.75	27.78	28.21	12.25	0.246

Table 26 to 29 shows the levels of lipid profile in Group I, II, III and IV respectively. There is no significant difference in levels of lipid profile between stressed and Non-stressed subjects of all the four groups.

Table No 30: Correlation between Cortisol and lipids, PSS in stressedand Non-Stressed groups of Group II (bank employees) participants.

Variables	Stressed (n=30) r-value	p-value	Non-stressed (n=67) r-value	p-value
Triglyceride	0.099	0.604	-0.021	0.863
HDL Cholesterol	-0.191	0.312	0.016	0.895
PSS	0.478	0.008**	0.041	0.740

* p<0.05, **p<0.01, ***p<0.001

Table No 31: Correlation between Cortisol and lipids, PSS in Stress and non-Stress as per PSS reference cut off value in Group III(bus drivers) participants

Variables	Stressed (n=36) r-value	p-value	Non-stressed (n=54) r-value	p-value
Triglyceride	0.158	0.357	0.120	0.387
HDL Cholesterol	0.032	0.854	-0.053	0.704
PSS	0.381	0.022*	0.180	0.194

* p<0.05, **p<0.01, ***p<0.001

The table no 30 and 31 show the Correlation between cortisol and lipids, PSS in bank employees and bus drivers respectively.It was observed that serum cortisol was positively correlated with PSS (0.478, p<0.05), (r=0.381, p< 0.05) in stressed group of bank employees and bus drivers respectively.

Table No 32: Correlation between Cortisol and lipid profile, PSS in Stress and non-Stress as per PSS reference cut off value in Group IV (police constables) subjects.

Variables	Stressed (n=41) r value	p-value	Non-stressed (n=67) r-value	p-value
Triglyceride	0.097	0.274	0.085	0.247
HDL Cholesterol	0.018	0.454	0.070	0.287
PSS	0.479	0.001**	0.191	0.061

* p<0.05, **p<0.01, ***p<0.001

The table 32 shows the Pearson correlation coefficient (r) between cortisol and lipids, PSS in police constables. It was observed that there was a positive correlation between Cortisol and PSS (r=0.479, p< 0.01) . No any such correlation was found in non stressed group of police constables.

4.1.4. Lipoprotein (a), Homocysteine & c-reactive protein: its concentration and relationship with stress in different occupational groups

Table No 33: Lipoprotein (a),Homocysteine and C - reactive protein level in different occupational Groups

Characteristics	Group I (N=110)	Group II (N=97)	Group III (N=90)	Group IV (N=108)	Total (N=405)	p value
Lipoprotein (a) mg/ dl	6.40±0.22	14.69±1.23[c]	15.10±0.72[e]	15.59±1.00[f]	12.77±0.47	0.000
Homocysteine µmol/l	5.43±0.22	11.92±0.74[b,c]	10.52±0.54[e]	9.96±0.55[f]	9.32±0.29	0.000
C-Reactive Protein mg/ dl	0.38±0.03	0.96±0.06[c]	0.80±0.09[e]	0.93±0.10[f]	0.76±0.04	0.000

[Values are expressed as mean±SEM. ANOVA followed by Post Hoc Tukey's multiple comparison test. Superscript 'a, b, c, d, e, f' express the significant difference between (1,2), (1,3), (1,4), (2,3), (2,4), (3,4) groups respectively.[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables].

Table 33 shows the values of Homocysteine, C-reactive protein, Lipoprotein (a).They were significantly raised in occupational groups comparing to control subjects.

Table No 34: Lipoprotein (a),Homocysteine & C-Reactive protein in stressed and non-stressed subgroups of Group I (Control group) participants

Characteristics	Stress PSS > 29 (n=22)		Non Stressed PSS <29 (no=88)		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	7.05	3.3	6.23	2.03	0.145
Homocysteine (µmol/l)	5.79	2.64	5.34	2.17	0.416
C-Reactive protein mg/ dl	0.37	0.16	0.38	0.33	0.932

Table No 35: Lipoprotein (a), Homocysteine & C-Reactive protein in stressed and non-stressed subgroups of Group II (Bank employees) participants

Characteristics	Stress PSS > 29 (n=30)		Non Stressed PSS <29 (no=67)		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	12.01	11.04	15.65	12.57	0.175
Homocysteine (µmol/l)	10.51	6.66	12.55	7.50	0.203
C-Reactive proteinmg/ dl	1.07	0.74	0.95	0.62	0.400

Table No 36: Lipoprotein (a), Homocysteine & C-Reactive protein in stressed and non-stressed subgroups of Group III (Bus drivers) participants

Characteristics	Stress PSS > 29 (n=36)		Non Stressed PSS <29 (no=54)		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	15.78	7.72	14.65	6.22	0.447
Homocysteine (µmol/l)	11.25	3.89	10.04	5.75	0.271
C-Reactive protein mg/ dl	1.02	1.15	0.66	0.60	0.055

Table No 37: Lipoprotein (a), Homocysteine & C-Reactive protein in stressed and non-stressed subgroups of Group IV (Police constables) participants

Characteristics	Stress PSS > 29 (n=41)		Non Stressed PSS <29 (no=67)		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	15.68	7.96	15.53	11.69	0.942
Homocysteine (µmol/l)	10.90	6.80	9.38	4.96	0.186
C-Reactive protein(mg/ dl)	0.93	1.09	0.93	1.02	0.993

Table 34 to 37 show the levels of Lipoprotein (a) ,Homocysteine & C-Reactive proteinin Group I, II, III and IV respectively. There is no significant difference in levels of Lp(a), Homocysteine and C-reactive protein between stressed and non-stressedsubjects of all the four groups

4.1.5. Discussion

Work-related stress can be because of poor work organization, poor management, unsatisfactory working conditions, and lack of support from colleagues and supervisors. Stress can have impact on overall health. The Causes of workplace stress vary greatly. In many cases, the origin of the stress is something that cannot be changed immediately. Problems at work are more strongly associated with health complaints than are any other life stressor more so than even financial problems or family problems. Occupational stress accounts for more than 10% of work-related health claims (Ruotsalainen JH et al., 2014).

Occupational stress and workplace health have become issues of great concern for the past many years. Given the important value of occupation in the society the amount of time spent at work and the current changes that are affecting the nature of work, it is not surprising that work stress appears to be increasing (Szymanski EM, 1999). Stress is an integral part of everyday life and simply cannot be avoided. People encounter stressful stimuli many times a day in their personal and social domains and in the work place as work is an essential aspect of human existence.

The stress experienced by different occupation types and job roles has been studied at other countries. However in India very few such studies have been undertaken.

This was a cross sectional study conducted to focus on effect of nature of work and associated risk factors which may lead to the development of metabolic syndrome. Our study has found most of the subjects from all the three occupations were having job related stress though the nature of work differs from one occupation to another. Working late hours, irregular shifts, sedentary working style and inadequate sleep are the common features of these occupations. Depending on the amount of time spent at the working place and nature of the work, the work stress increases and it has direct impact on health status leading to many health related complications.

In our study, we evaluated perceived stress among Control subjects, bank employees, bus drivers and police constables, including its sources and severity along with its correlation with other variables. The participants were categorized as 'stressed' and 'Non-stressed' based on the calculated score with the help of perceived stress scale (14 items). Out of 97 bank employees 30 subjects, Out of 90 bus drivers 36 subjects, out of 108 police constables 41 subjects and out of 110 control subjects 22 subjects were found to be under stress respectively. The serum cortisol level was measured in all the subjects. Serum Cortisol levels were higher in 'stressed' group of all the three occupational subjects. The values were statistically significant. But in control subjects there was no difference in the values of both stressed and Non-stressed. The similar observations were made in earlier studies also (Rosamond R et al., 1998).

The job stress has been associated with raised morning cortisol (Alderling M et al., 2006; Maina G et al., 2009) and increased cortisol secretion across the day (Kunz-Ebrecht SR et al., 2004). Jing Liao et al reported that the associations of work stress with cortisol were modest (Jing Liao et al., 2013).It was observed that higher cortisol concentrations measured in the hair of healthy and working middle-aged women were associated with higher perceived stress and generally poorer health and with depressiveness (Faresjo A et al.,2014). A study was conducted among irregular Shift Workers and regular daytime workers and found that salivary cortisol levels were found to be elevated in the subjects with irregular shift workers (Lindholm H et al., 2012)

There are three popular tools for measuring perceived stress: the Stress Appraisal Measure (SAM) (Peacock EJ et al., 1990), the Impact of Event Scale (IES) (Horowitz MJ et al., 1979) and the Perceived Stress Scale (PSS) (Cohen S. et al., 1983). Among these, PSS is the most widely used such as in studies assessing stressfulness of events, physical and psychiatric diseases and stress management programs (Leon KA et al., 2007, Marcus MT et al., 2003).

So far the scale has been translated in many languages such as Arabic, Swedish,Spanish, Chinese, Japanese and Turkish (Chaaya M et al., 2010, Eskin M et al., 1996, Remor E, 2006, Leung D et al., 2010, Mimura C et al., 2004, Orucu MC et al., 2008). In India also studies have been done using PSS to measure the perceived stress (Augustine LF et al., 2011, Gupta A, et al., 2014,Brahmbhatt KR et al .,2013). In our study, we provided the translated version of perceived stress scale prepared by us in local language (Kannada) to the participants if it was required.

In our study, we found a statistical significant positive correlation between Serum Cortisol and perceived stress scale, fasting blood Glucose and Glycosylated HbA1c in police constables and bank employees. The positive association was remained unchanged even after adjustment with the habits of smoking and alcohol consumption in police constables but not in bank employees. Where as in bus drivers a positive correlation between serum cortisol and PSS was observed and the association remained same even after adjusting the habits. This attributes to the fact that in bank employees, the habits had the impact on association between cortisol level and PSS.

Smoking increases heart rate and blood pressure as a result of constriction of blood vessels. It increases the concentration of fatty acids in blood and also the ability of blood platelets to adhere to each other and to the walls of blood vessels. Carbon monoxide in smoke reduces the oxygen carrying capacity of the blood. Nicotine causes stimulation and sedation of central nervous system depending upon the dose (Hammond D et al., 2004; Boudarene M et al., 2001). Nicotine causes stimulation and sedation of central nervous system depending upon the dose (Hammond D et al., 2004). The nicotine levels in the plasma correspond to the blood cortisol levels (Xue Y et al., 2010).it is well known that psychiatric stress also induces rise in cortisol levels (Boudarene M et al., 2001). In a study done 1st year and 3rd year medical students it is reported that smoking was not taken up to reduce stress level.

Also it shows that smoking causes metabolic changes leading to hypercortisolism (Tey R et al., 2014).

The presence of stress is known to affect a variety of endocrine, metabolic, behavioral and cardiovascular functions, which if persistent can lead to a variety of diseases later on in life. The association between stress responses and subsequent elevations in fasting serum lipids are explained by the authors that, Catecholamines stimulate lipolysis in adipose tissue, through activation of hormone-sensitive lipase, leading to the breakdown of triacylglycerol into fatty acids and glycerol. This effect is sensitized by cortisol (Brindley et al., 1993). Increased levels of fatty acids and cortisol lead to insulin insensitivity in tissues and promote increased triacylglycerol synthesis and apolipoprotein B secretion by the liver. These combined effects result in increased hepatic production and secretion of very low-density lipoprotein, which is ultimately converted to LDL, the principal carrier of cholesterol in the blood.

A study conducted by (Maryam S et al., 2010).demonstratedhigh stress levels in individuals with high Total cholesterol, high LDL-Cholesterol, and low HDL-Cholesterol compared to individuals with normal lipid profile. However study did not show any relationship between stress and high Triglyceride (Maryam Set al., 2010). In our study we did not find any relation between stress and lipids. Similarly a study undertaken to explore the association between plasma fat and glucose, cortisol and adrenocorticotropic hormone (ACTH) levels and genotypes of GR and ACTHR genes in healthy Chinese Han subjects found no significant difference between cortisol and plasma lipid , glucose levels (Lian YL et al., 2012). Previous reports suggested a causal relationship between cortisol and obesity (MarinP et al., 1992, Pasquali et al., 1996). However no correlation between BMI or waist circumference and cortisol was found in our study and by others also (Steptoe A et al., 2004, Abraham SB, et al., 2013).

C-reactive protein (CRP) is the prototype acute-phase protein, which can increase up to 1000-fold after the onset of a stimulus.Inflammation is one of the cornerstones in the etiology and pathogenesis of atherosclerosis, which led to worldwide attention being focused on CRP and its role in the process of atherosclerosis. it has been reported that an elevated CRP level is associated with an increased risk of occlusive arterial disease, especially acute coronary syndromes (Hans CA et al., 2002). Metabolic syndrome and depressive symptoms

are independently related to CRP (Bonnie A et al., 2013) One study reported about major depressive episode is strongly associated with elevated CRP in men aged 17 to 39 (Danner et al., 2003). In our study we could not find much difference in the C-reactive protein in the metabolic syndrome sub group of any occupational groups, studied,

4.2 METABOLIC SYNDROME IN DIFFERENT OCCUPATIONS

The participants were identified and classified as having metabolic syndrome and non-metabolic syndrome based on modified NCEP ATP III Criteria. (The five abnormalities , fasting blood glucose ≥ 100.0 mg/dl or on medication, Serum Triglyceride ≥ 150.0 mg/dl, HDL Cholesterol ≤ 40.0 mg/dl (for males), Waist circumference ≥ 90 Cm and blood pressure ≥ 130/85 mmHg or on medication.) The presence of any three abnormalities from these five suggests the presence of metabolic syndrome.

4.2.1 Metabolic and non-metabolic syndrome in Group I (control subjects)

Table No 38: Basic characteristics in the metabolic and non-metabolic syndrome in subgroups of Group I (Control subjects)

	Metabolic syndrome (no=13)		**Non-metabolic syndrome (no=97)**		
Variable	**Mean**	**S.D**	**Mean**	**S.D**	**p-Value**
Age(Years)	49.85	6.14	46.07	7.76	0.095
Height (Cm)	165.06	10.17	162.35	9.51	0.341
Weight (Kg)	58.38	5.55	60.36	3.31	0.068
BMI (Kg / m^2)	21.73	3.85	23.16	3.23	0.146
Waist circumference (Cm)	77.00	9.55	74.60	7.16	0.278
Hip circumference (Cm)	91.08	11.03	89.01	6.95	0.353
W / H Ratio	0.85	0.07	0.83	0.06	0.587
Systolic blood pressure (mmHg)	125.38	8.77	119.87	7.96	0.022*
Diastolic blood pressure (mmHg)	83.85	5.46	77.41	6.31	0.001**
Pulse rate (bpm)	80.92	8.07	80.36	6.95	0.783

* p<0.05, **p<0.01, ***p<0.001

Table 38depicts about basic characteristics of the subjects who served as controls. Though the both systolic and diastolic blood pressure levels were statistically significant, the values were within the normal limits.

Table No 39: Stress level in subjects with metabolic and non-metabolic syndrome in Group I (Control subjects)

Characteristics	**Metabolic syndrome (no=13)**		**Non-metabolic syndrome (no=97)**		
	Mean	SD	Mean	SD	p Value
Cortisol (µg/dl)	9.96	3.85	9.72	5.08	0.868
PSS	17.92	6.03	18.94	6.59	0.599

The table 39 shows the levels of Serum cortisol and PSS in both metabolic and non-metabolic syndrome subjects in Group I. There was no significant difference between the values of metabolic and non-metabolic syndrome groups.

Table No 40: Fasting blood glucose and other variables in subjects with metabolic and non-metabolic syndrome in Group I (Control group)participants.

Variables	**Metabolic syndrome (no=13)**		**Non-metabolic syndrome (no=97)**		
	Mean	SD	Mean	SD	p Value
Fasting blood glucose (mg/dl)	106.00	13.14	96.89	10.63	0.006**
Glycosylated HbA1c (%)	4.98	0.88	4.87	0.65	0.595
Insulin (µIU/ml)	6.28	7.29	5.81	5.99	0.797
HOMA-IR	1.74	2.35	1.43	1.61	0.541

* p<0.05, **p<0.01, ***p<0.001

The table 40 shows the Fasting blood glucose and other variables in subjects with metabolic and non-metabolic syndrome in Group I subjects. Impaired fasting glucose level was found in metabolic syndrome group which was statistically significant.

Table No 41: Lipid profile in subjects with metabolic and non-metabolic syndrome in Group I (Control group) participants

Characteristics	Metabolic syndrome (no=13)		Non-metabolic syndrome (no=97)		
	Mean	SD	Mean	SD	p-Value
Triglyceride (mg/dl)	146.85	27.01	114.61	26.30	0.000***
Total Cholesterol (mg/dl)	180.85	39.46	178.07	25.71	0.734
HDL Cholesterol (mg/dl)	40.08	3.90	48.32	5.93	0.000***
LDL Cholesterol (mg/dl)	111.40	40.28	106.83	26.44	0.586
VLDL Cholesterol (mg/dl)	29.37	5.40	22.92	5.26	0.000***

* p<0.05, **p<0.01, ***p<0.001

Table 41 shows the Lipid profile studied in the control subjects with metabolic and non-metabolic syndrome groups. Comparing to non-metabolic syndrome group, the raised Triglyceride and VLDL Cholesterol with lowered HDL cholesterol levels were found in metabolic syndrome group and were statistically significant. However the levels were within the prescribed normal limits.

Table No 42: CVD risk factors in metabolic and non-metabolic syndrome subgroups in Group I (Control subjects)

Characteristics	Metabolic syndrome (no=13)		Non-metabolic syndrome (no=97)		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	5.56	2.05	6.50	2.37	0.177
Homocysteine (µmol/l)	5.95	2.73	5.36	2.20	0.380
C-reactive protein(mg/ dl)	0.35	0.29	0.38	0.31	0.740

Table 42 depicts about CVD risk factors in subjects with metabolic and non-metabolic syndrome in Control subjects. We did not find any difference in the levels of Lp(a), Homocysteine and C-reactive protein between the metabolic and non-metabolic syndrome groups.

4.2.2 Metabolic and non-metabolic syndrome in Group II (Bank employees)

Table No 43: Basic characteristics in the metabolic and non-metabolic syndrome subgroups of Group II (Bank employees)

	Metabolic syndrome. (no=37)		**Non-metabolic syndrome (no=60)**		
Variables	**Mean**	**SD**	**Mean**	**SD**	**p-Value**
Age (years)	49.59	6.29	45.85	7.02	0.009**
Height (Cm)	168.31	8.80	164.58	10.50	0.074
Weight (Kg)	74.30	8.25	67.07	10.77	0.001**
BMI (Kg/ m^2)	26.38	3.69	24.98	4.81	0.132
Waist circumference (Cm)	95.92	6.63	88.50	10.08	0.000***
Hip circumference (Cm)	100.46	6.29	96.12	8.22	0.007**
W / H Ratio	0.97	0.05	0.92	0.07	0.001**
Systolic blood pressure (mmHg)	131.41	11.16	121.50	9.02	0.000***
Diastolic blood pressure (mmHg)	88.59	10.49	78.00	7.22	0.000***
Pulse rate	82.49	7.63	77.23	5.85	0.000***

*p<0.05, **p<0.01, ***p<0.001

Table 43 shows the baseline characteristics of the bank employees, classified as metabolic and non-metabolic syndrome subjects according to the NCEP ATP III guidelines. All the variables except Height and BMI showed statistically significant differences between subjects with and without metabolic syndrome.

Table No 44: Stress level in metabolic and non-metabolic syndrome subgroups of Group II (Bank employees) participants

Characteristics	**Metabolic syndrome. (no=37)**		**Non-metabolic syndrome (no=60)**		
	Mean	SD	Mean	SD	p Value
Cortisol (µg/dl)	16.63	6.68	15.61	6.00	0.436
PSS	23.32	6.29	25.53	6.61	0.107

Table 44 shows the levels of serum Cortisol and PSS levels in subjects with metabolic and non-metabolic syndrome in bank employees. There was no significant difference between the values of metabolic and non-metabolic syndrome groups.

Table No 45: Fasting blood glucose & insulin level in metabolic and non-metabolic syndrome subgroups of Group II (Bank employees)

Variables	**Metabolic syndrome. (no=37)**		**Non-metabolic syndrome (no=60)**		**p Value**
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	138.46	53.42	105.22	17.13	0.000***
Glycosylated HbA1c(%)	6.68	1.67	5.64	0.66	0.000***
Insulin (µIU/ml)	16.43	12.17	15.85	11.07	0.809
HOMA-IR	5.71	4.59	4.22	3.20	0.063

* p<0.05, **p<0.01, ***p<0.001

The table 45 describes the status of fasting blood glucose and other variables in subjects with metabolic and non-metabolic syndrome in Bank employees. There was a statistically significant difference in the levels of fasting blood glucose and Glycosylated HbA1c.Between the subjects sub grouped as metabolic and non-metabolic syndrome subjects.

Table No 46: Lipid profile in metabolic and non-metabolic syndrome subgroups of Group II (Bank employees) subjects

Characteristics	**Metabolic syndrome. (no=37)**		**Non-metabolic syndrome (no=60)**		
	Mean	SD	Mean	SD	p-Value
Triglyceride (mg/dl)	168.00	86.57	118.72	89.25	0.008**
Total Cholesterol (mg/dl)	174.08	24.02	186.97	33.20	0.043*
HDL Cholesterol (mg/dl)	36.08	7.31	42.07	8.79	0.001**
LDL Cholesterol (mg/dl)	104.40	22.16	121.16	33.74	0.009**
VLDL Cholesterol (mg/dl)	33.60	17.31	23.94	17.94	0.010*

*p<0.05, **p<0.01, ***p<0.001

Table 46 presents the values of lipid profile in the Group II Subjects with metabolic and non-metabolic syndrome. We found statistically significant lowered HDL Cholesterol values and higher values of serum Triglyceride, Total Cholesterol, LDL Cholesterol and VLDL Cholesterol in the metabolic syndrome group.

Table No 47: CVD risk factors in subjects with metabolic and non-metabolic syndrome in Group II (Bank employees)

Characteristics	**Metabolic syndrome. (no=37)**		**Non-metabolic syndrome (no=60)**		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	14.85	13.94	14.60	10.97	0.923
Homocysteine (µmol/l)	13.78	7.58	10.77	6.91	0.047*
C-reactive protein(mg/ dl)	1.01	0.61	0.97	0.69	0.794

* p<0.05, **p<0.01, ***p<0.001

Table 47 shows the CVD risk factors in subjects with metabolic and non metabolic syndrome in bank employees. We found there was a statistically significant rise in the Homocysteine level of the metabolic syndrome group.

4.2.3 Metabolic and non-metabolic syndrome in Group III (Bus drivers)

Table No 48: Basic characteristics in the group metabolic and non-metabolic syndrome in Group III (Bus drivers)

	Metabolic syndrome (no=33)		**Non-metabolic syndrome (no=57)**		
Variable	**Mean**	**S.D**	**Mean**	**S.D**	**p-Value**
Age(Years)	45.21	7.70	44.25	6.17	0.515
Height (Cm)	167.65	5.65	163.69	10.34	0.063
Weight (Kg)	71.48	6.43	65.28	11.36	0.005**
BMI (Kg / m^2)	25.42	1.78	24.43	4.39	0.217
Waist circumference (Cm)	99.09	9.16	85.91	13.80	0.000***
Hip circumference (Cm)	102.27	8.27	92.37	13.46	0.000***
W / H Ratio	0.97	0.05	0.93	0.01	0.049*
Systolic blood pressure (mmHg)	125.70	9.58	123.89	10.39	0.417
Diastolic blood pressure(mmHg)	83.21	8.99	79.58	7.34	0.040*
Pulse rate (bpm)	84.27	7.33	84.00	6.68	0.857

* p<0.05, **p<0.01, ***p<0.001

Basic characteristics of the bus drivers are summarized in Table 48. The weight, waist circumference, hip circumference, diastolic blood pressure levels were higher in participants with metabolic syndrome. The difference was statistically significant.

TableNo 49: Stress level in subjects with metabolic and non-metabolic syndrome subgroups of Group II (Bus drivers)

Characteristics	**Metabolic syndrome (no=33)**		**Non-metabolic syndrome (no=57)**		
	Mean	SD	Mean	SD	p Value
Cortisol (µg/dl)	24.09	10.99	18.90	9.61	0.022*
PSS	29.85	7.86	24.88	6.00	0.001**

* p<0.05, **p<0.01, ***p<0.001

Table 49 shows the levels of serum Cortisol and PSS levels in subjects with metabolic and non-metabolic syndrome in bus drivers. There was the significant difference in the values of Serum cortisol and PSS between metabolic and non-metabolic syndrome groups.

Table No 50: Fasting blood glucose and insulin indicesin metabolic and non-metabolic syndrome subgroups of Group III (Bus drivers)

Variables	**Metabolic syndrome (no=33)**		**Non-metabolic syndrome (no=57)**		**p Value**
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	141.55	69.86	87.56	15.75	0.000***
Glycosylated HbA1c(%)	6.58	1.32	5.26	0.54	0.000***
Insulin (µIU/ml)	18.07	8.95	7.85	5.70	0.000***
HOMA-IR	6.75	4.28	1.85	1.71	0.000***

* p<0.05, **p<0.01, ***p<0.001

Table 50 depicts about Fasting blood glucose and other variables in subjects with metabolic and non-metabolic syndrome in bus drivers. There was a statistically significant difference in the levels of all the parameters between the two groups.

Table No 51: Lipid profile in subjects with metabolic and non-metabolic syndrome in Group III (Bus drivers)

Characteristics	**Metabolic syndrome (no=33)**		**Non-metabolic syndrome (no=57)**		p-Value
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	204.03	111.26	116.25	33.88	0.000***
Total Cholesterol (mg/dl)	178.27	31.14	181.60	31.64	0.630
HDL Cholesterol (mg/dl)	38.79	8.80	44.21	10.08	0.012*
LDL Cholesterol (mg/dl)	98.68	36.77	114.14	33.02	0.041*
VLDL Cholesterol (mg/dl)	40.81	22.25	23.25	6.78	0.000***

*p< 0.05, **p<0.01, ***p<0.001

Table 51 shows the Lipid profile levels of the bus drivers with and without metabolic syndrome. The subjects with metabolic syndrome had shown statistically significant higher values of Triglyceride, LDL Cholesterol and VLDL Cholesterol with lowered HDL Cholesterol comparing to non-metabolic syndrome subjects.

Table No 52: CVD risk factors in subjects of metabolic and non-metabolic syndrome subgroups of Group III (Bus drivers)

Characteristics	**Metabolic syndrome (no=33)**		**Non-metabolic syndrome (no=57)**		p-Value
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	16.84	8.00	14.10	5.91	0.067
Homocysteine (µmol/l)	12.01	3.79	9.66	5.57	0.034*
C-reactive protein(mg/ dl)	0.87	0.90	0.76	0.87	0.591

* p<0.05, **p<0.01, ***p<0.001

Table 52 shows the CVD risk factors in subjects with metabolic and non metabolic syndrome in bus drivers. We found there was a statistically significant rise in the Homocysteine level in the metabolic syndrome group.

4.2.4 Metabolic and non-metabolic syndrome in Group IV (Police constables)

Table No 53: Basic characteristics in the group metabolic and non-metabolic syndrome in Group IV (Police constables)

	Metabolic syndrome (no=41)		**Non-metabolic syndrome (no=67)**		
Variable	**Mean**	**S.D**	**Mean**	**S.D**	**p-Value**
Age(Years)	44.44	9.60	40.78	10.64	0.075
Height (Cm)	168.88	5.92	172.01	4.44	0.002**
Weight (Kg)	77.44	6.55	74.49	6.18	0.021*
BMI (Kg / m^2)	27.15	1.77	25.01	2.17	0.000***
Waist circumference (Cm)	85.49	11.07	78.97	9.06	0.000***
Hip circumference (Cm)	86.54	14.35	78.57	13.57	0.005**
W / H Ratio	1.00	0.08	1.06	0.11	0.324
Systolic blood pressure (mmHg)	131.80	12.33	117.70	8.59	0.000***
Diastolic blood pressure (mmHg)	85.07	10.07	76.84	7.72	0.000***
Pulse rate (bpm)	83.59	4.42	81.67	5.68	0.068

*p<0.05, **p<0.01, ***p<0.001

The basic characteristics of the police constables studied are given in the Table 53. Except age, W/H ratio and pulse rate, rest of other results were higher in the metabolic syndrome group and were statistically significant comparing to non-metabolic syndrome.

Table No 54: Stress level in subjects of metabolic and non-metabolic syndrome subgroups of Group IV (Police constables).

Characteristics	**Metabolic syndrome (no=41)**		**Non-metabolic syndrome (no=67)**		
	Mean	SD	Mean	SD	p Value
Cortisol (µg/dl)	25.81	13.39	24.51	13.87	0.635
PSS	28.20	7.25	27.06	9.20	0.503

* p<0.05, **p<0.01, ***p<0.001

Table 54 shows the stress level in subjects with metabolic and non-metabolic syndrome in Police constables**.** There was no significant difference between the values of metabolic and non-metabolic syndrome groups.

Table No 55: Fasting blood glucose and insulin indices in subjects of metabolic and non-metabolic syndrome subgroups of Group IV (Police constables).

Variables	**Metabolic syndrome (no=41)**		**Non-metabolic syndrome (no=67)**		**p Value**
	Mean	SD	Mean	SD	
Fasting blood glucose (mg/dl)	133.46	53.93	103.85	36.29	0.000***
Glycosylated HbA1c (%)	6.34	1.61	5.60	0.79	0.002**
Insulin (µIU/ml)	17.18	12.64	13.05	11.97	0.091
HOMA-IR	6.08	5.44	3.90	4.54	0.027*

* p<0.05, **p<0.01, ***p<0.001

Table 55 presents the levels of fasting blood glucose and other variables in subjects with metabolic and non-metabolic syndrome in Police constables. Fasting blood glucose, Glycosylated HbA1c, and HOMA-IR were raised in the subjects with metabolic syndrome and were statistically significant.

Table No 56: Lipid profile in subjects with metabolic and non-metabolic syndrome in Group IV (Police constables)

Characteristics	**Metabolic syndrome (no=41)**		**Non-metabolic syndrome (no=67)**		p-Value
	Mean	SD	Mean	SD	
Triglyceride (mg/dl)	210.24	125.80	115.69	46.29	0.000***
Total Cholesterol (mg/dl)	213.63	61.78	192.85	51.40	0.062
HDL Cholesterol (mg/dl)	34.73	6.50	37.28	6.21	0.044*
LDL Cholesterol (mg/dl)	136.85	50.95	132.43	52.69	0.669
VLDL Cholesterol (mg/dl)	42.05	21.16	23.14	9.26	0.000***

* p<0.05, **p<0.01, ***p<0.001

Table 56 shows the values of lipid profile in the groups of metabolic syndrome and non-metabolic syndrome of police constables. We have noticed that with lowered HDL cholesterol, values of Triglyceride, VLDL cholesterol, Homocysteine of the subjects with metabolic syndrome were increased and the values were statistically significant.

Table No 57: CVD risk factors in subjects with metabolic and non-metabolic syndrome subgroups of Group IV (Police constables)

Characteristics	**Metabolic syndrome (no=41)**		**Non-metabolic syndrome (no=67)**		**p-Value**
	Mean	SD	Mean	SD	
Lipoprotein (a) (mg/dl)	16.81	11.15	14.85	9.91	0.344
Homocysteine (µmol/l)	12.50	5.98	8.40	5.04	0.000***
C-reactive protein(mg/ dl)	1.02	1.24	0.88	0.91	0.503

* p<0.05, **p<0.01, ***p<0.001

Table 57 shows the levels of CVD risk factors in subjects with metabolic and non- metabolic syndrome in Police constables. There was a statistically significant rise in the Homocysteine level of metabolic syndrome group.

4.2.5. CVD risk factors in subjects with metabolic and non-metabolic syndrome of occupational groups

Table No 58: CVD risk factors in the group of metabolic and non-metabolic syndrome of the three occupational groups.

CVD Risk factors.	Group II		Group III		Group IV	
Variables	Mets (no=37)	Non mets (no=60)	Mets (no=33)	Non mets (no=57)	Mets (no=41)	Non mets (no=67)
	%	%	%	%	%	%
Fasting blood glucose (100-125) mg/dl	24.2	22.8	24.3	21.7	24.4	6
Fasting blood glucose>125 mg/dl	42.4	5.3	37.8	5	41.5	14.9
Triglyceride. 150-200 mg/dl	30.3	7	24.3	6.7	29.3	9
Triglyceride. >200 mg/dl	36.4	8.8	29.7	8.3	51.2	9
Total Cholesterol 200-239 mg/dl	21.2	19.3	10.8	18.3	24.4	22.4
Total Cholesterol>240 mg/dl	3	7	2.7	5	22	11.9
HDL Cholesterol 40-31 mg/dl	45.4	28.1	64.8	26.7	80.5	37.3
HDL Cholesterol < 30 mg/dl	15.2	8.8	13.5	8.3	14.6	10.4

[Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

[Mets= Metabolic syndrome: Non Mets= Non Metabolic syndrome.]

Table N0: 58. Continued.

CVD Risk factors.	Group II		Group III		Group IV	
Variables	Mets (no=37)	Non mets (no=60)	Mets (no=33)	Non mets (no=57)	Mets (no=41)	Non mets (no=67)
	%	%	%	%	%	%
LDL Cholesterol 131-159 mg/dl	3	17.5	5.4	20	17.1	16.4
LDL Cholesterol. >160 mg/dl	6.1	12.3	5.4	11.7	24.4	22.4
Lipoprotein (a) >25.0 mg/dl	15.2	5.3	18.9	20	17.1	7.5
Homocysteine >14.0 µmol/l	27.3	12.3	48.6	21.7	39	17.9
BMI: 25-29.9 Kg / m^2	60.6	31.6	40.5	30	87.8	46.3
BMI: >30 Kg / m^2	6.1	10.5	18.9	10	9.8	3
Blood Pressure .>130/85 mmHg	24.2	14	35.1	13.3	19.5	4.5
Blood Pressure>140/90 mmHg	15.2	7	35.1	6.7	34.2	6
Waist circumference 90-102 Cm	63.6	17.5	67.5	28.3	39.0	13.4
Waist circumference > 102 Cm	27.3	31.6	24.3	8.3	4.9	3.0

[Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

[Mets= Metabolic syndrome: Non Mets= Non Metabolic syndrome.]

Table 58 shows the distribution of CVD risk factors in three occupational groups which are classified as metabolic and non-metabolic syndrome as per NCEP ATP III Criteria. The

values are expressed as percentage (%) distribution. Except for LP (a) and Homocysteine, the results of the rest of the parameters are presented in two grades of abnormality.

Table No 59: CVD risk factors (overall) in all the four studied groups.

Variables	Group I (no=110)	Group II (no=97)	Group III (no=90)	Group IV (no=108)
	%	%	%	%
Fasting blood glucose (100-125) mg/dl	31.8	21.6	12.2	12.9
Fasting blood glucose>125 mg/dl	8.2	16.5	17.7	25
Triglyceride. 150-200 mg/dl	12.7	12.4	17.7	16.6
Triglyceride. >200 mg/dl	0	16.5	13.3	25
Total Cholesterol 200-239 mg/dl	21.8	15.5	20	23.1
Total Cholesterol. >240 mg/dl	2.7	4.1	4.4	15.7
HDL Cholesterol. 40-31 mg/dl	15.5	43.2	32.2	60.1
HDL Cholesterol< 30 mg/dl	0	11.3	10	12.0
LDL Cholesterol 131-159 mg/dl	19.1	16.4	12.2	14.8
LDL Cholesterol>160 mg/dl	1.8	8.2	6.6	26.8
Lipoprotein(a) >25.0 mg/dl	0	19.5	8.8	12.9
Homocysteine >14.0 µmol/l	3.6	18.5	17.7	29.6
BMI; 25-29.9 Kg / m^2	27.3	32.9	38.8	62
BMI >30 Kg / m^2	4.5	15.5	8.8	14.8
BP .>130/85 mmHg	24.5	25.7	31.1	7.5
BP >140/90 mmHg	11.8	18.55	10.0	18.5
Waist circumference 90-102 Cm	6.4	48.4	30.0	19.4
Waist circumference > 102 Cm	2.0	11.3	17.8	2.8
Smoking habit.	16.9	16.0	23.8	47.9

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

Table 59 shows the distribution of CVD risk factors in all the four studied groups including controls.The values are expressed as percentage (%) distribution. Except for LP (a) and Homocysteine, the results of the rest of the parameters are presented in two grades of abnormality.

4.2.6 CVD risk factors as per age group

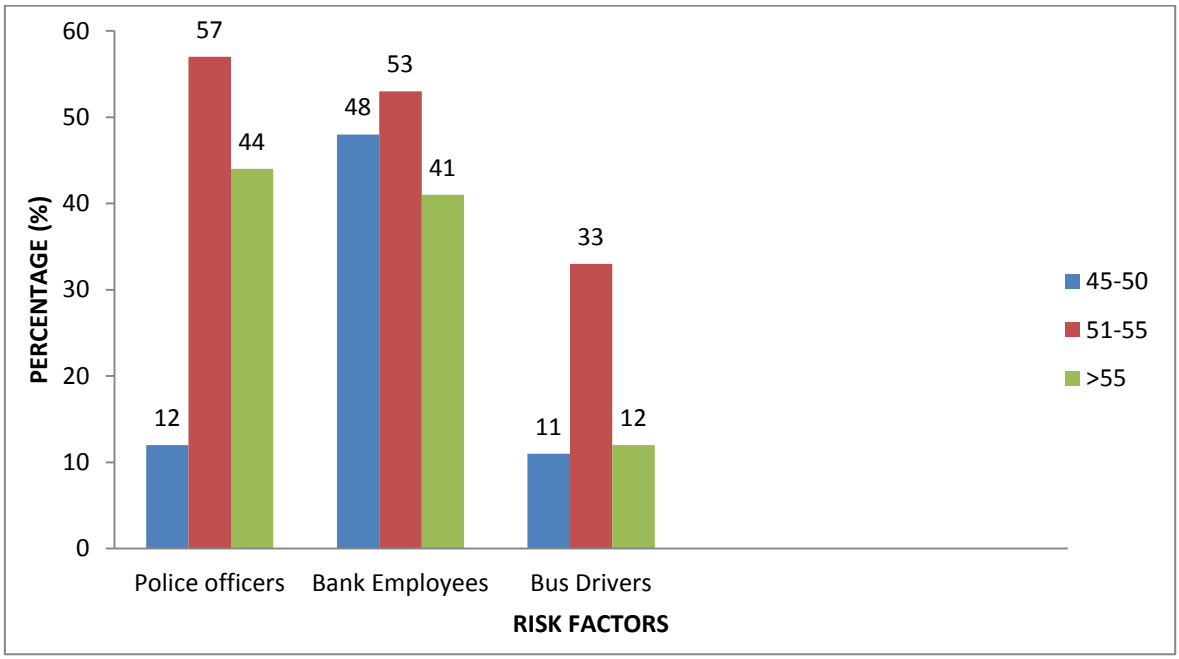

Fig No 3: CVD risk factors as per age group

4.2.7 Discussion

One of the objectives of this study was to determine the prevalence of metabolic syndrome and its associated risk factors among the subjects of three occupational groups. Totally 405 subjects participated in the study. Out of them, 124 (30.62 %) participants met the criteria of NCEP ATP III criteria and identified as having metabolic syndrome. Much of research work has been focused on life style factors which influence over the occurrence of metabolic syndrome (Peter M J et al., 2008). Limited research is available about metabolic syndrome prevalence or its components among employees (Nair C V, 2010, Davila E P, 2010) Studies suggest that the prevalence of Metabolic syndrome differs according to occupation type (Lin YC, et al., 2009).

Our study comprised three occupational groups. The basic and biochemical parameters were studied in all participants. The causes of obesity are multifactorial and may include genetic predisposition. Obesity results from a long-standing imbalance between energy intake and

energy expenditure, including energy utilization for basic metabolic processes and energy expenditure from physical activity. Obesity is becoming a worldwide epidemic .One of the tools to measure the obesity is BMI. It is considered as risk factor for Cardiometabolic diseases, including diabetes, hypertension, dyslipidemia, and coronary heart disease (CHD). The epidemiologic studies demonstrate a direct correlation between BMI and the risk of medical complications and mortality rate (Colditz GA et al., 1995, Calle EE et al., 1999). The BMI between 25 to 29.9 Kg / m^2 is considered as overweight and (BMI ≥30 Kg / m^2 as obesity. But as per NCEP ATP III, BMI is not one of the criteria for diagnosing the metabolic syndrome. A Chinese study reported that both BMI and the metabolic syndrome are independently associated with CHD (Yao He et al., 2007). In our study we included measuring waist circumference, BMI and waist to hip ratio to assess the obesity. We found that in both bank employees and bus drivers there was no difference in BMI values among the subjects with and without metabolic syndrome. However the police constables with metabolic syndrome had marginally raised BMI level which indicated overweight and statistically significant. The use of BMI measurement is suggested in the study done on adolescents (Katelynn EW et al., 2014).

Excess abdominal fat is associated with an increased risk of Cardiometabolic disease. However, precise measurement of abdominal fat content requires the use of expensive radiological imaging techniques. Therefore, waist circumference (WC) is often used as a surrogate marker of abdominal fat mass, because WC correlates with abdominal fat mass (Pouliot MC et al., 1994).

In our study, we found that in both bank employees and bus driver's waist circumference levels were elevated and the values were statistically significant. But in police constables though the difference in the waist circumference level between with and without metabolic syndrome subjects were statistically significant, they were within normal prescribed levels. The IDF definition differs from NCEP ATP III definition, in two key aspects. First, the IDF has lowered the threshold for waist circumference from 102 to 94 cm. (For Asians, 90 cm).Secondly; waist circumference is a required component of metabolic syndrome under the IDF criteria, rather than an optional component as used by the NCEP. In our study waist circumference was better indicator of obesity.

Even though BMI is commonly used for monitoring the occurrence of obesity in the population, it has numerous limitations. It does not provide any information on the distribution of the adipose tissue in the organism. BMI is a calculated statistical value which

does not take into consideration physiological differences in the proportions between the adipose and muscular tissues (Shields M et al., 2012). Besides, its value is affected by sex, age, constitution. Evidence from the conducted studies has revealed that abdominal obesity (assessed based on the waist circumference) plays a very important role in the development of metabolic disorders and in the assessment of cardiovascular risk. It is responsible for the development of insulin resistance which decreases the levels of the HDL-cholesterol fraction, increases the levels of Triglycerides, and leads to the development of arterial hypertension. All of the above-mentioned disorders contribute to metabolic syndrome and are related to the development of Type 2- diabetes and ischemic heart disease.

Waist to hip ratio is used to estimate the distribution of body fat and is called the index of central obesity, may be a quick and easy screening tool for assessing health risks. Waist to hip ratio is strongly associated with all factors for obesity and moreover, many studies have shown that WHR can help to predict morbidity and mortality, often better than BMI (Nambiar S et al., 2009). Studies have been done to compare body mass index (BMI), waist circumference (WC) and waist hip ratio (WHR) to identify the best predictor of metabolic syndrome (Abdulbari B et al., 2013). Excess abdominal fat, regardless of overall body fat, will predispose the subjects to obesity-related disease. To measure abdominal fat, WHR is utilized. But BMI can be better indicator of underweight rather than waist to hip ratio. In our study the waist to hip ratio levels were found to be significantly higher in the bank employees and bus driver subjects with metabolic syndrome. But the values were found to be raised above the cut off value mentioned in both the subjects with and without metabolic syndrome.

Hypertension is a very common condition which frequently remains undiagnosed until relatively late in its course, leading to a variety of other life-threatening conditions like kidney damage and heart failure. It is a very prominent feature of the metabolic syndrome. The cut off value suggested for the systolic and diastolic blood pressure as one of the NCEP ATP III criteria is 130/85 respectively. The establishment of hypertension as a component of the metabolic syndrome has enabled better insight into the condition and allowed for earlier detection and treatment. Though the cause of hypertension in the metabolic syndrome remains unclear, insulin resistance and central obesity have been recognized as the main factors involved in its pathophysiology (Lea D et al., 2008,Hidekatsu Y et al., 2008). The studies have revealed about coexistence of both hypertension and metabolic syndrome and

increased risk of CVD (Joseph Redon et al., 2008). The PAMELA (Pressioni Arteriose Monitorate ELoro Associazioni) population study revealed that high normal blood pressure values and hypertension were present in 80% of individuals with metabolic syndrome (MuleG et al., 2006). A study from Karachi, revealed that the subjects with metabolic syndrome with hypertension had higher insulin resistance than the non hypertensive (Khan SH et al., 2007).In our study we found that the systolic blood pressure in the subjects with metabolic syndrome of bank employees and police constables were higher and statistically significant. Whereas, the diastolic blood pressure was raised in the bank employees with metabolic syndrome. However, the total number ofhypertensive subjects in our study were bank employees (51.8%), bus drivers (38.7%), and police constables (28.3%).

The present study has revealed that dyslipidemia (high Triglyceride and low-HDL cholesterol) had the major role in predicting the metabolic syndrome. In all the three occupational groups it was found that elevated Triglyceride with lowered HDL Cholesterol were associated with the metabolic syndrome subjects. There was a wide difference among the levels of these two parameters between the subjects with and without metabolic syndrome and the difference was statistically significant. The guidelines given by various organizations have uniformly declared the cut off value for serum Triglyceride as ≥ 150.0 mg / dl. Except WHO, other organizations have adopted HDL Cholesterol cut off value as Male ≤ 40.0 mg/ dl and for female ≤ 50.0 mg/dl. WHO had suggested the cut off value for HDL Cholesterol as Male ≤ 35.0 mg/ dl and for female ≤ 39.0 mg/dl ((Ilanne et al., 2004). In a study from Wardha, India, it was concluded that measurement of Triglyceride is a rewarding screening parameter for assessment of cardio-metabolic risk in general population (Deshmukh PR et al., 2013). Increased prevalence rate of lowered HDL cholesterol has been reported (Enas et al., 1992).The authors concluded that low HDL cholesterol were a strong predictor of occurrence of myocardial infarction.

In a recent study on 9000 subjects (<40 years of age), it was reported that around 64.2% men and 33.8% women had abnormally low levels of HDL cholesterol (Sawant et., 2008). Our study shows around 37.8 % subjects had low HDL Cholesterol level between the ranges of 31 to 40 mg/ dl. In a study by Phase I of the Indian Council of Medical Research–India Diabetes (ICMR-INDIAB) study was conducted in a representative population of three states of India [Tamil Nadu, Maharashtra and Jharkhand]

and one Union Territory [Chandigarh], it was observed that hypertriglyceridemia was highest in Chandigarh (38.6%) and least in Maharashtra (22.8%) and the rates were significantly higher in urban compared to rural areas in Jharkhand and Chandigarh. In the study, the cut off value of ≥ 150.0 mg / dl was applied (Joshi SR et al., 2014).

In our study we found 28.4 % of subjects from all the study groups including control group had their Triglyceride level more than 150.0 mg/dl.Pre-diabetes represents an elevation of plasma glucose above the normal range but below that of clinical diabetes. Pre-diabetes can be identified as impaired fasting glucose (IFG). Any definition of pre-diabetes that is restricted to IGT(Impaired glucose tolerance) and/or IFG fails to include other risk factors for diabetes, such as, a family history of type 2- diabetes or the metabolic syndrome. The purpose of ATP III was to identify people at higher long-term risk for cardiovascular diseases (CVDs) and type 2- diabetes who deserve clinical lifestyle intervention to reduce risk. Hence the recommended cut off value for fasting blood glucose is ≥ 100.0 mg/dl or on treatment for type 2- diabetes. As per WHO guidelines, it is ≥ 110.0 mg/ dl.

Studies have shown that the prevalence of Type 2- diabetes is particularly high in South Asians, with the prevalence of insulin resistance in healthy, young lean Asian men being three to four times higher than lean men in other ethnic groups (Venkataraman R et al ., 2004; Mohan V et al ., 2008). In a study in South African Indian individuals with metabolic syndrome, elevated fasting blood glucose was found to be the most frequently occurring criterion (IDF, 87%; NCEP ATP III, 83%) (Ranjith N et al., 2008). In our study we found that there were 19.7 % subjects with impaired blood glucose level and 14.8 % subjects had their blood glucose level more than 126.0 mg/ dl. We also found that there was statistically significant differencein the fasting blood glucose levels between the subjects with and without metabolic syndrome of all the three occupational groups studied.

Measurement of glycosylated hemoglobin A1C is recommended for both (a) checking blood sugar control in people who might be pre-diabetic and (b) monitoring blood sugar control in patients with more elevated levels. Although glycosylatedHbA1c is not considered to be a diagnostic criterion for diabetes or pre-diabetes, it might provide a simple method of predicting metabolic syndromeorIFG (Impaired Fasting Glucose). One recent study examined the significance of glycosylated HbA1c as a surrogate for metabolic syndrome in high-risk African-Americans who were genetically predisposed to Type 2- diabetes.

They demonstrated that in subjects with increased glycosylated HbA1c, some, but not all, components of metabolic syndrome could be defined by glycosylated HbA1c (Osei et al., 2003). In a Korean study it was reported that glycosylated HbA1c of 5.45% predicted the presence of metabolic syndrome (Sung KCet al., 2007). In our study, GlycosylatedHbA1c was significantly raised in subjects with metabolic syndrome in all the three occupational groups. The mean value 6.14 % of HbA1c from our study may be considered as the predictive value for the presence of metabolic syndrome. in context to NCEP ATP III definition, it was suggested by (Mottillo S et al., 2010)that patients with the metabolic syndrome, but without Type 2- diabetes mellitus, are still at high risk for CVD mortality, myocardial infarction and stroke. So, metabolic syndrome does not require Type 2- diabetes mellitus in its definition in order to be closely associated with cardiovascular risk (Mottillo S et al., 2010).

The metabolic syndrome is associated with a 2-fold increase in cardiovascular outcomes. 'Traditional' risk factors such as hypertension, elevated cholesterol, smoking, and diabetes have long been linked to cardiovascular disease (CVD). As obesity rates soar, more and more patients are developing additional metabolic abnormalities that raise their CVD risk. Our study showed a high prevalence of cardio vascular risk factors like low HDL cholesterol, hypertension LDL cholesterol, smoking and others.

Overweight/obesity is an established risk factor for CVD and diabetes as reported earlier (King H et al., 1998). Our study showed a high prevalence of overweight/obesity. Obesity in terms of BMI > 30 Kg / $m^2$was 10.9% and waist circumference > 102 cm was 8.5 % .We found that subjects with HDL cholesterol less than 30 mg/ dl were 8.3% and between 31-40 mg/dl were 37%.It is in comparable to previous studies (Gupta R et al., 2009, Das M et al., 2011).We found the prevalence of hypertension was 14.7 % in the present study which is higher than other study (Gupta R et al ., 2009). The other findings in our study with respect to CVD risk factors were LDL cholesterol 15.6 %,Triglyceride> 200.0mg/dl were 13.7% and fasting blood glucose > 126.0 mg/dl 16.9 %.

4.3 INSULIN RESISTANCE (HOMA-IR)

In the present study we focused on insulin resistance which contributes to the clustering of the border line factors in the early stage of metabolic syndrome. We have used homeostasis model assessment for assessing insulin resistance.

HOMA-IR was calculated as

Fasting blood glucose (mg/dl) x Fasting serum insulin (µIU/ml) / 405.

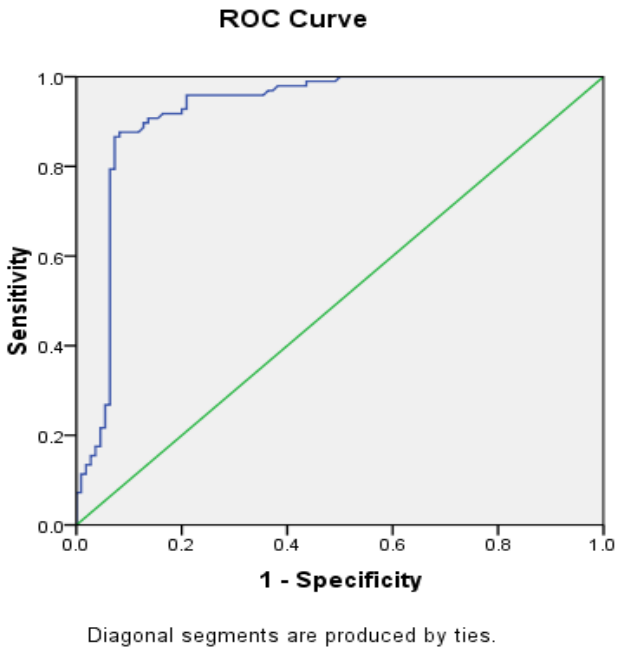

Fig No 4: ROC curve analyses of HOMA-IR values for Bank Employees

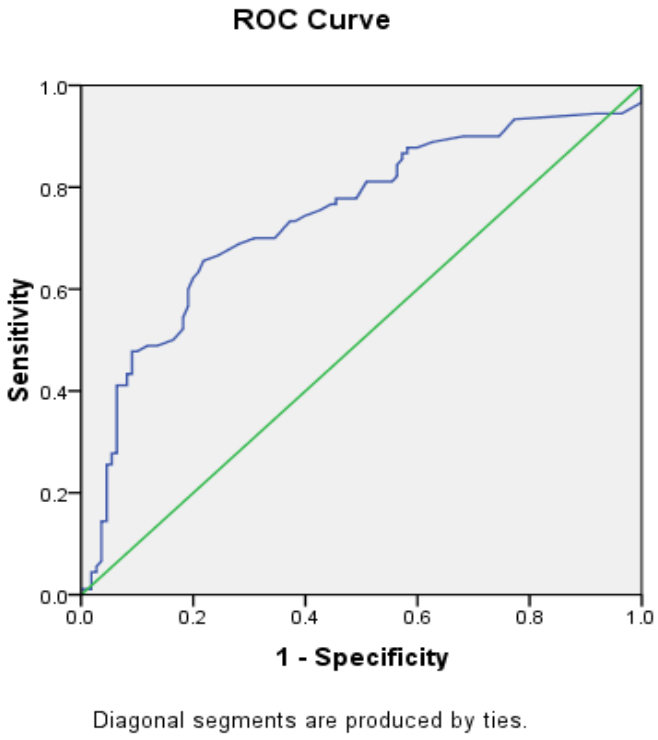

Fig No 5: ROC curve analyses of HOMA-IR values for Bus Drivers

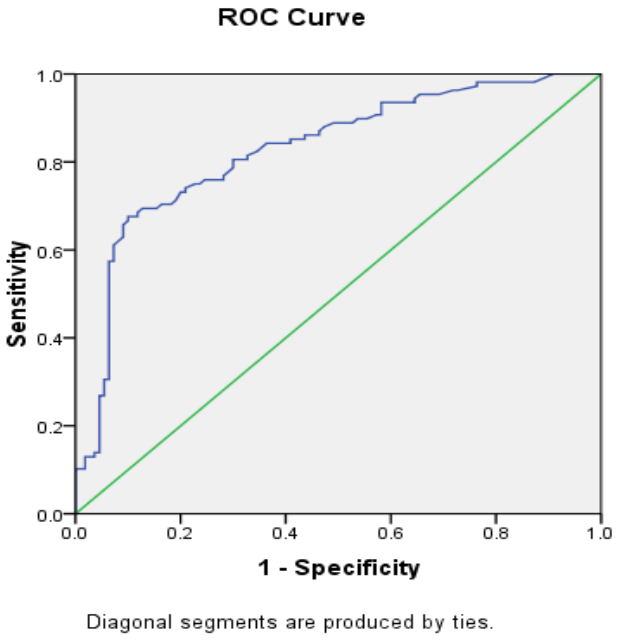

Fig No 6: ROC curve analyses of HOMA-IR values for Police constables

Table No 60: HOMA-IR for study groups

Groups	Area under curve for HOMA-IR ROC	SE
Bank employees	0.92	0.021
Drivers	0.68	0.04
Police constables	0.82	0.028

Figures 4 5 & 6 and table 60 shows the ROC curves and area under curve of HOMA-IR value in different study groups. It shows that insulin resistance is more evident among bank employees than drivers and police constables with compare to control population.

Table No 61: Distribution of HOMA-IR value by selected characteristics

Basic Variables	Bank Employees				Drivers				Police Constables				Controls			
	N	Mean	SD	p value	N	Mean	SD	p value	N	Mean	SD	p value	N	Mean	SD	p value
Age																
<=30	3	6.7	5.3	>0.05	3	1.1	0.5	>0.05	21	3.6	5.0	>0.05	1	0.6	NA	>0.05
31-40	9	2.7	0.9		18	3.4	4.3		26	5.0	5.6		23	1.4	1.6	
41-50	50	4.5	3.7		52	3.8	3.7		27	6.1	5.5		51	1.5	2.1	
>50	35	5.4	4.2		17	3.7	3.2		34	4.0	3.7		35	1.5	2.0	
BMI																
Normal	28	4.7	3.7	>0.05	17	2.7	2.9	>0.05	11	4.0	5.1	>0.05	54	1.6	2.1	>0.05
Overweight	18	3.5	2.2		26	4.5	4.3		22	3.7	4.6		18	1.0	0.3	
Obese	48	5.1	4.2		42	3.8	3.6		75	5.1	5.0		32	1.6	2.3	
Waist circumference																
<90cm	38	4.3	3.4	>0.05	41	2.4	2.7	<0.05	87	4.4	4.7	>0.05	110	1.5	1.9	NA
>=90cm	59	5.0	4.0		49	4.6	4.1		21	5.7	5.8		0	NA		
Waist hip ratio																
<0.9	24	4.0	3.4	>0.05	25	3.0	3.2	>0.05	7	7.7	8.1	>0.05	92	1.6	2.1	>0.05
>=0.9	73	5.0	3.9		65	3.8	3.9		101	4.5	4.6		18	1.1	0.3	
Stress																
No	67	4.2	3.5	<0.05	54	2.8	3.0	<0.05	67	4.3	4.6	>0.05	98	1.5	2.0	>0.05
Yes	30	5.9	4.2		36	4.8	4.3		41	5.3	5.4		1	0.6	NA	
BP																
>130/85	33	4.8	3.4	>0.05	26	4.3	3.7	>0.05	33	5.0	5.3	>0.05	13	2.7	3.3	>0.05
<=130/85	64	4.8	4.1		64	3.4	3.8		75	4.6	4.9		97	1.4	1.7	
Blood glucose																
<=99	59	3.1	2.1	>0.05	63	1.5	1.1	>0.05	68	3.4	4.3	>0.05	66	1.0	0.3	>0.05
100-125	21	6.6	3.9		11	6.8	1.2		14	8.4	5.5		35	1.2	0.7	
>125	17	8.5	4.9		16	10.0	2.7		26	6.1	5.2		9	6.6	4.2	

Table No 62: Distribution of HOMA-IR value by selected characteristics

Biochemical Variables	Bank Employees				Drivers				Police constables				Controls			
	N	Mean	SD	p value	N	Mean	SD	p value	N	Mean	SD	p value	N	Mean	SD	p value
HDL																
<40	51	4.9	4.0	>0.05	39	4.5	4.4	>0.05	84	5.1	5.3	>0.05	17	0.9	0.3	>0.05
>=40	46	4.6	3.6		51	3.0	3.0		24	3.5	3.7		93	1.6	2.1	
Cortisol																
<=25	90	4.5	3.5	<0.05	65	3.0	3.2	<0.05	58	5.4	5.9	<0.05	106	1.5	2.0	>0.05
>25	7	7.9	6.7		25	5.3	4.6		50	3.9	3.5		4	1.2	0.3	
CRP																
<1	58	4.6	3.7	>0.05	67	3.3	3.7	>0.05	83	4.4	4.9	>0.05	105	1.5	2.0	>0.05
>=1	39	5.1	4.1		23	4.6	3.7		25	5.7	5.4		5	1.2	0.5	
HCY																
<14	62	4.6	3.7	>0.05	72	3.3	3.7	>0.05	73	3.9	4.1	<0.05	110	1.5	2.0	>0.05
>=14	35	5.1	4.1		18	4.8	3.9		35	6.5	6.2		0	#	#	
LP(a)																
<=25	78	4.8	3.8	>0.05	82	3.4	3.6	>0.05	95	4.6	4.7	>0.05	110	1.5	2.0	>0.05
>25	19	4.9	3.9		8	6.4	4.1		13	5.9	7.0		0	#	#	
TRIG																
<=150	68	4.7	3.7	>0.05	62	2.9	3.0	>0.05	65	4.3	4.9	>0.05	93	1.5	2.0	>0.05
>150	29	5.0	4.2		28	5.3	4.7		43	5.4	5.2		17	1.6	1.9	
MA																
<20	67	4.8	4.0	>0.05	43	2.8	2.4	<0.05	23	5.0	6.3	>0.05	93	1.5	2.0	>0.05
20-30	12	4.3	3.2		30	3.8	4.4		41	4.4	4.3		15	1.6	1.8	
>30	18	4.9	3.8		17	5.4	4.8		44	4.8	4.9		2	1.0	0.4	
Metabolic Syndrome																
No	60	4.2	3.2	<0.05	57	1.8	1.7	<0.05	67	3.9	4.5	<0.05	97	1.5	1.9	>0.05
Yes	37	5.7	4.6		33	6.8	4.3		41	6.1	5.4		13	1.7	2.4	

Note: p-value of 0.05 or less was considered for statistical significance using χ^2 test of association.

Table 61 and 62 interprets the distribution of HOMA-IRvalues by selected characteristics. Mean HOMA-IR is higher among middle aged people in all groups. While among obese people, it is having higher values. With Waist circumference >=90 Cm. HOMA-IR values are significantly raised. Stress also plays significant role in increasing HOMA-IR values with presence of stress. With cortisol >25, the mean HOMA-IR is significantly higher among occupational groups. Microalbuminin significantly directly proportional to HOMA-IRvalues among bus drivers.

Table No 63: Correlation between HOMA-IR and other variables in different occupational groups

Variables	Group I		Group II		Group III		Group IV	
	r	p value	r	p value	r	p value	r	p value
Number	110		97		90		108	
Age.	-0.027	0.780	0.169	0.099	0.121	0.256	-0.027	0.781
Waist Circumference	0.103	0.285	0.101	0.327	.254*	0.016	0.040	0.682
Systolic blood pressure	0.050	0.601	0.047	0.646	0.145	0.172	0.120	0.216
Diastolic blood pressure	0.132	0.168	0.054	0.599	0.069	0.520	0.118	0.223
Fasting blood glucose	0.591***	0.000	0.574***	0.000	0.914***	0.000	0.275**	0.004
Glycosylated. HbA1C	0.563***	0.000	0.135	0.189	0.741***	0.000	0.083	0.393
Triglyceride	0.180	0.060	-0.025	0.812	0.393***	0.000	0.059	0.547
HDL Cholesterol	0.119	0.216	-0.037	0.716	-0.166	0.118	-0.019	0.847
Cortisol	-0.007	0.939	0.346**	0.001	0.227*	0.031	-0.173	0.074
Homocysteine	-0.067	0.489	0.051	0.623	0.240*	0.022	0.230*	0.017
Microalbumin/ gm of Creat.	-0.009	0.928	0.047	0.650	0.320**	0.002	-0.069	0.476

* p<0.05, **p<0.01, ***p<0.001

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police constables]

Table 63 shows that Fasting blood glucose and blood pressure were found to be significantly correlated with HOMA-IR among controls Cortisol and fasting blood glucose were significantly correlated with HOMA-IR among Bank Employees while Waist circumference, Triglyceride, Homocysteine,Microalbuminand fasting blood glucose were found to be significantly correlated with HOMA-IR among the bus drivers. Among policemen, Homocysteine and fasting blood glucose were found to be significantly correlated with HOMA-IR.

4.3.1 Discussion

Insulin resistance is a hallmark of obesity, diabetes and cardiovascular disease and leads to many abnormalities associated with Cardiometabolic syndrome. The quantitative measurement of insulin is not routinely used during biochemical investigations for diagnostics purposes. The emerging importance of insulin resistance has led to its wider application in research studies. The hyperinsulinaemic euglycaemic glucose clamp is the golden standard method for determination of insulin sensitivity (De fronzo RA et al., 1979). But it is impractical as it is laborious and time consuming. Insulin resistance is measured indirectly by HOMA-IR-IR. Majority of research workers consider HOMA-IR as the standard method for measuring insulin resistance (Singh B et al., 2010).

In the present study, HOMA-IR was taken for measuring insulin resistance. HOMA-IR was first developed in 1985 (Mathews et al., 1985). HOMA-IR is a model of the relationship of glucose and insulin dynamics that predicts fasting steady state of glucose and insulin concentration for a wide range of possible combinations of insulin resistance and β- cell function. The HOMA-IR model has proved to be a robust clinical and epidemiological tool for the assessment of insulin resistance (Singh B et al., 2010). In our study the participants were grouped as metabolic and non-metabolic syndrome subjects based on guidelines given by NCEP-ATP III. We found that the HOMA-IR level was significantly raised in the metabolic syndrome group of bus drivers and police constables. Similar findings were reported from other authors also (Maheria P et al., 2011, Chizumi Yet al., 2012).

Our study revealed that there was a positive correlation coefficient ($r= 0.254$, $p<0.05$) between HOMA-IR and waist circumference in the bus drivers. Other occupational groups did not show any such type of correlation in the study. However there are the reports which recommend the use of BMI and waist to hip ratio in the assessment of HOMA-IR. A study from Israel demonstrated a positive correlation between BMI and insulin (Assail et al. 2001). Another study from India, concluded that Increased waist to hip ratio and BMI were the clinical parameters for insulin resistance (HOMA-IR) (Chaudhari SP et al., 2012).

The correlation between metabolic syndrome and hypertension is reported though it is not the case always (Bjorntorp et al., 2000). Resistance to insulin mediated glucose disposal and compensatory hyperinsulinemia are common in patients with hypertension. However, not all hypertensive patients have the insulin resistance.

Several mechanisms appear to be involved in the link between hypertension and insulin resistance, involving sympathetic nervous system (Anderson, 1993), renal handling of sodium (Defronzo RA et al., 1976) and vasoconstrictor hormones(Galipeau D et al., 2001). In our study, we found significantly increased values of systolic and diastolic blood pressure in the bank employees, police constables and control subjects with metabolic syndrome.

However in control subjects with metabolic syndrome, the both systolic and diastolic blood pressure levels were within the normal limits. In our study we did not find any positive correlation coefficient between HOMA-IR and blood pressure (systolic and diastolic) of the participants of all the four study groups.

A positive correlation was found between fasting blood glucose and HOMA-IR among all the four groups and the relation was statistically significant. Similar findings were reported by others also (Aydin E, et al., 2014, Kohei O et al., 2013).In our study,there was a statistically significant positive coefficient correlation between Glycosylated HbA1c and HOMA-IR values in bus drivers and control subjects.Glycosylated HbA1c has the potential to reflect the history of mean insulin sensitivity over the preceding weeks or months, and it serves as a marker of insulin sensitivity in children who have a normal glucose tolerance (Borai A et al ., 2011).Glycosylated HbA1c levels can be used as a diagnostic tool for the early detection of insulin resistance.The screening of GlycosylatedHbA1c is beneficial to evaluate insulin sensitivity in the study population. Also it is a cheaper and easy method. Further there is no need of fasting sample.

Both high Triglycerides and low HDL-C are widely known to be associated with obesity and other features that define the metabolic syndrome. However, it is possible that much of the cardiovascular disease that is associated with the metabolic syndrome may be explained by the presence of insulin resistance (Grundy SM et al., 2004). The common underlying problem in development of insulin resistance is the body's excessive burden of fatty acids (Savage DB, 2007). In population studies, insulin resistance has been shown to independently predict both the development of a high-Triglyceride, low-HDL-C dyslipidemia and new cardio vascular disease in large general populations (Bonora E et al., 2007,Despres JP et al., 1996).

A study undertaken to examine the effect of insulin resistance (IR) in subjects without diabetes on the relationship of a dyslipidemia with high Triglycerides and low high-density

lipoprotein cholesterol (HDL-C) to the development of coronary heart disease (CHD), it was reported that incidences of coronary heart disease were more in presence of insulin resistance along with either lower HDL-Cholesterol or higher Triglyceride values(Sander J et al., 2011).

An Indian study undertaken to find association of obesity and insulin resistance with dyslipidemia in Indian women with polycystic ovarian syndrome, reported that Insulin resistance was associated with dyslipidemia in women with PCOS, independent ofobesity (Kalra A et al., 2006). In our study there was a significant rise in the level of Triglyceride accompanied with lowered HDL cholesterol level in the subjects with metabolic syndrome of bank employees, bus drivers and police constables. We found that there was a negative correlation between HOMA-IRand HDL cholesterol in the bank employees, bus drivers and police constables. There was a significant positive correlation between HOMA-IRand Triglyceride levels of bus drivers. Similar observations were reported by others also (Rashid S et al., 2003).

The area under curve (AUC) is a combined measure of sensitivity and specificity. AUC is a measure of overall performance of a diagnostic test and is interpreted as the average value of sensitivity for all possible values of specificity (Obuchowski NA., 2003). In our study,the area under curves in the ROC(Receiver operating characteristics)analysis was 0.92 for bank employees, 0.82 for police constables and for bus drivers 0.68. It indicates that insulin resistance was more prominent among bank employees than police constables and bus drivers with comparison to control subjects. There is a chance that present marker HOMA-IR is not 100 % capturing the prevalence of insulin resistance scenario.

4.4 BODY MASS INDICES IN DIFFERENT OCCUPATIONAL GROUPS

The obesity of the participants was assessed by measuring their waist circumference, waist to hip ratio and BMI was calculated by the formula

BMI= weight in kg / (height in meters) ^{2}.

And waist to hip ratio by

WHR (waist to hip ratio) = waist circumference (cm) / hip circumference.

The Correlation was calculated for few selected parameters in all the four groups. The participants who had their Waist circumference ≥ 90 cm, BMI ≥ 25 and WHR ≥ 0.90 were considered.

Table No 64: Pearson correlation coefficient (r) between waist circumference and selected Characteristics

	Group I (no=16)		Group II (no=41)		Group III (no=52)		Group IV (no=24)	
Variables	'r'	p Value	'r'	p Value	'r'	p Value	'r'	p Value
BMI	0.179	0.507	0.607	0.000***	0.123	0.386	0.435	0.034*
WHR	-0.376	0.152	0.481	0.000***	0.588	0.000***	-0.196	0.358
Systolic blood pressure	0.363	0.167	0.371	0.003**	0.233	0.112	0.214	0.315
Diastolic blood pressure	0.031	0.909	0.283	0.025*	0.347	0.012*	-0.017	0.937
Fasting blood glucose	-0.044	0.871	0.164	0.199	-0.017	0.905	-0.155	0.469
Triglyceride	-0.176	0.514	-0.075	0.558	-0.193	0.169	-0.233	0.273
HDL Cholesterol	0.314	0.237	0.176	0.167	0.001	0.997	-0.203	0.341
Insulin	-0.162	0.549	0.006	0.962	0.088	0.537	0.239	0.261
HOMA-IR	-0.172	0.525	0.089	0.489	0.023	0.869	0.174	0.415

* p<0.05, **p<0.01, ***p<0.001

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police Constables]

Table 64 shows the Correlation between waist circumference and selected characteristics in bank employees, bus drivers, police constables and controls. Statistically significant positive correlation was observed between waist circumference and BMI (r= 0.607, p< 0.001), waist

to hip ratio(r= 0.481, p< 0.001), systolic blood pressure(r= 0.371, p< 0.01), diastolic blood pressure (r=0.283, p< 0.05) in bank employees.

In bus drivers statistically significant positive correlation was found with waist to hip ratio, (r= 0.588, p<0.001) and diastolic blood pressure (r=0.347, p<0.05), and in police constables with BMI (r=0.435, p< 0.05). Also positive association between waist circumference and systolic blood pressure of bus drivers, police constables and in control subjects was observed.

Table No 65: Correlation between BMI and other variables

	Controls (no=32)		Bank employees (no=48)		Bus drivers (no=42)		Police constables(no=75)	
Variables	'r'	p Value	'r'	p Value	'r'	p Value	'r'	p Value
Waist Circumference	-0.071	0.698	0.344	0.017*	-0.077	0.628	0.453	0.000***
WHR	-0.190	0.298	0.159	0.280	-0.296	0.057	0.109	0.358
Systolic blood pressure	-0.135	0.460	0.199	0.174	-0.042	0.791	-0.177	0.129
Diastolic blood pressure	0.098	0.595	0.073	0.620	-0.091	0.508	-0.244	0.035*
Fasting Blood Glucose	-0.128	0.485	0.082	0.577	-0.236	0.133	-0.102	0.384
Triglyceride	-0.010	0.957	-0.014	0.924	-0.278	0.074	0.136	0.246
HDL Cholesterol	-0.043	0.186	0.185	0.208	0.130	0.410	-0.101	0.387
Insulin	-0.180	0.325	-0.012	0.890	-0.191	0.225	0.033	0.781
HOMA-IR	-0.181	0.321	0.044	0.766	-0.249	0.112	-0.015	0.896

* p<0.05, **p<0.01, ***p<0.001

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police Constables]

Table 65 depicts about Correlation between BMI and selected characteristics in bank employees, bus drivers, police constables and controls. There was a statistically significant positive correlation between BMI and waist circumference (r=0.344, p<0.05) of bank employees, waist circumference (r=0.453, p< 0.001) of police constables. There was a statistical significant negative correlation between BMI and diastolic blood pressure (r= -0.244, p< 0.05) in police constables.

Table No 66: Correlation between waist-hip ratio (WHR) and selected Parameters

	Group I (no=20)		Group II (no=76)		Group III (no=68)		Group IV (no=101)	
Variables	'r'	p Value	'r'	p Value	'r'	p Value	'r'	p Value
BMI	-0.123	0.606	0.139	0.230	-0.210	0.085	-0.283	0.019*
Waist Circumference	-0.037	0.877	0.218	0.059	0.178	0.147	-0.428	0.000***
Systolic blood pressure	-0.136	0.567	-0.027	0.819	0.014	0.909	0.051	0.613
Diastolic blood pressure	0.007	0.975	0.026	0.823	0.090	0.467	0.142	0.157
Fasting blood glucose	0.156	0.512	0.170	0.141	-0.075	0.542	0.015	0.878
Triglyceride	0.139	0558	0.151	0.194	-0.129	0.295	-0.132	0.187
HDLCholesterol	-0.185	0.436	0.158	0.172	-0.024	0.848	0.110	0.274
Insulin	-0.214	0.365	-0.072	0.537	-0.006	0.964	-0.083	0.409
HOMA-IR	-0.213	0.366	0.051	0.662	-0.061	0.622	0.042	0.674

* $p<0.05$, ** $p<0.01$, *** $p<0.001$

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police Constables]

Table 66 presents Correlation between waist to hip ratio and selected characteristics in bank employees, bus drivers, police constables and controls. It was observed that there was a statistically significant negative correlation between waist to hip ratio and waist circumference ($r= - 0.428$, $p<0.001$) in police constables.

4.4.1 Discussion

Obesity is an important determinant of cardiovascular disease (CVD). The central or visceral type is a predisposing factor for the development of Type 2- diabetes mellitus and hypertension. The prevalence of obesity has increased gradually among adolescents also (Yin-Ming Li et al., 2005).A recent report by the World Health Organization concluded that, where possible, abdominal obesity should also be measured and used in conjunction with BMI to assess and predict disease risk(WHO, 2011).In 2005, the International Diabetes Federation modified the metabolic syndrome criteria with an emphasis on abdominal adiposity as a necessary condition to be met, in addition to a minimum of two of the other four criteria (Alberti KG et al .,2006).

The risk factors mentioned for metabolic syndrome are potential for the development of CVD and type-2 diabetes. Of all the factors, obesity has been shown to be the most important index. Health professionals have noticed that overweight and obesity are closely related to the development of metabolic syndrome. At the same time, they have also noticed that many Asian races are more susceptible in developing abdominal obesity even with normal BMI. We found that there was there was a statistical significant Pearson correlation coefficient (r) between waist circumference and BMI (r= 0.607,p<0.001), WHR(r=0 .481,) p< 0.001), systolic blood pressure (r=0.371,p< 0.01), diastolic blood pressure (r= 0.283, p< 0.05) among bank employees. Similar positive correlation was found between waist circumference and waist to hip ratio (r=0.588, p<0.001 and diastolic blood pressure (r= 0.347, p< 0.05) in bus drivers.

Abdominal obesity is the most frequently observed component of metabolic syndrome. It was suggested that WC is a better predictor of metabolic risk factors for developing metabolic syndrome than BMI and propose thatmetabolic risk factors should be screened in the measured WC value in both genders exceeding 80 cm, regardless of BMI (Aye M et al., 2012). In another study it was concluded that waist circumference (WC) is a convenient measure of abdominal adipose tissue, which is a diabetes risk factor and is strongly linked to other cardiovascular disease (CVD) risk factors (Bouguerra et al., 2007). However, in a study conducted on south Indian population it was found that WHR be the best predictor for type-2 diabetes (Kaur P et al., 2008).

In our study We found that 10.9% subjects had BMI > 30 Kg / m2; 40.3% subjects had BMI between 25-29.9; 8.8% increased waist circumference > 102 cm and 26.1 % Waist circumference between 90 to 102 cm.The studies provide the evidence that measures of BMI andabdominal obesity are associated with increased prevalence of CVD risk factors (Margot S et al., 2012).

4.5 PREVALENCE OF METABOLIC SYNDROME

In the present study, we have adopted the NCEP ATP III guidelines to identify the participants with the metabolic syndrome. The presence of any three abnormalities out of five listed below.

1. Waist circumference: > 90.0 cm.
2. Blood pressure > 130 / 85 mmHg.
3. Serum Triglyceride: > 150.0 mg/dl.
4. Serum HDL Cholesterol : < 40.0 mg/dl
5. Fasting blood glucose: 100.0 mg /dl.

Table No: 67 Demographic characteristic of different study groups

Characteristics	Bank Employee (N=97)	Bus Drivers (N=90)	Police constables (N=108)	Control (N=110)	Total (N=405)	p value
Age (Years)	47.28±0.71[b,c]	44.60±0.71[d,e]	42.17±1.00[f]	46.52±0.73	45.11±0.41	0.000
Waist circumference (Cm)	91.71±0.99[b,c]	91.19±1.56[d,e]	81.33±1.00[f]	74.83±0.69	84.24±0.64	0.000
Systolic blood pressure mmHg	125.28±1.11[c]	124.56±1.06[e]	123.06±1.18	120.52±0.78	123.23±0.53	0.006
Diastolic blood pressure mmHg	82.04±1.02[c]	80.91±0.86	79.96±0.92	78.17±0.62	80.19±0.43	0.011
Fasting blood glucose (mg/dl)	117.90±3.95[c]	107.36±5.37	115.09±4.42[f]	97.96±1.08	109.39±1.98	0.001
Triglyceride (mg/dl)	137.52±9.24	148.43±8.81[e]	151.58±9.31[f]	117.94±2.68	138.38±3.97	0.009
HDL Cholesterol (mg/dl)	39.78±0.89[b,c]	42.22±1.05[d,e]	36.31±0.62[f]	47.38±0.60	41.46±0.44	0.000

Values are expressed as mean ± SEM. ANOVA followed by Post Hoc Tukey's multiple comparison test. Superscript 'a, b, c, d, e, f' express the significant difference between (1,2), (1,3), (1,4), (2,3), (2,4), (3,4) groups respectively.

The table 67 shows the demographic characteristics of the participants of different study groups. These were the components of metabolic syndrome involved in the study. There was a significant difference in the levels of the parameters on comparison to control group.

Fig No7: Prevalence of risk factors of metabolic syndrome in Bank Employees

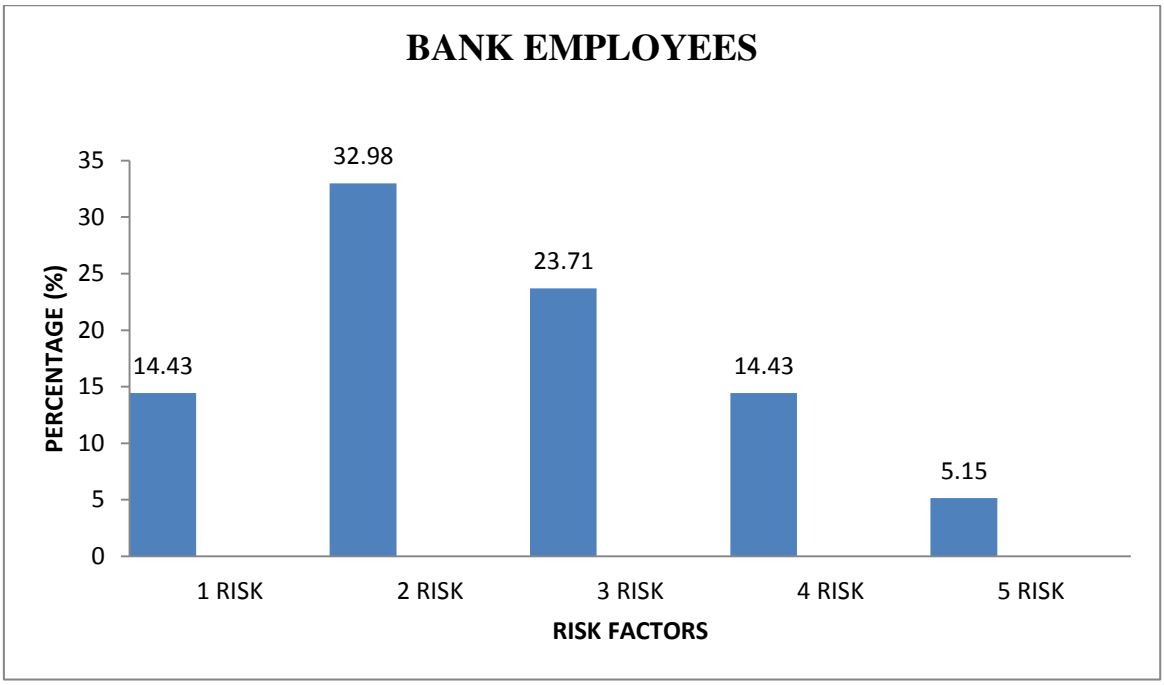

Fig No 8:Prevalence of risk factors of metabolic syndrome inBus drivers

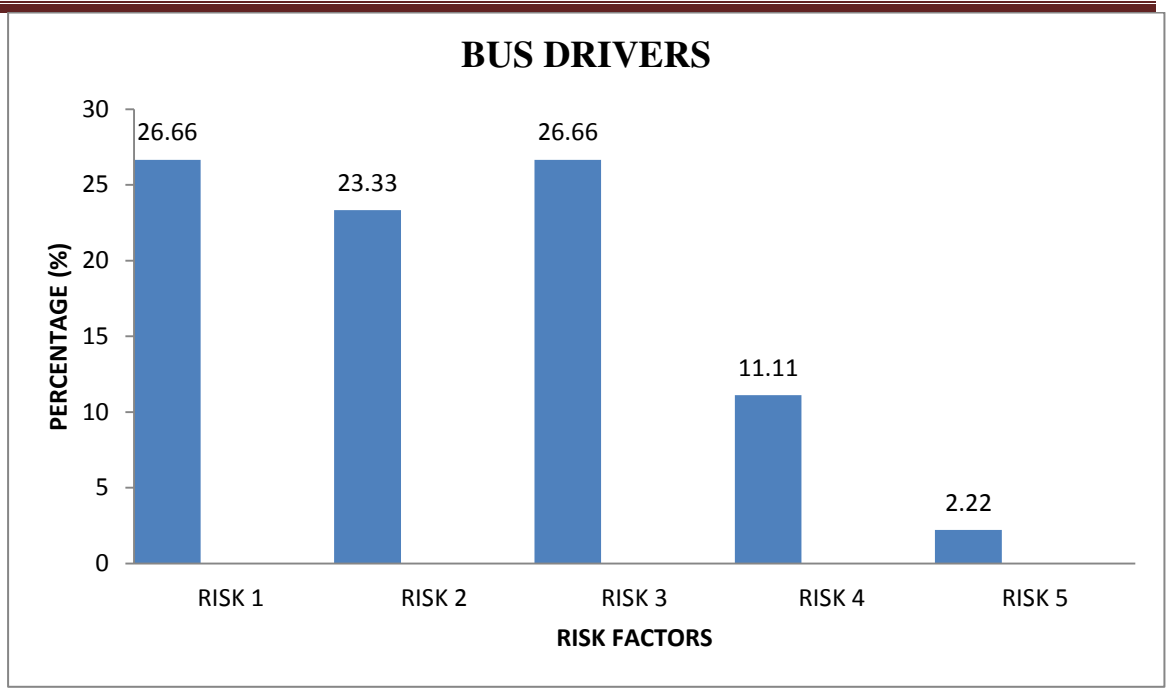

Fig No 9: Prevalence of risk factors of metabolic syndrome in police constables

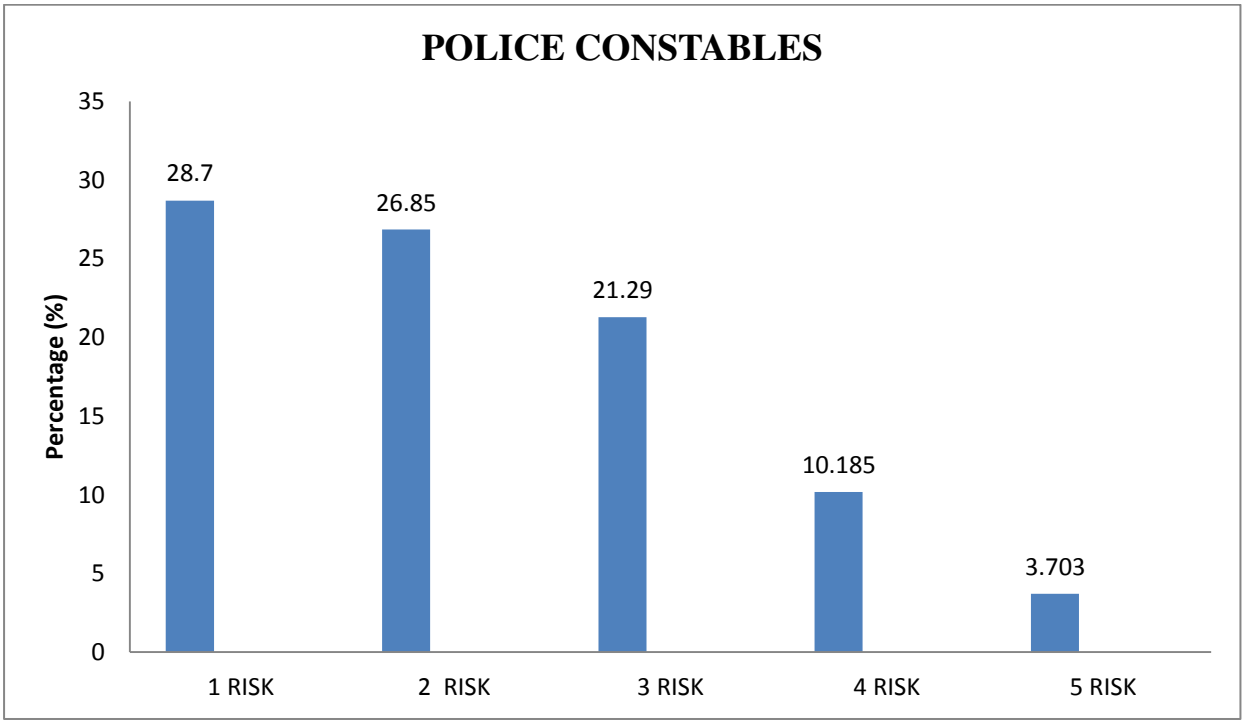

Fig No 10: Prevalence of risk factors of metabolic syndrome in control subjects

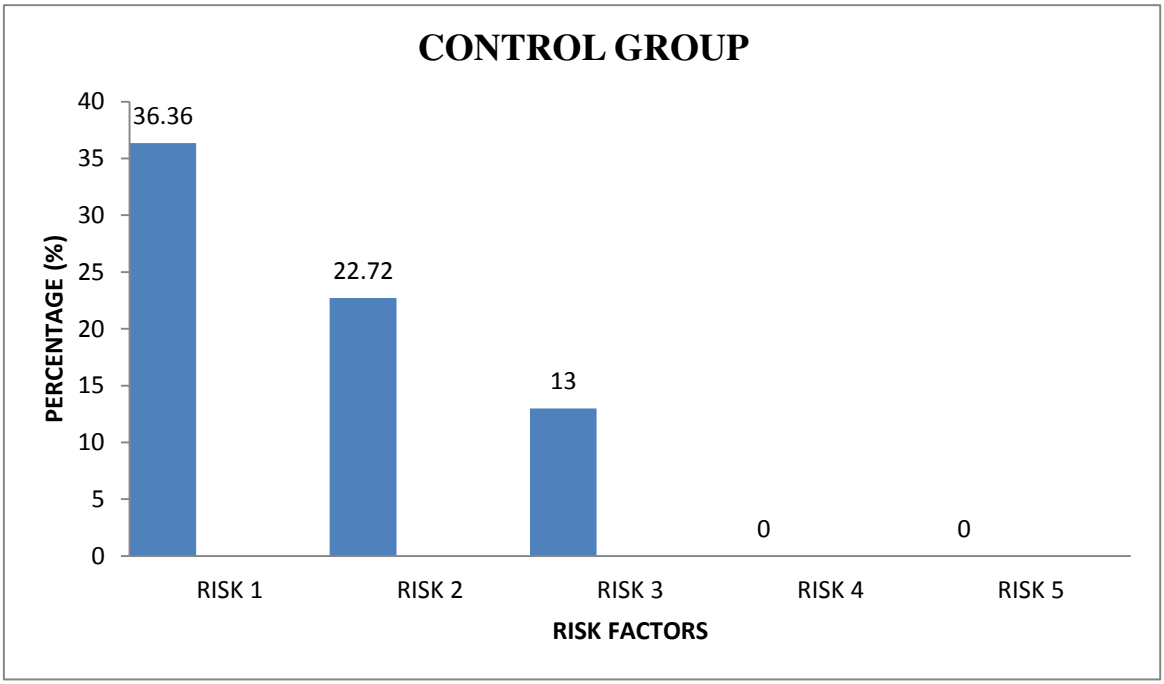

Figure 7 to 10 indicate the prevalence of clustering of risk factors(in percentage) in bank employees, bus drivers, police constables and control subjects respectively. All the four groups demonstrated the more prominence of one or two risk factor.

Fig No: 11. The prevalence rate of Metabolic syndrome as per NCEP ATP III.

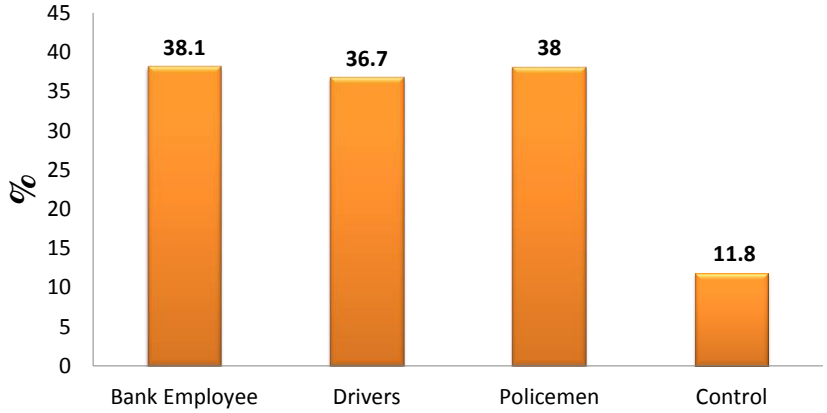

Fig No:12 Metabolic Syndrome rate by age groups among study groups

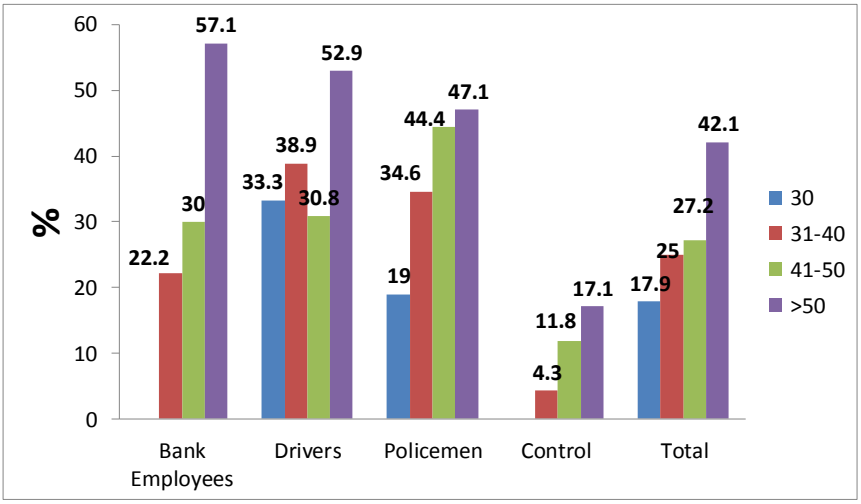

Fig no.13 The prevalence rate of Metabolic syndrome as per different Guidelines

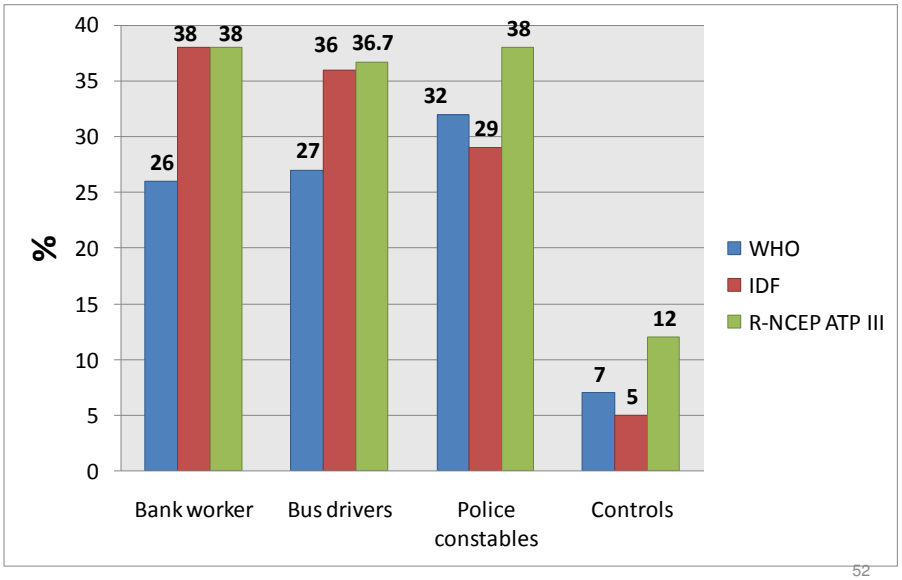

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Figure 11 depicts the prevalence rate of metabolic syndrome in all the four studied groups as per NCEP ATP III. Figure 12 shows the prevalence of metabolic syndrome as per Age groups in all the four studied groups. In all the four groups the prevalence rate of metabolic syndrome was found to be higher in the age group of above 50 years. Figure 13 indicate the prevalence rate of metabolic syndrome as per WHO, IDF, R-NCEP ATP III.The prevalence

rate was found comparatively to be in lowered in all the three occupational groups as per WHO Guidelines.

4.5.1 Microalbuminas a risk factor for metabolic syndrome.

Table No 68: Correlation between Microalbumin and other selectedvariables in all the studied groups.

Variables	**Group I (no=110)**		**Group II (no= 97)**		**Group III (no=90)**		**Group IV (no=108)**	
	'r'	p	'r'	p	'r'	p	'r'	p
Age	-0.007	0.943	0.226	0.026*	0.035	0.742	0.100	0.306
BMI	0.180	0.061	0.029	0.778	-0.063	0.557	-0.031	0.750
Waist Circumference	0.139	0.149	0.192	0.060	0.173	0.104	-0.054	0.582
Waist to hip ratio	0.070	0.469	0.115	0.262	0.138	0.195	0.152	0.116
Systolic Blood pressure	-0.022	0.818	0.227	0.025*	0.055	0.607	0.221	0.021*
Diastolic Blood pressure	-0.028	0.773	0.165	0.106	0.109	0.307	0.214	0.026*
Fasting Blood Glucose	0.139	0.147	0.147	0.149	0.355	0.001**	0.307	0.001**
Glycosylated HbA1c	-0.006	0.950	0.152	0.137	0.408	0.000***	0.303	0.001**
Triglyceride	-0.129	0.181	0.211	0.038*	0.545	0.000***	0.099	0.306
HDL Cholesterol	-0.013	0.895	0.035	0.733	-0.125	0.242	-0.114	0.242
Cortisol	0.023	0.814	0.026	0.803	0.103	0.334	0.212	0.028*
HOMA-IR-IR	-0.009	0.928	0.047	0.650	0.320	0.002**	-0.069	0.476

[Group I= Control, Group II=Bank employees, Group III= Bus drivers, Group IV= Police Constables]

Table 71 shows Correlation between Microalbumin and other selectedvariables in all the studied groups. There was no significant association between Microalbumin and the adiposity markers like BMI, WC and waist to hip ratio. A significant correlation was found between Microalbumin and systolic blood pressure (0.227, $p<0.05$) of bank employees and police constables (0.221, $p<0.05$) respectively. Fasting blood glucose and Glycosylated HbA1cwere found to be significantly correlated with bus drivers and police constables. Triglyceride and

HOMA-IR were significantly correlated with micro albumin among bus drivers. Serum Cortisol was found to be significantly correlated with Microalbumin in police constables.

4.5.2 Discussion

In our study, we found that the prevalence rate of Cardiometabolic syndrome was 38 %. The lowered HDL cholesterol raised waist circumference and elevated Triglyceride levels significantly contributed to an increased risk of Cardiometabolic syndrome among all the groups in our study. Low HDL Cholesterol is very common among Asian Indians and is corroborated also by others (Misra N et al., 2009)

The prevalence of metabolic syndrome is increasing exponentially in India, both in the urban and rural areas. The differences in the prevalence of metabolic syndrome between studies from Indian subcontinent may be attributed to different criteria employed, different age groups included, and different rates of prevalence of individual components of the metabolic syndrome. Earlier studies across urban south India documented that prevalence are ranging from 22.1% to 41% (Tharkar S et al., 2010; Vasan SK et al., 2011;Ramachandran A et al., 2003)which is comparable with our observation of 38.0%.

Studies have shown that the prevalence of the metabolic syndrome varies in different occupational groups (Sanchez-Chaparro MA et al., 2008; Davila EP et al., 2010). Among U.S. workers, the unadjusted and age-adjusted prevalences of the metabolic syndrome were greatest in “transportation and material occupations” and “food preparation and food service workers,” respectively (Davila EP et al., 2010).A study from Korea demonstrated differences in the prevalence of the metabolic syndrome by occupational group and identified the greatest risk for the metabolic syndrome in male non-manual workers (Ryu JY et al., 2013). A study concentrating on the factors like late working hours, shift duties, shorter sleep duration and others concluded that these may be important contributors to the metabolic syndrome among police officers(Violanti JM et al., 2009).Bank employees have sedentary lifestyle; relatively bettersocioeconomic condition and stressful job are subject to the risk of coronary heart disease(Laxmikant Lokare et al., 2012). A study conducted at Brazil on bank employees emphasized on urgent prevention and intervention program which are needed to curb the alarming increase in DM and pre-diabetes (Thierry G et al., 2014).

Prevalence of smoking and alcohol consumption was more in police constables comparing to other study groups in our study which was comparable with other studies (Tharkar S et al., 2008, ThayyilJ et al., 2012). The low level physical activity was found more among bus drivers in our study. It may be because of their unscheduled duty hours and as they leave

their home town on duty on several times in the week. Lack of awareness about importance of physical activity may be also another reason for similar findings found among the participants of other study groups.

Our results show that the prevalence of metabolic syndrome rises with age. This finding was more prominent in bank employees followed by bus drivers. We reported the prevalence rate of metabolic syndrome by applying NCEP ATP III guide lines. By applying IDF guideline the prevalence rate was also almost 38%.Using a similar definition (IDF), (Iwasaki T,et al .,2008) reported the prevalence of metabolic syndrome to be 58.5%. In our study, as per WHO guidelines, the prevalence rate of metabolic syndrome was between 26- 29 % in the three studied occupational groups. The WHO guideline suggests that the impaired glucose level should be one of the three criteria used to diagnose metabolic syndrome. In our study, comparing to control group the prevalence rate of metabolic syndrome is significantly high. We found that the NCEP ATP III or IDF are the better guidelines for the establishment of metabolic syndrome diagnosis.

The style of working differs from one occupation to another. But there was no much difference in the prevalence rate of metabolic syndrome among these occupational groups. A reason which can be speculated is that, as there is an option to select any three abnormalities out of five mentioned by NCEP ATP III, the difference in prevalence is minimized. However the existing individual risk factors should be assessed properly because each risk factor adds up to further complications.

The World Health Organization (WHO) has proposed a definition, which included the same components as the new NCEPATP III definition, with the addition of microalbuminuria (Alberti KG et al., 1998). Microalbuminuria is also known as a prognostic marker for cardiovascular risk in both diabetic and non-diabetic subjects (Hillege HL, et al., 2002). The metabolic syndrome is associated with an increased risk of both diabetes (Grundy SM, et al 2004) and cardiovascular disease (Lakka HM et al., 2002).The studies have investigated the relationship of microalbuminuria with the metabolic syndrome and its components (Palaniappan L et al., 2003, Hao Z et al., 2007). In the NHANES study, microalbuminuria was associated with high blood pressure and hyperglycemia ((Palaniappan L et al., 2003). In our study we found that there was a positive correlation between Microalbumin and systolic blood pressure among the bank employees and police constables. Fasting blood glucose and Glycosylated HbA1c significantly associated with Microalbumin in bus drivers and police

constables. An association between insulin resistance and microalbuminuria in Type 2-diabetes has often been found in cross-sectional studies (Hsu CC et al., 2011).

In our study there was a positive correlation between Microalbumin and HOMA-IR among bus drivers. We could not establish any association between adiposity markers like BMI, waist circumference, waist to hip ratio and Microalbumin in the whole study group collectively.

4.6 REFERENCES

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CHAPTER-V

SUMMARY AND CONCLUSION

CHAPTER V

SUMMARY AND CONCLUSION

5.1 SUMMARY AND CONCLUSION

The purpose of this study was to determine effect of occupational risk factors including stress in three different occupational groups.

We hypothesized that the stress at work place has a potential contribution towards the development of Cardiometabolic syndrome (Metabolic syndrome) and rate of prevalence differ from one occupation to another. We also hypothesized that rate of prevalence of metabolic syndrome differ as per the specific criteria applied while detecting the subjects with metabolic syndrome.

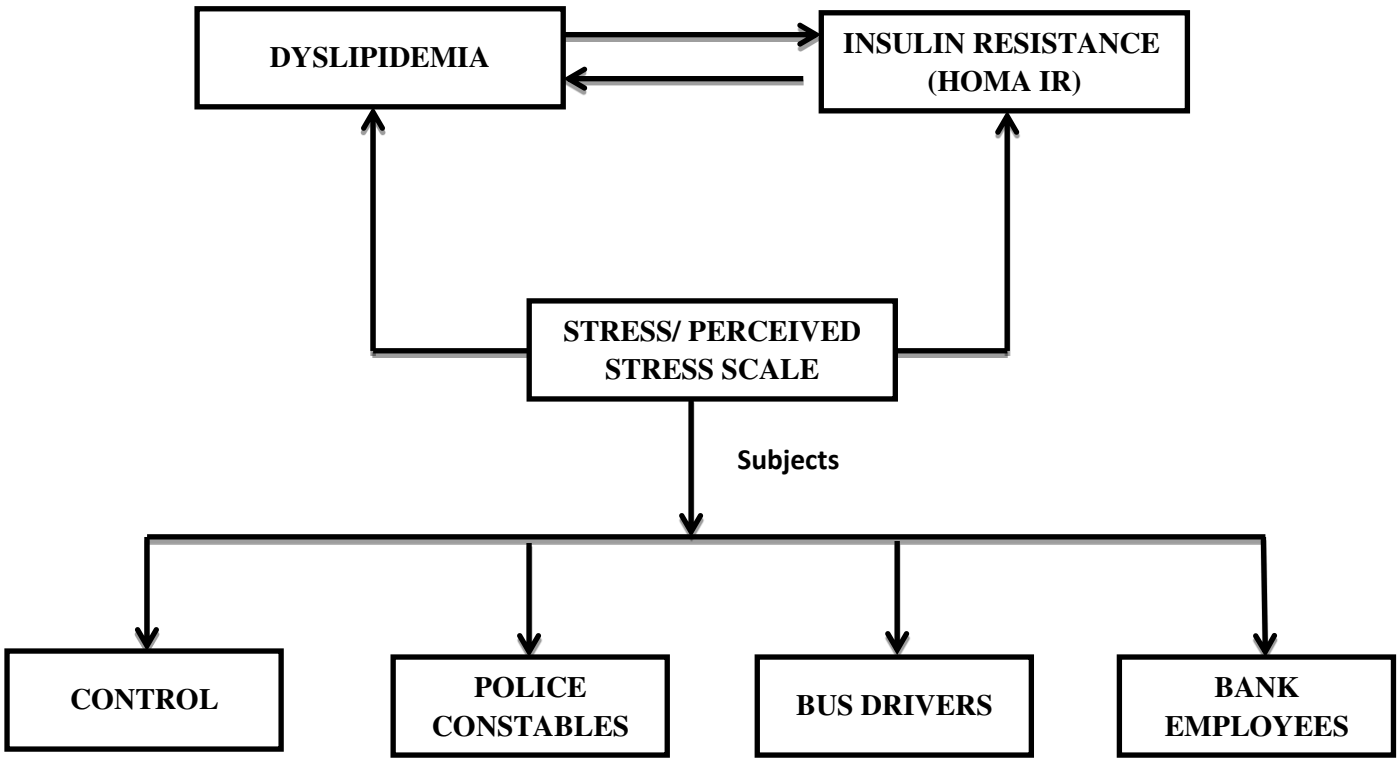

Figure No 14 : Summarized chart of occupational risk and stress which leads to metabolic syndrome

The objectives of the study were evaluated with a cross sectional study. The participants were bus drivers, bank employees and police constables. The subjects belonging to the occupations other than above three mentioned occupations were considered as control group. Totally 405 subjects took participation in this study.

The following parameters were tested for the study. Lipid profile: serum triglyceride, Total Cholesterol, HDL. Cholesterol, LDL Cholesterol, VLDL Cholesterol, Diabetic profile: fasting blood glucose, Glycosylated .HbA1c, fasting serum insulin, HOMA (Homeostasis model assessment), Stress assement: serum Cortisol and perceived stress scale (14-item questionnaire). Other parameters: Lp(a), Homocysteine, C-reactive protein and Microalbumin.

Anthropometric measurements, systolic blood pressure and diastolic blood pressure measurements were also the part of the study. Comparing to control group, the subjects from three occupations had elevated basic characteristics and biochemical parameters. The difference was statistically significant. We found that Serum cortisol ($p< 0.001$) and Perceived stress scale ($p < 0.001$) were increased significantly in the 'stressed' subjects of all the three occupations.

The stressed subjects from police constables group showed increased fasting blood glucose ($p<0.001$), glycosylated HbA1c ($p< 0.001$) and HOMA ($P< 0.05$) levels. Similarly the stressed subjects from bus drivers group presented raised fasting blood glucose, serum insulin and HOMA ($p< 0.05$).

A positive correlation coefficient (r) was observed between serum Cortisol and fasting blood glucose ($p< 0.001$), glycosylated HbA1c ($p<0.05$) in the group ' stressed' of bank employees. Similarly A positive correlation coefficient (r) was found between serum Cortisol and fasting blood glucose ($p< 0.001$), glycosylated .HbA1c ($p<0.05$) and perceived stress scale ($p< 0.001$) in the group 'stressed' of police constables.

There were no significant changes in serum triglyceride, Total cholesterol and HDL cholesterol in the participants classified as stressed and non-stressed from all the studied occupational groups.

In the present study, HOMA-IR was taken for measuring insulin resistance. Our study revealed that there was a positive correlation coefficient ($r= 0.254$, $p<0.05$) between HOMA-IR and waist circumference in the bus drivers. A positive correlation was found between fasting blood glucose and HOMA-IR among all the four groups and the relation was statistically significant. There was a statistically significant positive coefficient correlation between Glycosylated HbA1c and HOMA-IR values in bus drivers and control subjects ($p<0.001$).

In conclusion, we have observed that the serum Cortisol was associated significantly with perceived stress scale. We found a positive correlation coefficient between serum Cortisol and fasting blood glucose, glycosylated HbA1c in bank employees and police constables.

By adopting the NCEP ATP III guide lines, the participants were classified as subjects with metabolic and non-metabolic syndrome. In our study the overall prevalence of metabolic syndrome was 38 %.

Waist circumference level ($p< 0.001$) was significantly raised in the subjects with metabolic syndrome in bus drivers, bank employees and police constables. Systolic and diastolic blood pressure levels ($p< 0.001$) were significantly increased in the subjects with metabolic syndrome of bank service and police department.

Significantly increased levels of fasting blood glucose, glycosylated HbA1c, triglyceride ($p<0.001$) and lowered HDL cholesterol ($p< 0.05$) were found in subjects with metabolic syndrome subgroups of police constables, bank employees and bus drivers. In control subjects we could find statistically significant values of systolic, diastolic, triglyceride ($p< 0.05$) and lowered HDL Cholesterol ($p< 0.001$) level. However the values were within specified normal limits.

Our study findings reveal that the multiple risk factors of CVD were quite high among the participants. We found that 10.9% subjects had BMI > 30 Kg / m^2; 8.8 % increased waist circumference; 15.6 % elevated LDL cholesterol: 13.7 % raised triglyceride; 14.7 % hypertension and diabetes 16.9 %.

The association between HOMA-IR and waist circumference, fasting blood glucose can be considered as a predictive tool for insulin resistance, the precision of which requires further validation.

5.2 LIMITATION OF THE STUDY

The main limitation of this current study was that, only male participants from all the four groups were involved. So it may be incomplete information to draw the conclusion about the prevalence rate of metabolic syndrome in this region.

Another limitation of our study was that the workers from the three selected occupations are not certainly the representative sample of Bijapur working population as a whole. Therefore a more diverse population based study may shed light on the relationship between occupational stress and prevalence of metabolic syndrome.

5.3 FUTURE DIRECTIONS

1. Future research related to occupational health should examine the factors that may explain the higher likelihood of metabolic syndrome in these occupational groups and the occupations involving the female participants also.
2. There is some evidence suggesting that physical activity may positively influence obesity and the metabolic syndrome through its influence on the relationship between stress and these conditions. Future study should focus on finding the effect of physical activity on controlling the metabolic syndrome which otherwise is a pre-indication of type 2 diabetes and cardio vascular disease.
3. Our study indicates that while addressing the complications of job stress, there is a need of more adequate working health policy at all work places.
4. Future studies should provide suggestions, guidelines for reduction and management of occupational stress.

APPENDIX

APPENDIX -1

SAMPLE WRITTEN INFORMED CONSENT FORM

BLDE U'S SHRI B.M.PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE BIJAPUR

DEPARTMENT OF BIOCHEMISTRY

CONSENT FORM

Title of the project

The study on occupational risk factors in the manifestation of cardio metabolic syndrome in and around Bijapur

Principal investigators name: Sanjeev S Walvekar

1. PURPOSE OF RESEARCH: I have been informed that this study will assess the effect of stress in different occupations.
2. PROCEDURE: I understand that, the procedure of the study will involve recording of various physiological and biochemical parameters. The procedure will not interfere with any of my physiological parameter and they are non-invasive.
3. RISK AND DISOMFORTS: I understand determination of above mentioned tests will not cause any discomforts to me and do not involve any risk to my health.
4. BENEFITS: I understand that my participation in the study may have a direct benefits to me and also to the field of cardiovascular
5. CONFIDENTIALITY: I understand that medical information produced by this study will become part of institutional record and will be subject to confidentiality and privacy regulation of said institute. Information of a sensitive personal nature will not be a part of medical record, but will be stored in investigators research file and identified only by a code number.

   If the data are used for publication in the medical literature and teaching purposes no names will be used and other identities such as photographs, audio and video tapes will be used only with my special written permission. I understand I may see the photograph an the video tapes and have the audio tapes before giving permission.

6. REQUEST FOR MORE INFORMATION: I understand that I may ask more questions about the study at any time. Concerned researchers is available to answer my question or concerns, I understand that I will be informed of any significant new findings discovered during the course of this study which might influence my continued participants. If during the study or later, I wish to discuss my participants in all concerns regarding this study with a person not directly involved, I am aware that the social worker of the in available to talk to me. A copy of this consent form will be given to me to keep for careful re reading.
7. REFUSAL OR WITHDRAWAL OF PARTICIPANT: I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that researcher may terminate my participation in this study at any time after she/he has explained the reason for doing so and had helped arrange for my continued care by my physician or physical therapists if this is appropriate.
8. INJURY STATEMENT: I understand that in unlikely event of injury to me resulting directly from my participation in this study, if such injury were reported promptly, then medical treatment will be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to---------- (patients/relevant guardian) the purpose of the research, procedures required and possible risk and benefits to the best of my ability.

Investigator Date:

I confirm that _____ (Name of Principal Investigator) have explained to me the propose of research, the study procedures that I will undergo, and the possible risk and discomforts as well as benefits that I may experience. Alternative to my participation in the study have also been to give my consent form. Therefore I agree to give consent to participate s a subject and this research project

Participate Date

Witness to signature Date

APPENDIX II

PUBLICATIONS

1. **Sanjeev S. Walvekar,** Jeevan G. Ambekar , Basvaraj B. Devaranavdgi. Association of Obesity and Cardiometabolic Syndrome in Bank Employees: A Cross Sectional Study JKIMSU. Jan-Mar 2015, 4(1): 115-122.

2. **Sanjeev S. Walvekar**, Jeevan G. Ambekar, Basavaraj B. Devaranavadagi. Study on Serum Cortisol and Perceived Stress Scale in the Police Constables. Journal of Clinical and Diagnostic Research. 2015 Feb, Vol-9(2): BC 10-BC 14

ORIGINAL ARTICLE

Association of Obesity and Cardiometabolic Syndrome in Bank Employees: A Cross Sectional Study

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Abstract:

Background: Metabolic syndrome also referred as cardio metabolic syndrome is identified as the prestate for cardiovascular disease, type 2 diabetes and also for chronic kidney disease. Obesity is considered as the strongest component of metabolic syndrome as per the definition given by different organizations. The easiest way to measure obesity are waist circumference, Body Mass Index (BMI) and waist to hip ratio. *Aims and Objective:* To find the association between microalbuminuria and components of cardiometabolic syndrome in the bank employees and also the association between waist circumference, body mass index and components of cardiometabolic syndrome, microalbumin. *Material and Methods:* This was a cross sectional study involving 73 subjects working in a reputed bank. Their anthropometric measurements and biochemical parameters like fasting blood glucose, lipid profile, serum cortisol and microalbumin were measured. We defined microalbuminuria as a urinary albumin to creatinine ratio of 30 to 299 mg/gm. *Results:* out of 73 participants, in 33 participants at least 3-5 parameters of cardiometabolic syndrome were found to be present and were labeled as cardiometabolic syndrome patients. Waist circumference showed positive correlation with age (r=0.498), systolic blood pressure (r=0.500), diastolic blood pressure (r=0.476), fasting blood glucose (r=0.300), triglyceride (r=0.408) and microalbumin (r=0.409). Microalbumin also exhibited a significant positive correlation with age (r=0.404), waist circumference (r=0.419), fasting blood glucose (r=0.476) and HbA1c (r=0.466), while BMI showed negative correlation with microalbumin (r=-0.085). *Conclusions:* Obesity, one of the parameters of cardiometabolic syndrome measured as waist circumference, is indicative of the syndrome rather than BMI. Microalbumin may be considered as a promising parameter of cardiometabolic syndrome.

Keywords: Cardiovascular disease, Obesity, BMI, Microalbuminuria

Introduction:

Cardiometabolic syndrome (CMS) is an another name for metabolic syndrome which is a collection of symptoms like hyperglycemia, increased triglycerides, and decreased high-density lipoprotein cholesterol (HDL-C), abdominal obesity, and hypertension [1]. It is the syndrome, which increases the risk for cardiovascular disease and type 2 diabetes. The definition of this metabolic syndrome is explained by different organizations, like the World Health Organization, National Cholesterol Education Program's Adult Treatment Panel III (NCEP: ATP III), and International Diabetes Federation. Microalbuminuria is one of the components of the metabolic syndrome as per the WHO definition [2]. It is considered as an early marker of chronic kidney disease (CKD) and found to be associated with cardiovascular events [3]. The studies reported the relationship between metabolic syndrome and microalbuminuria [4, 5]. The early detection of microalbuminuria and timely therapeutic intervention will avert future complications like overt diabetic nephropathy [6].

Obesity is an important risk factor in the diagnosis of the metabolic syndrome. Anthropometric

parameters such as Body Mass Index (BMI), Waist Circumference (WC) are widely used for the measurement of visceral adiposity [7]. Both the parameters help to identify the subjects with CMS easily [8, 9]. BMI is commonly used to check the obesity. It is used to observe and monitor the obesity in the population. But there are certain limitations. It fails to give the information about the distribution of adipose tissue. The values are affected by age, sex and lean mass [10]. The cutoff values mentioned for BMI may underestimate the obesity and its related health risk factors. Waist circumference is associated with a raised risk of developing cardiovascular disease [11]. Application of waist circumference can be considered as a better indicator of obesity related risk factors than the BMI [12]. Bank employee's work is usually associated with job stress as there is the involvement of the factors like extended duty hours, sedentary working style, physical inactivity and confrontation with customers etc. so these workers are at the increased risk of developing the cardiometabolic syndrome.

We have undertaken a cross sectional study to find relationship between microalbuminuria and components of the cardiometabolic syndrome in bank employees and also to find the association between waist circumference, BMI and the components of cardiometabolic syndrome.

Material and Methods:

Study design:

The participants were from a reputed bank with its branches in this area and were in the age group of 30-60 years. Only male participants were selected for this study as it is a part of ongoing study, which includes only male subjects. The participant's anthropometric information like height, weight, waist circumference and hip circumference were recorded. The blood pressure was measured using sphygmanometer when participants were at rest in a sitting position for ten minutes. The study was reviewed and approved by the ethics committee on the research of BLDE University, Sri B. M. Patil Medical College, Hospital and Research center, Bijapur, Karnataka.

Inclusion and exclusion criteria:

The bank employees who expressed about their job related stress were randomly included in the study. The subjects underlying medical conditions like rheumatoid arthritis, tuberculosis and any other infective conditions were excluded from the study. None of the participants had any renal disorders. A total of 73 participants was included in this study.

Cardiometabolic syndrome:

The guidelines of the NCEP ATP III PANEL (National cholesterol education programme, adult training programme III panel) were followed [14]. Cardiometabolic syndrome was considered when the three or more of the following risk factors were present.

1. Blood pressure: $\geqslant$ 130/85 mmHg.
2. Waist circumference: $\geqslant$ 90 cm (specifically for Indians).
3. Triglyceride: $\geqslant$ 150.0mg/dl.
4. HDL-Cholesterol: $\leqslant$ 40.0 mg/dl.
5. Fasting blood glucose: $\geqslant$ 100.0 mg/dl.

Biochemical analysis:

The blood and urine samples of the participants were collected after overnight fasting. The serum cortisol was estimated by ELISA method. HbA1c was measured by chemiluminescent microparticle immunoassay on Abbott instrument. Micro-albumin was measured in the urine by the Turbidimetric immunoassay using the commercial kit supplied by ERBA. The Microalbumin values were expressed as mg per gram of creatinine.

Statistical analysis:

The data of the groups of cardiometabolic syndrome and non-cardiometabolic syndrome were expressed as mean ± SD. The Independent t-test was applied to find the significance between the two groups. The Pearson correlation coefficient was used to determine the correlation between the variables in this study.

Results:

Table 1 shows the anthropometric measurements of the subjects of both cardiometabolic syndrome (n=33) and non-cardiometabolic syndrome (n=40). Weight, BMI and Waist circumference measurements showed marked difference and were statistically significant. In both the groups, the rest of the variables did not show any significant difference.

Biochemical parameters of the individuals studied in the present study are given in the (Table 2).

HDL cholesterol levels decreased and the levels of the rest of the parameters increased in subjects with Cardiometabolic syndrome.

Table 3 shows the Pearson correlation coefficient analysis between variables of BMI and WC in subjects with cardiometabolic syndrome group.

Table 4 depicts the correlation coefficient between microalbumin and other variables in the subjects with cardiometabolic syndrome group.

Table 1: Anthropometric Parameters of the Cardiometabolic syndrome and Non Cardiometabolic syndrome

Sr. No.	Parameters	CMS Mean ± S.D (N=33)	Non CMS Mean ± S.D (N=40)	P value
1	Age (Years)	048.67±6.71	045.93±6.56	0.083
2	Height (cm)	168.64±8.33	168.15 ± 11.00	0.835
3	Weight (kg)	075.00± 8.78	068.55 ± 8.90	0.003**
4	BMI (kg/ m^2)	026.52 ± 3.88	024.48 ± 4.25	0.036*
5	WC (cm)	096.26 ±8.19	87.65 ± 12.37	0.0010**
6	HC (cm)	101.36 ± 8.00	095.58 ± 11.67	0.018*
7	Waist / Hip Ratio	00.95 ± 0.04	00.91 ± 0.06	0.013*
8	SBP (mmHg)	126.73 ± 9.82	124.75 ± 9.33	0.382
9	DBP (mmHg)	084.36 ± 8.86	080.38 ± 9.59	0.071

*Cardiometabolic syndrome- CMS, BMI- Body Mass Index, Systolic blood pressure- SBP, Diastolic blood pressure- DBP, WC-Waist Circumference, HP-Hip Circumference, * Significant at p<0.05 level, ** Significant at p<0.01 level*

Table 2: Biochemical Parameters of the Cardiometabolic syndrome and Non Cardiometabolic syndrome

Sr. No.	Parameters	CMS	Non CMS	P Value
1	FBSL (mg/dl)	113.94 ± 48.52	082.13 ± 16.75	0.002**
2	Gly. HbA1c (%)	006.02 ± 1.06	005.47 ± 0.37	0.003**
3	Micro albumin (mg/gm)	023.09 ± 15.42	015.50 ± 11.41	0.018*
4	Triglyceride (mg/dl)	178.09 ±105.24	102.90 ± 45.52	0.001**
5	T-Cholesterol (mg/dl)	162.48 ± 35.15	153.25 ± 29.08	0.23
6	HDL-Cholesterol (mg/dl)	034.42 ± 7.14	041.95 ± 9.50	0.003**
7	LDL-Cholesterol (mg/dl)	092.44 ± 30.60	090.72 ± 24.91	0.792
8	VLDL-Cholesterol (mg/dl)	035.62 ± 21.05	020.58 ± 9.11	0.001**
9	Serum Cortisol (µg/dl)	021.59 ± 7.14	015.06 ± 3.36	0.001**

*FBSL-Fasting Blood Sugar Level, Cardiometabolic syndrome- CMS, * Significant at p<0.05 level, ** Significant at p<0.01 level*

Table 3: Correlation Coefficient between Variables with CMS

Sr. No.	Parameters	BMI		WC	
		"r" Value	P value	"r" Value	P value
1	Age	0.19	0.144	0.498	0.002**
2	Waist to Hip Ratio	0.268	0.068	0.348	0.024*
3	SBP	0.333	0.029*	0.5	0.002**
4	DBP	0.193	0.141	0.476	0.003**
5	FBSL	-0.011	0.475	0.3	0.045*
6	Gly. HbA1c.	-0.12	0.253	0.223	0.106
7	Triglyceride	0.267	0.067	0.408	0.009**
8	Total Cholesterol	0.126	0.242	-0.146	0.209
9	HDL-Cholesterol	0.397	0.011	-0.293	0.049*
10	LDL-Cholesterol	-0.127	0.24	-0.522	0.001**
11	VLDL-Cholesterol	0.267	0.067	0.408	0.009**
12	Serum Cortisol	-0.211	0.12	-0.046	0.4
13	Microalbumin	-0.088	0.319	0.409	0.009**

*Cardiometabolic syndrome- CMS, BMI- Body Mass Index, WC-Waist Circumference, FBSL-Fasting Blood Sugar Level, Systolic blood pressure- SBP, Diastolic blood pressure- DBP, * Significant at p<0.05 level, ** Significant at p<0.01 level*

Table 4: Correlation Coefficient between Microalbumin and Variables in Cases with CMS

Sr. No.	Parameters	"r" value	P value
1	Age	0.404	0.010**
2	BMI	-0.085	0.319
3	WC	0.419	0.009**
4	HC	0.345	0.025*
5	Waist to Hip Ratio	0.14	0.219
6	SBP	0.216	0.113
7	DBP	0.119	0.255
8	FBSL	0.476	0.003**
9	Gly HbA1c.	0.466	0.003**
10	Triglyceride	0.182	0.156
11	Total Cholesterol	-0.16	0.187
12	HDL-Cholesterol	0.101	0.287
13	LDL-Cholesterol	-0.337	0.027*
14	VLDL-Cholesterol	0.182	0.156
15	Serum Cortisol	-0.56	0.379

*Cardiometabolic syndrome- CMS, BMI- Body Mass Index, WC-Waist Circumference, FBSL-Fasting Blood Sugar Level, Systolic blood pressure- SBP, Diastolic blood pressure- DBP, * Significant at p<0.05 level, ** Significant at p<0.01 level*

Discussion:

In this present cross sectional study the association between microalbumin and the components of cardiometabolic syndrome has been examined. In our study, we have used albumin to creatinine ratio technique to assess microalbuminuria which was used by others also [4]. The earlier studies had indicated that microalbuminuria being the sign of early onset of chronic kidney disease is associated with increased risk of the cardiovascular diseases, both in diabetic and non diabetic subjects [13,14]. Thus the cardiometabolic syndrome may be linked to these diseases through microalbumin. We have observed higher level of microalbumin in subjects with cardiometabolic syndrome compared to that of subjects without cardiometabolic syndrome in our study. Further, the Pearson correlation coefficient values indicated the positive correlation between microalbumin and the variables, age (r=0.404), waist circumference (r=0.419), hip circumference (r=0.345), fasting blood glucose (r=0.476) and Gly. HbA1c (r=0.466). Similar findings were reported by another study also [15]. In our study, out of 33 subjects with cardiometabolic syndrome, 11 subjects had their microalbumin value more than 30.0 mg /gm of creatinine.

The correlation coefficient value between microalbumin and BMI (-0.085, p=0. 319) suggests the good influence of waist

circumference on microalbumin (r=0. 419, p=0. 009). There are multiple, and variable definitions to explain the diagnosis of cardiometabolic syndrome. Commonly all have given importance to obesity. It is considered as a growing health problem, conferring excess risk for the development of type-2 diabetes and cardiovascular disease [16]. BMI, waist circumference and waist to hip ratio are the tools used to measure the obesity. The studies have indicated that the increased BMI levels are associated with hypertension, CVDs and type-2 diabetes [17, 18]. However, these results were challenged by others [19]. Compared to BMI and WHR (waist to hip ratio) WC is considered as a simple and easier obesity related anthropometric parameter because it has a single measurement whereas BMI and WHR are the measurements derived from two different parameters. In our study, we have assessed the components of cardiometabolic syndrome using both the BMI and WC. Our study indicates statistically significant positive correlation coefficient between WC and other parameters such as age (r=0.498), systolic blood pressure (r=0.500), diastolic blood pressure (r=0.476), blood glucose (F) (r=0.300) and triglycerides (r=0.408).Though positive correlation coefficient was found between same variables and BMI, only systolic blood pressure value(r=0.333) was statistically significant. Obesity, metabolic syndrome are considered as the independent risk factors for chronic kidney disease (CKD) and end stage renal disease (ESRD) [20]. Hsu *et al* reported the link between ESRD and BMI even after the risk factors like hypertension and diabetes were adjusted [21]. Similarly Kurella reported the association between CKD with the occurrence of metabolic syndrome [22].

The studies about the occurrence of renal injury being initiated by the obesity and metabolic syndrome are getting much research attention [23]. So there is a need to focus on the microalbuminuria, one of the risk factors of CKD as an independent risk factor of cardiometabolic syndrome. In our study there was a marked increase in the levels of all biochemical parameters except low HDL cholesterol in the subjects with the cardiometabolic syndrome than the subjects without cardiometabolic syndrome and most of the parameters were statistically significant. The association of dyslipidemia with metabolic syndrome is well documented and the findings of our study support the same [24]. We found elevated serum cortisol levels in subjects with cardiometabolic syndrome, which is supported by others [25]. However, we found a negative correlation of cortisol with BMI and waist circumference, and of microalbumin with BMI. Shivam Champaneri *et al* [26] reported that both BMI and WC were negatively correlated with cortisol and positively correlated after adjustments for gender, age, history of diabetes, socioeconomic status, steroids, hormone replacement therapy, and smoking habit.

The poor glycemic control and raised blood pressure are considered as the risk factors for microalbuminuria. [27]. In our study, we found a positive correlation between microalbumin and fasting blood glucose, blood pressure. The correlation with fasting blood glucose was statistically significant.

Conclusion:

The data presented in this cross sectional study of Bank employees, confirms the better association between cardiometabolic syndrome and the obesity measured as the waist circumference than the BMI. Microalbuminuria, already noted as a risk factor for CKD may be considered as one of the components listed to diagnose the cardiometabolic syndrome. The larger sample size could have projected the association between obesity and cardiometabolic syndrome more precisely.

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Original Article

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Biochemistry Section

Study on Serum Cortisol and Perceived Stress Scale in the Police Constables

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ABSTRACT

Introduction: The occupational stress may be more among the police constables. Under the stressed conditions, the body secretes more Cortisol. Elevated serum cortisol levels significantly correlate with the symptoms of metabolic Syndrome. Perceived stress scale (PSS) is the most widely used psychological tool for measuring the perception of stress. The objective of this study was to examine the association between perceived stress and Serum Cortisol and also to explore stress as an occupational risk factor which may lead to metabolic syndrome.

Materials and Methods: We measured Serum Cortisol, Lipid profile, Blood Glucose and HbA1c in both Police constables and the general population. Also to evaluate the occupational stress, the questionnaire consisting of Perceived stress scale -14 items was used.

Results: A positive correlation was found between Serum Cortisol and perceived stress scale, Blood Glucose, HbA1c. The biochemical parameters were found to be elevated in police constables compared to controls. It was found that among 108 policemen, 38% were confirmed with cardiometabolic syndrome.

Conclusion: The relation between Serum Cortisol and perceived stress scale indicates the severity of occupational stress the police constables are experiencing. So the occupation based health program for lifestyle changes, modification in job related rules and regulations will help to avert further complications and keep police personnel healthy.

Keywords: Cardiometabolic syndrome, Lipid profile, Occupation

INTRODUCTION

The police department is the one which has to provide a continuous service to the humankind. It is the occupation which consists of maximum stress. Polices play the role of maintaining law and order in the society despite many limitations. Police work is regarded as stressful because of the individual risk of exposure to violence and the everyday involvement in a variety of traumatic incidents. They should be prepared themselves mentally and physically for responding efficiently and even for the unforeseen and unpredictable incidents. Occupational stress and workplace health have become common issues of great concern. Depending on the amount of time spent at the working place and the nature of work, the work stress increases and it has the direct impact on the health status leading to many health related complications. Occupational stress can affect the health when the stress of the workplace exceed the employee's ability to have some control over their situation or to cope in other ways. Stress is defined as "the non-specific response of the body to any demand placed upon it" [1]. The stress induced physiologic changes are usually adaptable and compensative. However, when stressful events do occur more frequently and their intensity crosses certain limits, then the physiological changes become pathological in nature [2].

Under normal circumstances, the body maintains or regulates the normal cortisol levels. Most healthy adults have a high cortisol level in the morning and a low cortisol level at night. But under the condition of the more stressed, the body secretes more cortisol. Cortisol is frequently referred to as the "stress hormone" because it is also secreted in higher levels during the body's fight or flight response to stress. It is also responsible for several stress-related changes in the body. Studies have been done to find relation between cortisol and metabolic syndrome [3]. Because of enormous stress, police personnel suffer from the disorders such as psychological disorders, gastrointestinal disorders, Insomnia etc. [4-6]. The incidence of cardiovascular (CV) diseases is more prevalent in police personnel than the general population [7]. The metabolic syndrome is defined as the cluster of risk factors that increases the risk of heart disease and type 2 diabetes [8]. Dyslipidemia, raised blood pressure (BP), abdominal obesity, impaired glucose tolerance are the features of metabolic syndrome [9,10]. The studies also showed that morning cortisol levels are significantly correlated with the symptoms of metabolic syndrome, such as obesity, high blood pressure and a poor lipid profile [11-13]. Cortisol is released in response to Hypothalamic –pituitary adreno cortical axis (HPA) and catecholamines are released by the sympathetic adrenal medullary (SAM) system. These two hormones help to cope with any form of stress. However, a prolonged and continuous stress will interfere these functions and leads to increased risk for physical and mental disorder [14]. It is reported that the patients with metabolic syndrome show hyperactivity of hypothalamic-pituitary-adrenal (HPA) axis that leads to a state of hypercortisolism which may be due to chronic stress, contributing to the development of insulin resistance, Type 2 diabetes, visceral fat and obesity [15].

The reasons mentioned for the high prevalence of CVD among policemen include occupational stress, irregular food habits, inadequate sleep and unhealthy habits like smoking and alcohol drinking [16]. Therefore, this study was planned to investigate the level of stress and to estimate prevalence of metabolic syndrome, which is a good predictor of cardiovascular morbidity. It was planned to assess the stress with the use of perceived stress scale (14 items) and serum cortisol estimation. Also, it was aimed to explore the effect of stress on glucose and lipid levels. Workplace program to promote health and fitness among policemen are usually very less. Hence the present study would help to implement the interventions to provide good health to this one of the important occupational group.

MATERIALS AND METHODS

Study design: This study was a cross-sectional study designed to explore the effect of stress as an occupational risk factor which may lead to a cardiometabolic syndrome.

Participants: In our study there were 108 male police constables as the study group from the Bijapur, Karnataka state and age-matched 108 subjects from the general population were the controls. Out of 108 control subjects, only 12 subjects scored 'perceived stress scale' score more than 28 (cutoff value) and the rest 96 subjects were grouped as 'Non stressed'. Hence, the study of stress and non stress in the control group was not carried over.

The participants were informed of the purpose of the current study. The detailed information about their past medical history, habits of smoking, alcohol intake was recorded through interview based medical examinations. Height, weight, waist circumference and hip circumference were measured. Blood pressure was measured using a sphygmomanometer in a sitting position following the rest for ten minutes. The study was reviewed and approved by the Ethics Committee on the Research of BLDE University, Sri B.M. Patil Medical College, Hospital and Research Center, Bijapur, Karnataka state, India.

Inclusion and exclusion criteria: The police constables (Males) those who complained about job related stress were randomly included in the study. The constables underlying medical conditions like rheumatoid arthritis, tuberculosis and any other infective conditions were excluded from the study.

Measurement of perceived stress and the scoring: To evaluate the occupational stress, the Perceived Stress Scale (PSS) was used which contains 14 items or questionnaires. Perceived Stress Scale is the most validated psychological tool for measuring the perception of stress. It measures the degree to which situations in one's life are considered as stressful. The perceived stress is the one which measures the level of stress based on stressful incidents, capability to face them at an individual level. The questions in the PSS-14 items were asked about the feelings and thoughts of the constables during the past month. The PSS consisted of 14 items [17], seven positive and seven negative. The negative element was intended to assess the lack of control and the negative affective reactions, while the positive element measured the degree of the ability to cope with the existing stressors. Each item was rated on a five-point scale from 0 = 'never' to 4 = 'very often', covering the previous month. The PSS scores are obtained by reversing the responses (e.g., 0 = 4, 1 = 3, 2 = 2, 3 = 1 and 4 = 0) to the four positively stated items (items 4, 5, 7, and 8) and then summing across all the scale items. The scores ranged from 0 to 56, with higher scores indicating higher levels of perceived stress and the lower scores indicating lower levels of stress [18]. The PSS-Questionnaire-14 item translated into the local language (Kannada) was provided in case if it was required. The PSS score was divided into two sections. The score, 28 being the operational cutoff value of the upper bound and were labelled as 'stressed' and the score less than 28 as 'non stressed' respectively. This cut off value was selected in accordance with a similar study from Pakistan [19] and India [20].

Cardiometabolic syndrome: Diagnosis of the cardiometabolic syndrome was made using NCEP ATP III PANEL (National cholesterol education programme, adult training programme III) definition [21] and presence of metabolic syndrome was confirmed when three or more of the following risk factors were present: (1) Waist circumference: ≥90 cm (specific for Indians); (2) Blood pressure: ≥ 130/85 mmHg; (3) Triglycerides ≥ 150 mg/dl; (4) HDL Cholesterol ≤ 40 mg/dl; and (5) Glucose intolerance (Fasting serum glucose ≥ 100 mg/dl, or reported treatment for diabetes). Cardiometabolic syndrome was considered to be present if there was the presence of three or more above mentioned risk factors.

Biochemical analysis: Blood samples were collected from the subjects with their overnight fasting. Plasma glucose was estimated using glucose oxidize method [22]. The fasting serum sample was used for estimation of lipids, including total cholesterol [23], triglycerides [24] and high density lipoprotein cholesterol (HDL-C) [25]. All the biochemical estimations were done on the same day only. Serum cortisol was estimated by Elisa method [26]. HbA1c was measured by chemiluminescent microparticle immunoassay on Abbott instrument.

STATISTICAL ANALYSIS

Data were expressed as mean ± SD. The independent t-test was used to determine the significance between study and control groups. The Pearson correlation coefficient was used to determine the correlation between stress level and variables of cardiometabolic syndrome.

RESULTS

[Table/Fig-1] shows the baseline characteristics of Police constables and Control group participants. There was no significant difference in the age of participants between two groups, suggesting an age matched distribution of participants. The other characteristics like Height, Weight, BMI, Waist / Hip ratio, Perceived stress scale values were found to be higher in the cases comparing to control group and also statistically significant. The table also shows the percentage distribution of habits and prevalence rate of Hypertension, Type 2 diabetes and Cardiometabolic syndrome in both the groups.

[Table/Fig-2] Shows the values of lipid profile, Fasting blood glucose, HbA1c and Serum Cortisol of cases and controls. Patients presented significantly higher values of all Biochemical parameters in cases except HDL. Cholesterol which was lowered in cases compared to controls. The values were statistically significant.

[Table/Fig-3] Shows the values of PSS and Biochemical parameters studied in the subjects (cases) grouped as 'Stressed' and 'Non stressed' based on PSS score 28 as the cut off value. It was observed that the values of Fasting blood glucose, HbA1c, serum Cortisol and

Characteristics	Cases (No: 108)	Controls (No: 108)	p-value
	Mean ± S.D	Mean ± S.D	
Age (Years)	42.73 ± 10.10	43.23 ± 8.28	0.356
Height (Cm)	170.00 ± 5.25	162.50 ± 9.60	< 0.001
Weight (Cm)	75.11 ± 6.68	63.69 ± 4.40	< 0.001
BMI (kg / m^2)	26.00 ± 2.40	22.30 ± 1.90	<0.001
Waist / Hip ratio	1.14 ± 0.22	0.80 ± 0.07	< 0.001
Systolic blood pressure (mmHg)	123.06 ± 12.23	120.33 ± 8.16	0.061
Diastolic blood pressure (mmHg)	78.63 ± 13.63	76.11 ± 13.29	0.171
Pulse rate (per/min)	82.44 ± 6.67	80.33 ± 6.89	0.023
Perceived stress scale	27.49 ± 8.49	18.79 ± 6.56	<0.001
Smoking	52 [48.1%]	30 [27.8 %]	
Tobacco chewing	32 [29.6 %]	29 [26.9 %]	
Alcohol consumption	65 [60.2 %]	20 [18.5 %]	
Hypertension	22 (20.4%)	09 [08.3%]	
Type 2 Diabetes	24 (22.2%)	12 [11.1%]	
Cardiometabolic syndrome	41 (38.0%)	13 [12.0%]	

[Table/Fig-1]: Basic characteristics in cases and controls

Parameter	Cases (n=108)	Controls (n=108)	p-value
Fasting blood glucose (mg/dl.)	115.01 ± 45.92	97.90 ± 6.20	< 0.001
Gly.HbA1c (%)	5.8 ± 1.3	4.9 ± 0.5	< 0.001
Triglyceride.(mg/dl.)	151.58 ± 96.76	117.90 ± 28.37	< 0.001
Total Cholesterol. (mg /dl.)	199.43 ± 58.43	178.34 ± 27.70	< 0.001
HDL. Cholesterol.(mg /dl.)	36.31 ± 6.40	47.35 ± 6.30	< 0.001
LDL. Cholesterol. (mg /dl.)	132.79 ± 54.53	107.41 ± 28.35	< 0.001
VLDL.Cholesterol.(mg/dl.)	30.31 ± 19.35	23.58 ± 5.67	< 0.001
Cortisol (µg/dl)	25.00 ± 13.64	9.69 ± 4.94	< 0.001

[Table/Fig-2]: Biochemical parameters of the participants (cases and controls) variables are presented as mean ± standard deviation (SD). HDL-C: high density lipoprotein cholesterol; LDL-C: low density lipoprotein cholesterol; VLDL: very low density lipoprotein cholesterol

PSS were found to be increased in 'Stressed ' group comparing to 'Non stressed' group and the values were statistically significant. The values of Triglyceride, Total cholesterol, HDL cholesterol and VLDL cholesterol were found to be statistically insignificant though they were raised in the 'Stressed' group comparing to 'Non stressed 'group.

[Table/Fig-4] Depicts the correlation coefficient between cortisol and other variables in cases (Stressed and Non stressed) as per PSS reference cutoff value. It was observed that Waist/Hip ratio, Fasting blood glucose, HbA1c and PSS showed statistically significant positive correlation with Serum Cortisol.

[Table/Fig-5] Illustrates the correlation coefficient between serum cortisol and other variables in cases grouped as Stressed and Non stressed after excluding the subjects with the habits of Smoking, Tobacco chewing and Alcohol consumption. It was found that in the group 'Stressed' there was a positive correlation between Serum Cortisol and Waist/Hip ratio, Fasting blood glucose, HbA1c and PSS and also the values were statistically significant.

[Table/Fig-6] shows the results of multiple regression analysis, which is statistically significant and the association was found between serum cortisol and HbA1c ($\beta = 0.453$, $p < 0.001$), glucose ($\beta = 0.411$, $p = 0.001$) after adjusting age and waist to hip ratio.

Variables	Stressed (n=41)	Non stressed (n= 67)	p-value
Age (Years)	41.44 ± 10.34	42.61 ± 10.43	0.570
W/H Ratio	0.98 ± 0.08	0.97 ± 0.08	0.981
Systolic Blood pressure (mm Hg)	122.10 ± 11.92	123.64 ± 12.47	0.526
Diastolic Blood pressure (mm Hg)	77.71 ± 14.55	79.19 ± 13.12	0.584
Blood Glucose (Fasting) (mg /dl)	125.12 ± 45.98	105.94 ± 28.52	< 0.001
HbA1c (%)	6.02 ± 1.17	5.48 ± 0.60	< 0.001
Triglyceride (mg /dl)	163.73 ± 138.90	141.06 ± 61.30	0.246
Total cholesterol (mg /dl)	209.66 ± 67.03	193.16 ± 52.94	0.159
HDL cholesterol (mg /dl)	36.54 ± 8.04	36.18 ± 5.21	0.779
LDL cholesterol (mg /dl)	140.38 ± 57.69	128.77 ± 52.13	0.283
VLDL cholesterol (mg /dl)	32.75 ± 27.78	28.21 ± 12.25	0.246
Cortisol (µg/dl)	40.86 ± 11.01	14.09 ± 7.67	< 0.001
Perceived stress scale	35.78 ± 6.00	22.42 ± 5.13	< 0.001

[Table/Fig-3]: Demographic characteristics of police constables grouped as stressed and non stressed with PSS Score 28 as the cut off value

Variables	Stressed (n=41) ' r ' value	p-value	Non Stressed (n=67) ' r ' value	p-value
Age (Years)	0.038	0.406	0.180	0.073
W/H Ratio	0.408	0.004*	0.090	0.233
Systolic Blood pressure (mm Hg)	-0.005	0.488	0.152	0.109
Diastolic Blood pressure (mm Hg)	0.068	0.335	0.085	0.246
Blood Glucose (Fasting) (mg /dl)	0.357	0.011*	0.013	0.459
HbA1c (%)	0.424	0.003*	0.048	0.349
Triglyceride (mg /dl)	0.097	0.274	0.085	0.247
Total cholesterol (mg /dl)	-0.122	0.225	-0.144	0.123
HDL cholesterol (mg /dl)	-0.018	0.454	0.070	0.287
LDL cholesterol (mg /dl)	-0.185	0.123	-0.173	0.081
VLDL cholesterol (mg /dl)	0.097	0.274	0.085	0.247
Perceived stress scale	0.479	0.001*	0.191	0.061

[Table/Fig-4]: Correlation coefficient between Cortisol and other variables in cases (Non stressed and stressed as per PSS reference cut off value) [*= Statistically significant at 0.01]

DISCUSSION

Occupational stress is a result of the nature of work and working atmosphere [27] which leads to many disorders. Through the identification of risk factors and the introduction of appropriate measures, the stress and ill health in the workplace can be distinctly relieved. In our study, we evaluated perceived stress among police constables, including its sources and severity along with its correlation with other variables. The participants were categorized as 'stressed' and 'non stressed' based on the calculated score with the help of perceived stress scale (14 items). Out of 108 participants, 41 (38 %) subjects were found to be under stress. Since this study was undertaken with the aim of finding baseline information about the stress the policemen were reeling under, complete lifestyle aspects however were not assessed. This study revealed that there was a significant positive relationship between blood glucose, HbA1c and serum Cortisol among police constables. The findings from our study revealed that the participants in the study group with higher cortisol values were at higher risk for metabolic syndrome. The similar observations were made in earlier studies also [28].

We found that the most common manifestations of metabolic syndrome in police constables were Hypertriglyceridemia followed by low HDL Cholesterol. The other biochemical parameters studied also showed significantly elevated values compared to controls. It has been reported that blood pressure, triglyceride, and HDL-Cholesterol abnormalities were the most common metabolic abnormalities among the subjects in Taiwan [29]. Jovica jovanovic et al., reported that with the increase of occupational stress index, there was an increase in glucose, total cholesterol, LDL cholesterol and Triglyceride concentration in the stress exposed group [30].

Hypertension is an important CV disease risk factor and also one of the five components to define the metabolic syndrome [31]. In our study it was found that about 22.7 % police constables were hypertensive. Similarly, a study by Dr. Bakhtiyar Chaudhary et al., noted that prolonged exposure to work stress without proper coping

Variables	Overall (n=76)'r' value	p-value	Stressed (n=29) ' r ' value	p-value	Non stressed (n= 47) 'r ' value	p-value
Age (Years)	-0.016	0.445	0.206	0.142	0.095	0.262
W/H Ratio	0.395	0.000*	0.478	0.004*	-0.075	0.307
Systolic Blood pressure (mm Hg)	-0.082	0.240	-0.136	0.241	0.075	0.308
Diastolic Blood pressure (mm Hg)	-0.051	0.331	-0.003	0.493	0.038	0.401
Blood Glucose(Fasting) (mg /dl)	0.383	0.000*	0.343	0.034**	-0.021	0.443
HbA1c (%)	0.491	0.000*	0.407	0.014*	-0.033	0.414
Triglyceride (mg /dl)	0.071	0.272	-0.010	0.479	0.207	0.082
Total cholesterol (mg /dl)	-0.063	0.393	-0.220	0.125	-0.034	0.410
HDL cholesterol (mg /dl)	-0.193	0.047	-0.173	0.185	0.103	0.246
LDL cholesterol (mg /dl)	-0.060	0.303	-0.175	0.182	-0.109	0.233
VLDL cholesterol (mg /dl)	0.070	0.272	-0.010	0.479	0.207	0.082
Perceived stress scale	0.711	0.000*	0.500	0.003*	0.110	0.232

[Table/Fig-5]: Correlation coefficient between Cortisol and other variables in cases (Overall, stressed and Non Stressed after excluding the subjects with habits (Smoking, Tobacco chewing and Alcohol consumption) as per PSS reference cut off value) [*= statistically significant at 0.01] [**= statistically significant at 0.05]

Variables	Beta	't' value	p-value
HbA1c	0.453	4.072	0.001
Blood Glucose	0.411	3.526	0.001
Triglyceride	0.077	0.639	0.525
T.Cholesterol	0.086	0.680	0.499
HDL.cholesterol	-0.141	-1.143	0.257

[Table/Fig-6]: Multiple regression analysis between Serum Cortisol and other variables after adjusting Age and Waist to Hip ratio

strategies, may exist as a potential risk factor for hypertension and coronary artery disease [32].

In addition to increasing the risk of CV disease, the metabolic syndrome may hasten the development of stroke and complication of diabetes mellitus like diabetic nephropathy, retinopathy and neuropathy [33]. With respect to the NCEP-ATP III criteria, the type 2-diabetes mellitus subjects having already fulfilled one criteria and another two are needed to diagnose metabolic syndrome, suggesting that the patients with type 2 diabetes mellitus, exhibit the features of metabolic syndrome and it often results in hyperglycemia. In our study it was found that about 14.8 % were of impaired fasting glucose and 19.4 % cases were already having Type 2 diabetes. Waist Circumference has the strongest associations with health risk factors [34]. The increased waist circumference has been thought to be a predictor of CV disease and is an important diagnostic marker for the metabolic syndrome [35]. The recommended waist circumference cutoff values were 90 cm for Asian males and 80 cm for Asian females [36]. In our study about 27.8% polices had a waist circumference above 90 cm. The studies done on the lifestyles of policemen indicate increased rate of addiction to smoking and alcohol habits. Smoking is associated with an increased prevalence of metabolic syndrome, independent of sex and BMI class. This increased risk is mainly related to lower HDL cholesterol, and higher triglycerides and waist circumference [37]. Our study also showed that the alcohol and smoking habits were more among policemen followed by their greater triglyceride level, waist circumferences and lower HDL level.

Cortisol is an important regulator of the protein, glucose and lipid metabolism [38]. There was no significant correlation between serum cortisol and lipid profile in our study. But in our study, we found a positive correlation between Serum Cortisol and perceived stress scale, Blood Glucose and HbA1c, which corroborate the findings of other workers [39]. However, there are inconsistent reports about the correlation between Serum Cortisol and the parameters of metabolic syndrome [40]. Our study indicates that the scoring rate of perceived stress scale in police constables was higher than General Population and further it gives the impression about the occupational stress they were born. In earlier studies, the Perceived Stress Scale has been used involving police officers. Franke et al., found that increased PSS value was associated with CVD risk in police officers [41]. But Yoo and colleagues observed that the PSS was not significantly associated with the metabolic syndrome in law enforcement officers [42]. Though the results of each of these studies vary, these studies commonly used the cross-sectional study design. The usual general conception is that the policemen form a physically fit group in the society and based on this merit only they get the recruitment. However, it is not maintained in their service life by most of them. It was reflected with the reasons they mentioned like, long duty hours, irregular diet, limited choice of food while on duty, disrupted sleep time, etc. The studies done in association between metabolic syndrome and policemen, revealed about their nature of work, stress they face and other related factors. Physical Fitness was found to be ignored by policemen and less number of them are continuing to exercise daily. Stress is considered as the integral part of the life. The job related stress becomes a dominant aspect for some people and leads to the complications like coronary heart disease, diabetes mellitus and other health related disorders [43]. There is a need of serious attempt to reduce the job related stress, like the modifiable changes in the police department, improved training to them to have self efficacy and coping skills with the stress oriented situations [44]. The other aspects like arranging health checkups annually or biannually, also to instruct awareness and benefits of weight reduction, regular exercise and early diagnosis etc [45].

The findings of the present study performed among 108 police constables indicate that 38 % of the subjects have the metabolic syndrome. This is significantly higher than the prevalence of 13 % in the General population. The coexistence of other components of metabolic syndrome in this study is in the line of other studies revealed.

CONCLUSION

The findings of the current study emphasize the need to implement occupation based health program for lifestyle changes, modification in job related rules and regulations and other risk factors of Cardiometabolic syndrome. Also, the considerable amount of occupational stress found in this study among the police constables should trigger further work or research in this topic.

LIMITATIONS

The limitations of this study is that it was a cross-sectional design and perceived stress scale results were based on the self reported by the police constables.

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ಕೆಲವು ಕೆಲವು ಆಗು – ಹೋಗು ಘಟನೆಗಳಿಂದಾದ ಒತ್ತಡಗಳ ಅವಲೋಕನ ಮಾಪಕ

ಕಳೆದ ಒಂದು ವರ್ಷದಲ್ಲಿ ಕೆಲವು ಪ್ರಸಂಗಗಳಲ್ಲಿ ತಮಗೆ ಉಂಟಾದ ಅನಿಸಿಕೆ, ವಿಚಾರಗಳನ್ನು ಕುರಿತು ಈ ಕೆಳಗೆ 14 ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳಲಾಗಿದೆ. ಪ್ರತಿಯೊಂದು ಪ್ರಶ್ನೆಗೂ 5 ಬಗೆಯ ಉತ್ತರಗಳನ್ನು ನೀಡಲಾಗಿದ್ದು, ಸೂಕ್ತವಾದ ಒಂದು ಉತ್ತರವನ್ನು ಗುರುತುಹಾಕಿರಿ.

	ಇಲ್ಲವೇ ಇಲ್ಲ	ಬಹುತೇಕ ಇಲ್ಲ	ಕೆಲವು ಸಲ	ಹಲವು ಬಾರಿ	ತೀವ್ರ ಹಲವು ಬಾರಿ (ಬಹ ಸಲ)
1. ಕಳೆದ ತಿಂಗಳಿನಲ್ಲಿ ಕೆಲವು ಅನೀರೀಕ್ಷಿತ ಘಟನೆಗಳು ನಿಮ್ಮ ಮನಸ್ಸಿನ ಶಾಂತಿ ಹಾಳು ಮಾಡಿದವು ಎಂದು ಎಷ್ಟು ಸಲ ನಿಮಗೆ ಅನಿಸಿದೆ. ?	O	O	O	O	O
2. ಕಳೆದ ತಿಂಗಳಿನಲ್ಲಿ ಸಂಭವಿಸಿದ ನಿಮ್ಮ ಜೀವನದ ಕೆಲವು ಮಹತ್ವದ ಘಟನೆಗಳ ಮೇಲೆ ನಿಮ್ಮ ಹಿಡಿತ ಇರಲಿಲ್ಲ. ಹೀಗೆ ನಿಮಗೆ ಎಷ್ಟು ಸಲ ಅನಿಸಿತು. ?	O	O	O	O	O
3. ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ಎಷ್ಟು ಸಲ ಖಿನ್ನತೆ ಮಾನಸಿಕ ಒತ್ತಡ ಅನುಭವಿಸಿದ್ದೀರಿ. ?	O	O	O	O	O
4. ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ನೀವು ಎದುರಿಸಿದ ದೈನಂದಿನ ಉಪದ್ರವ ಹಾಗೂ ಸಮಸ್ಯೆಗಳನ್ನು ನೀಗಿಸುವಲ್ಲಿ ಎಷ್ಟು ಸಲ ಜಯಶಾಲಿಗಳಾಗಿದ್ದೀರಿ. ?	O	O	O	O	O
5. ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ನಿಮ್ಮ ಜೀವನದಲ್ಲಿ ಆದ ಕೆಲವು ಬದಲಾವಣೆಗಳಿಗೆ ಎಷ್ಟು ಸಲ ಸಮರ್ಪಕವಾಗಿ ಹೊಂದಾಣಿಕೆ ಮಾಡಿಕೊಂಡಿರುವಿರಿ. ?	O	O	O	O	O
6. ವೈಯಕ್ತಿಕ ಸಮಸ್ಯೆಗಳನ್ನು ಬಗೆಹರಿಸುವಲ್ಲಿ, ಹೋದ ತಿಂಗಳು ಎಷ್ಟು ಸಲ ನೀವು ಆತ್ಮ ವಿಶ್ವಾಸ ವ್ಯಕ್ತಪಡಿಸಿರುವಿರಿ. ?	O	O	O	O	O
7. ಎಲ್ಲವೂ ನೀವು ಅಂದುಕೊಂಡತೆ ಸರಾಗವಾಗಿ ನಡೆಯುತ್ತಿದೆ ಹೀಗೆ ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ಎಷ್ಟು ಸಲ ನಿಮಗೆ ಅನಿಸಿರುವುದು. ?	O	O	O	O	O
8. ನೀವು ಮಾಡಬೇಕಾದ ಕೆಲವು ಕೆಲಸಗಳನ್ನು ನೀವು ಸಮರ್ಪಕವಾಗಿ ಮಾಡಲು ಸಾಧ್ಯವಾಗಲಿಲ್ಲ. ಹೀಗೆ ಕಳೆದ ತಿಂಗಳು ನಿಮಗೆ ಎಷ್ಟು ಸಲ ಅನಿಸುರುವುದು. ?	O	O	O	O	O
9. ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ನಿಮ್ಮ ಜೀವನದಲ್ಲಿ ನಿಮ್ಮನ್ನು (ಸಿಟ್ಟಿಗೆಬ್ಬಿಸುವ) ಕೆರಳಿಸುವ ವಿಷಯಗಳ ಮೇಲೆ ನೀವು ಎಷ್ಟು ಸಲ ನಿಯಂತ್ರಣ ಸಾಧಿಸಲು ಸಾಧ್ಯವಾಯಿತು. ?	O	O	O	O	O
10. ನಾನು ಯಶಸ್ಸಿನ ತುತ್ತ ತುದಿಯಲ್ಲಿರುವೆ. ಹೀಗೆ ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ನಿಮಗೆ ಎಷ್ಟು ಸಲ ಅನಿಸಿತು. ?	O	O	O	O	O
11. ಕೆಲವು ಘಟನೆಗಳು ಸಂಭವಿಸಿದಾಗ್ ಅವುಗಳ ಮೇಲೆ ನೀವು ಹಿಡಿತ ಸಾಧಿಸಲು ಸಾಧ್ಯವಾಗಲಿಲ್ಲ ಹಾಗೂ ನೀವು ಕ್ರೋಧಗೊಂಡಿರಿ ಹೀಗೆ ಕಳೆದ ತಿಂಗಳಲ್ಲಿ ಎಷ್ಟು ಸಲ ಆಯಿತು. ?	O	O	O	O	O
12. ನೀವು ಮಾಡಬೇಕಾದ ಕೆಲವು ಕಾರ್ಯಗಳನ್ನು ಪೂರ್ಣಗೊಳಿಸುವ ಚಂತನೆಯನ್ನು ಹೋದ ತಿಂಗಳನಲ್ಲಿ ಎಷ್ಟು ಸಲ ಮಾಡಿರುವಿರಿ ?	O	O	O	O	O
13. ಸಮಯದ ಪೂರ್ಣ ಸದುಪಯೋಗ ಮಾಡುವುದರ ಬಗ್ಗೆ ಕಳೆದ ತಿಂಗಳು ನೀವು ಎಷ್ಟು ಸಲ ನಿಯಂತ್ರಣ ಸಾಧಿಸಿದ್ದೀರಿ. ?	O	O	O	O	O
14. ತೊಂದರೆಗಳು ಶೇಖರಣೆಯಾಗುತ್ತಿವೆ. ಅವುಗಳಿಂದ ನೀವು ಮುಕ್ತಿ ಪಡೆಯಲು ಸಾಧ್ಯವಾಗುತ್ತಿಲ್ಲ ಕಳೆದ ತಿಂಗಳು ನಿಮಗೆ ಎಷ್ಟು ಸಲ ಅನಿಸಿರುವುದು. ?	O	O	O	O	O

Perceived Stress Scale

The questions in this scale ask you about your feelings and thoughts during THE LAST YEAR. In each case, you will be asked to indicate your response by placing an "X" over the circle representing HOW OFTEN you felt or thought a certain way. Although some of the questions are similar, there are differences between them and you should treat each one as a separate question. The best approach is to answer fairly quickly. That is, don't try to count up the number of times you felt a particular way, but rather indicate the alternative that seems like a reasonable estimate.

	Never	Almost Never	Sometimes	Fairly Often	Very Often
	1	2	3	4	5
1. In the last month, how often have you been upset because of something that happened unexpectedly?	○	○	○	○	○
2. In the last month, how often have you felt that you were unable to control the important things in your life?	○	○	○	○	○
3. In the last month, how often have you felt nervous and "stressed"?	○	○	○	○	○
4. In the last month, how often have you dealt successfully with day to day problems and annoyances?	○	○	○	○	○
5. In the last month, how often have you felt that you were effectively coping with important changes that were occurring in your life?	○	○	○	○	○
6. In the last month, how often have you felt confident about your ability to handle your personal problems?	○	○	○	○	○
7. In the last month, how often have you felt that things were going your way?	○	○	○	○	○
8. In the last month, how often have you found that you could not cope with all the things that you had to do?	○	○	○	○	○
9. In the last month, how often have you been able to control irritations in your life?	○	○	○	○	○

	Never	Almost Never	Sometimes	Fairly Often	Very Often
	1	2	3	4	5
10. In the last month, how often have you felt that you were on top of things?	○	○	○	○	○
11. In the last month, how often have you been angered because of things that happened that were outside of your control?	○	○	○	○	○
12. In the last month, how often have you found yourself thinking about things that you have to accomplish?	○	○	○	○	○
13. In the last month, how often have you been able to control the way you spend your time?	○	○	○	○	○
14. In the last month, how often have you felt difficulties were piling up so high that you could not overcome them?	○	○	○	○	○

B.L.D.E.U'S SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586103

INSTITUTIONAL ETHICAL COMMITTEE

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BIJAPUR-586103

Ethical Committee
BIJAPUR-586103
OUT WARD
No 13/11
Date 05/04/11
Shri B. M. PATIL Medical College

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 31-03-2011 at 10-30am to scrutinize the Synopsis/Research projects of post graduate student/undergraduate student/Faculty members of this college from ethical clearance point of view. After scrutiny the following original/corrected & revised version Synopsis of the Thesis/Research project has been accorded Ethical Clearance.

Titie A Study on occupational risk factors in the manifestation of cardiometabolic syndrome in and around Bijapur

Name of P.G. /U.G.Student /Faculty member Mr. S.S. Walvekar
Dept of Biochemistry

Name of Guide Dr. J.G. Ambekar Prof of Biochemistry

DR.M.S.BIRADAR
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE

Following documents were placed before E.C.for securitization:
1) Copy of Synopsis/Research project
2) Copy of informed consent form
3) Any other relevant document's