“Investigation of prediabetes and obesity prevalence and their association with various risk factors”

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In Partial Fulfillment of the Requirements For

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In
Pharmacy

BY
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Under the Guidance of
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Certificate

This is to certify that the thesis entitled **"Investigation of prediabetes and obesity prevalence and their association with various risk factors"** has been prepared by **Ms. Bhoomi Arora** with registration number **(12EXTPHDP74)** under my supervision and guidance. The thesis is her own original work completed after careful research and investigation. The work of the thesis is of the standard expected of a candidate for Ph.D. Programme in **Pharmacy** and I recommend that it be sent for evaluation.

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Abstract

Aim and Objectives:

With rapid economic developments, the increased prevalence of prediabetes and obesity has become major health crisis globally. These lifestyle chronic disorders are the major cause of cardiovascular complications. The aims of the current study were to determine prevalence of prediabetes and obesity as well to explore their association with various risk factors. This help us to develop management strategies by early diagnosis and early interventions in the form of education regarding lifestyle modifications.

Materials and methods:

This was an observational, cross-sectional, multicentric study. The study included 2,412 participants of age ≥12 years to 55 years of both the genders. The participants were further divided into three groups: 12-17, 18-35 and 36-55 years. Subjects from different zones in the Ahmedabad city were included to avoid bias and to get an equal distribution of subjects by socioeconomic state, ethnic variability and gender.

The questionnaire was assessed for life style, physical activity, dietary habit, family histories and social factors that influence physical and psychosocial health of subject. Clinical examination of the subjects was carried out by taking their blood samples and were selected as prediabetic based on Fasting blood sugar ≥100 mg/dL and <126 mg/dL. The subjects with BMI 25 – 29.9 kg/m^2 were considered obese and BMI $\geqslant$ 30 kg/m^2 were considered obese. Blood lipid profile, vitamin D level, insulin level and C-reactive protein were analysed for significance with prediabetes and obesity.

Results:

The prevalence of prediabetes was found significantly higher among 36-55 years (33.19%) and 18-35 years (28.81%) age group compared to 12-17 years (5.09%). The prediabetes prevalence

was remarkably found higher among overweight and obese participants than healthy weight. Family history of diabetes was found significantly associated with incident prediabetes among 18-35 and 36-55 years old subjects. The incidence of pre-hypertension and hypertension was shown an association with prediabetes with higher mean fasting blood sugar level among pre-hypertensive and hypertensive participants. Physical activity postulated a protective effect on prevention of prediabetes in current study. It was also noticed that participation in indoor and outdoor games reduces the risk of prediabetes in 18-35 and 36-55 years old subjects. Higher prevalence of non-vegetarian dietary form reported among prediabetes participants in all the age groups. Once in a week frequency of junk food habit was noted significantly higher among 18-35 and 36-55 years old participants. Everyday habit of eating sweets was considered as predisposing factor for incident prediabetes in all age groups. Subjects with medium level of stress has significant association with prediabetes among participants of all age groups. The prevalence of prediabetes was noted higher among upper and upper middle socioeconomic class. The mean values of lipid, vitamin D, insulin and C-reactive protein were noted significantly higher among prediabetics than normal subjects.

The current study noted 16.23%, 18.51% and 26.22% prevalence of overweight while 5.48%, 5.15% and 5.39% prevalence of obesity among 12-17, 18-35 and 36-55 years participants, respectively. The prevalence of pre-hypertension and hypertension was significantly higher among overweight and obese subjects than healthy weight. Physical activity shown protective effect on BMI in current study in all three age groups. It was further noted that participants who did not play either indoor or outdoor games had higher BMI. The dietary type whether vegetarian, non-vegetarian or eggetarian had no significant association with BMI in all participants of three age groups. The everyday junk food eating habit established an association with overweight and obesity among 18-35 and 36-55 years age groups. Whilst prevalence of overweight and obese was higher among subjects who had everyday sweet eating habit in all three age groups. Level of stress had an association with obesity in school going children. In all three age groups, the overweight and obese subjects were found higher from upper socioeconomic class. The study established and association of lipid abnormalities, vitamin D deficiency, hyperinsulinemia and elevated C-reactive protein with overweight and obesity in all three age groups.

Conclusion:

Hence, from the above study it was concluded that the life style disorders such as prediabetes and obesity found to have an association with metabolic syndrome and cardiovascular complications. Proper health education, and awareness creation programmes needs to be done for lifestyle modification, as part of intervention.

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Declaration

I, **Ms. Bhoomi Arora**, registered as Research Scholar, bearing Registration No. **12EXTPHDP74** for Doctoral Programme under the Faculty of **Pharmacy** of Nirma University do hereby declare that I have completed the course work, pre-synopsis seminar and my research work as prescribed under R. Ph.D. 3.5.

I do hereby declare that the thesis submitted is original and is the outcome of the independent investigations / research carried out by me and contains no plagiarism. The research is leading to the discovery of new **correlation of scientific facts** already known. This work has not been submitted to any other University or Body in quest of a degree, diploma or any other kind of academic award.

I do hereby further declare that the text, diagrams or any other material taken from other sources (including but not limited to books, journals and web) have been acknowledged, referred and cited to the best of my knowledge and understanding.

Date: 11/4/19

Ms. Bhoomi Arora

I endorse the above declaration made by the student.

Dr. Snehal S. Patel

Acknowledgement

It is a moment of gratification and pride to look back with a sense of contentment at the long traveled path, to be able to recapture some of the fine moments and to be able to thank infinite number of people who have supported and encouraged me throughout my work and helped me to reach the stage of writing this page. Although the list of individuals I want to thank extends beyond the limit of this format, I would like to mention and especially thank some of them for the dedication, prayers and support extended by them.

Getting such help, I feel, is comparable to our human body. **Almighty** has given us a wonderful body in which all the organs and organ systems have their own functions. These systems work synchronously to help us lead a healthy life. Similarly, my project work was like a human body where many people helped me in some or the other way for its successful completion. At this juncture, I would like to express my deep gratitude to one and all.

Nervous system is the system that controls and integrates the functions of the human body. From the day one of my Ph.D course, I was highly motivated by one person who always stands tall and inspires others to rise above all. Her immense knowledge over the subject is tremendous. The brain is the seat from which many thoughts and ideas emerges. This seat of ideas for my work is my mentor and esteemed guide **Dr. Snehal S. Patel**, Assistant Professor, Institute of Pharmacy, Nirma University. Her innovative ideas, thoughts, concepts and notions made my work very interesting and at the same time enjoyable. Brain detects and processes the sensory information from inside and outside the body. Similarly, she detected this research topic and constantly helped me to process this work properly. With a deep sense of gratitude, I take this opportunity to thank her sincerely.

Nerve fibres are next important part of the nervous system. **Prof. Manjunath Ghate**, Director, Institute of Pharmacy, Nirma University and **Dr. Jigna Shah,** Head of Department, Institute of Pharmacy, Nirma University, are like nerve fibres. I extend my special thanks for all the support, guidance, efforts and care that were provided during the course work.

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Homeostasis is the mechanism regulating the integrity of the body fluids and is the key to longevity of life. I take this privilege and grateful to **Dr. K. N. Patel**, Principal, SAL Institute of Pharmacy, Ahmedabad. His helping and caring nature has helped me a lot during my entire project work. He has encouraged and supported me during my Ph.D work.

Blood is specialized connective tissue, which helps in transportation of gases and nutrients and maintaining water and acid-base balance in the body. I express my heart full gratitude to my husband **Dr. Saurabh S. Arora** and my son **Master Kiaansh** for supplying all nutrients. Their patience, love and encouragement have upheld me, particularly in those many days in which I spent more times with my computer than with them during weekends and holidays in completing this dissertation. Thank you for being there always for me.

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Skeletal system composed of bones, cartilages, ligaments and other tissues that perform essential functions for the human body. For me it was **Mr. Surendrakumar Arora, Mr. Tarun Arora** and **Mrs. Lovely Arora,** who supported me throughout my Ph.D work.

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Bhoomi Arora

Date:

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List of Abbreviations

Abbreviation	Full name
↑	Increased
↓	Decreased
25(OH)D	25-hydroxyvitamin D
ACTH	Adrenocorticotropic hormone
ADA	American Diabetes Association
Ang-II	Angiotensin – II
ANS	Autonomic nervous system
APO-A	Apolipoprotein A
APO-B	Apolipoprotein B
APO-C	Apolipoprotein C
BMI	Body mass index
CDC	Centre for disease control and prevention
CETP	Cholesterol ester transfer protein
CRH	Corticotrophin releasing hormone
CRP	C-reactive protein
DASH	Dietary approach to stop hypertension
DM	Diabetes mellitus
DPP-4	Dipeptidyl peptidase – 4
FFA	Free fatty acids
foxP3	Forkhead box P3
GIP	Gastric inhibitory peptide
GLP-1	Glucagon like peptide-1
HbA1C	Haemoglobin A1C
HDL	High density lipoprotein
HDL-C	High density lipoprotein cholesterol
HGP	Hepatic glucose production
HPA	Hypothalamic pituitary adrenal axis
Hs-CRP	High sensitivity C-reactive protein

IAP	The Indian academy of paediatrics
ICMR INDIAB	The Indian council of medical research – India diabetes
IFG	Impaired fasting glucose
IgE	Immunoglobulin E
IGT	Impaired glucose tolerance
i-IFG	Isolated impaired fasting glucose
i-IGT	Isolated impaired glucose tolerance
IL	Interleukin
IL-1Ra	Interleukin-1 receptor antagonist
IOTF	International obesity task force
LDL	Low density lipoprotein
MCP-1	Monocyte chemoattractant protein-1
MGRS	Multicentre growth reference study
NCHS	National centre for health statistics
NGT	Normal Glucose
OECD	Organization for economic co-operation and development
OGTT	Oral Glucose Tolerance Test
OPD	Outpatient department
PNS	Parasympathetic nervous system
PVN	Para ventricular nucleus
RAAS	Renin angiotensin aldosterone system
SAM	Sympathetic adrenomedullary system
SES	Socioeconomic status
SNS	Sympathetic nervous system
SSMM	Sweet, solid fat, meat and mayonnaise
TGF	Transforming growth factor
T_H	T- helper cell
TNF	Tumor necrosis factor
T_{REG}	T regulatory cell
VFL	Vegetables, fruits, legumes
VLDL	Very low density lipoprotein

WC	Waist circumferences
WHO	World health organization
WHR	Waist to hip ratio

1. Introduction

Diabetes, a lifestyle chronic disorder is one of the important public health problems posing a serious threat to the population globally due to its steady increase over the past few decades. Its global prevalence is estimated to be 422 million adults and it is still rapidly increasing at alarming rate (Global report on Diabetes, WHO, 2016). Rapid socioeconomic transition with urbanization and industrialization are the main causes for the global diabetes epidemic (Pradeepa et al, 2017). Irrefutably, majority of diabetes mellitus cases are of the type 2. In India, the diabetes epidemic is on a steep rise with 41 million patients which has experienced a paradigm shift in its prevalence in the socioeconomic and geographic strata as well as in the age group of occurrences (Joshi et al, 2007).

This chronic disorder has caused 1.5 million deaths across the globe in the year of 2012 (Global report on Diabetes, WHO, 2016) including its major complications like hypertension, heart attack, stroke, renal failure, retinopathy, leg amputation; additionally, it also brings economic burden to the people with diabetes and their families. India leads the prevalence of obesity with 20 million Indians either being obese or abdominally obese. The increased prevalence of diabetes also reflects an increase in the associated risk factors such as obesity or overweight. Evidence from clinical practice and literature suggest that the most common risk factors associated with type 2 diabetes are obesity, overweight, family history of diabetes, genetic and metabolic factors, ethnicity, unhealthy diet and physical inactivity (Joshi et al, 2007). Mitigating these major risk factors greatly help in preventing the type 2 diabetes and its complications. These risk factors should be alleviated at an early age when lifestyle habits are formed to avert the risk of type 2 diabetes later in life.

The Finnish Diabetes Prevention Study demonstrated that continuing comprehensive and adaptive recommendations on diet and physical activities reduced the incidence of new-onset diabetes by 58%as compared to those who received generalized instructions to maintain the lifestyle (Tuomilehto et al,2001).While the Da Qing trial conducted inn 1986 on 110,660 men and women from 33 health care clinics in the city of Da Qing, China, demonstrated that the

regulated diet, physical activity or the combination of both considerably curbs the incidence of diabetes (Pan et al,1997).

World Health Organization(WHO) defines prediabetes as a state of intermediate hyperglycaemia using two specific parameters, impaired fasting glucose (IFG) defined as fasting plasma glucose(FPG) of 6.1-6.9 mmol/L (110 to 125 mg/dL) and impaired glucose tolerance (IGT) defined as 2 h plasma glucose of 7.8-11.0 mmol/L (140-200 mg/dL) after ingestion of 75 g of oral glucose load or a combination of the two based on a 2 h oral glucose tolerance test (OGTT) (Report of WHO/IDF, 2006).

While the American Diabetes Association (ADA) defines it as (ADA, 2014);

- IGT of 140-200 mg/dL,
- IFG of 100-125 mg/dL and
- hemoglobinA1c(HbA1c) based criteria of a level of 5.7% to 6.4%.

Impaired fasting glucose (IFG) is a condition in which fasting blood glucose levels are higher than normal but not high enough to be diagnosed as type 2 diabetes. The liver is mainly responsible for keeping a proper supply of glucose in the blood when not eaten for several hours. In impaired fasting glucose, the liver does not respond normally to the hormone insulin and glucose is released immensely into the bloodstream from the liver overnight. That results in high glucose in blood on waking. This is called ‘hepatic insulin resistance’. In impaired glucose tolerance (IGT), blood glucose levels are higher than normal and higher than in impaired fasting glucose but still not high enough to be diagnosed as type 2 diabetes. IGT is diagnosed when the blood glucose level at 2 hours during an Oral Glucose Tolerance Test (OGTT) is higher than the normal range but not high enough to diagnose type 2 diabetes.

Most effective approach of prevention of diabetes is early diagnosis. Weir’s proposal on diabetes progression explains the correlation of the beta cell dysfunction with the stages of progression of disease (Weir et al, 2007). In the stage 1 of diabetes, insulin resistance is

compensated by the increase in insulin release resulting into normal glycaemic levels and an increase in the beta cell mass. Stage 2 diabetes marks an increase in glucose levels up to 5.0-6.5 mmol/L and decrease in the beta cells resulting in the disturbance of the regulations of glucose levels, hence no longer insulin resistance is compensated at this stage. Stage 3 of diabetes is a brief period of decompensation where the glucose levels rise steeply to the level of actual diabetes at stage 4. Stage 5 diabetes marks a significant reduction in beta cell mass progressing to ketosis. Condition of prediabetes is met when both the first two stages have extensively occurred (Tabak et al, 2012). Development of diabetes from stage 3 to stage 4 translates into development of prediabetes into diabetes. Treating prediabetes is to reverse the progression from stage 3 to stage 2 or stage 1. Several studies have demonstrated the reversal of this progression from an individual being prediabetic to normoglycemic (Weir et al, 2007).

Prediabetes is a common disorder in most populations. Prediabetes is a condition that has been projected as a precursor to diabetes (Nathan et al, 2007). The reported prevalence of pre-diabetes appears to vary among populations with different ethnic background. Three hundred and fourteen million people are currently affected with prediabetes all over the world, and by 2025, it is estimated that approximately 500 million people will have pre-diabetes (Boyle et al, 2001). The current estimates are that up to 70% of pre-diabetic subjects eventually get diabetes (Sahai et al, 2011).

Hence prediabetes is a condition that marks the rise of blood glucose levels above normal but below the blood glucose levels of diabetes onset. There is a marked evidence of prevalence of end organ damage associated with the prediabetes. The associated damages with prediabetes include retinopathy, nephropathy, neuropathy, hypertension and macrovascular diseases (Tabák et al, 2012).

Around 5-10% of prediabetic subjects are converted to diabetes annually. Though the conversion rate of individuals from prediabetes to diabetes changes with population characteristics and the criteria used to define prediabetes (Nathan et al, 2007; Forouhi et al, 2007). According to an ADA expert panel, up to 70% of individuals with prediabetes will eventually develop diabetes (Eikenberg et al, 2013). Gerstain et al shown the incidence rate of

diabetes was found to be4-6% for isolated IGT, for isolated IFG 6-9% and for both IGT and IFG was 15-19% (Gerstein et al, 2007).

In the Diabetes Prevention Program (DPP) Outcomes Study, the incidence of diabetes was noted to be 11% in the control group (Knowler et al, 2009). The progression of prediabetes to type 2 diabetes has been examined in many populations with varying results. In general, epidemiological studies indicate that ~ 25% of subjects with IFG or IGT progress to type 2 diabetes in 5 years, whereas about ~ 50% remain pre-diabetic and 25% revert to normal (Meigs et al, 2003).

In an 11 year follow-up study among adults with IGT in Mauritius, 46% developed diabetes, 28% remained unchanged, and 26% reverted to normal. Those with the combination of IFG and IGT develop type 2 diabetes at approximately twice the rate as do individuals who manifest a single abnormality (Shaw et al, 1999). In comparison with adults who have normal glucose tolerance, people with impaired fasting glucose have a two- to three-fold increased prospective risk of cardiovascular events, which is most marked in younger subjects (Sahai et al, 2011).

A prevalence study by Dasappa et al suggested, prevalence of prediabetes in India is ranges between 2-29% and which is higher among females. Study also found that prevalence of prediabetes is increases with the increasing the age and also significantly associated with family history of diabetes (Dasappa et al, 2015). Several studies suggested that elevation of blood pressure is significantly higher in individuals with impaired glucose tolerance and diabetics compared to normal individuals.

The risk factors associated with prediabetes such as age, sex, ethnicity, fasting glucose, systolic blood pressure, HDL cholesterol, BMI and history of diabetes in parents or siblings has been shown to have better predictive value than either IFG or IGT (Stern et al, 2002).

The basic strategy to prevent and manage diabetes is to prevent the population with prevalent risk factors transitioning from prediabetes to diabetes. Once the prediabetes condition is diagnosed, effective lifestyle and pharmacological intervention must be planned to stabilize the progression. Several studies have exhibited a drop-in progression of diabetes with the

improvement in the lifestyle and drug intervention (Avery et al, 2012; Angermayr et al, 2010; Schellenberg et al, 2013; Yuen et al, 2010; Brand et al, 2014; Yang et al, 2014).

Excess body weight, abdominal obesity and physical inactivity are the major predisposing lifestyle factors contributing to the occurrence of prediabetes. World Health Organization designates obesity to be the most common but disregarded public health problem posing a serious threat to both the developed and the developing countries.

Developing countries like India is undergoing a transitional economy with increased urbanization, socioeconomic development and globalization of food market which has resulted in a dramatic change in lifestyle, consisting of physical inactivity, diet rich in fat, sugar and salt, coupled with a high level of mental stress (Goran et al, 2008). This has led to increased incidence of lifestyle diseases like hypertension, type 2 diabetes mellitus, dyslipidaemia, obesity and ischemic heart diseases. Studies have shown that most people with prediabetes develop type 2 diabetes within 10 years, unless they lose 5 to 7 percent of their body weight, by making changes in their diet and level of physical activity (Perreault et al, 2012; Knowler et al, 2002**;** Knowler et al, 2009). People with prediabetes also are at increased risk of developing cardiovascular disease, metabolic syndrome and polycystic ovarian syndrome in female at younger age (Bhardwaj et al, 2008).

The Finnish Diabetes Prevention Study (Lindstro et al, 2003) and the US Diabetes Prevention Program (Bray et al, 2002) with a 3-year follow-up found a risk of diabetes decreases after interventions aimed at weight loss, dietary change and increase in physical activity. Hence the primary and the most effective strategy to manage and prevent prediabetes would be to focus on the abdominal adiposity from the early childhood.

It is imperative, therefore, to screen and treat prediabetes by basic lifestyle interventions at an early formative and habit forming age. Subsequently it would reduce the economic burden of diabetes across the globe by reducing the cost to diagnose, treat and manage the complications arising from diabetes. Moreover, there is a dire need to develop clinical guidelines for the treating doctors which would aid in a uniform approach to treat and manage prediabetes; these

guidelines would provide an endpoint to manage prediabetes so that the patients not responding to lifestyle intervention could be put to pharmacological intervention.

The occurrence of diabetes in Indians is almost a decade earlier than in the western population (Ramaiya et al, 1990). Most people with prediabetes are usually asymptomatic. People may have for several years without noticing anything. Recent research has shown that some long term damage to the cardiovascular system could start even in the prediabetic stage. It is beneficial to identify subjects with prediabetes early helps in appropriate management, thereby reducing both the incidence of diabetes, and related cardiovascular and microvascular complications (Nayak et al, 2011).

The data on prevalence of prediabetes in school and college students as well as in adults is scanty. There are hardly any studies providing prevalence of prediabetes among school, college students and in adults in our country. It is now very well known that there is an epidemic of diabetes in West Indian population and onset of type 2 diabetes occurs at early age in Indian population (Taranikanti et al, 2014).

The present study is planned to study the prevalence of prediabetes in apparently healthy school and college-going students and in adult population. The study will provide evidence for association of prediabetic status with obesity, socioeconomic status, sedentary behavior and physical activity in Ahmedabad's population and these findings can emphasize the need to begin awareness for effective prevention of overweight and obesity by modifying associated lifestyle factors of students which is an increasingly prevalent condition. This information clearly dictates that clinicians must intervene at the prediabetic stage to prevent development of diabetes and a host of complications rather than ignoring prediabetes.

Insufficient studies on prediabetes in central India and high projected prevalence and high conversion rate of prediabetes to diabetes (70%) generates the rationale behind the research. Fasting blood glucose levels of 100–125 mg/dl as impaired fasting glucose and/or 2-h postprandial blood glucose levels of 140–199 mg/dl after a load of 75 g oral glucose as impaired glucose tolerance were the criteria for prediabetes diagnosis according to American Diabetes Association.

Therefore, objectives of our studies were;

1. To investigate the prevalence of prediabetes and obesity in Ahmedabad's population.

2. To study association of prediabetes with basal metabolic index (BMI). Association of prediabetes and BMI with prehypertension, hypertension, family history of diabetes, physical activity, leisure activities such as indoor and outdoor games, playing on laptop/mobile and watching television, dietary form, frequency of junk food eating habit, frequency of sweet eating habit, stress level, socioeconomic status, hyperinsulinemia, lipid abnormalities such as hypercholesterolemia, hypertriglyceridemia, low HDL-C and dyslipidemia, vitamin-D deficiency and elevated C-reactive protein.

3. To develop management strategy by early diagnosis and early intervention in the form of health education and to prevent preventable disease and save life from chronic non communicable diseases.

2. Review of literature

2.1 Prediabetes

Prediabetes broadly refers as an intermediate stage between completer normal blood glucose level and the clinical stage of type 2 diabetes mellitus by considering two parameters impaired fasting glucose (IFG) and impaired glucose tolerance (IGT).

World Health Organization (WHO) defined IFG of 6.1 – 6.9 mmol/ L (110 – 125 mg/dL) and IGT defined as 2 hour plasma glucose of 7.8 – 11.0 mmol/L (140-200 mg/dL) after ingestion of 75 gm glucose load (Oral Glucose Tolerance Test) (World Health Organization, 2006).

The American Diabetes Association (ADA), had same cut off value for IGT (140 -200 mg/dL) but has lower cut off value for IFG than WHO (100 – 125 mg/dL) and has additional hemoglobin A1c (HbA1c) based criteria of a level of 5.7% to 6.4% to define prediabetes (American Diabetes Association, 2014).

Table 2.1 ADA and WHO classification of diabetes and prediabetes

Parameters	Category	ADA Definition	WHO definition
Fasting plasma glucose (FPG)	Impaired Fasting Glucose (IFG)	100 – 125 mg/dL	140 – 200 mg/dL
2 hour glucose tolerance test (OGTT)	Impaired Glucose Tolerance (IGT)	110 – 125 mg/dL	140 – 200 mg/dL
HbA1c	-	5.7% - 6.4%	-

2.1.1 Pathophysiology of Prediabetes

The pathophysiology of prediabetes relies on two important conditions: insulin resistance and beta cell dysfunction.

1. Insulin resistance

Subjects with isolated impaired fasting glucose (IFG) or isolated impaired glucose tolerance (IGT) shown to have a mild increase in fasting plasma glucose concentration compared to subjects with normal glucose tolerance (NGT) (Abdul-Ghani et al, 2006). Usually this increased level of fasting plasma glucose is more in subjects with isolated IFG than isolated IGT. Subjects with IFG have high fasting plasma glucose level and thus have the highest level of fasting insulin (Abdul-Ghani et al, 2006). This greater level of insulin among subjects with prediabetes indicates state of insulin resistance. Subjects with isolated IGT have increased insulin resistance in skeletal muscles, whilst subjects with isolate IFG have normal or near-normal insulin sensitivity in skeletal muscles (Abdul-Ghani et al, 2009).

Insulin resistance is manifested in tissues such as skeletal muscles, liver and adipocytes (Groop et al, 1989).

Insulin resistance in skeletal muscles

During prandial state, insulin suppresses the hepatic glucose production (HGP). Subjects with isolated IFG, in the presence of hyperinsulinemia basal HGP decreases in presence of hyperinsulinemia which indicates severe hepatic resistance to insulin (Jani et al, 2008).

During fasting or post-absorptive state, in the presence of fasting hyperinsulinemia basal HGP remains normal or slightly decreased. Subjects with isolated IGT also have insulin resistance but the severity of insulin resistance is higher in isolated IFG (Abdul-Ghani et al, 2006).

Insulin resistance in liver

Insulin inhibits lipolysis (Groop et al, 1989). Subjects with IGT have higher fasting plasma free fatty acids levels (FFA) which suggests greater rate of lipolysis in adipocytes. Increased fasting plasma free fatty acids in presence of fasting hyperinsulinemia indicates resistance state of insulin for inhibiting lipolysis in adipocytes. Despite hyperinsulinemia, postprandial

suppression of plasma free fatty acid is impaired (Reaven et al, 1988). Plasma free fatty acid level continue to rise causes insulin resistance in skeletal muscles and hepatocytes (DeFronzo et al, 2004).

Whereas subjects with IFG have fasting plasma free fatty acid level similar to that of subjects with normal glucose tolerance, indicating normal or near-normal rate of lipolysis in subjects with IFG (Abdul-Ghani et al, 2008). Subjects with IFG have normal rate of lipolysis in presence of fasting hyperinsulinemia. On note, under normal fasting insulin level, IFG subjects have increased insulin resistance and thus resistant to antilipolytic activity of insulin (Abdul-Ghani et al, 2009).

Insulin in adipocytes

Both the subjects with IFG and IGT have adipocyte insulin resistance. Subjects with IFG have severe hepatic insulin resistance with near-normal skeletal muscle insulin sensitivity whereas IGT subjects have mild hepatic insulin resistance with marked increase in skeletal muscle insulin sensitivity (Abdul-Ghani et al, 2009).

The increased insulin resistance in subjects with prediabetes appears with obesity, hypertension and dyslipidemia, which are risk factors for cardiovascular disorders.

2. Beta cell dysfunction

Insulin resistance mainly occurs in the early history of type 2 diabetes mellitus whilst progressive beta cell dysfunction is the prime factor responsible for development and progression to type 2 diabetes mellitus (Kahn SE, 2003). Glucose stimulated insulin secretion is impaired in both IGT and IFG (Abdul-Ghani et al, 2006; Weyer et al, 1999; Festa et al, 2004; Haeften et al, 2002; Pimenta et al, 2002). Under normal conditions, ingestion of glucose mainly occurs through gastrointestinal tract, where incretin hormones; glucagon-like peptide – 1 [GLP-1] and gastric inhibitory peptide [GIP] were secreted which acts on beta cells to potentiates glucose-stimulated insulin secretion (Laakso M, 2008). The rate of insulin secretion determines the impairment in beta cells in subjects with IFG and IGT. In prediabetes subjects, decrease in level of incretin hormone or resistance to incretin hormones results in declining beta cell glucose sensitivity and impaired insulin secretion (Figure 2.1).

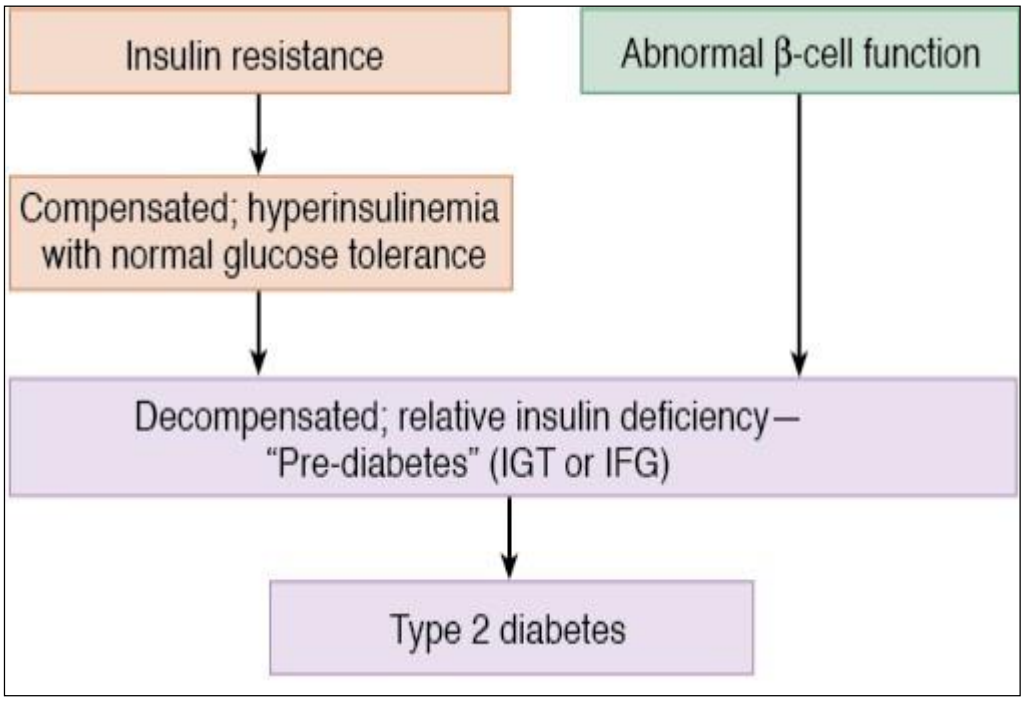

Figure 2.1 Pathophysiology of prediabetes (Tintinalli JE and Meckler JD, 2011)

An epidemic of chronic disease like diabetes threatens the health of large numbers of individuals in developed and developing countries like India.

2.1.2 History of diabetes

The physician ARETAEUS of Cappadocia (Ca. 81-138 A.D.) stated "Diabetes is a mysterious illness.

Ebers Papyrus, written in 1500 BC and published by Egyptologist Georg Ebers in 1874, from various diseases, a condition of "too great emptying of the urine" – refers to diabetes mellitus. Egyptian physicians were using wheat grains, fruit, and sweet beer as a treatment of diabetes mellitus (Papaspyros NS, 1964; Ancient Egyptian Medicine).

Physicians in India at around the same time named diabetes mellitus as "madhumeha" or "honey urine" as they found that the urine from people with diabetes attracted ants and flies. Additionally patients with "madhumeha" suffered from extreme thirst and foul breath noted by Indian physicians (Papaspyros NS, 1964). The ancient Indian physician, Sushruta, and the

surgeon Charaka (400–500 A.D.) were able to identify the two types, later to be named Type 1 and Type 2 diabetes (Tipton MC, 2008).

For the first time term “diabetes” (in Greek means “to pass through”) was used by Apollonius of Memphis in around 230 BC. Apollonius and his scientist reported diabetes a disease of the kidneys (Papaspyros NS, 1964).

Aulus Cornelius Celsus in 30 BC–50 AD published the first complete clinical description of diabetes (Medvei VC, 1993; Southgate TM, 1999).

Two scientists; Aretaeus of Cappadocia physician from Greek who worked in Rome and Alexandria in the second century AD, distinguished diabetes mellitus and diabetes insipidus for the first time (Medvei VC, 1993; Sanders LJ, 2002).

Aretaeus and the Roman physician Galen both observed diabetes as a rare disease and encountered only two such cases in his entire career (Sanders LJ, 2002). Galen reported diabetes as weakness of the kidney. He named diabetes as “diarrhea of the urine” (Medvei VC, 1993).

Sushruta and the surgeon Charaka (400–500 A.D.), two Indian physicians, first differentiated two types of diabetes, observing that among thin individuals developed diabetes at a younger age in contrast to heavier individuals had a later onset of diabetes and lived longer period of time after the diagnosis (Medvei VC, 1993). Later to be named type 1 and type 2 diabetes

In seventh century AD in China, LiHsun found that diabetics have more prone to develop boils and lung infections and he further prescribe avoidance of sex and wine as treatment for diabetes (Medvei VC, 1993).

Avicenna, or Ibn-Sina (980– 1037 AD), a court physician to Caliphs of Baghdad, compiled an exhaustive medical text (“Canon Avicennae”), which included detailed description of diabetes. Sweet urine and increased appetite, and complications, such as diabetic gangrene and sexual dysfunction, were described as clinical features by Avicenna (Medvei VC, 1993).

2.1.3 Diabetes mellitus as seen in the ancient Ayurveda medicine

The understanding of diabetes mellitus in Ayurvedic medicine dates back to the treatises of Sushrutha and Charaka (400 BC). The complete pathophysiology of the disease was discovered by Wagbhat during 800 BC. Ayurvedic physicians considered diabetes as a urinary disease and described it as “Madhumeha”. It was included under “vat prameh” considering it as incurable. Sushrutha also noted that sedentary persons, averse to exercise and who eat foods promoting obesity would develop Pramah (Diabetes).

Different precipitating causes of diabetes like hereditary nature and genetic predisposition were known to ancient Ayurvedic physicians like Charaka and Sushrutha which is evident by the fact that Sushrutha classified diabetes into two groups. One is due to hereditary factor present from birth, and the other is due to violating the rules of healthy living (S. S. Ajagaonkar, 1972).

2.1.4 Global Estimation

Globally, the number of people with diabetes is expected to almost double in the next two decades, increasing from 194 million in 2003 to 380 million in 2025 (International Diabetes Federation, 2006).

Approximately 24 million Americans have diabetes and the prevalence of type 2 diabetes continues to increase, is expected to exceed 366 million worldwide by 2030 (Wild S, 2004).

Boyle et al. warned the prevalence of total diabetes (diagnosed and undiagnosed) in the United States will increase from about 1 in 10 adults in the years 2010 to between 1 in 5 and 1 in 3 adults in 2050 (Boyle JP, 2010).

International Diabetes Federation projects an increase in prevalence of prediabetes to 471 million globally by 2035 (International Diabetes Federation, 2013).

According to study published by Wise J, 35% of adults in England had prediabetes in 2011, up from just 12% in 2003 (Wise J, 2014).

2.1.5 Prevalence of prediabetes

2.1.5.1 National studies

Table 2.2 Description of prevalence of prediabetes in India

Year	Author	Area of study	Age	Prevalence of prediabetes	
				Urban	Rural
2018	Bisht et al	Himachal Pradesh	30-70 Years	25.0%	
2017	Anjana et al. ICMR-INDIAB study	Andhra Pradesh	≥ 20 Years	11.1%	9.6%
		Bihar		15.5%	9.3%
		Gujarat		8.4%	11.5%
		Karnataka		14.1%	10.2%
		Punjab		8.6%	7.9%
		Arunachal Pradesh		14.2%	12.3%
		Assam		13.6%	11.6%
		Manipur		7.2%	7.5%
		Meghalay		7.4%	10.6%
		Mizoram		6.2%	5.8%
		Tripura		16.2%	14.2%
2017	Pandey et al.	Kanpur	17-19 Years	32.1% (Overall)	
2017	Tripathy et al.	Punjab	18-69 Years	6.3% (Overall)	

2017	Patel et al	Raipur	≥ 18 Years	18.3% (Overall)	
2017	Dasappa et al	Bangalore	≥ 35 Years	11.57%	
2017	Choudhary et al.	Jaipur	10-18 Years	13.89% (IGT)	
2017	Singh PS	Neighbouring districts of Etawah and Mainpuri	> 25 Years	-	10.04%
2016	Tripathy et al	Punjab	18-69 Years	6.3%	
2016	Sultana et al	Bhavani Nagar, Hyderabad	> 20 Years	3.5%	-
2015	Dasappa et al.	Bangalore	≥ 35 Years	11.57%	-
2015	Ingole et al	Wardha	> 25 Years	16.7%	
2015	Muthunarayanan et al.	Tamil Nadu	> 20 Years	-	8.5%
2015	Thomas et al	Uttar Pradesh	> 18 Years	10.6%	
2015	Deepa et al	Chennai	≥ 20 Years	37.9%	
		Delhi		47.6%	
		Karachi		31.1%	
2014	Taranikanti et al	South India	14-18 Years	6.89%	
2012	Joshi et al.	Multicentric	≥ 18 Years	18.4% (Overall)	

2012	Veera RB	Visakhapatnam	18-40 Years	15.4% (Overall) 12.2% (IFG) 3.26% (IGT) 2.4% (IFG and IGT)	
2011	Anjana et al. ICMR-INDIAB study	Tamil Nadu	≥ 20 Years	8.3%	
		Maharashtra		12.8%	
		Jharkhand		7.1%	
		Chandigarh		14.6%	
2011	Ravikumar *et al*	North Indian city	> 20 Years	13.2%, 15.4	
2008	Mohan et al.	South India		10.1%	-
2008	Zargar et al	Kashmir Valley	20-40 Years	11.9% (IFG) 2.0% (IGT)	
2007	Raghupathy *et al*.	Vellore and nearby village, Tamil Nadu	26-32 Years	18.9% (IGT) 3.8% (IFG)	14.3% (IGT) 3.4% (IFG)
2007	Kokiwar et al.	Nagpur district	≥ 30 Years	5.96% (IGT) 3.57% (IFG)	
2005	Basavanagowdappa et al.	Suttur village, Karnataka	> 25 Years	2.82% (IFG, Male) 2.78% (IFG, Female)	
2004	Sadikot et al	108 centers of India	> 25 Years	4.8%	2.5%
2003	Mohan et al	Chennai, South India	≥ 20 Years	5.9% (IGT)	

2003	Ramachandran *et al.*	Urban India	20-75 Years	8.7% (IFG) 8.1% (IGT)
2003	Snehalatha *et al*	Urban Southern India	< 40 Years	13.1% (IGT)
			≥ 40 Years	15.7% (IGT)
2003	Gupta et al	Western India	> 20 Years	5.2% (IFG)
2001	Ramachandran et al	National Urban Diabetes Study	> 20 Years	14% (IGT)

2.1.5.2 International studies

Table 2.3 Description of international studies demonstrating prevalence of prediabetes

Year	**Author**	**Area of study**	**Age**	**Prevalence of prediabetes**	
				Urban	**Rural**
2018	Aldossari et al	Al-Kharj, Saudi Arabia	≥ 18 Years	27.6% (Male)	
2018	Basit et al	Pakistan	≥ 20 Years	15.5%	13.9%
2017	Aladeniyi et al	Nigeria	19-76 Years	11.7%	
2016	Bahijri et al	Saudi Arabia	≥ 18 Years	11.9%	
2016	Ghoraba et al	Saudi Arabia	18-65 Years	23.6%	
2015	Al-Azzawi OF	Iraq	30-75 Years	33.66%	
2014	Manasour et al	Iraq	≥ 19 Years	29.1%	
2013	Buckley et al	Ireland	≥ 45 Years	19.8%	

2013	Xu et al	China	> 18 Years	50.1% (Estimated)
2013	Akter et al	Bangladesh	> 35 Years	22.4%
2012	Feng et al	Han Chinese	≥ 20 Years	18.96%
		Manchu Chinese		19.36%
		Korean Chinese		20.47%
2011	Khambalia et al	Nauru, Central Pacific Ocean	15-64 Years	6.39%
2010	Lu et al.	Han Chinese	13-18	3.5%
2010	Yang et al	China	≥ 20 Years	15.5%
2009	Zhang et al	China	≥ 20 Years	18.5%
2008	Jimeno et al	Philippine	≥ 20 Years	2.7% (IFG) 7.0% (IGT)
2008	Manasour et al	Iraq	≥ 20 Years	2.02% (IFG)

2.1.6 Transition from prediabetes to diabetes

People with prediabetes are at increased risk of developing diabetes. The conversion rate varies from population to population and region to region (Shah SN, 2018). The conversion rate from prediabetes to diabetes was 11.7% among Jordanian patients in 20118 (Al-dajah et al, 2018). In 2017, Wu et al reported that 33.7% (96 out of 285 participants) of prediabetes participants transitioned to overt diabetes (Wu et al, 2017). Anjana et al in 2015 demonstrated that the incidence of diabetes among NGT participants at baseline would increase to 23.9 per 1,000 person-years whereas incidence of diabetes among those with prediabetes at baseline to 84.2 per 1,000 person-years (Anjana et al, 2015). In 2011, the ICMR-INDIAB study, among both

urban and rural population of 4 major states of India identified a prediabetes to diabetes conversion rate, ranging between 7.1 % - 15.2% (Anjana et al, 2011). Overall, 8.1% of subjects with added IFG (initial abnormal fasting glucose was 100–109 mg/dl) within a mean of 41.4 months, and 24.3% of subjects with original IFG (initial abnormal fasting glucose was 110–125 mg/dl) at a rate of 5.56% per year progressed to diabetes, reported in 2007 (Nichols et al, 2007).

2.1.7 Risk factors associated with prediabetes

2.1.7.1 Body Mass Index (BMI):

Obesity is one of the most conspicuously visible yet most neglected global health problem today. It is a major concern of public health as it is related to multiple metabolic disorders (Guh et al, 2009). Many studies have shown trends of increasing prevalence of prediabetes among obese adults. The American Diabetes Association (ADA) suggests periodic glucose screening for people of age 45 or above, especially for those with body mass index 25.0 kg/ m^2 or above (ADA, 2002).

In 2018, Aldossari et al, conducted a population-based cross-sectional study on 381 Saudi adult males and found 27.6% prevalence of prediabetes. According to this study, 31.43% overweight and 49.52% obese participants had prediabetes whereas 28.57% overweight and 54.29% obese participants had diabetes.

In 2017, Amiri et al, in Tehran Lipid and Glucose Study on 5568 subject of age more than 20 years reported 23.6% prevalence of prediabetes. This study found prediabetes among 50.4% overweight and 29.4% obese men whereas prediabetes prevalence among overweight and obese women was 38.1% and 47.7%, respectively. This study concluded increased prevalence of prediabetes among overweight and obese men than women.

In 2017, a population based cohort study on 1,765 healthy participants including 446 males and 1,371 females was conducted by Haghighatdoost et al. Out of 1,765, data from 960 were subjected to statistical analysis. This study noted that BMI and waist circumferences (WC) were

the significant predictor of diabetes but not the prediabetes in either of the genders (Haghighatdoost et al, 2017).

Man et al, carried out a study on 1,137 participants with mean age of 55 (10) years without diabetes (DM) at baseline from the Singapore Malay Eye Study in 2017. According to this study, 20.4% participants had prediabetes. Further it was found that higher BMI was independently associated with incident prediabetes (Man et al, 2017).

In 2017, Abtahi et al in a cross- sectional study among 3,115 teachers residing in Shiraz aged 21- 73 years reported 47.2% prevalence of impaired fasting glucose. In addition, this study has shown a relationship between higher range of BMI and prevalence of diabetes and prediabetes (Abtahi et al, 2017).

In 2016, Rahmanian et al, carried out a study on Iranian urban population. This study included 788 participants with 360 men and 428 women between the ages 30-85 years. The study reported higher prediabetes prevalence among participants with higher BMI and the association between prediabetes and BMI was significant in women (Rahmanian et al, 2016).

In 2015, Muthunarayan et al among 544 individuals over the age of 20 years of Tamil Nadu reported the prediabetes (47.9%) and diabetes (56.4%) participants had significantly higher BMI than normal participants (37.7%). This study further found significantly higher waist hip ratio (WHR) among prediabetic (37%) and diabetic (47.3%) compared to normal individuals (28.2%).

In 2015, a study was carried out on Taiwanese women of age between 40 years and 64 years by Wu et al. This study included 8,580 non-smoking, non-drinking and non-areca nut chewing women without history of diabetes mellitus. The prevalence of prediabetes was noted 21.7% and found body mass index as one of the risk factor for prediabetes (Wu et al, 2015).

In 2015, Dasappa et al, in a cross-sectional study on slums of Bangalore in the age group of 35 years and above found 11.9% prediabetes and 14.6% diabetes prevalence among participants

with BMI 25 kg/m^2 and above. This study showed statistically significant association between BMI and prevalence of diabetes and pre-diabetes (Dasappa et al, 2015).

2.1.7.2 Hypertension

Hypertension and diabetes are the two growing health problems for mortality in developing nations (WHO, 2010). Diabetes and hypertension are also known to coexist in 40 to 60% type 2 diabetes patients (Sowers et al, 2001; Arauz-Pacheco et al, 2002). Together hypertension and diabetes mellitus can cause increased peripheral vascular resistance (Talukdar et al, 2017). Additionally, retention of sodium ion and increase in exchangeable sodium ion may lead to hypertension in diabetes patients (Talukdar et al, 2017). Insulin resistance or hyperinsulinemia can be considered as one of the main pathogenesis of hypertension (Talukdar et al, 2017).

Govindarajan et al reported 25-47% participants with hypertension have either insulin resistance or impaired glucose tolerance (Govindarajan et al, 2006). Together, elevated blood pressure and diabetes, increases the risk of retinopathy, nephropathy, and cardiovascular disease (Sowers et al, 2001; Bakris et al, 2002). The diabetic subjects have 1.5-2.0 times higher prevalence of hypertension than those without diabetes (Arauz-Pacheco et al, 2002; Arauz-Pacheco et al, 2004). The Professional Practice Committee of the American Diabetes Association recommended a blood pressure goal of < 130/80 mmHg for adult patients with diabetes (Standards of medical care in diabetes, 2007).

In 2016, Manjavkar et al, in a cross-sectional study on 100 hypertensive patients attending medicine OPD at postgraduate institute at Mumbai city of age 40 years and above found 43% prevalence of prediabetes and 16% diabetes prevalence. This study showed increased risk of prediabetes and diabetes among individuals with known cases of hypertension.

Framingham study shown higher rates of mortality for all causes and cardiovascular events among participants with hypertension and diabetes mellitus compared to normotensive subjects with diabetes mellitus, suggesting greater risk of cardiovascular complications with coexistent diabetes mellitus and hypertension (Chen et al, 2011).

2.1.7.3 Family History

An emerging line of research into the pathophysiology of type 2 diabetes has identified family history of diabetes as a key risk factor for developing diabetes. (Tsenkova et al, 2016). Family history of diabetes represents genomic information and the complex interplay between genes, shared environments and behaviors, and epigenetic effects. Inheritable type 2 diabetes mellitus ranges from 20% to 80% and studies shown hereditary comes from variety of population, family and twin-based studies (Meigs et al., 2000; Poulsen et al., 1999). According to Tillil et al, risk of type 2 diabetes increases to 40% for individuals who have one parent and 70% for if both parents have diabetes (Tillil et al, 1987). Individuals who have first degree relatives with type 2 diabetes are about three times higher risk for developing diabetes than without a positive family diabetes history (Florez et al, 2003).

2.1.7.4 Physical Activity

The global epidemic of non-communicable diseases such as diabetes is driven by the globalization of western culture and lifestyle. Physical inactivity is the fourth risk factor for global mortality and is estimated for 6% death globally. Furthermore, physical inactivity is estimated to be the main cause of approximately 27% of diabetes and 30% of ischemic heart diseases cases (WHO, 2017). Moreover, with physical activity, improved insulin sensitivity and glucose tolerance as well delayed onset of diabetes among subjects with prediabetes was noted in several randomized controlled trials (Knowler et al, 2002; Lindström et al, 2003; Malin et al, 2012; Mensink et al, 2003).

There are few mechanisms explains the possible association between physical activity and the risk of prediabetes. Obesity is considered as one of the major risk factor for prediabetes (Mayega et al, 2013; Díaz-Redondo et al, 2015). Physical activity increases total energy expenditure, which can help to improve energy balance or lose weight and prevent obesity (Mozaffarian et al, 2011). Moreover, physical activity shown to reduce blood glucose level and helps to improve insulin sensitivity among subjects with or without diabetes (Boulé et al, 2001; Duncan et al, 2003; Mayer-Davis et al, 1998).

Leisure-time physical activities such as sports, exercise and recreational activity, etc. have more advantages than work-related physical activity (Jia et al, 2017). Several studies reported high levels of sedentary time, low levels of daily movement, and little moderate to vigorous physical activity are associated with poor glycemic control (Helmerhorst et al., 2009; Mayer-Davis et al., 1998; Colberg, 2012; Assah et al., 2008).

In 2017, of the 8,204 subjects without diabetes and self-reported prediabetes, high level of total leisure-time physical activity and low level of vigorous leisure-time physical activity were inversely associated with the risk of prediabetes. Among the subjects under the age of 45 years, high level of total leisure-time physical activity and low and high level of vigorous leisure-time physical activity were associated with a decreased risk of prediabetes. Whereas, in the 45 to 65 age group, only high level of total leisure-time physical activity had protective effect on prediabetes (Jia et al, 2017).

In 2017, Færch et al found improvements in insulin sensitivity and β-cell function due to a 5-year increase in moderate-to-vigorous-intensity physical activity. However, physical activity was not associated with reversion to normal glucose tolerance. But, higher amounts of physical activity shown reversion to normal glucose tolerance among women with i-IFG (isolated IFG) or i-IGT (isolated IGT). Furthermore, this study also found only 4.4% HbA1c-defined prediabetes subjects reverted to normoglycemia, and physical activity was not associated with improvement in glycemic markers (Færch et al., 2017).

In 2014, Farni et al noted a negative association between measures of physical activity and the prevalence of prediabetes among 20,470 middle-aged US adult population independent of adiposity (Farni et al, 2014).

2.1.7.5 Nutritional factor

India has different cuisine depending on their social, religion and cultural identity (Vecchio et al, 2014) as well as local agricultural practices and availability of diverse foods (Subramanyam et al, 2010). India is undergoing a significant nutrition transition from traditional diets to ‘Westernized’ food (Mohan V, 2004).

Traditional food pattern has beneficial effect on diabetes mellitus and glucose metabolism, which significantly reduces the risk of developing type 2 diabetes. High consumption of food rich in high monounsaturated fatty acids, dietary fibres, antioxidants and polyphenols such as vegetables, fruits, legumes, nuts, fish, cereals and oil prevent weight gain and exert a protective effect on the development of type-2 diabetes (Asif M, 2014). Supporting this fact Gonzalez et al reported that diet which include high content of whole cereals, vegetable and fruits, adequate amount of proteins, and low intake of trans fatty acids improve insulin sensitivity which results in better glycemic control in diabetic patients (Gonzalez et al, 2008). Whole grains are rich in dietary fiber, starch, antioxidants, phenolic compounds, lignin which have been linked to reduced risk of obesity, insulin resistance, dyslipidemia, high cholesterol, and heart disease (Misra et al, 2009). A paleolithic diet which consist of lean meat, fish, shellfish, fruits and vegetables, roots, eggs and nuts, but not grains, dairy products, salt or refined fats, and sugar was associated with marked improvement of glucose tolerance but did not find significant improvement in glucose tolerance among control subjects though decreases in weight and waist circumference (Holt et al, 1994; Nicholson et al, 1999; UK Prospective Diabetes Group, 1998).

In 2017, Corsino et al found that The Dietary Approaches to Stop Hypertension (DASH) dietary pattern which includes high intake of fruits, vegetables, whole grains, and low-fat dairy products was originally developed to treat hypertension but also found effective in management of insulin resistance and diabetes among among Hispanics/Latinos of US (Corsino et al, 2017).

In 2016, Rosemary et al identified that high-fat and high-sugar foods such as sweets, snacks and animal products significantly increases body size, whilst fruit, snacks and meat were associated with significantly smaller body size. Furthermore, dietary pattern including rice and pulses associated with a lower risk of diabetes or pre-diabetes whereas sweets and snack associated with higher risk of diabetes or pre-diabetes (Rosemary et al, 2016).

In 2016, Bagheri et al examined the relationship between two types of dietary pattern; the vegetables, fruits and legumes (VFL) dietary pattern and the sweet, solid fat, meat and mayonnaise (SSMM) dietary pattern and prediabetes among 150 prediabetic subjects and 150 healthy weight subjects. The VFL dietary pattern was found to be negatively associated with

lower pre-diabetes, whereas SSMM dietary pattern was positively associated with pre-diabetes (Bagheri et al, 2016).

In 2013, Viscogliosi et al reported that Mediterranean Diet (Med Diet), based on the consumption of minimally processed foods, including most of the dietary protective factors, such as vegetables, fruits, unrefined grains, fish, vegetable proteins from pulses, vegetable fats mainly from olive oil, moderate consumption of red wine, and more rarely poultry may protect against metabolic syndrome, prediabetes, and microinflammation in subjects free of diabetes and clinical cardiovascular diseases.

2.1.7.6 Stress

Stress is associated with heart rate variability and cortisol/dehydroepiandrosterone sulfate ratio (Loerbroks et al, 2010; Gadinger et al, 2011). It consists of an interrelated response from the sympathetic adrenomedullary system (SAM) and the hypothalamic pituitary adrenal axis (HPA). Initially, SAM releases epinephrine and nor-epinephrine. On repeated stress, HPA comes into play and increases level of cortisol, which raises blood glucose levels by stimulating hepatic gluconeogenesis, and inhibiting the action of insulin (Bergmann et al, 2014). The neuroendocrine system regulates glucose uptake, release and storage. Upon sustained stress this system activates and increases metabolic activity without corresponding need, which may predispose the development of diabetes (Balanos et al, 2010; Felšöci et al, 2011). Activated neuroendocrine system also increases accumulation of abdominal fat (Bjorntorp P, 1991). Chronic stress also activates the innate immune system, resulting in increased levels of inflammatory cytokines such as interleukin 6, which are involved in mediating insulin resistance (Pickup, 2004).

Another plausible mechanism is that during stress numerous metabolic changes occurs including vasoconstriction in the peripheral vascular system, increased heart rate, increased muscle activity, and increased production of stress hormones, which elevate blood glucose levels as extra energy required to combat stress (Lustman et al, 1995).

Psychological distress symptoms, such as stress, anxiety, depression, feelings of hopelessness, and sleep disturbances interfere with long-term lifestyle changes and also found to reduce

adherence to pharmacological treatment. Several studies reported psychological factors such as self-efficacy (Ikeda et al, 2003), self-esteem (Kneckt et al, 2001), and social support (Glasgow et al, 1989) were found to be associated good glycemic control; whereas factors such as stressful life events (Aikenset al, 1997), daily environmental stressors (Toobert et al, 1991), and diabetes-related distress (Polonsky et al, 1995) were associated with poor glycemic control.

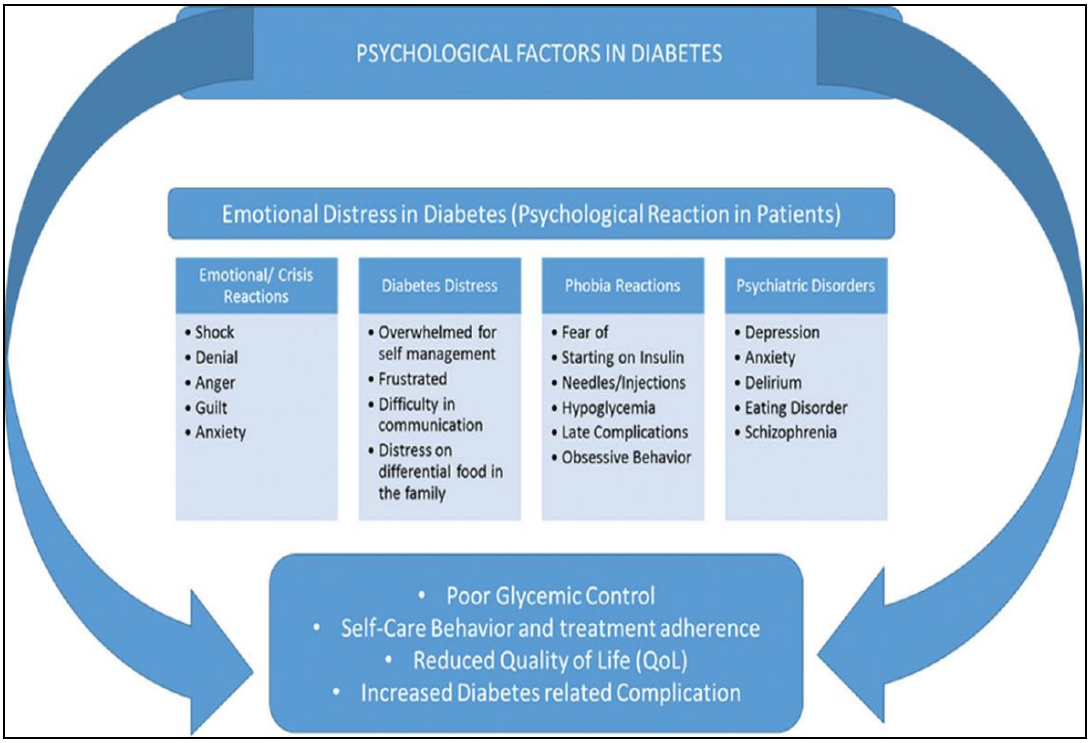

Figure 2.2: Psychological factors and reactions with negative outcomes in patients with diabetes (Kalra et al, 2018)

In 2018, a cross-sectional study was carried out on 1,300 male office workers of in Seoul, Korea. This study found that subjects with high scores on the subscales of occupational stress (interpersonal conflict, job insecurity, and occupational climate) had a high risk for prediabetes (Ryu et al, 2018).

In 2016, large national cohort study on 1.5 million young men found that low stress resilience is associated with a higher risk of developing type 2 diabetes. This study further shown stress resilience in late adolescence decreases the risk of type 2 diabetes risk in adulthood (Crump et al, 2016).

In 2014, Virtanen et al reported that among participants with prediabetes, 40.9% of those with psychological distress compared with 28.5% without distress developed diabetes during the follow-up. Furthermore, this study found that psychological distress is associated with a doubling of diabetes risk in a high-risk populations (Virtanen et al, 2014)

2.1.7.7 Socioeconomic status and education

Diabetes mellitus is no longer the rich man's disease. It has also become a problem of middle and lower income class of the society.

Socioeconomic condition in early life have an effect on biological, behavioural and social factors which act as a mechanism for link between socioeconomic status and type 2 diabetes mellitus (Kuh et al, 2003). Children belonged to lower socioeconomic class are more likely to have low education and thus maintain their low socioeconomic status in adulthood (Galobardes et al, 2004). Studies shown that children of low socioeconomic class are at increased risk of developing childhood obesity (Shrewsbury et al, 2008) and maintain their label of obesity though adulthood, which puts them at high risk of developing type 2 diabetes mellitus in later life (Juonala et al, 2011).

In 2017, Rathinavelu et al conducted a study on subjects of 18 years or above and had shown a significant association between occupation and DM, education and DM, income of head of the family and DM. This study further reported that participants with low income have a higher prevalence of diabetes than wealthy participants (Rathinavelu et al, 2017).

In 2017, a workplace cross-sectional survey on 4,828 public service workers of Nigeria was carried out by Aladeniyi et al. The researchers found higher prevalence of diabetes among individuals with lower education (Aladeniyi et al, 2017).

In 2017, Derks et al has conducted a cross-sectional study on data from The Maastricht Study. The aim of the study was to examine the association of socioeconomic conditions in early life with prediabetes and type 2 DM in adulthood. The researchers found that the participants with low early life socioeconomic conditions significantly more often had prediabetes or type 2 DM, compared to participants with high early life socioeconomic conditions (Derks et al, 2017).

In 2017, the ICMR-INDIAB study found higher prevalence of diabetes in the more economically developed states. Furthermore, within states diabetes was more common in individuals of medium or high socioeconomic status than in individuals of low socioeconomic status. The study further found that the prevalence of diabetes was higher in individuals of low socioeconomic status in the urban areas of seven states, whilst in rural areas, the prevalence of diabetes was higher in individuals of higher socioeconomic status in all the states of India studied including Tamil Nadu, Jharkhand, Chandigarh and Maharashtra (Anjana et al, 2017).

In 2015, Rahmanian et al conducted study on 788 subjects of 30-85 years of age group of Iran. The author reported that pre-diabetic subjects were low educated than normoglycemic subjects. The educational level was found inversely associated with prediabetes in this study (Rahmanian et al, 2016).

2.1.7.8 Lipid abnormalities

Lipid abnormalities are common among individuals with type 2 diabetes mellitus and prediabetes (Mooradian, 2009; Santos-Gallego et al, 2014). “Diabetic dyslipidemia” is commonly used term for lipid abnormalities in diabetes patients. It is characterized by high total cholesterol, high triglycerides, low high density lipoprotein cholesterol and increased levels of small dense low density lipoprotein (LDL) particles (Goldberg, 2001). Dyslipidemia increases macrovascular complications in prediabetes and diabetics.

Lipid abnormalities among diabetes patients could be due to defects in insulin action and hyperglycemia (Goldberg, 2001). Type 1 diabetes, is associated with insulin deficiency. Hypertriglyceridemia and low levels of high density lipoprotein (HDL) are commonly seen among individuals with poorly controlled type 1 diabetes and ketoacidosis (Ginsberg, 1996). Exogenous insulin therapy in such patients may correct elevated glucose level, which in turn normalize HDL and triglycerides level.

Likewise in type 1 diabetes, the commonly occurring lipid abnormalities in type 2 include hypertriglyceridemia and reduced plasma HDL cholesterol. Additionally in type 2 diabetes, low density lipoprotein are converted to smaller, lipoproteins termed small dense low density

lipoproteins (Krauss, 1994). But unlike type 1 diabetes, these abnormalities are not usually fully corrected with glycemic control. Furthermore, type 2 diabetes dyslipidemia is often seen among prediabetes subjects, patients with insulin resistance but normal indexes of plasma glucose (Haffner et al, 2000). Hence, abnormalities in insulin action but not hyperglycemia are associated with lipid abnormality. The plausible factors responsible for diabetic dyslipidemia are insulin effects on liver apoprotein production, regulation of lipoprotein lipase, actions of cholesteryl ester transfer protein, and peripheral actions of insulin on adipose and muscle (Goldberg, 2001).

In 2018, a population-based cross-sectional study on Bangladeshi subjects was carried out by Bhowmik et al. This study found higher prevalence of hypercholesterolemia, hypertriglyceridemia and low HDL-C among participants with type 2 diabetes mellitus and prediabetes than those with normal glucose tolerance (Bhowmik et al., 2018).

In 2018, Wankhade et al., conducted a cross-sectional study among 300 male employees from a packaging and binding industry in Maharashtra, India. This study found abnormal triglyceride level was associated with high blood sugar level and hypertension. Further the researchers noted that abnormal total cholesterol level was associated with high body fat percentage and hypertension whereas abnormal HDL level was associated with hypertension and high cholesterol/HDL ratio (Wankhade et al, 2018).

In 2017, Balgi et al. carried out a cross-sectional, case control study on patients of age > 18 years attending K. R. Hospital, Mysore. This study included 100 cases and 100 age and sex matched control. The mean values of total cholesterol, LDL, triglyceride, and VLDL were significantly higher whereas HDL was significantly lower in prediabetes participants as compared to normal healthy participants (Balgi et al, 2017).

In 2017, Veeramalla et al conducted study on 100 diabetes and 100 non-diabetes healthy participants attended tertiary care hospital in India. The aim of the study wad to identify lipid levels among diabetes and non-diabetes participants. The study identified higher prevalence of elevated total cholesterol, triglycerides and VLDL was noted among diabetes participants than non-diabetes participants. Whilst HDL was significantly low among diabetes than non-diabetes participants (Veeramalla et al, 2017).

In 2017, Anisa Tia et al, among 69 patients of prediabetes reported weak negative correlation between HbA1c level and lipid profile including triglyceride and total cholesterol. Further it was documented that lipid abnormalities among prediabetes participants may be due to absence of insulin resistance or other factors compared to HbA1C level (Anisa Tia et al, 2017).

In 2016, a case control cross sectional study reported that total cholesterol, low density lipoprotein, triglyceride, very low density lipoprotein, TG/HDL ratio and LDL/HDL ratio were significantly raised in prediabetic individuals as compared to normal healthy subjects, whereas high density lipoprotein (HDL) was significantly lower in prediabetic individuals as compared to normal healthy subjects (Kansal et al, 2016).

2.1.7.9 Vitamin D deficiency

Vitamin D deficiency is a global health problem affecting people of all the age group. Several factors are responsible for hypovitaminosis D; inadequate cutaneous production from 7-dehydrocholesterol, low dietary intake or impaired intestinal absorption of vitamin-D. The newer factors such as obesity and high levels of environmental pollution have been identified to be linked with vitamin D deficiency (Savastano et al, 2013).

Vitamin D is a crucial and essential micronutrient for human health. It is associated with many nonskeletal effects, including its potential in pancreatic insulin synthesis and secretion as well as insulin action (Rosen et al, 2012) because vitamin D response element in the human insulin gene promoter and the transcriptional activity of the human insulin gene caused by 1, 25-dihydroxyvitamin D, the active form of vitamin D (Gedik et al, 1986).

Few studies evidenced that subjects with severe vitamin-D deficiency have the highest insulin resistance (Holick et al, 2004; Dutta et al, 2013). Epidemiological study reported that vitamin D is common amongst individuals with diabetes (Holick et al, 2007). Whilst many studies suggest that low level of vitamin D is inversely associated with impaired glucose tolerance and diabetes (Pittas et al, 2010; Forouhi et al, 2012; Deleskog et al, 2012). Moreover, studies demonstrated that increased intake of vitamin D was significantly associated with a lower risk of type 2 diabetes (Pittas et al, 2006). Further, another study shown that vitamin D and calcium

supplementation can even improve glucose homeostasis in adults with impaired fasting glucose (IFG) (Pittas et al, 2007). The low level of vitamin D may affect glucose homeostasis and parathyroid concentration among prediabetes subjects (Karras et al, 2018; Gandhe et al, 2013).

In 2018, Gao et al. in China conducted study on 490 non-prediabetes and non-diabetes participants. The study found that low 25(OH)D levels were significantly associated with the onset of prediabetes and type 2 diabetes mellitus in the Chinese population. Further it was suggested that 25(OH)D is independently predictive in the development and pathophysiology of hyperglycemia, including both prediabetes and type 2 diabetes mellitus (Gao et al, 2018).

In 2018, Ayhan et al, carried out a study on 247 patients og age between 20-65 years attending Istanbul Haseki Training and Research Hospital. According to this study, the mean plasma 25[OH]D level (25.7±14.9 nmol/l) was statistically lower among prediabetes participants than the control group (31.4±17.8 nmol/l). This study further noted that serum low 25[OH]D level correlated with insulin resistance and metabolic parameters in prediabetic patients (Ayhan et al, 2018).

In 2018, Park et al conducted study on participants from the Rancho Bernardo Study, a population-based cohort of primarily older, middle-income, community-dwelling Caucasian adults living in a southern California suburb. The researchers found that higher 25(OH)D concentrations (>30 ng/ml) were associated with lower hazard ratios (HR) for diabetes: 30–39 ng/ ml, HR = 0.31; for 40–49 ng/ml, HR = 0.29; for>50 ng/ml, HR = 0.19. All HRs are compared to <30 ng/ml. The author further reported that 25(OH)D concentrations were more weakly inversely associated with pre-diabetes risk, and the trend was not significant (Park et al, 2018).

In 2016, a study on 202 obese Swedish children between 4.5 and 17.9 years of age was carried out by Ekbom et al. The researchers reported 9.1% prevalence of IFG. The prevalence of IFG was 16.7%, which was found higher among vitamin D deficient children than non-deficient children (5.3%). This indicate that vitamin D deficiency was associated with an increased risk of a disturbed glucose metabolism (Ekbom et al, 2016).

In 2016, Srinath et al, included 40 prediabetes and 40 healthy participants of South India in a case control study. This study found around 72.5% prediabetes and 35% of healthy participants had vitamin D levels less than 20 ng/ml whereas, 5% of the prediabetes and 12.5% of the healthy participants had vitamin D above 30 ng/ml and this difference was statistically significant. Overall mean vitamin D levels in prediabetes and healthy participants was 17.09±5.89 ng/ml and 23.67±11.02 ng/ml respectively. This shown high prevalence of hypovitaminosis D exist among prediabetes than healthy participants.

2.1.7.10 Inflammation

Studies have found the link between an inflammation and predictive risk of development of type 2 diabetes mellitus (Hotamisligil et al, 2006; Bertoni et al, 2010; Barzilay et al, 2001). Type 2 diabetes mellitus is associated with rise in proinflammatory cytokines such as interleukin- (IL-) 1, IL-6, tumor necrosis factor- (TNF-) α, C-reactive protein (CRP), transforming growth factor- (TGF-) β, and leptin or the fall in anti-inflammatory cytokines including, interleukin-1 receptor antagonist (IL-1Ra), IL-4, IL-10, IL-13, and adiponectin. This increase or decrease in inflammatory cytokines is a crucial step in glucotoxicity and lipotoxicity induced mitochondrial injury, oxidative stress, and beta cell apoptosis occurs in type 2 diabetes mellitus (Banerjee et al, 2014; Brunetti et al, 2014; Saxena et al, 2013). Moreover, these pro- and anti-inflammatory cytokines can enhance insulin resistance directly in adipocytes, muscle, and hepatic cells which may lead to systemic disruption of insulin sensitivity and impaired glucose homeostasis (Arora et al, 2011).

In 2019, Kato et al conducted study on 4,362 subjects of Ota Memorial Hospital, Ota City, Gunma, Japan. In this study, researchers found that subjects with impaired IFG, impaired IGT or combined IFG/IGT had higher median levels of serum CRP (0.70 mg/L, 0.70 mg/L, and 0.70 mg/L, respectively) than subjects with normal glucose tolerance (0.50 mg/L). This study further demonstrated presence of inflammation in prediabetes subjects (Kato et al, 2019).

In 2017, Brahimaj et al found wide range of inflammatory markers for phase-specific prediction of progression to type 2 DM and identified EN-RAGE, IL-13 and IL-17 as novel inflammatory markers. Higher EN-RAGE levels were associated with an increased risk of incident

prediabetes. Higher IL-13 levels were associated with a decreased risk of prediabetes, incident type 2 DM as well as it also indicates need for insulin therapy. Higher IL-17 levels were associated with a decreased risk of incident type 2 DM (Brahimaj et al, 2017).

In 2016, Wang et al carried out a study on 7,054 rural residents aged 50–75 years from the city of Huzhou, china. According to this study, the median levels of IgE in type 2 diabetes mellitus subjects (40 IU/L) were significantly higher compared to subjects with normal glucose (18 IU/L) level and prediabetes (20 IU/L). Median levels of CRP, tryptase, IL-4 and IL-10 in prediabetes subjects or type 2 diabetes mellitus subjects were significantly higher compared with subjects with normal glucose level. However, this study did not find significant differences in IL-6, TNF-α, and Foxp3 (Wang et al, 2016).

2.2 Overweight and Obesity

The prevalence of obesity is increasing worldwide at higher rate. It is a disorder of all the age groups and now on rise in low and middle income countries also. It is the main risk factor for many non-communicable disease and it itself is a non-communicable disease (Purnell et al, 2018). WHO has described obesity as a chronic disease of the developed or developing countries, which is replacing the more traditional public health concerns, such as undernutrition and infectious diseases (WHO, 1998).

Obesity is defined as accumulation of abnormal or excessive fat in adipose tissue to the extent that deteriorates the health and well-being (Garrow et al, 1988; WHO Technical Report Series; 2000). The distribution of excess fat in the body mainly around the waist and truck (abdominal or central obesity; male or apple shaped) and peripherally (gynoid obesity; female or pear pattern) have their own health implication. Central obesity is associated with higher morbidity and mortality rate than peripheral distribution of fat (Kissebah et al, 1994).

WHO in 2016 reported that more than 1.9 billion (39%) adults of age 18 years and older were overweight. Of these, over 650 million adults (13%) were obese. From 1975 to 2016, the worldwide prevalence of obesity among adults nearly tripled. Overweight or obesity among children under the age of 5 years was estimated to be 41 million. Nearly 50% of children under the age of 5 years were overweight or obese in Asia. Around 340 million children and

adolescents of age 5 to 19 years were found overweight and obese. The prevalence of overweight and obesity increased from 4% in 1975 to 18% in 2016 (WHO, 2016).

According to Organization for Economic Co-operation and Development (OECD) updates (2015), the prevalence of obesity among adults across the OECD countries was 19.5%. The rate of prevalence was the lowest nearly 6% in Korea and Japan and the highest 30% in Hungary, New Zealand, Mexico and the United States. In addition, this report further noted that more than one in four adults was obese in Australia, Canada, Chile, South Africa and the United Kingdom. The prevalence of overweight or obesity at the age of 15 was 10% in Denmark and 31% in United States. The increased prevalence of obesity is expected to be high in the United States (47%), Mexico (39%) and England (35%), whereas the increase is expected to be weaker in Italy (13%) and Korea (9%) in 2030 (OECD Health Statistics, 2017). India is the third largest number of obese people after the United States of America and China (Mehta et al, 2016).

Obesity is a central player for the pathophysiology of many chronic conditions such as type 2 diabetes mellitus, cardiovascular diseases (CVD) including hypertension, stroke and coronary heart disease as well as gall bladder disease, certain cancers (endometrial, breast, prostate, colon) and non-fatal conditions like gout, respiratory conditions, gastro-esophageal reflux disease, osteoarthritis and infertility (Ofei et al, 2005; Redinger et al, 2007).

2.2.1 Pathophysiology of Obesity:

Obesity involves an epidemiological triad between genetic susceptibility and environmental / lifestyle adaptation. Genetic susceptibility deal with the genes encoding for the circulatory protein like leptin and melanocortin, wherein leptin is responsible for relaying the information to the central nervous system about the abundance of energy stores, suppression of food intake and permit energy expenditure. Absence of leptin culminates into obesity due to increased appetite and food intake, although many obese individuals have high circulating levels of leptin that can be regarded as leptin resistance (Gadde et al, 2018).

Environmental and life style adaptation also have an important role to play in the pathogenesis of obesity due to the effect of energy balance involving both energy intake as well as energy

expenditure as prime factor for its maintenance. Energy intake is primarily controlled by the central nervous system, especially the hypothalamus also known as the master nutrient sensor that integrates with other areas of the brain such as the cortex and the limbic system involved in processing the external sensory stimuli (taste, visual, olfactory and auditory) especially in the presence of food and also with hindbrain involved with satiation. The interaction of these three areas of the brain to a very greater extent influence the food intake with respect to ingestion or rejection / stopping of food intake that directly influences the endocrine and autonomic nerves to release hormones in connection with gut to digest and absorb the nutrients (Gadde et al, 2018).

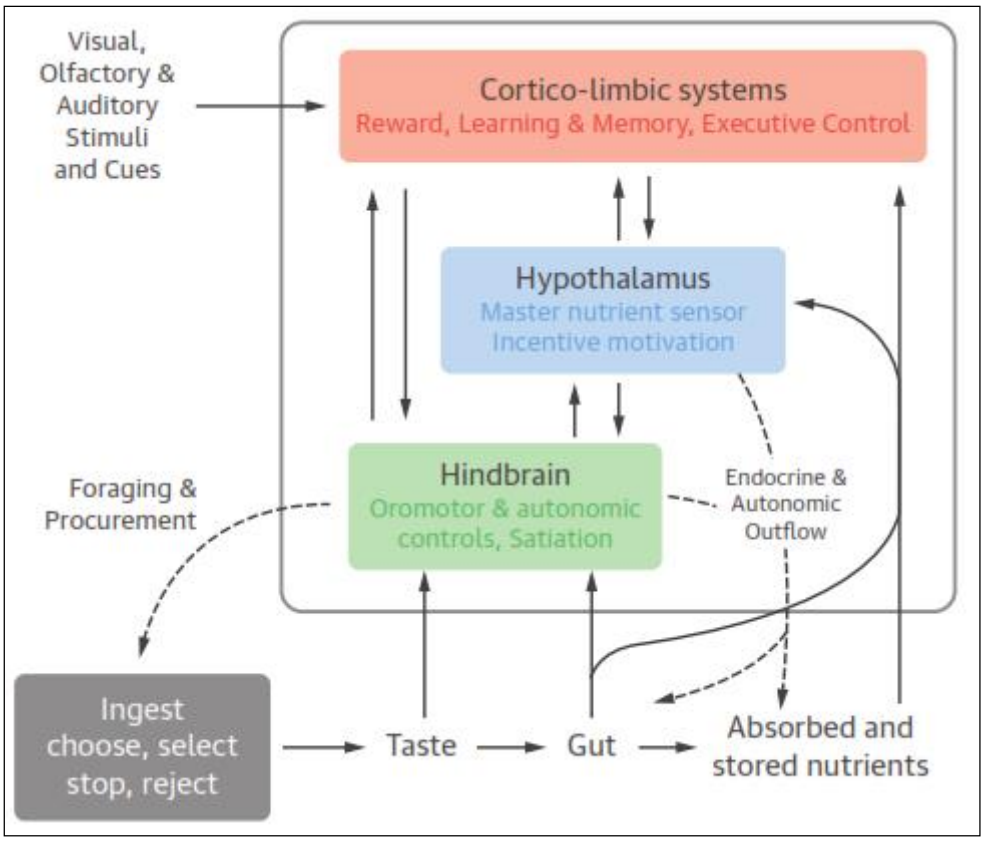

Figure 2.3 Neural pathways and system controlling ingestive behavior and energy balance (Gadde et al, 2018)

Energy expenditure is also considered a vital factor in energy balance which is closely related to the food intake and the ability to regulate body weight and obesity. Thus, all these factors

play a central role is affecting the food behavior and thereby contributing in the pathogenesis of obesity (Gadde et al, 2018).

2.2.2 Measurement of BMI

Though excess body weight is associated with adiposity, weight alone cannot justify obesity, because height is associated with it (Troiano et al, 1999). Body Mass Index (BMI) is defined as weight (kg)/height squared (m^2); which is the most frequently used measure of weight in relation to height (Sweeting et al, 2007). Due to considerable change in BMI with age, the cutoffs of BMI are different for children and adolescents compared to adults.

Among adults, a WHO Expert Committee on Physical Status, in 1993, classified BMIs as follows: < 18.5 as ‘underweight’; 18.5-24.9 as ‘healthy weight’; 25–29.9 as 'overweight' (or 'pre-obese'); 30–34.9 as 'obese class I'; 35–39.9 as 'obese class II'; and over 40 as 'obese class III'.

To assess physical growth in infants, children and adolescents, 2000 Centers for Disease Control and Prevention (CDC) Growth Charts are used to describe and interpret the classification of overweight and obesity. This growth chart is the revised version of the growth charts developed by the National Center for Health Statistics (NCHS) in 1977. BMI-for-age (Weight (kg)/Height (m^2)) is a tool used to screen children of 2 to 20 years of age to classify BMI as follows: ≥ 95^{th} percentile as ‘obese’; ≥ 85^{th} to < 95^{th} percentile as ‘overweight’; ≥ 5^{th} to < 85^{th} percentile as ‘healthy weight’ and < 5^{th} percentile as ‘underweight’.

World Health Organization (WHO) published new growth standards for children under the age of 5 years in 2006 which are being adopted in many countries including India as a global single standard of childhood growth. Standard growth chart has 7 percentile lines and include 3^{rd}, 10^{th}, 25^{th}, 50^{th}, 75^{th}, and 97^{th} percentiles. These percentiles are standard for height and weight charts. Any individual who is below 3^{rd} and above 97^{th} percentile is considered out of normal range. For the BMI charts, however, there are 85^{th} and 95^{th} percentile lines which indicate overweight and obesity cut offs. Proportion charts use Z score lines instead of percentile lines and discrepancy of more than 2Z scores in the upper and lower segment is considered abnormal.

The correlation between Z scores and percentiles described in the recent WHO Multicenter Growth Reference (MGRS) 2006 study, which is as follows (Khadilkar et al, 2011).

Table 2.4 Correlation between percentiles and Z scores for World Health Organization chart (Khadilkar et al, 2011)

Z-score	Exact percentile	Rounded percentile
0	50	50
-1	15.9	15
-2	2.3	3
-3	01	1
1	84.1	85
2	97.7	97
3	99.9	99

Table 2.5 Growth parameters and their interpretation for the World Health Organization charts (Khadilkar et al, 2011)

Z Score (Percentile)	Length/height for age	Weight for age	BMI for age
> 3 (99)	May be abnormal	May be abnormal (Use BMI)	Obese
> 2 (97)	Normal	Use BMI	Overweight
> 1 (85)	Normal	Use BMI	Risk of overweight
0 (50)/	Normal	Use BMI	Normal
< -1 (15)	Normal	Normal	Normal
< -2 (3)	Stunted	Underweight	Wasted
< -3 (1)	Severely Stunted	Severely underweight	Severe wasted

2.2.3 Prevalence of overweight and obesity

2.2.3.1 National studies

Table 2.6 Description of prevalence of overweight and obesity in India

Year	Author	Area of study	Age	Prevalence			
				Overweight		Obesity	
				Boys	Girls	Boys	Girls
2018	Kumar et al	Chandigarh, India	11-16 years	2.4%	2.4%	12.1%	6.1%
2018	Raj et al	Tamil Nadu slum resident area, India	> 18 years	21.4%	43.9%	9.7%	13.7%
				36.5%		12.4%	
2018	Gupta et al	Nainital District, Uttarakhand State, North India.	> 60 years	18%		4.6%	
2017	Meharda et al.	Udaipur, India	14-16 years	8.20%		2.40%	
2017	Eshwar et al	Rajkot, Gujarat, India	8-18 years	IAP standard: 19.1% IOTF standard: 15.8% WHO standard: 15.3%		IAP standard: 14% IOTF standard: 5.1% WHO standard: 11.1%	
2017	Mishra et al	Sambalpur district, Odisha	10-12 years	5.6%	7.4%	3.4%	3.3%
2017	Sharma et al	Bikaner, Rajasthan, India	16-19 years	10%		4.54%	

2016	Prasad et al	Pondicherry, South India		10-18 years	14%		
2016	Girdhar et al	Ludhiana, India		20-60 years	12.7% (BMI: 23-24.9)		29.6% (BMI: > 25)	
2016	Chandrakala et al	Bangalore, India		12-18 years	5.1%	8.2%	1.5%	5.9%
2010	Goyal et al	West India		12-18 years	14.3%	2.9%	9.3%	1.5%
2014	Rajkamal et al	Puducherry, India		> 60 years	41.4%		4.5%	
2014	Jagadesan et al	Chennai, India	Gov. School	6-11years	1.6%	2.6%	0.3%	0.4%
			Private School		16.2%	13.7%	4.2%	3.9%
			Gov. School	12-17 years	3.6%	4.1%	0.4%	1.1%
			Private School		17.9%	19.2%	4.6%	4.6%
2014	Jiwane et al	Maharastra, India		5-19 years	5.08%		3.43%	
2013	Shah et al	Mehsana, Gujarat, India		10-12 years	34.82 %	32.95%	13.39 %	7.95%
2013	Selvaraj et al	Kanchipuram district, Tamil Nadu, India		19-23 years	24.3%		8.6%	
2013	Sen J	Jalpaiguri, West Bengal, India		20-60 years	23.67 %	20.33%	9.67%	29.33 %
2012	Alok et al	Surat, Gujarat, India		14-16 years	Rural: 25.8% Urban: 26.3%		Rural: 12.8% Urban: 14.6%	
2012	Kamath et al	Bangalore, India		12-15 years	10%		5%	

2012	Mandal et al	Kolkata, India	12-18 years	28.5%		4.2%	
2010	Goyal et al	West India	12-18 years	14.3%	2.9%	9.3%	1.5%

2.2.3.2 International studies

Table 2.7 Description of international studies demonstrating prevalence of overweight and obesity

Year	**Author**	**Area of study**	**Age**	**Prevalence**			
				Overweight		**Obesity**	
				Boys	**Girls**	**Boys**	**Girls**
2018	Liu et al	Chinese rural adults	18-79 years	34.97%		General obesity: 16.82% Abdominal obesity: 43.71%	
2018	Chowdhury et al	Bangladesh	15-46 years	-	28.37 %	-	10.77 %
2018	Jakab et al	Jász-Nagykun-Szolnok county, Hungary	3-18 years	13.4%		6.6%	
2017	Hu et al	Southern China	≥ 15 years	25.9%	25.7%	8.4%	7.6%
2017	Do et al	Vietnam	3-6 years	16.7%		4.5%	
2017	de Araujo	Brazil	2-5 years	4.2%		0%	
2017	Razzak et al	United Arab Emirates	-	-		16-28.4%	
2017	Snook et al	NHANES data, United States	20-59 years	68.20%			

2017	Afthentopoulou et al	Athens, Greece	4-8 years	18.1%	18.3%	5.6%	8.8%
2016	Wang et al	Jilin Province, Northeast China	18-79 years	34.3%	30.2%	16.3%	12.8%

2.2.4 Risk factors associated with overweight and obesity:

2.2.4.1 Socioeconomic status:

In the most developing nations like India, where there is problems like undernutrition and anemia, the country also witnessed the problems of overweight and obesity (Gouda et al, 2014). Hence, the country is burdened with two different nutrition related health problems (Kennedy et al, 2006). The urban population in India is around more than 30% and it expected to increase to 900 million or 55% by 2050 (India, Ministry of Home Affairs, 2011; United Nations, World Urbanization Prospects, 2008). As India is undergoing a rapid and unplanned urbanization, the intra-urban socioeconomic discrepancies are rising, and thus inequity in health among urban population has become an apparent challenge. On one hand, education was a primary indicator associated with adiposity in the developed countries while on the other, it was socio-economic in developing countries (Sobal et al, 1989).

Social and economic development and ongoing epidemiological transition leads to disease transition from communicable to non-communicable diseases. In socioeconomically primitive culture; risk factors for non-communicable disease such as smoking, physical inactivity, diets rich in carbohydrate and calories but low in fibers and stress are more in high socioeconomic status subjects (Yusuf et al, 2001).

It is plausibly said that socioeconomic status is a proximal risk factor which affects the underweight, overweight and obesity (Martinez et al, 2000). Families belonging to higher socioeconomic class likely to have different lifestyle including changes in diet, food consumption pattern, public services including health care and transportation as well physical activities. Socioeconomic markers such as education, income, and occupation-based wealth indices are associated with obesity (Zaman et al, 2012; Kinra et al, 2010). Low socioeconomic

subjects have limited food intake and excessive manual labour, hence it becomes difficult for them to achieve net positive energy intake required to maintain or gain weight (Zaman et al, 2012; Humphreys et al, 1998; Agrawal et al, 2011). These associations explains that social determinants play a role in both over- and undernutrition (Sobal et al, 1989).

In 2018, Ahmad et al conducted study on 3,798 school adolescents of 12-year age in Terengganu, Malaysia. This study found adolescents from high SES has strong prediction for obesity whereas adolescents with a medium SES level had a minor increase in their risk of obesity. Further this study shown significant association of high household income group with obesity, whilst household size showed an inverse relationship with the BMI status among adolescents.

In 2018, Luhar S et al, studied the trends in the socioeconomic patterning of overweight/obesity in India. The study used nationally representative data from India collected in 1998/1999, 2005/2006 and 2015/2016 of 15-49 years old women and 15-54 years old men. This study found overweight/obesity prevalence was consistently highest among higher socioeconomic position individuals (Luhar et al, 2018).

In 2018, a community based cross-sectional study on adults of age 20 years and above at Srikot, Uttarakhnad, India was carried out by Rautel et al. It was found that 80.2% obese participants were of upper socioeconomic class and 19.8% obese participants were of middle socioeconomic class. This study further reported 34.9% obesity among employed and 65.1% among unemployed/ students/ homemaker participants (Rautela et al, 2018).

A study on 800 school girls (6-10 years) of Jaipur city was carried out by Singh et al in 2018. This study found significant relationship between obesity and socioeconomic status among children of private school whereas, insignificant association between obesity and socioeconomic status was noted among children of government school (Singh et al, 2018).

In 2015, Taneja M et al, performed a cross sectional survey study among 5,993 school going adolescents (10-19 years) of Ambala district, Haryana, India. The family income in this study defined the economic class into 4 categories such as, category I: Monthly income of ≤ 12,000

rupees, category II: Rs 12,001–60,000, category III: Rs. 60,001–1,20,000 and Category IV: >Rs 1,20,000. This study noted higher prevalence of overweight (11.7%) and obesity (3.4%) among participants with family income of more than 1,20,000 Rs, following 9.9% overweight and 2.3% obesity among category III income group. Whilst overweight (3.3%) and obesity (0.4%) was noted the lowest among participants belonging to category I. The study found significant association between obesity and socioeconomic conditions (Taneja et al, 2015).

2.2.4.2 Family history of obesity

Family history and personal genomics plays vital role in development of chronic disease (Khoury et al, 2010). The family history of obesity are associated with environmental and biological risk factors (Chen et al, 2005). In terms of environmental risk, parents, particularly mother, inculcate eating habits, lifestyle and their perception of weights to their children (Powers et al, 2006; Chen et al, 2005). In terms of its biological nature, the genetic makeup of overweight/obesity parents, especially when both parents are overweight/obese, may predispose their children to developing this condition (Berkowitz et al, 2005).

The role of family history of obesity in development of obesity was explained by Niranjan et al in 2016. This study was conducted on 280 medical students of Rewa, Central India and reported higher prevalence of overweight and obesity among participants with family history of obesity. The association between family history of obesity and higher BMI was noted significantly (Niranjan et al, 2016).

An observational study conducted in school adolescents between the ages 11 to 19 years of Bangalore, India by Vedavyathi et al in 2016. The prevalence of obesity among male and female gender was 6.9% and 4.2%, respectively. Family history of obesity was present among 9.9% participants, of which 22.97% participants were obese. Further, this study found that the family history of obesity was significantly associated with obesity (Vedavyathi et al, 2016)

Sharma M (2014) conducted a study on school children of 10-18 years. The prevalence of overweight and obesity was 12% and 4.5%, respectively. Further the incidence of obesity/overweight was found to be significantly higher in those with family history of obesity (Sharma M, 2014).

2.2.4.3 Physical activity:

Globally, the level of physical activity declines in recent decades (Ng et al, 2012). Physical activity on regular basis shown to overcome many of the health problems associated with being overweight or obese (Blair et al, 1999). WHO (2014) demonstrated that increased prevalence of non-communicable disease such as hypertension and diabetes are linked with increased obesity prevalence.

The Physical Activity Guidelines for Americans (2008) suggests at least 150 minutes per week of moderate-intensity activity or 75 minutes per week of vigorous intensity aerobic physical activity. To promote weight loss, more than 300 minutes per week of moderate intensity activity is needed (Kushner et al, 2012).

Physical activities such as walking, sports, gardening, climbing stairs or household chores should be included in daily routine life. Colleges and schools should be encouraged to provide physical knowledge and provide facilities outdoor sports activities. Other forms of physical activity such as Indian dance forms or martial arts should be promoted. Regular practice of yoga reduces obesity and helps to control over mind. Together exercise and dietary modification is the most beneficial approach for the treatment of obesity than exercise alone.

In India, with urbanization, industrialization and mechanization, individual's work load has decreased, whereas sedentary activities and energy intake have increased. The decreases in physical activity and increase in sedentary behavior among children may predicts their body mass in late adolescence. Television watching time, sleeping pattern in afternoon, and decreased participation in sports as well as exercise have been associated with childhood obesity since reduction in physical activity results in decreased resting metabolism (Goyal et al, 2010). The short sleep duration is also found to have an association with risk of childhood obesity (Brown et al, 2015). Among all sedentary behaviors, watching television is more strongly associated with obesity (Tremblay et al, 2011).

In 2018, Arifa et al, carried out a cross sectional community based study on school going children of both sex aged 6-14 years in rural and urban areas of Jammu. According to this study,

about one third of overweight and obese were noted to indulge in low physical activity and were 6 times a higher risk. More than 90% overweight and obese children had sedentary behaviour and were 2.2 times at higher risk (Arifa et al, 2018).

In 2017, a comparative, cross-sectional study on 600 children of class VI to X of two government and two private schools was carried out by Panda et al. This study found that children spend plenty of time in watching television and playing video games. Furthermore, the study observed around 55% of both obese and non-obese children were used to watch television and play video games. However, the association of watching television and obesity was not found significant (Panda et al, 2017).

In 2016, Tripathy et al, conducted a household survey in the state of Punjab among 5,127 individuals of 18-69 years of age. The researchers found extreme low level of recreational physical activity in India with 90% of population in both urban and rural areas reporting no physical activity during leisure time (Tripathy et al, 2016).

A significant positive association between sedentary lifestyle and overweight, obesity, high waist to hip ratio and high waist circumferences was noted by Singh et al (2015) in a community-based, cross sectional study on 1,047 participants of 25-64 years age group of urban area of Ludhiana, India (Singh et al, 2015).

2.2.4.4 Nutritional Factor:

An imbalance between energy intake and energy expenditure results in obesity (Seidell, 1998). When energy expenditure is less in compared to energy intake, a state of positive energy balance occurs which results in an increase in body mass, of which 60% to 80% is usually body fat (Hill et al, 1996).

Three major dietary patterns; ‘cereals-savoury foods’ (cooked grains, rice/rice-based dishes, snacks, condiments, soups, nuts), ‘fruit-veg-sweets-snacks’ (Western cereals, vegetables, fruit, fruit juices, cooked milk products, snacks, sugars, sweets) and ‘animal food’ (red meat, poultry, fish/seafood, eggs) were identified through principal components analysis (PCA) of dietary intake data in a large cross-sectional study of rural and urban populations in India. Positive

graded associations were found between the 'animal-food' pattern and obesity risk factors. Whereas, moderate intake of the 'cereals-savoury foods' pattern was associated with reduced odds of obesity and central obesity risk (Satija et al, 2015).

The developing countries like India undergoes globalization which brings nutritional transition and new eating practices driven by desires for status and convenience. These have led to dietary and lifestyle changes that encourage the consumption of high- value added foods, including processed foods and food consumed outside the home (Keshari et al, 2016). Furthermore, the introduction of fast-food chains and Westernized dietary habits providing meals with empty calories with no nutritional value seems to be an important cause for increasing prevalence of obesity. This high-energy density food is rich in fat, mainly industrially produced trans fatty acids. High intake of trans fat may produce abdominal obesity which is an important marker in the metabolic syndrome, type 2 diabetes and cardiovascular disease (Bhattacharjee et al, 2017).

In 2018, Rautel YS et al carried out a community based cross sectional study on adults of age 20 years and above at Srikot, Uttarakhnad, India. The overweight and obesity was found 14.8% and 55.5%. Furthermore, the study found 93% obese study participants reported consumption of junk food. The primary reasons stated for consumption of junk food were due to taste (46.9%) and convenient (50.3%) (Rautela et al, 2018).

In 2018, a study on 13–15-year-old adolescents from schools of Aligarh, India was carried out by Failzi et al. According to this study, the prevalence of overweight or obesity was significantly higher (29.2%) among those who did not consume vegetables daily, compared to those who consumed vegetables once daily (7.8%). Similarly, more than two-fifths of the studied population did not eat fruits every day and the prevalence of overweight and obesity was noted higher among those who did not eat fruits daily (23.8%) compared to those who consumed fruits twice or more daily (14.1%). The higher frequency of soft drink intake was noted higher among overweight and obese participants who consumed soft drinks twice or more in a day (22.8%), compared to who did not consume soft drinks daily (12.4%) and who did not consume soft drinks (9.7%) (Failzi et al, 2018).

In 2018, Shete et al, conducted a study on adolescent school children (11-16 years) of Kohlapur, Maharashtra, India. According to this study 12% overweight participants has daily eating junk food habit whereas 100% obese respondents were having junk food more than once in a week. Moreover, association between frequency of eating junk food and body mass index was found highly significant (Shete et al, 2018).

In 2016, Saranya et al, in a cross-sectional study on medical students of South India found nearly one quarter of the students who consumed junk foods everyday were found to be overweight or obese. Furthermore, higher prevalence of overweight and obese subjects reported to consume carbonated drinks everyday (47.2%) and the significant association was found between consumption of carbonated drinks and overweight/obesity (Saranya et al, 2016).

A cross-sectional analytical study on adult population of Jamnagar city, Gujarat, India was carried out by Vadera et al in 2010. This study observed that dietary constitutes also affect the weight status. Mean oil consumption was significantly higher among overweight groups. The consumption of vegetables was significantly lower in overweight subjects than non-overweights. Further it was found that increased frequency of taking vegetables and fruits decreases the risk of overweight and increased intake of fried food increases the risk of overweight. The prevalence of overweight was noted higher with higher frequency of eating in restaurants and intake of fast food.

2.2.4.5 Stress

The two major components of stress are: rapid activation of the autonomic nervous system (ANS), which encompasses the sympathetic and parasympathetic nervous system (PNS), followed by activation of the hypothalamic-pituitary-adrenal (HPA) axis (Bose et al, 2009).

Stress can be caused by either external stressor or by internal stressors. Upon the action of stressor, catecholamines such as epinephrine and norepinephrine, which are associated with “fight or flight” response, produced in nervous system and in the adrenal medulla. These catcholamines are associated to increase in heart rate and stroke volume that results in vasoconstriction of blood vessels of skin and gut (Eline et al, 2018). Additionally, epinephrine

also stimulates glycogenolysis causing increase in serum glucose levels providing energy (Gunnar et al, 2007).

Catecholamines are also associated with hypothalamic-pituitary-adrenal (HPA) axis with cortisol, responds immediately and supports the action of catecholamines. Stressors provoke the release of corticotropin-releasing-hormone (CRH) from the para-ventricular nucleus (PVN) of the hypothalamus which in turn stimulates the synthesis of adrenocorticotropic hormone (ACTH) (Herman et al, 2016; Nicolaides et al, 2015). The CRH release depends on the duration, intensity and feedback of stressor. ACTH activates the production of glucocorticoids, mainly cortisol from the adrenal cortex (Eline et al, 2018). Glucocorticoids are known to increase the consumption of foods enriched in fat and sugar and causes weight gain.

It was reported that, increased amount of stress and emotions affects eating behaviors in human (Lattimore et al, 2004; O'Connor et al, 2008; Wallis et al, 2009). Supporting this, a study found that sadness favored eating of high fat/sweet which gives pleasure and satisfaction for food, whereas happy state of mind favored intake of dried fruit (Garg et al, 2007). This eating behavior may slowly lead to obesity which includes cortical and subcortical pathway. This pathway involve learning and memory of satisfaction and pleasure eating, habit formation and decreased cognitive control. As the stress hormones elevates, the intake of pleasant-tasting food increases and hence resulting in accumulation of fat which may serve as feedback signals that reduce perceived stress (Pecoraro et al, 2006). This mechanism reinforces stress-induced feeding behavior.

In 2018, Gudegowda et al performed a cross-sectional study among 424 medical students of Bangalore Medical College and Research Institute. This study reported that 74.5% overweight/obese participants often feel stress during exams and among them 56.0% eat more during exams (Gudegowda et al, 2018).

Singh et al (2018), studied an association between stress and obesity among female teachers aged 30-59 years from five colleges of Jalandhar, Punjab. The researchers found a positive and significant correlation of stress with the consumption of traditional savoury snacks, western fast food, higher frequency of eating out, emotional eating. Further, the study shown that adiposity,

eating behaviours and food choices were influenced by stress and a lesser concern about body shape among working women (Singh et al, 2018).

In 2017, Goswami et al conducted study on 138 medical students of Assam and found a strong correlation between psychological stress and body weight, demonstrating higher the psychological stress more is the body weight (Goswami et al, 2017).

2.2.4.6 Hypertension and insulin resistance:

Progression from normotensive to hypertensive among obese participants results from combination of various factors including environmental (diet content, physical activity, level of stress), physiological, and genetic. The association of hypertension and obesity was first demonstrated prospectively in the Framingham Heart Study in 1967 (Kannel et al, 1967). Even before the Framingham Heart Study, researchers explained the plausible pathogenesis of hypertension in obese patients by linking the cardiovascular and metabolic complications of upper-body obesity (Vague, 1956). Coetaneous studies reported an association between obesity and metabolic abnormalities such as insulin resistance and hypertriglyceridemia (Kissebah et al, 1982; Krotkiewski et al, 1983). Together abdominal obesity, hypertension, insulin resistance and hypertriglyceridemia are the predisposing factors for later development of metabolic syndrome and cardiorenal syndrome (Alberti et al, 1998; Alberti et al, 2009).

The pathogenesis of obesity-related hypertension includes, central obesity, sympathetic nervous system (SNS) over-activation, increase renin-angiotensin-aldosterone system (RAAS) activity and increased renal sodium absorption (Landsberg et al, 2013).

Sympathetic nervous system over-activation:

Obesity is associated with increased activity of sympathetic nervous system particularly of the heart, kidneys and skeletal muscle and with dysfunction of baroreflex that leads to alteration in blood pressure (Rumantir et al, 1999; Grassi et al, 2000; Hall et al, 2003). The causes for activation of the sympathetic nervous system in obesity remain uncertain, but the assumed mechanisms include hyperinsulinemia and/or insulin resistance; leptin or other adipokines; renin–angiotensin; lifestyle factors (Lambert et al, 2010). Insulin is now acknowledged as one

the risk factor for pathophysiology of obesity-induced hypertension because insulin stimulates the sympathetic nervous system and obese patients have increased sympathetic nervous system activity. Hence insulin-mediated sympathetic nervous system over-activation is considered as a likely factor in the pathogenesis of high BP in the setting of central obesity (Landsberg et al, 2013). Similarly, leptin is a potent appetite suppressant and it also stimulates the sympathetic nervous system (Tang-Christensen et al, 1999; Haynes et al, 1997). Generally, obese individuals have leptin deficiency, but small minority of obese individuals have elevated leptin levels (Kennedy et al, 1997; Mantzoros et al, 1999). Leptin has shown an elevation in blood pressure (Shek et al, 1998) by stimulating sympathetic nervous system (Dunbar et al, 1997).

Additionally, the elevated level of renal norepinephrine indicates, increased renal sympathetic nervous system activity among obese patients (Rumantir et al, 1999). The cardiac sympathetic nervous system activity suppress among normotensive obese individuals, whereas cardiac sympathetic nervous system activity elevates among hypertensive obese individuals (Rumantir et al, 1999). Hence, increases in both renal and cardiac sympathetic nervous system activity might be one of the mechanism that leads to the development of hypertension among obese patients. However, other studies suggest that not only sympathetic nervous system activation lead to the development of hypertension, but increased α-adrenergic-mediated vascular tone also reported in overweight individuals with hypertension (Egan et al, 1989).

Increased renin-angiotensin-aldosterone system (RAAS) activity:

The renin-angiotensin system is mainly involved in the development of hypertension through two systems including tissue and circulating. Adipose tissues are the major site for production of angiotensinogen, angiotensin (Ang) I and angiotensin II, which enters the circulation. These RAAS components once produced are taken up by the cells, in which Ang-II receptors are overexpressed. Angiotensinogen production causes adipocyte hypertrophy that leads to elevation of blood pressure through the action of Ang-II, which induces systematic vasoconstriction, direct sodium and water retention and increased aldosterone production.

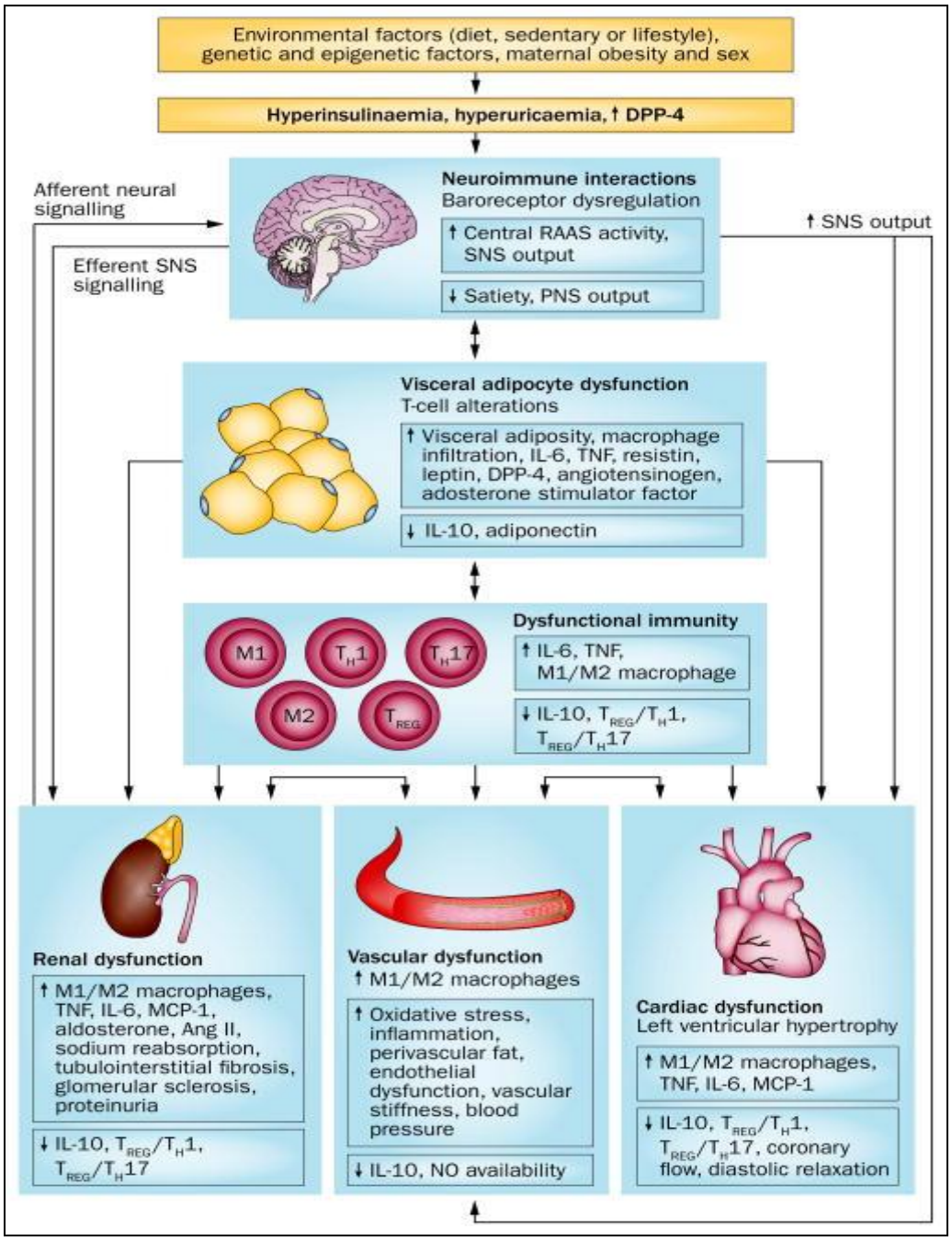

Figure 2.4 Role of obesity in development of hypertension (DeMarco et al, 2014)

Consequently, high salt-sensitive blood pressure condition is produced at high rate in obese patients which is not suppressed by volume expansion **(**Shu-Zhong et al, 2016).

Another mechanism of RAAS activation includes chronic elevation of sympathetic tone that causes renal vasoconstriction and renin-dependent chronic hypertension. Once the blood

pressure rises in obese patients, the arterial blood pressure controlling mechanism such as diuresis and natriuresis activates. However, abnormalities in these mechanisms tend to raise blood pressure, which increase excretion of sodium and water through pressure natriuresis and diuresis. This helps in expansion of extracellular fluid volume, resulting in a hypertensive adjustment of the pressure natriuresis **(**Shu-Zhong et al, 2016). Obesity also causes changes in intrarenal forces that may contribute to increases tubular reabsorption and sodium retention. This may occurs due to histological changes in the inner renal medulla, including large increase in the number of intestinal cells and extracellular matrix between the tubules, which would tend to increase renal intestinal tissue fluid hydrostatic pressure and cause compressive force on the tubules and vasa recta (Hall, 1997).

In 2018, a community based cross-sectional study conducted in rural field of Andhra Pradesh by Department of community medicine, Dr. Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation. This study reported significantly higher prevalence of hypertension (15.1%) among participants with generalized obesity (BMI $\geq$ 25 kg/m^2) compared to non-obese participants (5.9%). Additionally, 15.8% obese participants (both generalized obesity and abdominal obesity) had hypertension which was noted higher compared to non-obese participants (6.00%). However, this study did not find significant association between abdominal obesity and hypertension (Undavalli et al, 2018).

In 2017, Singh et al, in a community based cross-sectional study among 25-64 years old participants of Varanasi found the significant association between BMI and hypertension. Further, the study noted overweight and obesity (both BMI and abdominal obesity) were major predisposing factors for development of hypertension. Additionally, the risk of hypertension was more than twofold high among overweight subjects and more than threefold high among obese subjects (Singh et al, 2017).

In 2017, Prasad et al carried out a cross sectional study on 18-80 years of subjects of Tamil Nadu. This study found higher prevalence of stage I, II and pre-hypertension among obese (24%, 8.4%, and 45%) and overweight (16.12%, 12.9% and 45.16%) participants. Further, mean systolic and diastolic blood pressure were noted significantly higher among obese participants than normal BMI participants (Prasad et al, 2017).

In 2016, a cross-sectional study was carried out on 500 subjects of a rural area of Ranchi district of Jharkhand by Kumar et al. According to this study, out of 21 obese participants (BMI ≥ 27.50 kg/m^2), 12 (57.1%) had hypertension and 9 (42.9%) were non-hypertensive. Further, the prevalence of hypertension was noted higher among both male and female subjects with abdominal obesity than those without abdominal obesity. However, the association between abdominal obesity and hypertension was found significant for male subjects only (Kumar et al, 2016).

2.2.4.7 Lipid abnormalities:

The commonly seen lipid abnormalities among obese patients are elevated triglyceride, VLDL, Apo B, and non-HDL cholesterol levels, low levels of HDL cholesterol and Apo A-I, whilst levels of LDL cholesterol remains normal (Bay et al, 2013; Grundy, 2004; Franssen et al, 2011; Xiao et al, 2016).

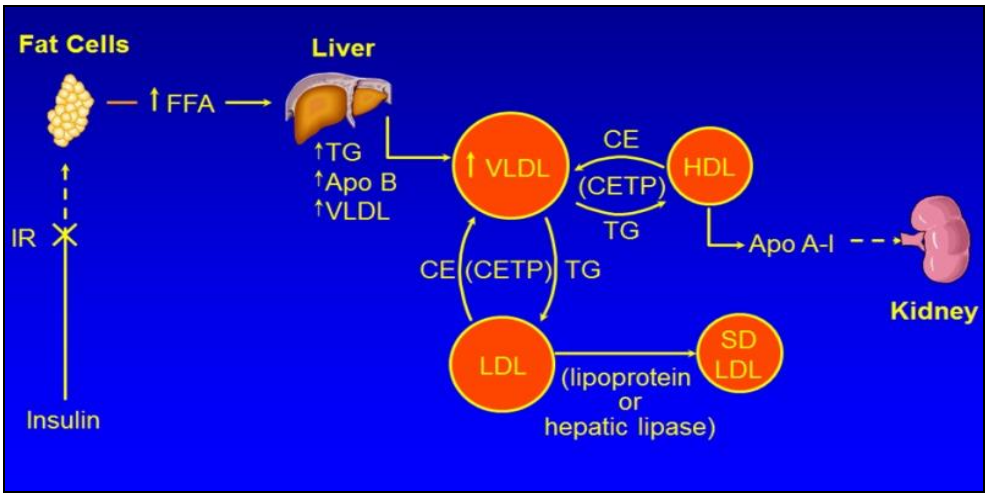

Figure 2.5 Pathophysiology of dyslipidemia of obesity **(**Feingold et al, 2018)

The different abnormalities that causes dyslipidemia in obese patients include the combination of the greater delivery of free fatty acids to the liver from increased total and visceral adiposity, insulin resistance and a pro-inflammatory state, induced by macrophages infiltrating fat tissue. Out of these abnormalities, overproduction of VLDL particles by the liver is an important contributor to the elevation in serum triglyceride levels (Bay et al, 2013; Xiao et al, 2016; Klop et al, 2013; Bjornson et al, 2017; Yu et al, 2005). Elevated levels of triglycerides prevents the

intrahepatic degradation of Apo B-100 that increases formation and secretion of VLDL. The three sources of fatty acids in liver may be altered in obese patients. First, the flux of fatty acids from adipose tissue to the liver is increased. Second, de novo fatty acid synthesis is increased that may be mediated by hyperinsulinemia in patients with insulin resistance. Third, uptake of triglyceride rich lipoproteins by the liver. The intestinal fatty acid synthesis is enhanced and accompanied by the enhanced secretion of chylomicrons in obesity. This elevated chylomicrons increases the delivery of fatty acids to the liver (Bay et al, 2013; Bjornson et al, 2017; Yu et al, 2005). These three pathways increases the hepatic fatty acids synthesis of triglycerides in the liver and protects Apo B-100 from degradation that results in the increased formation and secretion of VLDL (Xiao et al, 2016; Yu et al, 2005). Additionally, the ability of insulin to suppress Apo B secretion is diminished in patients with obesity and marked insulin resistance (Bjornson et al, 2017; Yu et al, 2005). Lastly, calorie rich food increases circulating triglycerides which results in increase in chylomicron triglyceride levels and/or providing fatty acids to the liver or dietary carbohydrate enhancing de novo hepatic lipogenesis (Feingold et al, 2018).

Along with overproduction of triglyceride rich lipoproteins by the liver and intestine, the metabolism of these triglyceride rich lipoprotein is also altered that results in increase in triglyceride levels. Expression of Apo C-III levels are inhibited by insulin (Bjornson et al, 2017). Moreover, obese subjects are insulin resistant, that accounts for higher level of Apo C-III levels in obese patients (Chan et al, 2002; Bjornson et al, 2017). Apo C-III is inhibits the activity of lipoprotein lipase and hence reduce the clearance of triglycerides rich lipoprotein. Moreover, Apo C-III also inhibits the cellular uptake of triglyceride rich lipoproteins (Feingold et al, 2018). Lastly, if insulin resistance is severe then insulin dependent lipoprotein lipase may not be stimulated properly, which may also decrease the clearance of triglyceride rich lipoproteins (Klob et al, 2013). Hence, a decrease in clearance of triglyceride rich lipoproteins results in the elevation in serum triglyceride levels in obese patients.

The elevation in triglyceride rich lipoprotein levels in turn has effects on other lipoproteins. The triglyceride rich lipoprotein and VLDL exchange triglycerides for cholesterol from LDL and HDL, which is mediated by cholesterol ester transfer protein (CETP). Increase in the levels of triglyceride rich lipoproteins also increases CETP mediated exchange that results in increase in

the triglyceride content and decrease in the cholesterol content of both LDL and HDL. In addition, obesity also increases the activity and mass of CETP (Franssen et al, 2011). Hence CETP-mediated exchange decreases the HDL cholesterol levels when triglyceride levels are high and the increase in HDL cholesterol when triglyceride levels decrease. The triglyceride on LDL and HDL undergoes hydrolysis by hepatic lipase and lipoprotein lipase and produces small dense LDL and small HDL particles (Bay et al, 2013; Grundy et al, 2004; Klop et al, 2013). Furthermore, in obese patients the hepatic lipase activity increases which clears triglyceride from LDL and HDL and produces small lipoprotein particles (Bay et al, 2013; Grundy et al, 2004; Klop et al, 2013). The Apo A-I has less affinity for small HDL particles, gets dissociated and cleared by kidneys (Bay et al, 2013). These results in low levels of Apo A-I and HDL in obese patients.

In 2018, Wankhade et al in a cross-sectional study of 300 male employees from a packaging and binding industry in Maharashtra found that 18.7%, 31.3% and 54.5% participants with high BMI had hypercholesterolemia, hypertriglyceridemia and abnormal cholesterol/HDL ratio, respectively compared to those with normal BMI. Further, the study also noted higher prevalence of hypercholesterolemia (18.6%), hypertriglyceridemia (30.8%) and abnormal cholesterol/HDL ratio (51.1%) among participants with high body fat% as compared to those with normal body fat% (Wankhade et al, 2018).

In 2015, a cross-sectional study on 400 subjects attending the medical outpatient department of a private medical college hospital at Salem was conducted by Ranganathan et al. According to this study the prevalence of hypercholesterolemia (59%), HDL-C of <30 mg/dl (42%), LDL-C of >130 mg/dL (41%), very low-density lipoprotein (VLDL)-C of >40 mg/dL (56%) and TG >150 mg/dL (55%) were noted higher among high BMI participants than normal BMI participants. In addition, the mean total cholesterol, LDL cholesterol, VLDL cholesterol and triglycerides levels were also found significantly higher among participants with higher BMI as compared to those with normal BMI in both male and female. Whereas, the levels of HDL-C did not find significantly different between higher and normal BMI groups.

In 2014, the ICMR-INDIAB study was conducted on participants of age more than 20 years in four regions of India including Tamil Nadu, Maharashtra, Jharkhand and Chandigarh. In all the four regions, subjects with any lipid abnormalities, had significantly higher BMI ($p<0.001$) and

waist circumference ($p<0.05$) (except females in Jharkhand) compared to those with no lipid abnormality (Joshi et al, 2014).

2.2.4.8 Inflammation

Obesity is associated with a chronic and low grade inflammation which is involved in the pathogenesis of several chronic diseases, such as type 2 diabetes, hypertension, atherosclerosis, fatty liver, cancer, asthma, and sleep apnea (Ye J, 2013). These chronic conditions are characterized by increase of cytokines and serum concentrations of acute-phase reactants (markers of active inflammation) such as fibrinogen, C-reactive protein (CRP), complement, serum amyloid A, haptoglobin, sialic acid and low albumin concentrations (Pickup, 2014). CRP is a classic sensitive acute-phase reactant which is very sensitive systemic marker of inflammation. Under normal condition, the levels of CRP remains in low concentration but, its concentration rapidly increases on variety of stimuli (Pearson et al, 2003; Pepys et al, 2003).

Visceral adipose tissue may produces inflammatory mediators which in turn produces acute-phase reactants in hepatocytes and endothelial cells (Jacobs et al, 2009). This explains the relationship between obesity and inflammation. In fact, because it has been shown that adipocytes express and secrete TNF-alpha, adipose body mass may be an important mediator to explain the relation between obesity and inflammation (Kern et al, 1995). Some studies have shown elevated level of hs-CRP among subjects with normal BMI but abdominal adiposity. This describes that abdominal adiposity is associated with elevation of CRP levels, independent of body mass index (BMI) (Lapice et al, 2009).

In 2017, Lavanya et al conducted a cross sectional study showing an association between overweight, obesity and serum CRP level in adults of 20-70 years. This study showed that hs-CRP values were higher in overweight (75%) and obese (93.5%) subjects as compared to those with normal BMI (1.1%).This study further found that in both female and male subjects, high waist circumferences was associated with high hs-CRP levels.

In 2016, a study on 1,432 patients of more than 35 years of age of Telangana, India, was conducted by Ramdas et al. This study reported normal levels of CRP up to class I obesity.

Among patients of the class II and III obesity, the elevated levels of CRP were noted. Only about 4.5% of the patients with normal BMI had more than 10 mg/L CRP levels whilst 42.8% of the patients under the class III obese BMI had abnormal CRP levels (more than 10 mg/L) (Ramdas et al, 2016).

In 2015, a study on 88 participants of age between 20-60 years of Karnataka reported 2.40 ± 1.00 mg/L mean level of CRP among participants with BMI < 23 kg/m^2, whereas higher level of CRP (4.04 ± 2.03 mg/L) was noted among participants with BMI > 23 kg/m^2 (Sadanand et al, 2015).

In 2014, Dayal et al has conducted study on 6 to 16 years children of Paediatric Endocrinology clinic of a tertiary care hospital in Northern India. In this study, the mean CRP levels among overweight and obese children was significantly higher (3.92 ± 2.20 mg/L) as compared to normal BMI children. In addition, this study has found a strong positive correlation between BMI and hsCRP levels in obese children but correlation between WHR and hsCRP was not significant although subjects with lower WHR had lower hsCRP levels (Dayal et al, 2014).

3. Materials and Methods

3.1 Type of Study

Observational Study

3.2 Study Design

Cross-sectional, multicentric study

3.3 Study Population

- Gender: Both
- Age group: ≥ 12 Years to 55 Years – divided into three categories:
  1. 12 to 17 years: School going children
  2. 18 to 35 years: Young aged group
  3. 36 to 55 years: Adult aged group

3.4 Study Duration

3 years and 1 month: December 2013 to December 2016

3.5 Study centres

- For 12 to 17 Years children, data were collected from six different schools;
  1. St. Anns School, Sabarmati (Private school)
  2. Sakar School, New C G Road (Private school)
  3. Gayatri and Navsarjan School, Ranip (Private school)
  4. GLS School, Law Garden (Private school)
  5. Kendriya Vidhyalaya, Vastrapur (Government school)
  6. Kendriya Vidhyalaya, Sahibaug (Government school)

- For 18 to 35 and 36 to 55 years old subjects, data was collected by conducting health camps at colleges and of six different regions of Ahmedabad.

Name of the colleges from where data was collected:

1. SAL Institute of Pharmacy, Opp. Science City, Ahmedabad
2. SAL Institute of Engineering, Opp. Science City, Ahmedabad
3. Arihant School of Pharmacy and Bio-Research Institute, Adalaj, Ahmedabad
4. Shree Swaminarayan Sanskar Pharamcy College, Zundal, Ahmedabad

Name of the six regions of Ahmedabad from where data was collected

1. Chandkheda
2. Vastrapur
3. Shahibaug
4. Science City
5. Naroda
6. Anandnagar

3.6 Study Sample Size

3.6.1 Sample size calculation:

Sample size for this study was calculated from the first study of India conducted by Anjana et al in 2011 on rural and urban population of four states; Tamil Nadu, Maharashtra, Jharkhand and Chandigarh. This study was published in Diabetologia; stating the overall prevalence of prediabetes among Tamilnadu, Maharashtra, Jharkhand and Chandigarh were 8.3%, 12.8%, 8.1% and 14.6%, respectively.

The present study has considered prevalence of prediabetes of Tamilnadu state (8.3%) to calculate sample size for current study.

The sample size was calculated by single cross-sectional survey formulae. (Mahajan BK, 1999)

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

Where,

Z = 1.96 for 95% confidence level

P= prevalence of prediabetes of Tamilnadu (8.3%)

d=allowable error (1.2%)

$$n = \frac{(1.96)^2 (0.083)(1-0.083)}{(0.012)^2}$$

$$n = 2030$$

The estimated sample size calculated from above equation is 2030. But to increase the power of the study current study included total 2412 participants of school going children, young aged and adult aged subjects from different zones in the city.

These subjects were further divided as:

- ✓ 12 to 17 years: 456
- ✓ 18 to 35 years: 1010
- ✓ 36 to 55 years: 946

3.7 Selection Criteria

3.7.1 Inclusion Criteria:

- Age Group: ≥ 12 Years to 55 Years
- Gender: Both
- Newly diagnosed case of prediabetes

- Newly diagnosed case of pre-hypertension and hypertension
- For School going children (subjects below the age of 18 years): only those were included whose legal guardian or parents signed consent form.
- For Adult subject (subjects above the age of 18 years): only those were included who had given his/her voluntary consent for participation in study.
- Only those schools and colleges were included in the study whose head has given consent to carry out the study related procedure in the school and college.
- Only those subjects were included who were able to understand, and both willing and physically able to comply with study related procedures.
- Only those participants were included who were able and willing to provide written informed consent for themselves.

3.7.2 Exclusion Criteria:

- Subject with any chronic ailments like;
  - ✓ COPD
  - ✓ Asthma
  - ✓ Cancer
  - ✓ Hyperthyroidism or hypothyroidism,
  - ✓ Hypocalcaemia,
  - ✓ Sarcoidosis,
  - ✓ Acute or chronic kidney disease, or
  - ✓ Significant chronic medical condition that would interfere with study participation
- Pregnant or Lactating women
- Subject under any drug therapy were excluded from the study

3.8 Methodology

The study was previously approved by Institutional Ethics Committee with protocol number IEC/NU/V/IP/01.

This study included total 2412 subjects of both gender in the age group of ≥12 years to 55 years, which were further divided in to three groups, i.e. school going children (12 year to 17 years), young aged (18 years to 35 years) and adult aged (36 years to 55 years) from different zones in the city to get an equal distribution of subjects by socioeconomic state, ethnic variability and gender.

Before carrying out any study related activity, complete study procedure was explained to the subject in the language which he/she understood by principal investigator and where applicable legally acceptable representative (LAR) and/or impartial witness present when principal investigator was explaining study to the subject. The subjects were ensured for their strict confidentiality. For school going children in the age group of 12–17 years, after selecting and finalizing the tools for data collection, the principal investigator visited the schools for taking prior permission from the headmasters/ headmistress of the schools for collecting the anthropometric measurements and other investigation. Subsequently, the investigator discussed in detail about investigation with heads of the respective schools sought the permission from them for collecting the necessary data and the subjects (students) were explained about the nature and purpose of the study (Annexure-III). For young aged and adult aged subjects informed consent form was obtained from subject for anthropometric measurements and other investigation whereas for school children informed consent was taken from their parents or legally acceptable representative (Annexure-II). Once voluntarily consent from subjects was obtained, all study related procedure was started. A complete medical history and physical examination was conducted by skilled persons using established guidelines.

Anthropometric data like height and weight were measured with stadiometer and a digital weighing scale respectively. Then, body weight (kg) was divided by height (m^2) to obtain body mass index. The information collection proforma contained details about the participation in exercise, indoor games, outdoor games, playing on laptop or mobile and watching television with either yes or no answer. Dietary type was classified as vegetarian (vegetables only), non-vegetarian (Vegetables, eggs and non-vegetables) and eggetarian

(vegetables and eggs) diet. Frequencies of junk food and sweet eating habit were categorized as everyday, once in a week, once in fifteen days and once in a month. Stress level was determined by asking questions with five options; never, rarely, sometimes, often and very often. Each options caries different score. The score of all questions was calculated and level of stress was categorized as no stress at all, less level of stress, medium level of stress and high level of stress. Socioeconomic class was defined using education, occupation and monthly family income score described by Gurura et al. (Gururaj et al, 2014). Clinical examination of the subjects was carried out by taking their BP measurements. BP was recorded in sitting position in right arm by auscultatory method using a standard mercury sphygmomanometer with the subject seated and the arm extended over the table at the level of heart.

Standardized protocol was used for all interviews and examinations. The questionnaire was assessed for life style, physical activity and social factors that influence physical and psychosocial health of subject. Socioeconomic status (upper, upper middle, lower middle, upper lower & lower), participation in sports, physical exercise (participation in exercise, participation in games), dietary form (vegetarian, non-vegetarian or eggetarian food), frequencies of having junk food and sweet eating habit (everyday, once in a week, once in fifteen days, once in a month), family history of diabetes, hypertension, thyroid and obesity and stress level of subject (no Stress, less level of stress, medium level of stress, high level of stress) also was assessed (Annexure-I).

Subjects prior to study were informed to come for lab investigation on empty stomach (not to eat anything after 10 pm).Blood samples of overnight fasted subjects were collected by trained lab technician to analyze;

- ✓ Fasting blood sugar (FBS),
- ✓ Lipid profile,
- ✓ Insulin,
- ✓ Vitamin D and
- ✓ C-reactive protein

3.8.1 Procedure for height measurement:

- Subject was instructed to remove shoes, heavy outer clothing, and barrettes.
- He/she stands with the back as straight as possible. Weight should be evenly distributed on both feet.
- Position the subject with heels close together, legs straight, arms at sides, and shoulders relaxed. Buttocks and shoulders should touch the wall.
- He/she was instructed to look straight ahead with head erect.
- Then by using measuring tap height of the subject was measured in centimeter unit.

3.8.2 Procedure for weight measurement:

- Zero the scale before the subject steps on the scale.
- Subject was instructed to remove shoes and bulky clothing (no jackets).
- Subject was instructed empty out pockets of any objects (keys, change, and wallet).
- Subject was instructed to stand with both feet on the center of the scale, and not touch other objects or persons.
- Then weight was recorded in kilogram unit.
- At the end of measuring and recording the weight, return the scale to the "zero" position.

3.8.3. Body Mass Index (BMI) calculation:

3.8.3.1 Body mass index calculation on subjects above the age of 18 years

BMI of all subjects above the age of 18 years was calculated by following formula;

$$\frac{\text{Weight (in Kg)}}{\text{Height (in m}^2\text{)}}$$

In this study we have used BMI classification for Asian population of age above 18 years recommended by World Health Organization (WHO, 1999). Overweight and obesity were defined by WHO as BMI 25 to 29.9 and BMI greater than or equal to 30, respectively.

Table 3.1 BMI classification for subjects above the age of 18 years recommended by World Health Organization

Sr. No	BMI (kg/m^2)	BMI Classification
1	< 18.5	Underweight
2	18.5 to 24.9	Normal
3	25 to 29.9	Overweight
4	≥ 30.0	Obese

3.8.3.2 Body mass index calculation for subjects of age 2 to 17 years

BMI-for-age reference curve was used to assess weight in relation to stature for children between the age of 2 to 18 years as recommended by Centres for Disease Control and Prevention (CDC) (Ogden CL, 2002). Once the BMI is calculated for a child below the age of 18 years, then the subject was classified as follows.

Table 3.2 BMI-for-age reference curve recommended by Centres for Disease Control and Prevention

Percentile Range	BMI Classification
< 5^{th} percentile	Underweight
≥ 5^{th} percentile to < 85^{th} percentile	Normal
≥ 85^{th}percentile and < 95^{th} percentile	Overweight
≥ 95^{th} percentile	Obese

Overweight and obesity were defined as BMI-for-age ≥ 85 and ≥ 95 percentiles respectively.

3.8.4 Procedure for measurement of blood pressure:

- Blood pressure was taken from the arm (brachial artery) from all respondents in the first encounter by using calibrated digital sphygmomanometer.
- Blood pressure measurement was done in a sitting position with the arm supported and repeated after 5 minutes; the average of the two measurements was taken as a blood pressure.
- The systolic blood pressure of 120–139 mm Hg and/or diastolic blood pressure of 80–89 mm Hg was provided the diagnosis of pre-hypertension
- The systolic pressure of above or equal to 140 mmHg and diastolic pressure above or equal to 90 mmHg was regarded as a high blood pressure.
- Those who found to have high blood pressure were referred to a physician for further evaluation and possible treatment.

3.8.4.1 Classification of Blood Pressure

The Seventh Report of the Joint National Committee on Prevention, Detection and Treatment of High Blood Pressure (JNC 7) criteria used for classification of Blood Pressure (Chobanian et al, 2003).

Table 3.3 Classification of hypertension as per JNC 7 criteria

Systolic Blood Pressure (mmHg)	Diastolic Blood Pressure (mmHg)	Classification
< 120	< 80	Normal
120-139	Or 80-89	Prehypertension
140-159	Or 90-99	Stage 1 Hypertension

≥160	Or ≥ 100	Stage 2 Hypertension

3.8.5 Biochemical Tests

After an overnight fasting, blood samples for lipid profiles, vitamin D levels, C-reactive protein, insulin and blood glucose was collected. Five millilitres of venous blood was taken from the antecubital fossa and placed in empty sterile tubes.

3.8.5.1 Measurement of fasting blood glucose level:

Test Principle: Enzymatic reference method with hexokinase

- ✓ Hexokinase (HK) catalyzes the phosphorylation of glucose by ATP to form glucose-6-phosphate and ADP.

$$\text{Glucose + ATP} \xrightarrow{\text{Hexokinase}} \text{G-6-P + ADP}$$

- ✓ Glucose-6-phosphate dehydrogenase oxidizes glucose-6-phosphate in the presence of NADP to gluconate-6-phosphate. No other carbohydrate is oxidized. The rate of NADPH formation during the reaction is directly proportional to the glucose concentration and is measured photometrically.

$$\text{G-6-P + NADP}^{+} \xrightarrow{\text{G-6-PDH}} \text{Gluconate-6-P + NADPH +H}^{+}$$

Name of instrument: Roche Cobas c111 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ COBAS c 111 Glucose HK Liquid – 100 tests
- ✓ R1 - TRIS buffer: 100 mmol/L, pH 7.8; Mg^{2+}: 4 mmol/L; ATP: >1.7 mmol/L; NADP: >1.0 mol/L; preservative.

- ✓ SR - HEPES buffer: 30 mmol/L, pH 7.0; Mg^{2+}: 4 mmol/L; HD (yeast): >130 ukat/L; G-6-PDH (E. coli): >250 ukat/L; preservative.

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum
- ✓ Plasma: Li-heparin, K_3-EDTA, NaF/Na_2-EDTA, NaF/Citrate/Na_2-EDTA, KF/Na_2-EDTA or NaF/K-oxalate plasma

The stability of glucose in specimens is affected by storage temperature, bacterial contamination, and glycolysis. Plasma or serum samples should be separated from the cells or clot within an hour of being drawn. Specimens that cannot be separated from the cells within one hour should be placed on ice or refrigerated.

NOTE: Tests cannot be added on to specimens that were iced.

When blood is drawn and stands uncentrifuged at room temperature, the average decrease in serum glucose is ~ 7% in 1 hour. This decrease is the result of glycolysis. (Even NaF does not prevent glycolysis within the first few hours when left at room temperature.) The rate of in vitro glycolysis is higher in the presence of leukocytosis or in patients with increased hematocrits.

Stability: (If separated from cells): 8 hours at 20-25oC, 24 hours at 2-8oC, 72 hours at 2-8^{o}C.

Assay procedure:

Table 3.4 Assay of fasting blood glucose estimation

Application for serum and plasma	
Measuring mode:	Absorbance
Absorbance calculation mode:	Endpoint

Wavelength A/B:	340/409 nm	
Calculation first/last (Serum, plasma):	16/37	
Unit:	mmol/L	
Pipetting parameters		
R1	150 µL	
Sample	2 µL	20 µL (H_2O)
SR	30 µL	
Total volume	202 µL	

Calculation:

The COBAS C 111 analyzer automatically calculates the analyte concentration of each samples.

Conversion factors:

mmol/L X 18.02 = mg/dL

mmol/L X 0.1802 = g/L

mg/dL X 0.0555 = mmol/L

Measuring range:

Serum, plasma and urine: 0.11 – 40 mmol/L (1.98 – 720 mg/dL)

Determine samples having higher concentrations via the rerun function. Dilution of samples via the rerun function is a 1:10 dilution. Results from samples diluted using the rerun function are automatically multiplied by a factor 10.

Lower limits of measurement:

Lower detection limit of the test: 0.11 mmol/L (1.98 mg/dL)

Expected values:

For serum glucose levels, we referred to American Diabetes Association guidelines (2006).

Table 3.5 Classification of prediabetes and diabetes according to American Diabetes Association guideline

Serum Glucose Level	Classification
< 100 mg/dL	Normal
100-125 mg/dL	Prediabetes
>126 mg/dL	Diabetes

Subjects with fasting blood glucose more than 100 mg/dL and less than 126 mg/dL were considered as having prediabetes whereas fasting blood sugar level more than 126 mg/dL was referred as diabetes in current study.

3.8.5.2 Measurement of lipid Profile:

3.8.5.2.1 Measurement of total cholesterol level:

Test Principle: Enzymatic colorimetric method

Cholesterol esters are cleaved by the action of cholesterol esterase to yield free cholesterol and fatty acids. Cholesterol oxidase then catalyzes the oxidation of cholesterol to cholest-4-en-3-one and hydrogen peroxide. In the presence of peroxidase, the hydrogen peroxide formed effects the oxidative coupling of phenol and 4-aminoantipyrine to form a red quinone-imine dye.

$$\text{Cholesterol esterase} + H_2O \xrightarrow{CE} \text{Cholesterol} + \text{RCOOH}$$

$$\text{Cholesterol} + O_2 \xrightarrow{CHOD} \text{Cholest-4-en-3-one} + H_2O_2$$

$$2H_2O_2 + \text{4-AAP} + \text{Phenol} \xrightarrow{\text{POD}} \text{Quinone-imine dye} + 4\ H_2O$$

The color intensity of the dye formed is directly proportional to the cholesterol concentration. It is determined by measuring the increase in absorbance.

Name of instrument: Roche Cobas c111 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ R1 - PIPES buffer: 225 mmol/L, pH 6.8; Mg^{2+}: 10 mmol/L; sodium cholate: 0.6 mmol/L; 4-aminoantipyrine: ≥ 0.45 mmol/L; phenol: ≥ 12.6 mmol/L; fatty alcohol polyglycol ether: 3%; CE (pseudomonas spec.): ≥ 25µkat/L (≥ 1.5 U/mL); CHOD (E.coli): ≥ 7.5 µkat/L (≥ 0.45 U/mL); POD (horseradish): ≥ 12.5 µkat/L (≥ 0.75 U/mL); stabilizers; preservatives.

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum
- ✓ Plasma: Li-heparin, K_3-EDTA plasma.

NOTE: The use of EDTA plasma leads to slightly lower values. Do not use citrate, oxalate or fluoride.

Fasting or non-fasting samples can be uses.

Centrifuge samples containing precipitates before performing the assay.

Assay procedure:

Table: 3.6 Assay of cholesterol estimation

Application for serum, plasma and urine		
Measuring mode:	Absorbance	
Absorbance calculation mode:	Endpoint	
Wavelength A/B:	512/659 nm	
Calculation first/last:	6/37	
Unit:	mmol/L	
Pipetting parameters		
R	47 µL	70 µL (H_2O)
Sample	2 µL	23 µL (H_2O)
Total volume	142 µL	

Calculation:

The COBAS C 111 analyzer automatically calculates the analyte concentration of each samples.

Conversion factors:

mmol/L X 38.66 = mg/dL

mmol/L X 0.3886= g/L

mg/dL X 0.0259 = mmol/L

Measuring range:

0.25 – 20.7 mmol/L (9.7 – 800 mg/dL)

Determine samples having higher concentrations via the rerun function. Dilution of samples via the rerun function is a 1:10 dilution. Results from samples diluted using the rerun function are automatically multiplied by a factor 10.

Lower limits of measurement:

Lower detection limit of the test: 0.25 mmol/L (1.98 mg/dL)

Expected values:

For serum cholesterol, we referred to NCEP - ATP III (National Cholesterol Education Program, 2002) guidelines. Hypercholesterolemia is defined as total cholesterol level more than 200 mg/dL in current study.

Table 3.7 Classification of hypercholesterolemia

Total Cholesterol	Classification
< 200 mg/dL	Desirable
200-239 mg/dL	Borderline High
≥ 240 mg/dL	High

3.8.5.2.2 Measurement of triglycerides level:

Test Principle: Enzymatic colorimetric method

$$\text{Triglycerides} + 3\ H_2O \xrightarrow{\text{LPL}} \text{Glycerol} + 3\ \text{RCOOH}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow[Mg^{2+}]{\text{GK}} \text{Glycerol-3-phosphate} + \text{ADP}$$

$$\text{Glycerol-3-phosphate} + O_2 \xrightarrow{\text{GPO}} \text{Dihydroxyacetone phosphate} + H_2O_2$$

$$H_2O_2 + \text{4-aminophenazone} + \text{4-cholorophenol} \xrightarrow{\text{Peroxidases}} \text{4-(p-benzoquinone-monoamino)-phenazone} + 2\ H_2O + HCl$$

Triglycerides are esters of the trihydric alcohol glycerol with 3 long-chain fatty acids. They are partly synthesized in the liver and partly ingested in food.

The enzymatic triglycerides assay as described by Eggstein and Kreutz still required saponification with potassium hydroxide. Numerous attempts were subsequently made to replace alkaline saponification by enzymatic hydrolysis with lipase. Bucolo and David tested a lipase/protease mixture; Wahlefeld used an esterase from the liver in combination with a particularly effective lipase from Rhizopus arrhizus for hydrolysis.

This method is based on the work by Wahlefeld using a lipoprotein lipase from microorganisms for the rapid and complete hydrolysis of triglycerides to glycerol followed by oxidation to dihydroxyacetone phosphate and hydrogen peroxide. The hydrogen peroxide produced then reacts with 4-aminophenazone and 4-cholorophenol under the catalytic action of peroxidase to form a red dyestuff (Trinder endpoint reaction). The color intensity of the red dyestuff formed is directly proportional to the triglyceride concentration and can be measured photometrically.

Name of instrument: Roche Cobas c111 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ R1 - PIPES buffer: 50 mmol/L, pH 6.8; Mg^{2+}: 40 mmol/L; sodium cholate: 0.20 mmol/L; ATP: ≥ 1.4 mmol/L; 4-aminophenazone: ≥ 0.13 mmol/L; 4-cholorophenol: 4.7 mmol/L; LPL (pseudomonas spec.): ≥ 83 µkat/L; GK (Bacillus stearothermophilus): ≥ 3 µkat/L; GPO (E.coli): ≥ 41 µkat/L; POD (horseradish): ≥ 1.6 µkat/L; stabilizers; preservatives.

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum
- ✓ Plasma: Li-heparin, K_3-EDTA plasma.

NOTE: EDTA tubes that are less than ½ full may cause a negative bias for triglycerides results.

Patients should refrain from eating for 10 to 14 hours before blood is drawn. Samples must be drawn in a soap and glycerol free collection device.

Centrifuge samples containing precipitates before performing the assay.

Assay:

Table 3.8 Assay of triglyceride estimation

Application for serum, plasma and urine		
Measuring mode:	Absorbance	
Absorbance calculation mode:	Endpoint	
Wavelength A/B:	512/659 nm	
Calculation first/last:	6/21	
Unit:	mmol/L	
Pipetting parameters		
R	12 µL	
Sample	2 µL	28 µL (H_2O)
Total volume	150 µL	

Calculation:

The COBAS C 111 analyzer automatically calculates the analyte concentration of each samples.

Conversion factors:

mmol/L X 88.5 = mg/dL

mg/dL X 0.0113 = mmol/L

Measuring range:

0.1 – 10 mmol/L (8.85 – 885 mg/dL)

Determine samples having higher concentrations via the rerun function. Dilution of samples via the rerun function is a 1:10 dilution. Results from samples diluted using the rerun function are automatically multiplied by a factor 10.

Lower limits of measurement:

Lower detection limit of the test: 0.1 mmol/L (8.85 mg/dL)

Expected values:

For serum triglyceride, we referred to NCEP - ATP III (National Cholesterol Education Program, 2002) guidelines. Hypertriglyceridemia is defined as total triglyceride level more than 150 mg/dL in current study.

Table 3.9 Classification of hypertriglyceridemia

Triglycerides	**Classification**
< 150 mg/dL	Normal
150-199 mg/dL	Borderline High
200-499 mg/dL	High
≥500 mg/dL	Very High

3.8.5.2.3 Measurement of HDL-C level:

Test Principle: Homogenous enzymatic colorimetric method

Non-HDL lipoproteins such as LDL, VLDL and chylomicrons are combined with polyanions and a detergent forming a water-soluble complex. In this complex the enzymatic reaction of CHER and CHOD towards non-HDL lipoproteins is blocked.

Finally only HDL particles can react with CHER and CHOD. The concentration of HDL-cholesterol is determined enzymatically by CHER and CHOD.

Cholesterol esters are broken down quantitatively into free cholesterol and fatty acids by CHER.

$$\text{HDL-cholesterol} + H_2O \xrightarrow{\text{CHER}} \text{HDL-cholesterol} + \text{RCOOH}$$

In the presence of oxygen, cholesterol is oxidized by cholesterol oxidase to Δ^4-cholestenone and hydrogen peroxide.

$$\text{HDL-cholesterol} + O_2 \xrightarrow[Mg^{2+}]{\text{CHOD}} \Delta^4\text{-cholestenone} + H_2O_2$$

In the presence of peroxidase, the hydrogen peroxide generated reacts with 4-amino-antipyrine and EMSE to form a dye. The color intensity of this dye is directly proportional to the cholesterol concentration and is measured photometrically.

$$2H_2O_2 + \text{4-amino-antipyrine} + \text{EMSE} + H^+ + H_2O \xrightarrow{\text{Peroxidase}} \text{Colored pigment} + 5\ H_2O$$

Name of instrument: Roche Cobas c111 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ R1 - TAPSO buffer: 62.1 mmol/L, pH 7.77; polyanions: 1.25g/L; EMSE: 1.08 mmol/L; ascorbate oxidase (cucurbita): ≥ 50 ukat/L; peroxidase (horseradish): >166.7 ukat/L; detergent; BSA: 2.0 g/L: preservative. R2 –
- ✓ SR - Bis-Tris buffer: 20.1 mmol/L, pH 6.70; cholesterol esterase (microorganism): ≥ 7.5 ukat/L; cholesterol oxidase (recombinant E. coli): ≥ 7.17 ukat/L; cholesterol

oxidase (microorganism): ≥ 76.7 ukat/L; peroxidase (horseradish): >333 ukat/L; 4-aminoantipyrine: 1.48 mmol/L; BSA: 3.0 g/L; detergents; preservatives.

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum
- ✓ Plasma: Li-heparin, K_2 and K_3-EDTA plasma.

NOTE: EDTA tubes that are less than ½ full may cause a negative bias for triglycerides results.

Fasting and non-fasting samples can be used. Specimens should preferably by analyzed on the day of collection.

Centrifuge samples containing precipitates before performing the assay.

Assay procedure:

Table 3.10 Assay of HDL-C estimation

Application for serum, plasma and urine		
Measuring mode:	Absorbance	
Absorbance calculation mode:	Endpoint	
Wavelength A/B:	583/659 nm	
Calculation first/last:	16/37	
Unit:	mmol/L	
Pipetting parameters		
R	120 µL	

Sample	2.5 µL	7 µL (H_2O)
SR	40 µL	
Total volume	169.5 µL	

Calculation:

The COBAS C 111 analyzer automatically calculates the analyte concentration of each samples.

Conversion factors:

mmol/L X 38.66 = mg/dL

mg/dL X 0.0259 = mmol/L

Measuring range:

0.08 – 3.88 mmol/L (3.09 – 150 mg/dL)

Determine samples having higher concentrations via the rerun function. Dilution of samples via the rerun function is a 1:10 dilution. Results from samples diluted using the rerun function are automatically multiplied by a factor 10.

Lower limits of measurement:

Lower detection limit of the test: 0.08 mmol/L (3.09 mg/dL)

Expected values:

For serum HDL-cholesterol, we referred to NCEP - ATP III (National Cholesterol Education Program, 2002) guidelines. HDL-C less than 40 mg/dL in men and less than 50mg/dL in women was considered as abnormal in current study.

Table 3.11 Classification of low level of HDL-C in men and women

Classification	HDL Cholesterol (For men)	HDL Cholesterol (For women)
Low	< 40 mg/dL	< 50 mg/dL
High	≥60 mg/dL	≥60 mg/dL

3.8.5.2.4 Definition of dyslipidemia

According to NCEP - ATP III (National Cholesterol Education Program, 2002) guidelines, dyslipidemia is defined by presence of hypercholesterolemia and low HDL-C levels. The current study has referred NCEP – ATP III guideline for classification of dyslipidemia.

3.8.5.3 Measurement of Vitamin D Levels:

Test principle: Enzyme Linked Fluorescent Assay (ELFA)

The assay principle combines an enzyme immunoassay competition method with a final fluorescent detection (ELFA).

The Solid Phase Receptacle (SPR) serves as the solid phase as well as the pipetting device for the assay.

Reagents for assay are ready to use and pre-dispensed in the sealed reagent strips. All the assay steps are performed automatically by the instrument. The reaction medium is cycled in and out of the SPR several times. The sample is mixed with pre-treatment reagent to separate vitamin D from its binding protein.

The pre-treated sample is then collected and transferred into the well that contains an alkaline phosphatase (ALP) labelled anti-vitamin D antibody (conjugate). During the final detection step, the substrate (4-methyl-umbelliferyl phosphate) is cycled in and out of the SPR. The conjugate enzyme catalyzes the hydrolysis of this substrate into a fluorescent product (4-methyl-umbelliferone), the fluorescence of which is measured at 450 nm. The intensity of the fluorescence is inversely proportional to the concentration of vitamin D antigen present

in the sample. At the end of the assay, results are automatically calculated by the instrument in relation to the calibration curve stored in memory, and then printed out.

Name of instrument: BioMérieux's VIDAS 25 OH Vitamin D Total (VITD)

Reagents:

- ✓ VITD strips: Stabilizer of human origin – consists of 10 wells covered with a labeled, foil seal.

Description of VITD strips

Wells	Reagent
1	Sample well
2	Conjugate: TRIS, NaCl + anti-vitamin D antibody conjugated with alkaline phosphate + stabilizer of human origin + preservative
3	Pre-treatment solution: TRIS, NaCl + dissociation agent + surfactant + methanol
4-5-6	Empty well
7-8-9	Wash buffer: TRIS, NaCl + preservative + surfactant
10	Reading cuvette with substrate: 4-methyl-umbelliferyl phosphate (0.6 mmol/L) + diethanolamine (DEA) (0.62 mol/L or 6.6%, pH 9.2) + 1 g/L sodium azide

- ✓ VITD SPRs: Interior of SPR coated with vitamin D
- ✓ C1: 25-(OH) vitamin D diluted in human serum + preservative (Control)
- ✓ S1: 25-(OH) vitamin D diluted in human serum + preservative (Standard)

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum
- ✓ Plasma: Li-heparin

NOTE: Do not use EDTA tubes.

Results and interpretation:

Once the assay is completed, results are analyzed automatically by the computer. Fluorescence is measured twice in the Reagent strip's reading cuvette for each sample tested.

The first reading is a background reading of the substrate cuvette before the SPR is introduced into the substrate. The second reading is taken after incubating the substrate with the enzyme remaining on the interior of the SPR. The RVF (Relative Fluorescence Value) is calculated by subtracting the background reading from the final result. This calculation appears on the result sheet.

The results are automatically calculated using calibration curves which are stored by the instrument (4-parameter logistics model) and are expressed in ng/mL or nmol/L. Assay results should be used in conjugation with other clinical or laboratory data to assist the clinician in making individual patient management decisions.

Measurement range:

The VIDAS 25 OH Vitamin D Total measurement range extends from 8.1 ng/mL up to 126.0 ng/mL. Values below the lower limit of the measurement range are reported as < 8.1 ng/dL. Values above the upper limit of the measurement range are reported as > 126.0 ng/dL.

Expected values:

As recommended by US Endocrine Society Guideline, vitamin D status graded as deficiency, insufficiency & sufficiency (Holick MF, 2011). Subjects with vitamin D level less than 20 ng/dL were considered as vitamin D deficient in this study.

Table 3.12 Classification of vitamin D deficiency

Vitamin D Level	Classification
< 20 ng/dL	Deficiency
20-30 ng/dL	Insufficiency
> 30 ng/dL	Sufficiency

3.8.5.4 Measurement insulin level:

Test Principle: Sandwich principle. Total duration of assay: 18 minutes.

1st incubation: Insulin from 20 µL sample, a biotinylated monoclonal insulin‑specific antibody, and a monoclonal insulin‑specific antibody labelled with a ruthenium complex) form a sandwich complex.

2nd incubation: After addition of streptavidin-coated microparticles, the complex becomes bound to the solid phase via interaction of biotin and streptavidin.

The reaction mixture is aspirated into the measuring cell where the microparticles are magnetically captured onto the surface of the electrode. Unbound substances are then removed with ProCell/ProCell M. Application of a voltage to the electrode then induces chemiluminescent emission which is measured by a photomultiplier.

Results are determined via a calibration curve which is instrument specifically generated by 2‑point calibration and a master curve provided via the reagent barcode or e‑barcode.

Name of instrument: Roche Cobas e 411 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ M - Streptavidin-coated microparticles (transparent cap), 1 bottle, 6.5 mL: Streptavidin-coated microparticles 0.72 mg/mL; preservative.

- ✓ R1 -Anti-insulin-Ab~biotin (gray cap), 1 bottle, 10 mL: Biotinylated monoclonal anti-insulin antibody (mouse) 1 mg/L; MESb) buffer 50 mmol/L, pH 6.0; preservative.
- ✓ R2 -Anti-insulin-Ab~Ru(bpy) (black cap), 1 bottle, 10 mL: Monoclonal anti-insulin antibody (mouse) labeled with ruthenium complex 1.75 mg/L; MES (2-morpholino-ethane sulfonic acid) buffer 50 mmol/L, pH 6.0; preservative.

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum collected using standard sampling tubes or tubes containing separating gel.
- ✓ Li-heparin, K3-EDTA and sodium citrate plasma.

NOTE: Hemolysis interferes, as insulin-degrading peptidases are released from erythrocytes. Centrifuge samples containing precipitates before performing the assay.

Calculation:

The analyzer automatically calculates the analyte concentration of each sample (either in µU/mL or pmol/L).

Conversion factors:

µU/mL x 6.945 = pmol/L

pmol/L x 0.144 = µU/m

Measuring range:

0.2-1000 µU/mL or 1.39-6945 pmol/L (defined by the lower detection limit and the maximum of the master curve). Values below the lower detection limit are reported as < 0.2 µU/mL (< 1.39 pmol/L). Values above the measuring range are reported as > 1000 µU/mL (> 6945 pmol/L).

Lower limits of measurement:

Lower detection limit of the test Lower detection limit: 0.2 µU/mL (1.39 pmol/L)

Expected value:

We referred "Williams Textbook of Endocrinology, 12th edition by Melmed S et al published in 2011" for hyperinsulinemia. A fasting insulin level < 25 mIU/L (< 174 pmol/L) is considered normal in current study.

3.8.5.5 Measurement of C-reactive protein

Test Principle: Particle enhanced turbidimetric method

Human CRP agglutinates with latex particles coated with monoclonal anti-CRP antibodies. The precipitate is determined turbidimetrically.

Name of instrument: Roche Cobas c111 Analyser (Roche Diagnostics International Ltd, Rotkreuz, Switzerland)

Reagents:

- ✓ R1 – TRIS buffer with bovine serum albumin and immunoglobulins (mouse); preservative
- ✓ SR – Latex particles coated with anti-CRP (mouse) in glycine buffer, preservative

Specimen collection and preparation:

For specimen collection and preparation only use suitable tubes or collection containers. Only the specimens listed below were tested and found acceptable:

- ✓ Serum: Separate immediately from clot and analyse promptly.
- ✓ Plasma: Li-heparin, K_3-EDTA plasma.

Assay:

Table 3.13 Assay of C-reactive protein estimation

Application for serum, plasma and urine	
Measuring mode:	Absorbance

Absorbance calculation mode:	Kinetic	
Wavelength A/B:	552 nm	
Calculation first/last:	17/25	
Antigen excess check	No	
Unit:	mg/L	
Pipetting parameters		
R1	82 µL	48 µL (H_2O)
Sample	2.5 µL	30 µL (H_2O)
SR	28 µL	14 µL (H_2O)
Total volume	204.5 µL	

Calculation:

The COBAS C 111 analyzer automatically calculates the analyte concentration of each samples.

Conversion factors:

mg/L X 9.52 = nmol/L

mg/dL X 95.2 = nmol/L

mg/L X 0.1 = mg/dL

mg/dL X 10 = mg/L

mg/dL X 0.01 = g/L

g/L X 100 = mg/dL

Measuring range:

1 - 200 mg/L (0.1 – 20 mg/dL)

Determine samples having higher concentrations via the rerun function. Dilution of samples via the rerun function is a 1:10 dilution. Results from samples diluted using the rerun function are automatically multiplied by a factor 10.

Lower limits of measurement:

Lower detection limit of the test: 1.0 mg/L (0.1 mg/dL)

Expected values:

According to American Heart Association, the following guidelines are recommended for the assessment of cardiovascular risk in regards to hs-CRP levels. Subjects with CRP more than 3 mg/L were considered as having elevated CRP in current study.

Table 3.14 Classification of cardiovascular risk based on Hs-CRP level

Hs-CRP Level	**Classification**
< 1 mg/L	Low risk of cardiovascular disease
1 – 3 mg/L	Medium risk of cardiovascular disease
> 3 mg/L	High risk of cardiovascular disease

3.9 Data coding

After collection of all data from subjects, data were entered in M.S Excel 2010. After entering all data, incomplete, incorrect and inaccurate data were identified by data validation tool and these data were replaced or modified with correct data. Once the data was cleaned, data coding has been done for all variables. After completion of data coding, data were transferred to SPSS 20 for analysis and interpretation of the data.

3.10 Statistical Analysis

In this study, we have calculated frequencies and percentage for qualitative data. The Statistical Package for the Social Sciences (SPSS 20) is the software used to run tests of agreement and concordance of screening methods for prediabetes. The analysis is stratified by age group to observe differences in distributions of prediabetes and body mass index status by screening method. The results of biochemical parameters, body mass index and blood pressure were expressed as mean ± SEM. Comparison of categorical data between

groups was done by Pearson's Chi-square test. Whilst comparison of mean values between groups were tested by one-way analysis of variance (ANOVA). Significance for each analysis is set at a p-value of 0.05.

4. Result

4.1. Distribution of subjects according to age group

The current study have enrolled total 2412 participants from different regions of Ahmedabad city and out of them 456 (18.91%) participants were school going children aging between 12 years to 17 years, 1,010 (41.87%) participants were of 18 to 35 years age group and 946 (39.22%) participants were of 36 to 55 years age group. (Table 4.1)

Table 4.1 Distribution of subjects in three age groups

Age Group	N
12 Y - 17 Years	456
18 Y - 35 Years	1,010
36 Y - 55 Years	946
Total	**2412**

4.2 Prevalence of prediabetes and diabetes

4.2.1 Prevalence of prediabetes and diabetes among subjects of 12-17 years, 18-35 years and 36-55 years age group

Prevalence of prediabetes in school going children was 5.09% followed by 28.81% and 33.19% in 18-35 year and 36-55 years age group, respectively. Diabetes was found 11.78% among 18-35 years age group participants and 17.44% among 36-55 years age group participant. None of the school going participants reported to have diabetes. (Table 4.2)

Table 4.2 Prevalence of prediabetes and diabetes among subjects of 12-17 years, 18-35 years and 36-55 years age group

Blood Parameter	Age Group		
	12-17 Years (N = 456)	**18-35 Years (N = 1,010)**	**36-55 Years (N = 946)**
Prediabetes (FBS >100 mg/dL)	23 (5.09%)	291 (28.81%)	314 (33.19 %)
Diabetes (FBS >125 mg/dL)	0 (0.00%)	119 (11.78 %)	165 (17.44%)

4.2.2 Mean fasting blood sugar level among subjects of 12-17 years, 18-35 years and 36-55 years age group

In current study we noted mean fasting blood sugar among 12-17 years old participants was 83.4±1.063 mg/dL, among 18-35 years old it was found 94.7±0.831 mg/dL, whilst the highest mean level of fasting blood sugar (108.3±1.011 mg/dL) among all three age group was found in 36-55 years old participants. Further, it was found that mean fasting blood sugar level was increasing with age, the strong association was found between mean fasting blood sugar level with age demonstrating risk of prediabetes and diabetes increases with age. (Table 4.3)

Table 4.3 Mean fasting blood sugar level among subjects of 12-17 years, 18-35 years and 36-55 years age group

Parameter	Age Group			P value
	12-17 Years (N = 456)	**18-35 Years (N = 1,010)**	**36-55 Years (N = 946)**	
Mean fasting blood sugar ± SEM (mg/dL)	83.4 ± 1.063	94.7 ± 0.831	107.3 ± 1.011	0.000*

Analysed by One-way ANOVA followed by post-hoc test. * indicates statistical significance at p value less than 0.05.

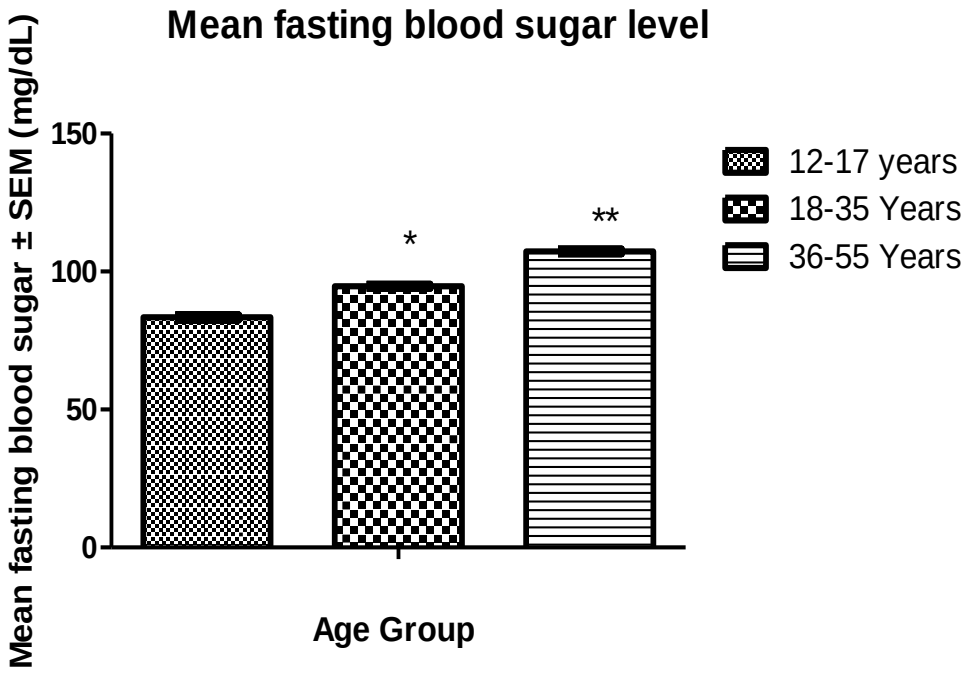

Figure 4.1 Mean fasting blood sugar level among subjects of 12-17 years, 18-35 years and 36-55 years

4.2.3 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (12-17 years age group)

The current study found mean fasting blood sugar among prediabetes subjects was 112.4±1.689 mg/dL, whilst among non-prediabetics it was noted 82.6±0.418 mg/dL. Prediabetics of the age group 12-17 years reported significantly higher mean fasting blood sugar than non-prediabetics. (Table 4.4)

Table 4.4 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (12-17 years age group)

12 to 17 Years Age Group			
	Prediabetes subjects (N = 23)	**Normal subjects (N = 433)**	**P value**
Mean fasting blood sugar ± SEM (mg/dL)	112.4±1.689	82.6±0.418	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05. Mean fasting blood sugar was significantly higher among prediabetes subjects.

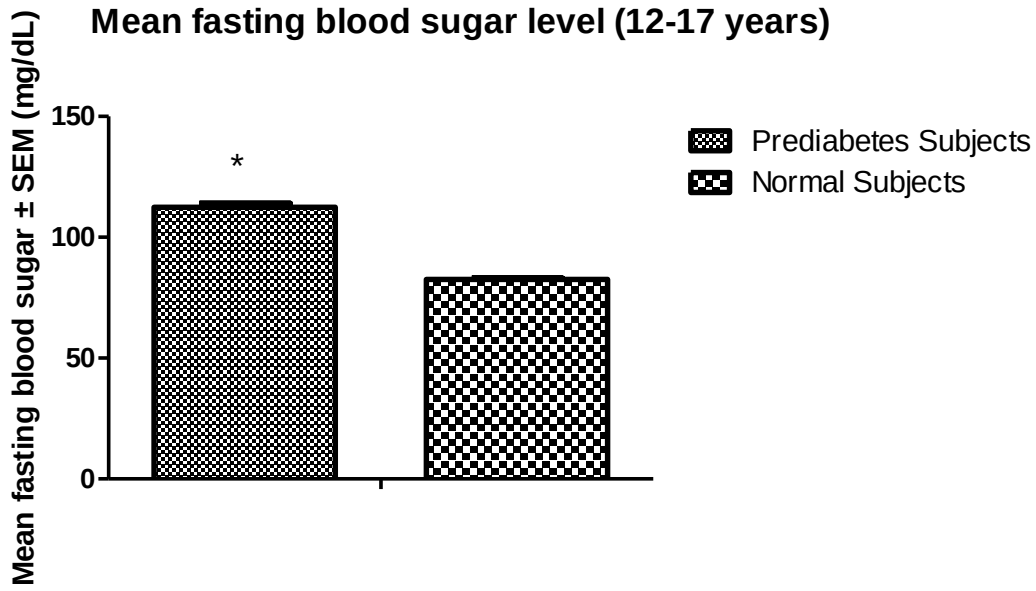

Figure 4.2 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (12-17 years age group)

4.2.4 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (18-35 years age group)

In current study on 18-35 years age group, we noted mean fasting blood sugar among prediabetics and non-prediabetics was 116.2±0.410 mg/dL and 88.1±0.276 mg/dL,

respectively. The mean fasting blood sugar level was noted significantly higher among subjects with prediabetes than without prediabetes. (Table 4.5)

Table 4.5 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (18-35 years age group)

18 to 35 Years Age Group			
	Prediabetes subjects (N = 291)	**Normal subjects (N = 719)**	**P value**
Mean fasting blood sugar ± SEM (mg/dL)	116.2±0.410	88.1±0.276	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

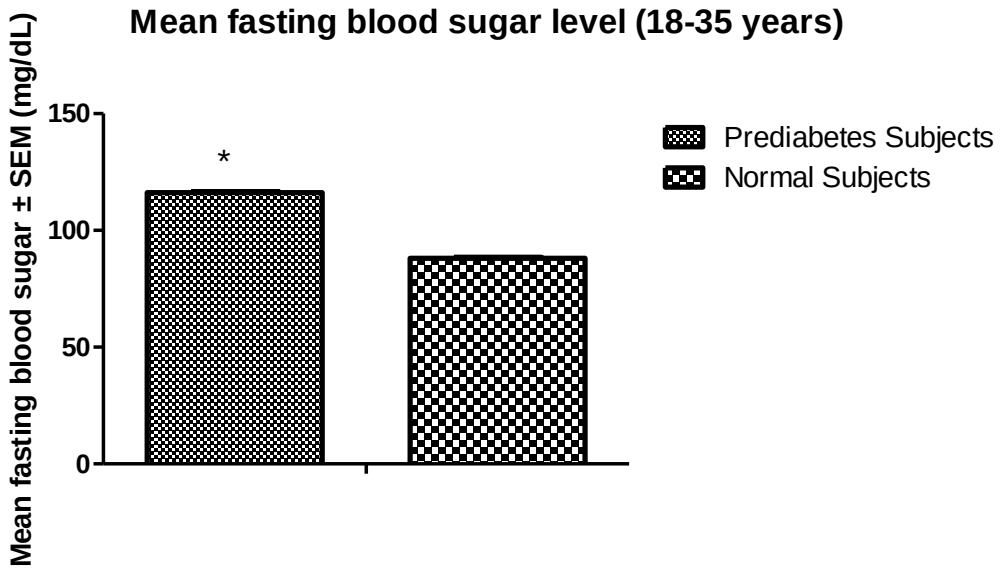

Figure 4.3 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (18-35 years age group)

4.2.5 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (36-55 years age group)

The prediabetes subjects of 36-55 years age group had 116.9±0.344 mg/dL mean fasting blood sugar level which was found significantly higher compared to normal subjects (90.2±0.302 mg/dL) in present study. (Table 4.6)

Table 4.6 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (36-55 years age group)

36 to 55 Years Age Group			
	Prediabetes subjects (N = 314)	**Normal subjects (N = 632)**	**P value**
Mean fasting blood sugar ± SEM (mg/dL)	116.9±0.344	90.2±0.302	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0

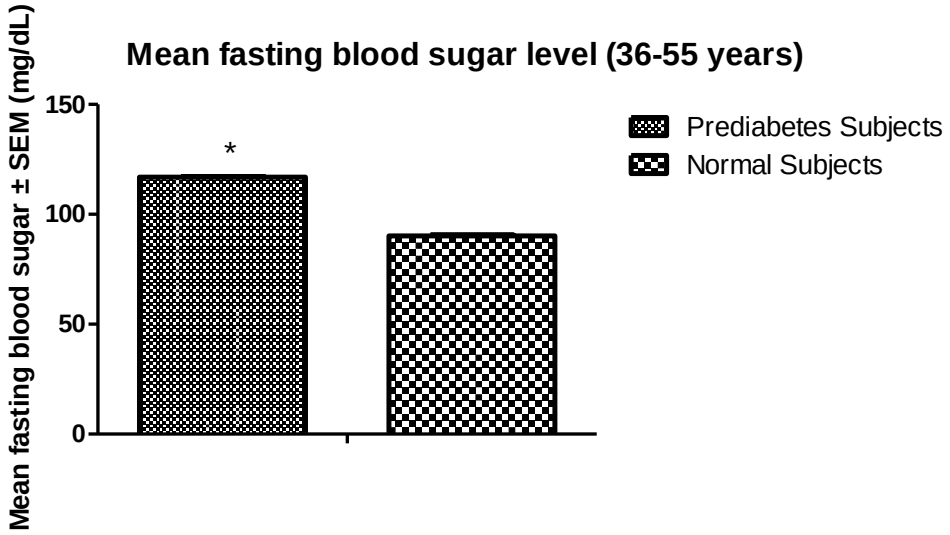

Figure 4.4 Mean fasting blood sugar level among subjects with prediabetes and without prediabetes (36-55 years age group)

4.3 Prediabetes and its associated condition

4.3.1 Association of body mass index (BMI) with prediabetes

4.3.1.1 Association of body mass index (BMI) with prediabetes (12-17 years age group):

Among subjects of 12-17 years, percentage prevalence of underweight, healthy weight, overweight and obese was 17.11%, 61.18%, 16.23% and 5.48%, respectively (Table 4.7).

In this age group of present study, we found that prevalence of prediabetes was noted the highest among obese (28.0%) participants following overweight (6.75%) compared to healthy weight (3.94%) subjects. Whereas prediabetes prevalence was 0.00% among underweight participants in the same age group. (Table 4.8)

The mean BMI among subjects with prediabetes was documented 25.6±0.688 kg/m^2 compared to 21.3±0.082 kg/m^2 in prediabetes free subjects. The high value of mean BMI

among prediabetics found significant. This findings implies that in age group 12-17 years, higher BMI is associated with prediabetes.

Table 4.7 Prevalence of prediabetes among subjects with different BMI classification (12-17 years age group)

12 to 17 Years Age Group			
BMI Classification	**Total (N = 456)**	**Prediabetes subjects**	**P value**
Underweight	78 (17.11%)	0 (0.00%)	0.012*
Healthy weight	279 (61.18%)	11 (3.94%)	
Overweight	74 (16.23%)	5 (6.75%)	
Obese	25 (5.48%)	7 (28.0%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.8 Mean BMI among subjects with prediabetes and without prediabetes (12-17 years age group)

12 to 17 Years Age Group			
	Prediabetes subjects (N = 23)	**Normal subjects (N = 433)**	**P value**
Mean BMI ± SEM (kg/m^2)	25.6±0.688	21.3±0.082	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

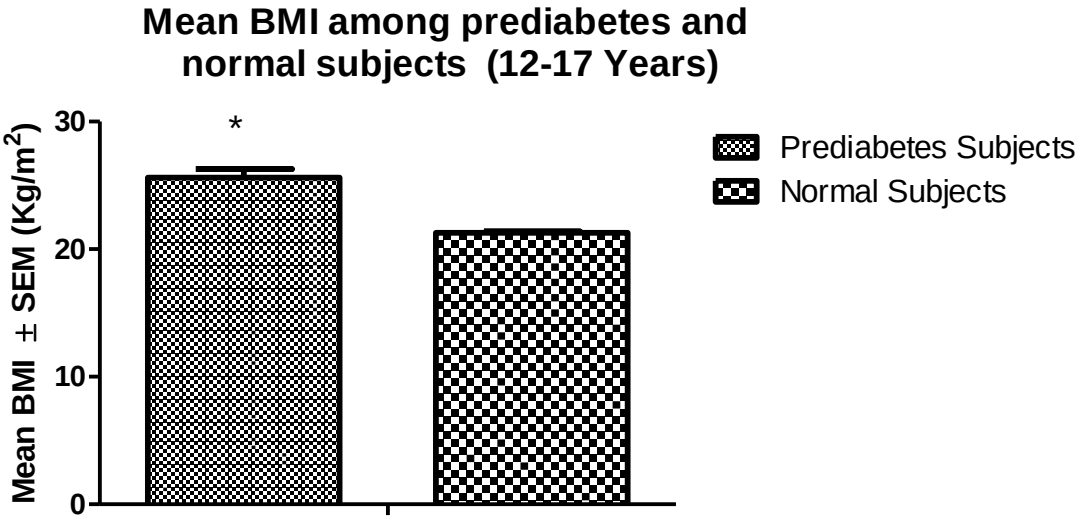

Figure 4.5 Mean BMI among subjects with prediabetes and without prediabetes (12-17 years age group)

4.3.1.2 Association of body mass index (BMI) with prediabetes (18-35 years age group):

The present study showed 6.83%, 69.50%, 18.51% and 5.15% prevalence of underweight, healthy weight, overweight and obese, respectively among subjects of 18-35 years.

In the same age group, prevalence of prediabetes was lowest among healthy weight subjects (22.64%). Whist prevalence of prediabetes among overweight and obese was indistinguishably similar (55.08% and 55.76%). Here in the same age group none of the underweight subjects reported to have prediabetes. (Table 4.9)

In present study, mean BMI among prediabetics was reported 25.8±0147 kg/m^2 which was higher than non-prediabetes subjects (22.3±0.123 kg/m^2) and indicated significant association of higher BMI with prediabetes in 18-35 years age group. (Table 4.10)

Table 4.9 Prevalence of prediabetes among subjects with different BMI classification (18-35 years age group)

18 to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Prediabetes subjects**	
Underweight	69 (6.83%)	0 (0.00%)	< 0.0001*
Healthy Weight	702 (69.50%)	159 (22.64%)	
Over Weight	187 (18.51%)	103 (55.08%)	
Obese	52 (5.15%)	29 (55.76%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.10 Mean BMI among subjects with prediabetes and without prediabetes (18-35 years age group)

18 to 35 Years Age Group			
	Prediabetes subjects (N = 291)	**Normal subjects (N = 719)**	**P value**
Mean BMI ± SEM (kg/m^2)	25.8±0.147	22.3±0.123	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

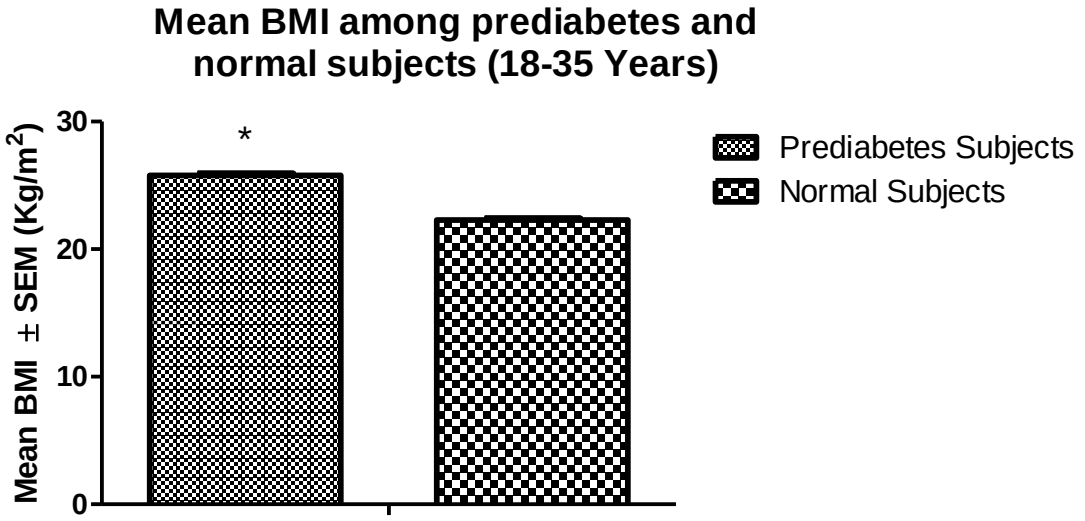

Figure 4.6 Mean BMI among subjects with prediabetes and without prediabetes (18-35 years age group)

4.3.1.3 Association of Body mass index (BMI) and prediabetes (36-55 years age group)

In the present study, the subjects of 36-55 years old had the prevalence of underweight 4.76%, healthy weight 63.64%, overweight 26.22% and obese 5.39%.

Among the subjects of same age group, we noted that risk of prediabetes gradually increases with BMI as prevalence of prediabetes among healthy weight subject was 24.25% which was less than overweight (55.64%) and obese (58.82%). (Table 4.11)

The value of mean BMI was found high among prediabetes participants (25.7±0.220 kg/m^2) when compared with prediabetes free participants (22.6±0.127 kg/m^2) and showed significant association of prediabetes with BMI in 36-55 years age group. (Table 4.12)

Table 4.11 Prevalence of prediabetes among subjects with different BMI classification (36-55 Years Age Group)

36 to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Prediabetes subjects**	
Underweight	45 (4.76%)	0 (0.00%)	< 0.0001*
Healthy Weight	602 (63.64%)	146 (24.25%)	
Overweight	248 (26.22%)	138 (55.64%)	
Obese	51 (5.39%)	30 (58.82%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.12 Mean BMI among subjects with prediabetes and without prediabetes (36-55 years age group)

36 to 55 Years Age Group			
	Prediabetes subjects (N = 314)	**Normal subjects (N = 632)**	**P value**
Mean BMI ± SEM (kg/m^2)	25.7±0.220	22.6±0.127	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

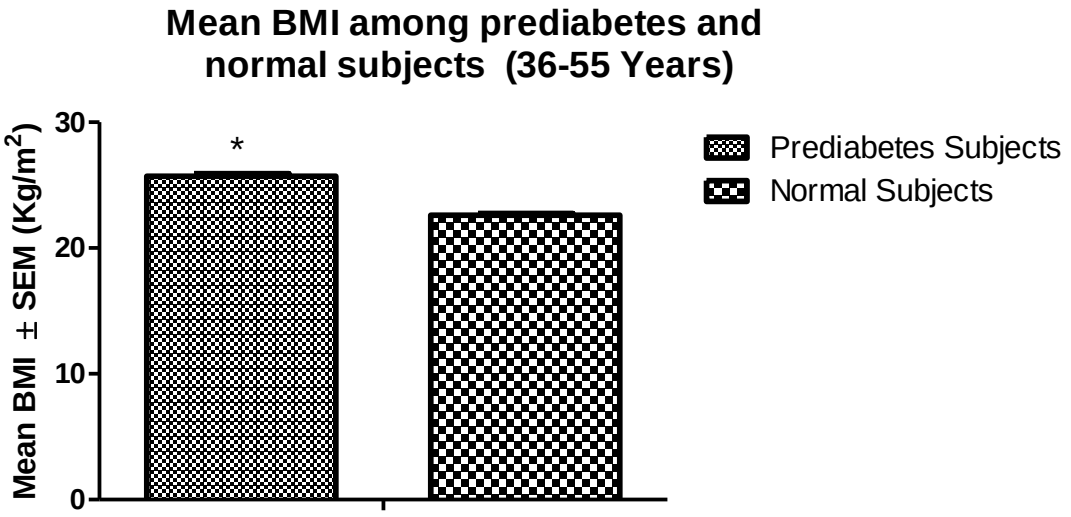

Figure 4.7 Mean BMI among subjects with prediabetes and without prediabetes (36-55 years age group)

From findings of all three group it can be concluded that the prevalence of overweight was higher in 36 to 55 years age group (26.22%) followed by 18 years to 35 years age group (18.61%) and 12 to 17 years age group (16.23%). Whereas percentage of obese participants was higher in 12-17 years age group (5.48%) followed by 36-55 years age group (5.39%) and 18-35 years age group (5.15%). The percentage of healthy weight was higher in 18-35 years age group (69.50%) compare to other two age groups. It was observed that % of underweight participant was higher in school going children (17.11%) compare to adults. Further we found that there was approximately 6 times higher chances to develop a prediabetes in subjects who were overweight or obese compared to subjects who were underweight or normal weight. We found the positive relationship between BMI and prediabetes in all the age groups in present study.

4.3.2 Association of family history of diabetes, obesity, thyroid and hypertension with prediabetes

4.3.2.1 Association of family history of diabetes, obesity, thyroid and hypertension with prediabetes (12-17 years age group)

In current study among 12-17 years old participants, 35.53% were found to have family history of diabetes and 36.18% participants were found to have family history of

hypertension. In the same age group prevalence of family history of obesity and thyroid was 43.20% and 7.89% respectively.

In present study it was found that positive family history of diabetes was present in 3.70% prediabetic participants. Similarly, family history of obesity, thyroid and hypertension was positive among 7.10%, 5.55% and 3.03% prediabetes participants. The risk of prediabetes was found significantly associated with family history of diabetes, thyroid and hypertension whilst not associated with family history of obesity in 12 to 17 years age group. (Table 4.13)

Table 4.13 Prevalence of prediabetes among subjects with positive family history of diabetes, obesity, thyroid and hypertension (12-17 Years Age Group)

12 to 17 Years Age Group				**P value**
	Family history	**Total (N = 456)**	**Prediabetes subjects**	
Family History of Diabetes	Present	162 (35.53%)	6 (3.70%)	0.022*
	Absent	294 (64.47%)	17 (5.70%)	
Family History of Obesity	Present	197 (43.20%)	14 (7.10%)	0.297
	Absent	259 (56.79%)	9 (3.47%)	
Family History of Thyroid	Present	36 (7.89%)	2 (5.55%)	<0.0001*
	Absent	420 (91.10%)	21 (5%)	
Family History of Hypertension	Present	165 (36.18%)	5 (3.03%)	0.0007*
	Absent	291 (63.81%)	18 (6.18%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.2.2 Association of family history of diabetes, obesity, thyroid and hypertension with prediabetes (18-35 years age group)

In current study in the age group of 18-35 years, 38.22%, 39.20%, 41.18% and 13.96% participants were reported family history of diabetes, hypertension, obesity and thyroid respectively. In this age group prevalence of family history of thyroid was to be highest among all three age group.

In the same age group, it was documented that 48.44% subjects with prediabetes had positive family history of diabetes, 43.75% prediabetes subjects had positive family history of obesity, 34.75% prediabetes subjects positive family history of thyroid and 46.46% prediabetes subjects positive family history of hypertension. We found prevalence of prediabetes was significantly associated with positive family history of diabetes, obesity, thyroid and hypertension which suggested that the risk of prediabetes increases with positive family history of such diseases among subjects of 18-35 years. (Table 4.14).

Table 4.14 Prevalence of prediabetes among subjects with positive family history of diabetes, obesity, thyroid and hypertension (18-35 years age group)

18 to 35 Years Age Group				**P value**
	Family history	**Total (N = 1,010)**	**Prediabetes subjects**	
Family History of Diabetes	Present	386 (38.22%)	187 (48.44%)	< 0.001*
	Absent	624 (61.78%)	104 (16.66%)	
Family History of Obesity	Present	416 (41.18%)	182 (43.75%)	< 0.001*
	Absent	594 (58.81%)	109 (18.35%)	
Family History of Thyroid	Present	141 (13.96%)	49 (34.75%)	< 0.001*
	Absent	869 (86.03%)	242 (27.84%)	

Family History of Hypertension	Present	396 (39.20%)	184 (46.46%)	< 0.001*
	Absent	614 (60.79%)	107 (17.42%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.2.3 Association of family history of diabetes, obesity, thyroid and hypertension with prediabetes (36-55 years age group)

In present study, the prevalence of family history of diabetes in 36-55 years age group was 37.10%, hypertension was 43.70%, obesity was 38.79% and thyroid was 10.78%. Prevalence of family history of hypertension was highest in this age group compared to two other groups.

The present study documented 73.78% prediabetes participants had positive family history of diabetes, 62.39% prediabetes subjects had positive family history of obesity, 40.19% prediabetes subjects positive family history of thyroid and 68.35% prediabetes subjects positive family history of hypertension. Here in this group we also found prevalence of prediabetes was significantly associated with positive family history of diabetes, obesity, thyroid and hypertension. This result suggested that the risk of prediabetes increases among subjects with positive family history of diabetes, obesity, thyroid and hypertension among subjects of 36-55 years. (Table 4.15)

Table 4.15 Prevalence of prediabetes among subjects with positive family history of diabetes, obesity, thyroid and hypertension (36-55 years age group)

36 to 55 Years Age Group				**P value**
	Family history	**Total (N = 946)**	**Prediabetes subjects**	
Family History of Diabetes	Present	351 (38.22%)	259 (73.78%)	< 0.001*
	Absent	595 (61.78%)	55 (9.24%)	

Family History of Obesity	Present	367 (38.79%)	229 (62.39%)	< 0.001*
	Absent	579 (61.20%)	85 (14.68%)	
Family History of Thyroid	Present	102 (10.78%)	41 (40.19%)	< 0.001*
	Absent	844 (89.21%)	273 (27.84%)	
Family History of Hypertension	Present	414 (43.76%)	283 (68.35%)	< 0.001*
	Absent	532 (56.23%)	31 (17.42%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.3 Association of blood pressure and prediabetes

4.3.3.1 Association of blood pressure and prediabetes (12-17 years age group)

In present study we found among the subjects of 12-17 years, 12.28% prevalence of pre-hypertension and 5.04% prevalence of hypertension. Whilst prevalence of subjects with normal blood pressure was the highest (82.68%).

The present study also documented, 23.21% and 34.78% prevalence of prediabetes among subjects with pre-hypertension and hypertension, respectively. Whereas, 0.53% subjects with normal blood pressure reported to have prediabetes. The prevalence of hypertension was noted higher than pre-hypertension in subjects with prediabetes in the age group of 12-17 years. We found elevated blood pressure reported to have significant association with incidence prediabetes. (Table 4.16)

The mean fasting blood sugar level in pre-hypertensive subjects was 97.3±1.363 mg/dL while higher mean fasting blood sugar level was found in hypertensive subjects (103.2±2.606 mg/dL). We noted that the mean fasting blood sugar level was less among subjects with normal blood pressure compared to pre-hypertensive and hypertensive subjects. This findings implies significant association of prehypertension and prediabetes as well hypertension and prediabetes. (Table 4.17, Table 4.18)

Table 4.16 Prevalence of prediabetes among subjects with pre-hypertension, hypertension and normal blood pressure (12-17 Years age group)

12 to 17 Years Age Group			
Blood Pressure	**Total (N = 456)**	**Prediabetes subjects**	**P value**
Pre-Hypertension	56 (12.28%)	13 (23.21%)	0.018*
Hypertension	23 (5.04%)	8 (34.78%)	
Normal	377 (82.68%)	2 (0.53%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.17 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (12-17 years age group)

12 to 17 Years Age Group			
Mean fasting blood sugar ± SEM (mg/dL)	**Pre-hypertension (N = 13)**	**Normal blood pressure (N = 2)**	**P value**
	97.3±1.363	88.7±0.556	0.032*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

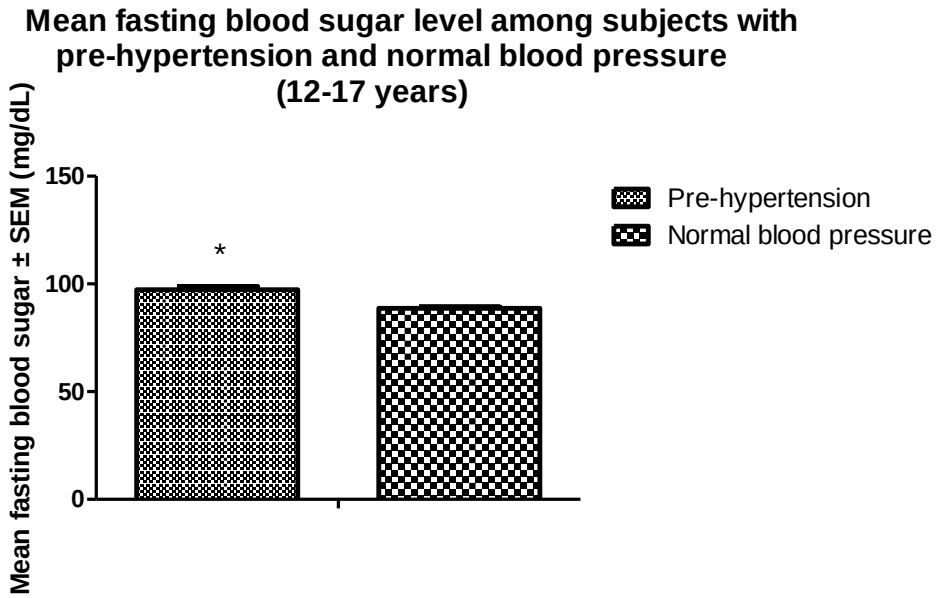

Figure 4.8 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (12-17 years age group)

Table 4.18 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (12-17 years age group)

12 to 17 Years Age Group			
Mean fasting blood sugar ± SEM (mg/dL)	**Hypertension (N = 8)**	**Normal blood pressure (N = 2)**	**P value**
	103.2±2.606	88.7±0.556	0.028*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

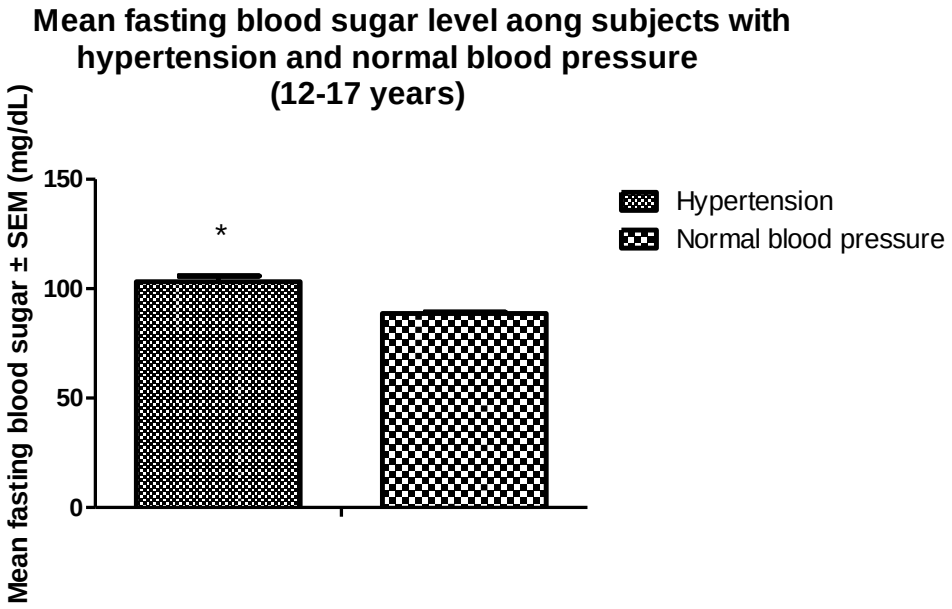

Figure 4.9 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (12-17 years age group)

4.3.3.2 Association of blood pressure and prediabetes (18-35 years age group)

The prevalence of pre-hypertension, hypertension among subjects of 18-35 years age group was 27.62% and 22.87%, respectively. Whereas in the same age group 49.50% participants reported to have normal blood pressure.

In the same age group, we found 44.44% and 32.46% prevalence of prediabetes among subjects with pre-hypertension and hypertension respectively whilst among subjects with normal blood pressure it was noted less (18.4%) compared to other two. Elevated blood pressure was found significantly associated with prediabetes. (Table 4.19)

The mean fasting blood sugar level was the highest among subjects with hypertension (108.1±0.691 mg/dL) and pre-hypertension (103.4±0.335 mg/dL) compared to normal blood pressure (91.3±0.152 mg/dl). We further noted that mean level of blood sugar increased with blood pressure. We found mean blood pressure had significant association with prediabetes in this age group. (Table 4.20, Table 4.21)

Table 4.19 Prevalence of prediabetes among subjects with pre-hypertension, hypertension and normal blood pressure (18-35 Years age group)

18 to 35 Years Age Group			
Blood Pressure	**Total (N = 1,010)**	**Prediabetes subjects**	**P value**
Pre-hypertension	279 (27.62%)	124 (44.44%)	0.002*
Hypertension	231 (22.87%)	75 (32.46%)	
Normal	500 (49.50%)	92 (18.4%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.20 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (18-35 years age group)

18 to 35 Years Age Group			
Mean fasting blood sugar ± SD (mg/dL)	**Pre-hypertension (N = 124)**	**Normal blood pressure (N = 92)**	**P value**
	103.4±0.335	91.3±0.152	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

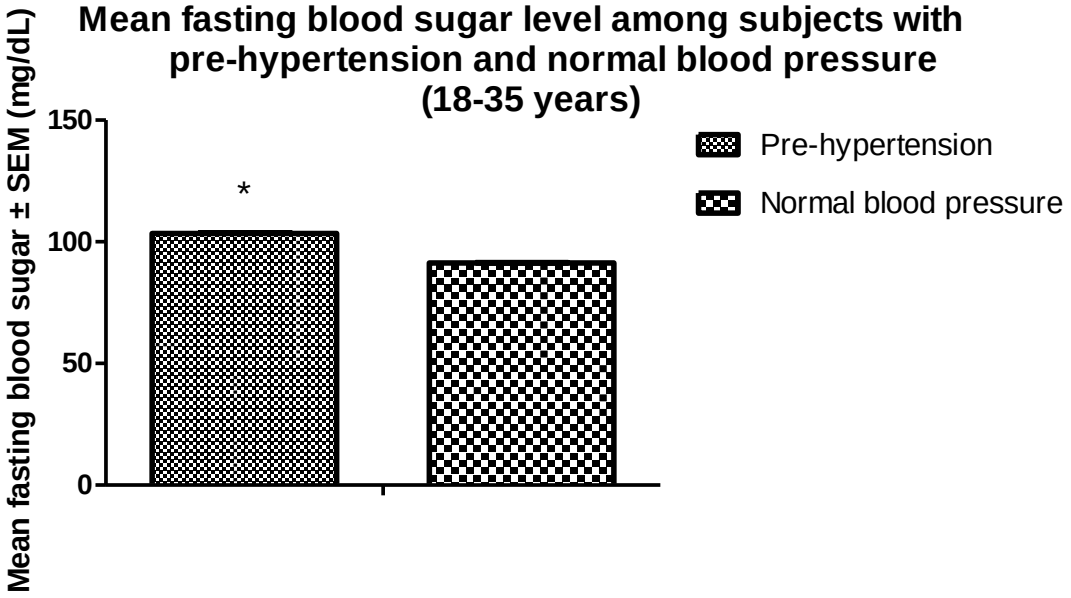

Figure 4.10 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (18-35 years age group)

Table 4.21 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (18-35 years age group)

18 to 35 Years Age Group			
Mean fasting blood sugar ± SEM (mg/dL)	**Hypertension (N = 75)**	**Normal blood pressure (N = 92)**	**P value**
	108.1±0.691	91.3±0.152	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

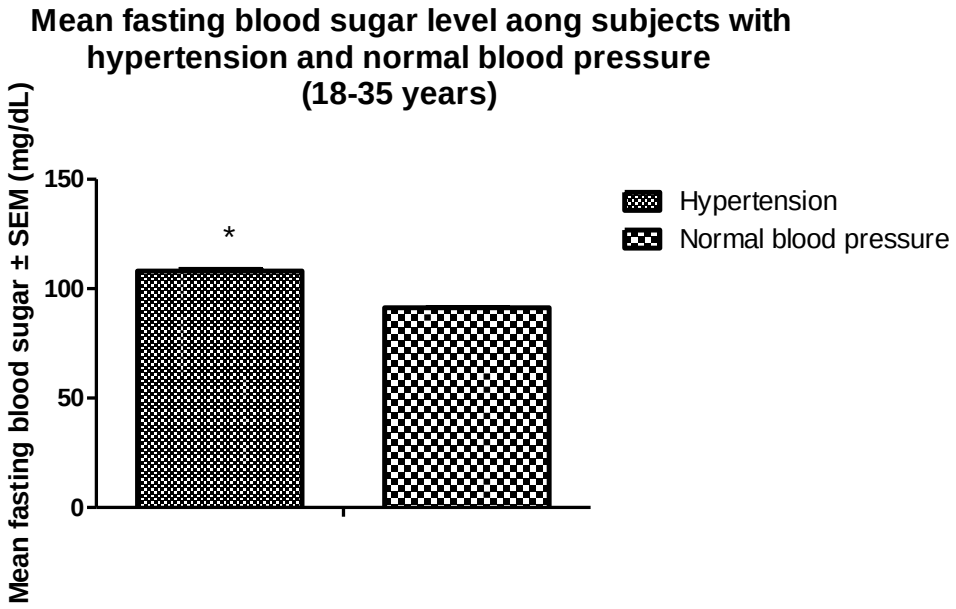

Figure 4.11 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (18-35 years age group)

4.3.3.3 Association of blood pressure and prediabetes (36-55 years age group)

The prevalence of prehypertension was 29.28% and hypertension was 31.92% among subjects of 36-55 years age group. Whereas 38.79% participants were found to have normal blood pressure.

The current study revealed that the prevalence of prediabetes among age group of 36-55 years was nearly equal in participants with hypertension and pre-hypertension (42.05% and 44.40% respectively). Whereas 17.43% subjects with prediabetes had normal blood pressure which is found lower than other two. Elevated blood pressure was found significantly associated with incidence prediabetes. (Table 4.22)

In the same age group, the mean fasting blood sugar level was greater in pre-hypertensive subjects (106.8±0.216 mg/dL) than subjects with normal blood pressure. Similarly, mean fasting blood sugar level was higher in subjects with hypertension (115.6±0.340 mg/dL) than with normal blood pressure. We found significant association of mean fasting blood sugar with prediabetes. (Table 4.23, Table 4.24)

Table 4.22 Prevalence of Prediabetes among subjects with pre-hypertension and hypertension (36-55 years age group)

36 to 55 Years Age Group			
Blood Pressure	**Total (N = 946)**	**Prediabetes subjects**	**P value**
Pre-hypertension	277 (29.28%)	123 (44.40%)	< 0.001*
Hypertension	302 (31.92%)	127 (42.05%)	
Normal	367 (38.79%)	64 (17.43%)	

Analysed by One way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.23 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (36-55 years age group)

36 to 55 Years Age Group			
Mean fasting blood sugar ± SEM (mg/dL)	**Pre-hypertension (N = 123)**	**Normal blood pressure (N = 64)**	**P value**
	106.8±0.216	97.2±0.355	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

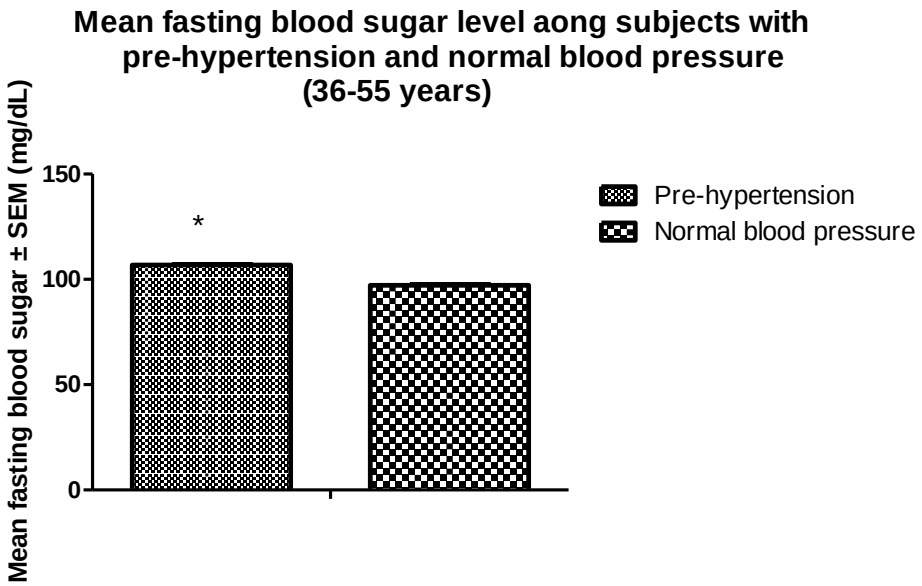

Figure 4.12 Mean fasting blood sugar level among subjects of pre-hypertension and normal blood pressure (36-55 years age group)

Table 4.24 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (36-55 years age group)

36 to 55 Years Age Group			
Mean fasting blood sugar ± SEM (mg/dL)	**Hypertension (N = 127)**	**Normal blood pressure (N = 64)**	**P value**
	115.6±0.340	97.2±0.355	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

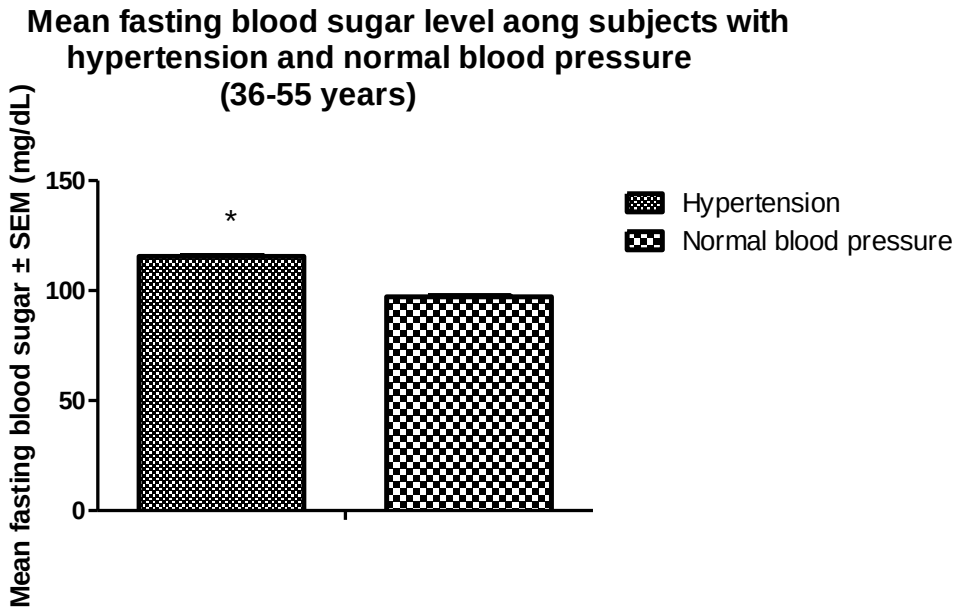

Figure 4.13 Mean fasting blood sugar level among subjects of hypertension and normal blood pressure (36-55 years age group)

In the ultimate analysis among all three age groups, it was observed that prevalence of pre-hypertension and hypertension was higher than the normal blood pressure in subjects with prediabetes. Furthermore, the mean fasting blood sugar level was greater among hypertensive participants following pre-hypertensive and normal.

4.3.4 Association of participation in exercise with prediabetes

4.3.4.1 Association of participation in exercise with prediabetes (12-17 years age group)

The present study observed that 66.67% school going children of aged 12-17 years age group participating in exercise, whilst 33.33% participants denied their participation in exercise.

In this study, in the age group of 12-17 years, we found 7.89% prediabetes participants denied to take part in exercise whereas 3.61% prediabetes participants reported their participation in exercise. It was clear from the observation that no participation in exercise was the risk factor for prediabetes and these two were significantly associated. (Table 4.25)

Table 4.25 Prevalence of prediabetes among subjects who participate or did not participate in exercise (12-17 years age group)

12 to 17 Years Age Group			P value
Participation in Exercise	**Total (N = 456)**	**Prediabetes subjects**	
Yes	304 (66.67%)	11 (3.61%)	0.043*
No	152 (33.33%)	12 (7.89%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

4.3.4.2 Association of participation in exercise with prediabetes (18-35 years age group)

In current study we found 32.48% participants of age group 18-35 years participate in exercise while 67.52% participants were denied to perform any form of exercises.

In the same age group, 35.77% participants with prediabetes were not involved in any kind of exercise or physical activity whereas 14.32% prediabetes participants were found to take part in exercise. This association was found statistically significant, indicating the risk of prediabetes would be diminished with active participation in exercise. (Table 4.26)

Table 4.26 Prevalence of prediabetes among subjects who participate or did not participate in exercise (18-35 years age group)

18 to 35 Years Age Group			P value
Participation in Exercise	**Total (N = 1,010)**	**Prediabetes subjects**	
Yes	328 (32.48%)	47 (14.32%)	< 0.001*
No	682 (67.52%)	244 (35.77%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

4.3.4.3 Association of participation in exercise with prediabetes (36-55 years age group)

Among participant of age group 36-55 years, we found 22.20% participating in exercise whereas 77.80% denied their participation in exercise.

In the same age group, we found 37.63% prevalence of prediabetes who did not participate in exercise or physical activity whereas 17.61% prevalence of prediabetes in participants who vigorously participated in exercise. The association of prediabetes and physical exercise was found statistically significant in current study. (Table 4.27)

Table 4.27 Prevalence of prediabetes among subjects who participate or did not participate in exercise (36-55 years age group)

36 to 55 Years Age Group			P value
Participation in Exercise	**Total (N = 946)**	**Prediabetes subjects**	
Yes	210 (22.20%)	37 (17.61 %)	< 0.001*
No	736 (77.80%)	277 (37.63%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

4.3.5 Association of participation in games and leisure activities with prediabetes

4.3.5.1 Association of participation in games and leisure activities with prediabetes (12-17 years age group)

In current study we found 26.75% and 56.36% participants of 12-17 years age group participate in indoor and outdoor games respectively. Whilst we noted 100% subjects of same age group gave their confirmation for playing on laptop/mobile and watching television regularly.

In the same age group we identified higher prevalence of prediabetes among subjects who participate in indoor game whereas indistinguishable prevalence of prediabetes was found among subjects who participate outdoor games. Participation in either indoor or outdoor games found to have no significant association with prediabetes development among 12-17 years old subjects. (Table 4.28)

Table 4.28 Prevalence of prediabetes among subjects who indulge in games and leisure activities (12-17 years age group)

12 to 17 Years Age Group				P value
Parameter		**Total (N = 456)**	**Prediabetes subjects**	
Indoor games	Yes	122 (26.75%)	9 (7.37%)	0.297
	No	334 (73.24%)	14 (4.19%)	
Outdoor games	Yes	257 (56.36%)	13 (5.05%)	0.532
	No	199 (43.64%)	10 (5.02%)	
Playing on laptop/mobile	Yes	456 (100%)	23 (5.04%)	< 0.001*
	No	0 (0%)	0 (0%)	
Watching television	Yes	456 (100%)	23 (5.04%)	< 0.001*
	No	0 (0%)	0 (0%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.5.2 Association of participation in games and leisure activities with prediabetes (18-35 years age group)

In the age group of 18 to 35 years, we noted 12.28% and 16.93% subjects participates in indoor and outdoor games respectively. Here in this group also we found 100% subjects of

same age group gave their consent for playing on laptop/mobile and watching television regularly.

In current study among the subjects of same age group we identified higher prevalence of prediabetes among subjects who was not participating in indoor and outdoor games. Our findings shown no participation in indoor as well outdoor games found to have significant association with prediabetes development. (Table 4.29)

Table 4.29 Prevalence of prediabetes among subjects who indulge in games and leisure activities (18-35 years age group)

18 to 35 Years Age Group				**P value**
Parameters		**Total (N = 1,010)**	**Prediabetes subjects**	
Indoor games	Yes	124 (12.28%)	23 (18.54%)	< 0.001*
	No	886 (87.72%)	268 (30.24%)	
Outdoor games	Yes	171 (16.93%)	43 (25.14%)	< 0.001*
	No	839 (83.06%)	248 (29.55%)	
Playing on laptop/mobile	Yes	1,010 (100%)	291 (28.81%)	< 0.001*
	No	0 (0%)	0 (0%)	
Watching television	Yes	1,010 (100%)	291 (28.81%)	< 0.001*
	No	0 (0%)	0 (0%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.5.3 Association of participation in games and leisure activities with prediabetes (36-55 years age group)

We found 6.23% and 8.24% subjects participates in indoor and outdoor games respectively among the subjects of 36-55 years age group. In this group we noted 81.71% prevalence for playing on laptop/mobile which was 100% in other two groups. Hundred percent subjects of this group confirmed they watch television regularly.

In current study among the subjects of same age group we identified low prevalence of prediabetes among subjects who participate in both indoor as well as outdoor games. In this age group, the prevalence of prediabetes was higher (40.23%) among subjects who plays on laptop/mobile compared to subjects who did not play on laptop/mobile (1.73%). Not participating in indoor and outdoor games and playing on laptop/mobile or watching television found to have significant association with prediabetes development. (Table 4.30)

Table 4.30 Prevalence of prediabetes among subjects who indulge in games and leisure activities (36-55 years age group)

36 to 55 Years Age Group				**P value**
Parameters		**Total (N = 946)**	**Prediabetes subjects**	
Indoor games	Yes	59 (6.23%)	11 (18.64%)	< 0.001*
	No	887 (93.76%)	303 (34.16%)	
Outdoor games	Yes	78 (8.24%)	19 (24.35%)	< 0.001*
	No	868 (91.75%)	295 (33.98%)	
Playing on laptop/mobile	Yes	773 (81.71%)	311 (40.23%)	< 0.001*
	No	173 (18.28%)	3 (1.73%)	

Watching television	Yes	946 (100%)	314 (33.19%)	< 0.001*
	No	0 (0%)	0 (0%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.6 Association of dietary type with prediabetes

4.3.6.1 Association of dietary type with prediabetes (12-17 years age group)

In present study, the subjects of 12-17 years with vegetarian diet were reported higher in prevalence (83.11%), followed by eggetarian (11.40%) and non-vegetarian (5.48%).

Among the participants of same age group, we found that prevalence of prediabetes among vegetarian subjects was 3.16%, non-vegetarian subjects was 24% and eggetarian subject was 9.61%. It was justified from these observations that prediabetes was the highest among subjects with non-vegetarian dietary forms than eggetarian and vegetarian. The dietary type was not significantly associated with incident prediabetes in this age group. (Table 4.31)

Table 4.31 Prevalence of prediabetes among subjects with different dietary type (12-17 years age group)

12 to 17 Years Age Group			**P value**
Dietary Type	**Total (N = 456)**	**Prediabetes subjects**	
Vegetarian	379 (83.11%)	12 (3.16%)	0.153
Non-Vegetarian	25 (5.48%)	6 (24.00%)	
Eggeterian	52 (11.40%)	5 (9.61%)	

Analysed by One-way chi-square test for goodness of fit.

4.3.6.2 Association of dietary type and prediabetes (18-35 years age group)

The prevalence of vegetarian diet was 57.92%, non-vegetarian 7.23% and eggetarian 34.85% among participants of 18-35 years age group.

The present study in 18-35 years age group documented, 21.53% prediabetes participants were vegetarian, 56.16% prediabetes participants were non-vegetarian and 35.27% prediabetes participants were eggetarian. We found greater number of subjects with prediabetes among non-vegetarian dietary type following eggetarian and vegetarian. The association of type of diet and risk of prediabetes was found statistically significant. (Table 4.32)

Table 4.32 Prevalence of prediabetes among subjects with different dietary type (18-35 years age group)

18 to 35 Years Age Group			**P value**
Diet Type	**Total (N = 1,010)**	**Prediabetes subjects**	
Vegetarian	585 (57.92%)	126 (21.53%)	< 0.001*
Non-Vegetarian	73 (7.23%)	41 (56.16%)	
Eggeterian	352 (34.85%)	124 (35.27%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.6.3 Association of dietary type and prediabetes (36-55 years age group)

In current study, we noted higher prevalence of vegetarian dietary type (70.93%) followed by eggetarian (23.15%) and non-vegetarian (5.92%) among subjects of 36-55 years age group.

Among the subjects of same age group, we found higher prevalence of prediabetes in non-vegetarians (66.07%) following eggetarian (36.07%) and vegetarian (29.50%). Correspondingly to 18-35 years age group, in this group subjects we found significant association of type of diet with risk of prediabetes. (Table 4.33)

Table 4.33 Prevalence of prediabetes among subjects with different dietary type (36-55 years age group)

36 to 55 Years Age Group			**P value**
Diet Type	**Total (N = 946)**	**Prediabetes subjects**	
Vegetarian	671 (70.93%)	198 (29.50%)	< 0.001*
Non-Vegetarian	56 (5.92%)	37 (66.07%)	
Eggeterian	219 (23.15%)	79 (36.07%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.7 Association of frequency of junk food eating habit with prediabetes

4.3.7.1 Association of frequency of junk food eating habit with prediabetes (12-17 years age group)

The present study showed once in a week frequency of junk food habit was higher (50.44%) following once in fifteen days (19.52%). Additionally, it was remarked that once in a month frequency (16.23%) of eating junk was higher in comparison to everyday (13.82%).

In present study it was noted that in 12-17 years age group 1.58% prediabetes participants had everyday junk food eating habit which was declining among once in week (4.34%). Once in 15 days (7.86%) and once in month (6.75%) junk food habit frequency was found unremarkably different. Among these frequencies of eating junk food habit we observed lowest in everyday whilst we did not find any significant difference among other three

frequencies. The frequency of junk food habit and risk of developing prediabetes was found statistically non-significant in this age group. (Table 4.34)

Table 4.34 Prevalence of prediabetes among subjects with different frequency of junk food habit (12-17 Years age group)

12 to 17 Years Age Group			P value
Frequency of Junk Food Eating	**Total (N = 456)**	**Prediabetes subjects**	
Everyday	63 (13.82%)	1 (1.58%)	0.059
Once in a Week	230 (50.44%)	10 (4.34%)	
Once in 15 Days	89 (19.52%)	7 (7.86%)	
Once in a Month	74 (16.23%)	5 (6.75%)	

Analysed by One-way chi-square test for goodness of fit.

4.3.7.2 Association of frequency of junk food eating habit with prediabetes (18-35 years age group)

In current study, in the age group of 18-35 years, the frequency of once in a week frequency of junk food eating habit was the highest (62.18%) following once in 15 days (18.42%) and everyday (13.76%). Whereas subjects who had habit of eating junk food once in a month was lowest (5.64%).

In the same age group, we noted highest participants (41.87%) with once in a week junk food eating habit had prediabetes. Whereas 14.38% participants with everyday junk food eating habit and 4.30% participants who had habit of eating junk food once in fifteen days found to have prediabetes. Another observation in this context was that none of the participants with once in a month junk food eating habit had prediabetes. Here in this group we noted frequency of junk food habit is significantly associated with risk of developing prediabetes. (Table 4.35)

Table 4.35 Prevalence of prediabetes among subjects with different frequency of junk food habit (18-35 years age group)

18 to 35 Years Age Group			P value
Frequency of Junk Food Eating	**Total (N = 1,010)**	**Prediabetes subjects**	
Everyday	139 (17.76%)	20 (14.38%)	< 0.001*
Once in a Week	628 (62.18%)	263 (41.87%)	
Once in 15 Days	186 (18.42%)	8 (4.30%)	
Once in Month	57 (5.64%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.7.3 Association of frequency of junk food eating habit with prediabetes (36-55 years age group)

In present study we found once in a week frequency of junk food habit was the highest (52.11%) among all. Prevalence of frequencies such as once in fifteen days, once in a month and everyday was 22.52%, 14.54% and 10.48%, respectively.

It was observed in 36-55 years of age group, greater number of subjects who ate junk food everyday and once in a week had prediabetes; though the prevalence of prediabetes among these frequencies was equally similar (46.07% and 47.05% respectively). Whilst risk of prediabetes among subjects who had junk food in the frequencies of once in fifteen days and once in a month was low respectively, 12.20% and 6.52%. This data revealed that frequencies of junk food habit showed significant association with risk of developing prediabetes. (Table 4.36)

Table 4.36 Prevalence of prediabetes among subjects with different frequency of junk food habit (36-55 years age group)

36 to 55 Years Age Group			P value
Frequency of Junk Food Eating	**Total (N = 946)**	**Prediabetes subjects**	
Everyday	102 (10.78%)	47 (46.07%)	< 0.001*
Once in a Week	493 (52.11%)	232 (47.05%)	
Once in 15 Days	213 (22.52%)	26 (12.20%)	
Once in Month	138 (14.59%)	9 (6.52%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.8 Association of frequency of sweet eating habit with prediabetes

4.3.8.1 Association of frequency of sweet eating habit with prediabetes (12-17 years age group)

Among the subjects of 12-17 years, once in a week frequency of sweet eating habit was highest (47.37%) following everyday (22.59%), once in fifteen days (17.11%) and once in a month (12.94%) was lowest among all.

In present study it was found that in the same age group, occurrence of prediabetes in subjects with frequency of sweet eating habit everyday, once in a week, once in fifteen days and once in a month was 7.76%, 5.09%, 3.84% and 1.69% respectively. A frequency of sweet eating habit and risk of developing prediabetes was found significantly associated. These findings indicate that frequencies of habit of eating sweet can be considered as predisposing factor for the incident prediabetes at early age group. (Table 4.37)

Table 4.37 Prevalence of prediabetes among subjects with different frequency of sweet eating habit (12-17 years age group)

12 to 17 Years Age Group			P value
Frequency of Sweet Eating	**Total (N = 456)**	**Prediabetes subjects**	
Everyday	103 (22.59%)	8 (7.76%)	0.012*
Once in a Week	216 (47.37%)	11 (5.09%)	
Once in 15 Days	78 (17.11%)	3 (3.84%)	
Once in Month	59 (12.94%)	1 (1.69%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.8.2 Association of frequency of sweet eating habit with prediabetes (18-35 years age group)

In present study, we noted prevalence of once in a week frequency was the highest (53.17%) following everyday (26.24%), once in fifteen days (13.76%) and once in a month (6.83%) among subjects of 18-35 years age group.

In the same age group, we found 35.84% prediabetes participants had habit of eating sweet everyday, which was the highest among all. Prevalence of prediabetes in subjects with once in a week and once in fifteen days was reported 29.79% and 20.14%, respectively. While prevalence of prediabetes in subjects who ate sweet once in a month was the lowest (11.59%). Here we noted frequency of sweet eating habit was significantly associated with incident prediabetes. (Table 4.38)

Table 4.38 Prevalence of prediabetes among subjects with different frequency of sweet eating habit (18-35 years age group)

18 to 35 Years Age Group			P value
Frequency of Sweet Eating	**Total (N = 1,010)**	**Prediabetes subjects**	
Everyday	265 (26.24%)	95 (35.84%)	< 0.001*
Once in a Week	537 (53.17%)	160 (29.79%)	
Once in 15 Days	139 (13.76%)	28 (20.14%)	
Once in Month	69 (6.83%)	8 (11.59%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.8.3 Association of frequency of sweet eating habit with prediabetes (36-55 years age group)

Among subjects of 36-55 years, we found high prevalence of once in a week frequency of sweet eating habit (48.52%). The prevalence of everyday and once in fifteen days frequencies of sweet eating habit was respectively 24.21% and 19.66%. Whereas only 7.61% subjects reported to have once in a month habit of eating sweets.

In present study, high prevalence of prediabetes was found in subjects who had everyday and once in a week sweet eating habit (42.79%, 40.74% respectively). Prevalence of prediabetes was 11.82% and 9.72% respectively among subjects with once in a week, once in fifteen days and once in a month sweet eating habit. This data revealed that frequency of sweet eating habit has significant association with risk of prediabetes. (Table 4.39)

Table 4.39 Prevalence of prediabetes among subjects with different frequency of sweet eating habit (36-55 years age group)

36 to 55 Years Age Group			P value
Frequency of Sweet Eating	**Total (N = 946)**	**Prediabetes subjects**	
Everyday	229 (24.21%)	98 (42.79%)	< 0.001*
Once in a Week	459 (48.52%)	187 (40.74%)	
Once in 15 Days	186 (19.66%)	22 (11.82%)	
Once in Month	72 (7.61)	7 (9.72%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.9 Association of stress level with prediabetes

4.3.9.1 Association of stress level with prediabetes (12-17 years age group)

Among subjects of 12-17 years age group, majority of subjects categorized into no stress level (84.87%) category following less level of stress (11.84%) and medium level of stress (3.29%) category. None of the participants found to have high level of stress in current study.

In present study in the same age group, occurrence of prediabetes in subjects with medium level of stress was the highest (13.33%), followed by less level of stress (9.25%) and lowest among subject with no stress (4.13%). Here we found level of stress was significant with incident prediabetes. (Table 4.40)

Table 4.40 Prevalence of prediabetes among subjects with different level of stress (12-17 years age group)

12 to 17 Years Age Group			P value
Stress Level	**Total (N = 456)**	**Prediabetes subjects**	
No stress	387 (84.87%)	16 (4.13%)	< 0.001*
Less level of stress	54 (11.84%)	5 (9.25%)	
Medium level of stress	15 (3.29%)	2 (13.33 %)	
High level of stress	0 (0.00%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.9.2 Association of stress level with prediabetes (18-35 years age group)

The current study showed the highest percentage prevalence of no stress level (74.15%) category following less level of stress (19.21%) and medium level of stress (6.63%) category. None of the participants found to have high level of stress in the age group of 18-35 years.

In the same age group, we found 26.30% subjects with prediabetes were reported to have no stress. The difference in the level of stress from less stress to medium level of stress was not mammoth; less level of stress 35.05% and medium level of stress 38.80%. It could be established by this observation that the level of stress was significantly associated with risk of developing prediabetes. (Table 4.41)

Table 4.41 Prevalence of prediabetes among subjects with different level of stress (18-35 years age group)

18 to 35 Years Age Group			P value
Stress Level	**Total (N = 1,010)**	**Prediabetes subjects**	
No stress	749 (74.15%)	197 (26.30%)	< 0.001*
Less level of stress	194 (19.21%)	68 (35.05%)	
Medium level of stress	67 (6.63%)	26 (38.80%)	
High level of stress	0 (0.00%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.9.3 Association of stress level with prediabetes (36-55 years age group)

In the age group of 36-55 years, majority of subjects were identified in no stress category (57.82%) following less level of stress (38.90%) and medium level of stress (3.28%) category. None of the participants found to have high level of stress in current study.

In present study, high prevalence of prediabetes was found in subjects who had medium level of stress (45.16%). We further noted that risk of prevalence of prediabetes was declined as stress level decreased. Prevalence of prediabetes was 39.13% and 28.51% respectively among subjects with less level of stress and no stress at all. This data revealed that stress level has significant association with incident prediabetes. (Table 4.42)

Table 4.42 Prevalence of prediabetes among subjects with different level of stress (36-55 years age group)

36 to 55 Years Age Group			P value
Stress Level	**Total (N = 946)**	**Prediabetes subjects**	
No stress	547 (57.82%)	156 (28.51%)	< 0.001*
Less level of stress	368 (38.90%)	144 (39.13%)	
Medium level of stress	31 (3.28%)	14 (45.16%)	
High level of stress	0 (0.00%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

One of the paramount reasons of greater number of the subjects under no stress level category could be we have followed questionnaire of western countries to evaluate stress level, which mainly focused on family issues and isolation from the society. Such predicaments are less common in India compared to western countries.

4.3.10 Association of socioeconomic status with prediabetes

4.3.10.1 Association of socioeconomic status with prediabetes (12-17 years age group)

In the age group of 12-17 years, the prevalence of upper, upper middle, lower middle, upper lower and lower socioeconomic status was 17.98%, 62.72%, 5.04%, 12.94% and 1.32%, respectively. In this age group we found the highest participants of upper middle class following upper, upper lower and lower middle class. We found lowest prevalence for lower socioeconomic class in current study in this age group.

The present study stated in the age group of 12-17 years, prevalence of prediabetes was the highest among upper socioeconomic class (10.47%). Whilst prevalence of prediabetes was virtually indistinguishable in upper middle (4.54%) and lower middle (4.34%)

socioeconomic class. Furthermore, none of the participants was reported to have prediabetes among upper lower and lower socioeconomic class. In this age group found significant association of socioeconomic class and risk of prediabetes. (Table 4.43)

Table 4.43 Prevalence of prediabetes among subjects with different socioeconomic class (12-17 years age group)

12 to 17 Years Age Group			P value
Socioeconomic Status	**Total (N = 456)**	**Prediabetes subjects**	
Upper	82 (17.98%)	9 (10.97%)	< 0.001*
Upper middle	286 (62.72%)	13 (4.54%)	
Lower middle	23 (5.04%)	1 (4.34%)	
Upper lower	59 (12.94%)	0 (0.00%)	
Lower	6 (1.32%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.10.2 Association of socioeconomic status with prediabetes (18-35 years age group)

In current study among the subjects of 18-35 years, we found highest prevalence of upper middle socioeconomic class (46.73%) following upper (34.26%), upper lower (11.29%), lower middle (4.85%) and lower (2.87%).

In the same age group, prevalence of prediabetes was found the highest (33.81%) among upper socioeconomic class subjects following upper middle (30.72%) and lower middle (28.57%) socioeconomic class. Whilst the prevalence among upper lower class prediabetics was lowest (13.15%). Prevalence of prediabetes was absent among lower socioeconomic class subjects. It can be re-established socioeconomic class had significant association with risk of developing prediabetes. (Table 4.44)

Table 4.44 Prevalence of prediabetes among subjects with different socioeconomic class (18-35 years age group)

18 to 35 Years Age Group			P value
Socioeconomic Status	**Total (N = 1,010)**	**Prediabetes subjects**	
Upper	346 (34.26%)	117 (33.81%)	< 0.001*
Upper middle	472 (46.73%)	145 (30.72%)	
Lower middle	49 (4.85%)	14 (28.57%)	
Upper lower	114 (11.29%)	15 (13.15%)	
Lower	29 (2.87%)	0 (0.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.10.3 Association of socioeconomic status with prediabetes (36-55 years age group)

In the age group of 36-55 years, highest prevalence of upper middle socioeconomic class (54.65%) was reported following upper (32.14%) and upper lower (8.14%). We noted prevalence of lower middle (2.96%) and lower (2.11%) socioeconomic class was nearly similar in this age group.

In current study, high prevalence of prediabetes was found in subjects belonged to upper (38.48%) and upper middle socioeconomic class (33.65%), followed by lower middle socioeconomic class (25%), upper lower (18.18%) and lowest in lower (10%) socioeconomic class subjects. In the age group of 36-55 years, we found socioeconomic status has significant association with prediabetes incident. (Table 4.45)

Table 4.45 Prevalence of prediabetes among subjects with different socioeconomic class (36-55 years age group)

36 to 55 Years Age Group			P value
Socioeconomic Status	**Total (N = 946)**	**Prediabetes subjects**	
Upper	304 (32.14%)	117 (38.48 %)	< 0.001*
Upper middle	517 (54.65%)	174 (33.65%)	
Lower middle	28 (2.96%)	7 (25.00%)	
Upper lower	77 (8.14%)	14 (18.18 %)	
Lower	20 (2.11%)	2 (10 %)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.3.11 Association of lipid abnormalities with prediabetes

4.3.11.1 Association of lipid abnormalities with prediabetes (12-17 years age group)

None of the subjects in the age group of 12-17 years found to have any of the lipid abnormalities.

However, the current study shown elevated mean values of cholesterol level and triglyceride level among prediabetics than non-prediabetics; 177.9±3.503 mg/dL mean cholesterol level and 128.5±2.523 mg/dL mean triglyceride level among prediabetics. Whereas, mean level of HDL-C among prediabetic was 47.7±0.897 mg/dL which was reported higher among non-prediabetics (50.3±0.250 mg/dL). This findings implies that though lipid abnormalities were absent in early age, higher mean values of cholesterol and triglycerides as well as lower level of HDL-C were found significantly associated with prediabetes. (Table 4.46)

Table 4.46 Mean lipid profile level among subjects with prediabetes (12-17 years age group)

12 to 17 Years Age Group			P value
Mean Lipid Levels (mg/dL)	**Prediabetes subjects (N = 23)**	**Normal subjects (N = 433)**	
Mean cholesterol level ± SEM	177.9±3.503	146.9±0.831	< 0.0001*
Mean triglyceride level ± SEM	128.5±2.523	119.1±0.519	< 0.0001*
Mean HDL-C level ± SEM	47.7±0.897	50.3±0.250	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

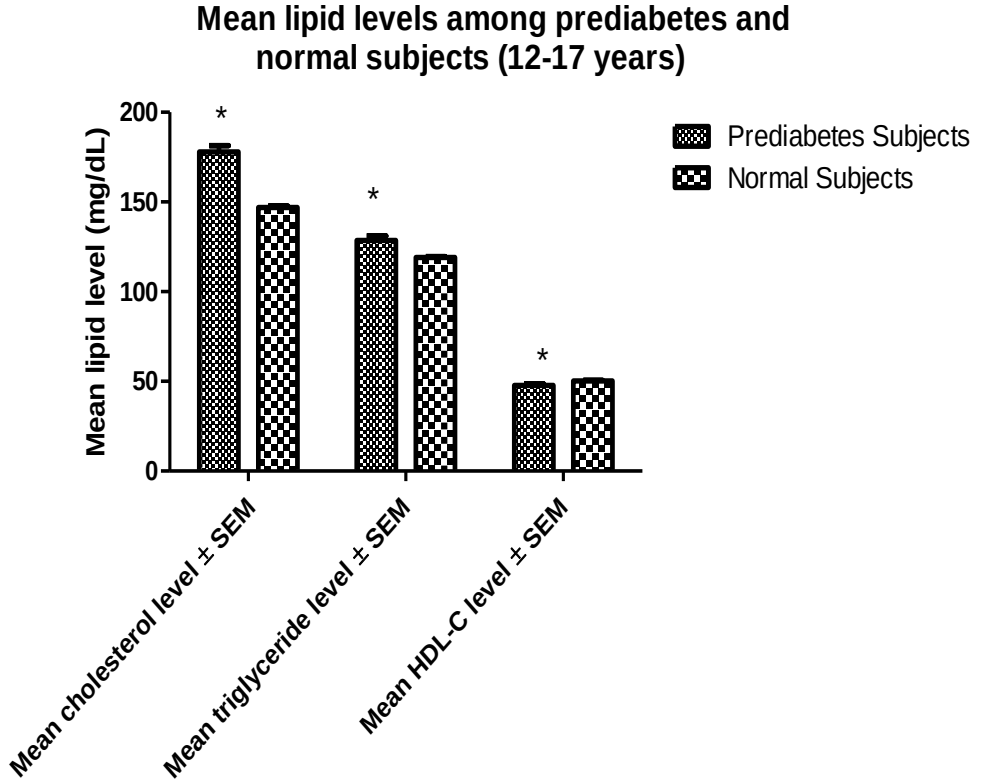

Figure 4.14 Mean lipid profile level among subjects with prediabetes (12-17 years age group)

4.3.11.2 Association of lipid abnormalities with prediabetes (18-35 years age group)

In current study, hypercholesterolemia, hypertriglyceridemia, low HDL-C level and dyslipidemia among subjects of 18-35 years was 13.56%, 16.14%, 27.13% and 12.97%, respectively.

Lipid abnormalities such as hypercholesterolemia, hypertriglyceridemia, low HDL-C level and dyslipidemia in prediabetes subjects was 34.02%, 29.89%, 44.32% and 27.83% respectively. The percentage prevalence of all lipid abnormalities was found highest among prediabetics than non-prediabetics. Hypercholesterolemia and hypertriglyceridemia noted to have significant association with prediabetes demonstrating risk of elevation of total cholesterol and triglycerides increases among prediabetes participants. (Table 4.47, Table 4.48, Table 4.49, Table 4.50)

The mean cholesterol, mean triglycerides and mean HDL-C level among prediabetes subjects was 191.1±1.512 mg/dL, 147.1±1.155 mg/dL and 41.8±0.592 mg/dL, respectively. We noted that the mean value of these lipid profiles were higher among prediabetics compared to non-prediabetics. The association of mean lipid levels was found significantly associated with prediabetes in 18-35 years age group for all lipid profile. (Table 4.51)

Table 4.47 Prevalence of hypercholesterolemia among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group				**P value**
Lipid Abnormality	**Total (N = 1,010)**	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	
Hypercholesterolemia	164 (16.23%)	99 (34.02%)	65 (9.04%)	0.008*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.48 Prevalence of hypertriglyceridemia among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group				**P value**
Lipid Abnormality	**Total (N = 1,010)**	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	
Hypertriglyceridemia	163 (16.14%)	87 (29.89%)	100 (13.90%)	0.004*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.49 Prevalence of low HDL-C among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group				P value
Lipid Abnormality	**Total (N = 1,010)**	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	
Low HDL-C	274 (27.13%)	129 (44.32%)	145 (20.16%)	0.934

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.50 Prevalence of dyslipidemia among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group				P value
Lipid Abnormality	**Total (N = 1,010)**	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	
Dyslipidemia	131 (12.97%)	81 (27.83%)	72 (10.01%)	0.467

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.51 Mean lipid levels among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group			
Mean Lipid Levels (mg/dL)	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	**P value**
Mean cholesterol level ± SEM	191.1±1.512	171.1±0.742	< 0.0001*
Mean triglyceride level ± SEM	147.1±1.155	128.9±0.552	< 0.0001*
Mean HDL-C level ± SEM	41.8±0.592	44.8±0.306	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

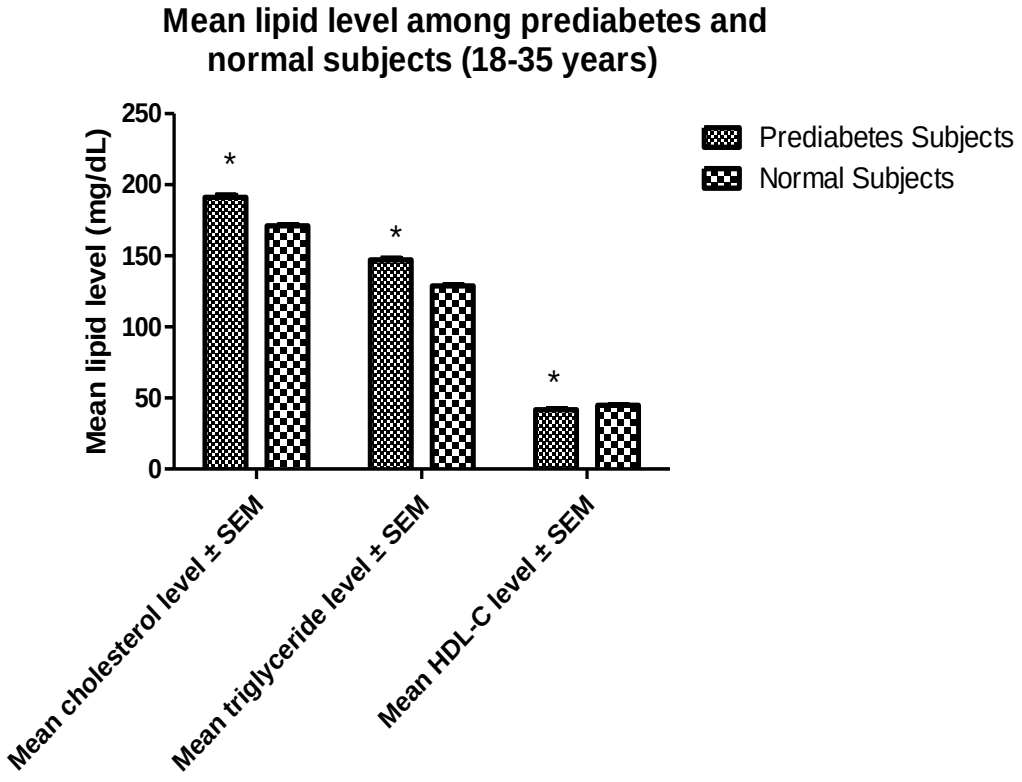

Figure 4.15 Mean lipid profile level among subjects with prediabetes (18-35 years age group)

4.3.11.3 Association of lipid abnormalities with prediabetes (36-55 years age group)

Among subjects of 36-55 years, we noted 29.28%, 32.98%, 40.80% and 26.64% prevalence of hypercholesterolemia, hypertriglyceridemia, low HDL-C and dyslipidemia, respectively.

In present study, prevalence of hypercholesterolemia among subjects with prediabetes was 39.17%, hypertriglyceridemia 36.6%, low HDL-C 45.22% and dyslipidemia was 35.98%. The percentage prevalence of lipid abnormalities in the age group of 36-55 years was found lower among normal subjects compared to prediabetics. Hypertriglyceridemia and low level of HDL-C was found significantly associated with prediabetes whilst hypercholesterolemia and dyslipidemia were insignificant with prediabetes. (Table 4.52, Table 4.53, Table 4.54, Table 4.55).

In current study the mean cholesterol level was 203.3±1.479 mg/dL, mean triglycerides was 155.6±1.665 mg/dL and mean HDL-C level was 37.9±0.547 mg/dL among prediabetics. These mean value of the lipid profiles were higher among prediabetics compared to non-prediabetes subjects. The association of mean lipid levels was found significantly associated with prediabetes in 36-55 years age group. (Table 4.56)

Table 4.52 Prevalence of hypercholesterolemia among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group				
Lipid Abnormality	**Total (N = 946)**	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Hypercholesterolemia	277 (29.28%)	123 (39.17%)	154 (24.36%)	0.063

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.53 Prevalence of hypertriglyceridemia among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group				
Lipid Abnormality	**Total (N = 946)**	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Hypertriglyceridemia	312 (32.98%)	115 (36.6%)	197 (31.17%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.54 Prevalence of low HDL-C among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group				
Lipid Abnormality	**Total (N = 946)**	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Low HDL-C	386 (40.80%)	142 (45.22%)	244 (38.60%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.55 Prevalence of dyslipidemia among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group				
Lipid Abnormality	**Total (N = 946)**	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Dyslipidemia	252 (26.64%)	113 (35.98%)	139 (21.99%)	0.101

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.56 Mean lipid level among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group			
Mean Lipid Level (mg/dL)	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Mean cholesterol level ± SEM	203.3±1.479	179.6±0.609	< 0.0001*
Mean triglyceride level ± SEM	155.6±1.665	133.2±0.728	< 0.0001*
Mean HDL-C level ± SEM	37.9±0.547	43.1±0.342	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

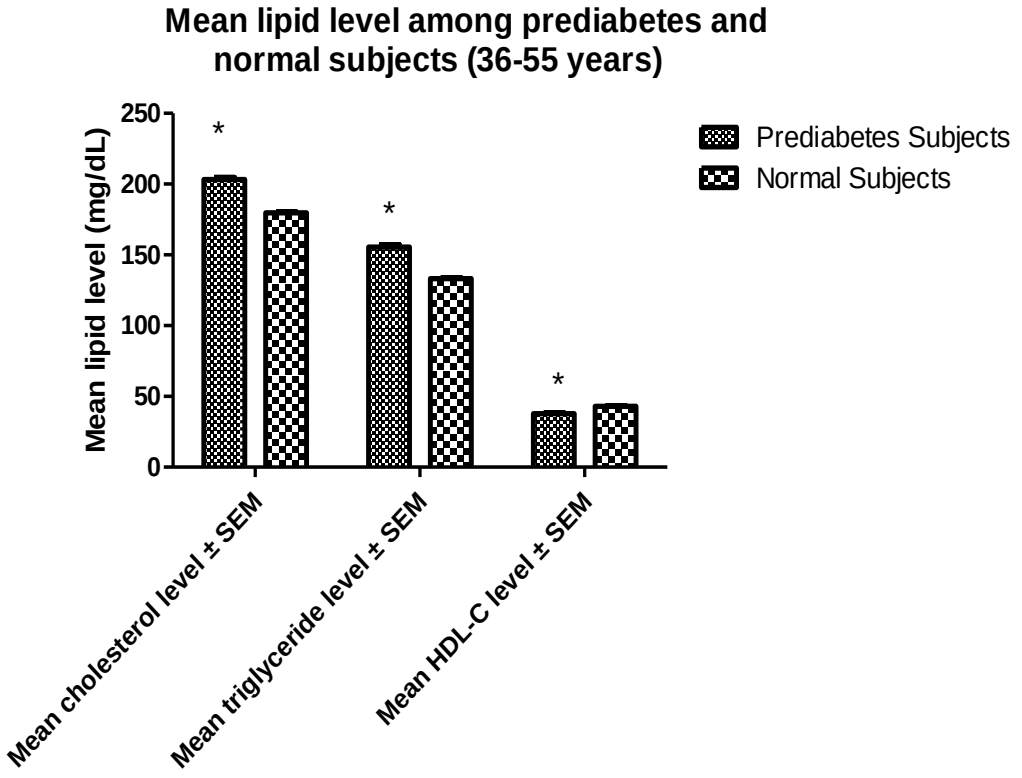

Figure 4.16 Mean lipid profile level among subjects with prediabetes (36-55 years age group)

4.3.12 Association of vitamin D deficiency with prediabetes

4.3.12.1 Association of vitamin D deficiency with prediabetes (12-17 years age group)

In present study, we found 55.26% prevalence of vitamin D deficiency among subjects of 12-17 years of age group.

In the same age group, 86.95% subjects with prediabetes had vitamin D deficiency whereas 53.57% non-prediabetes subjects had vitamin D deficiency. It implies that prevalence of vitamin D deficiency was higher among prediabetics than non-prediabetics. Further vitamin D deficiency was found significantly associated with prediabetes explaining risk of vitamin D deficiency increases among prediabetes participants than normal subjects. (Table 4.57)

The mean vitamin D level was 18.7±0.563 ng/dL among subjects with prediabetes whereas among non-prediabetics the mean vitamin D level was higher (21.8±0.235 ng/dL). By

referring this point it was justified that, vitamin D deficiency was significantly associated with prediabetes. (Table 4.58)

Table 4.57 Prevalence of vitamin-D deficiency among prediabetes and normal subjects (12-17 Years age group)

12 to 17 Years Age Group				
	Total (N = 456)	**Prediabetes subjects (N = 23)**	**Normal subjects (N = 433)**	**P value**
Vitamin D Deficiency	252 (55.26%)	20 (86.95%)	232 (53.57%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.58 Mean level of vitamin D among prediabetes and normal subjects (12-17 years age group)

12 to 17 Years Age Group			
	Prediabetes subjects (N = 23)	**Normal subjects (N = 433)**	**P value**
Mean Vitamin D level ± SEM (ng/dL)	18.7±0.563	21.8±0.235	0.002*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

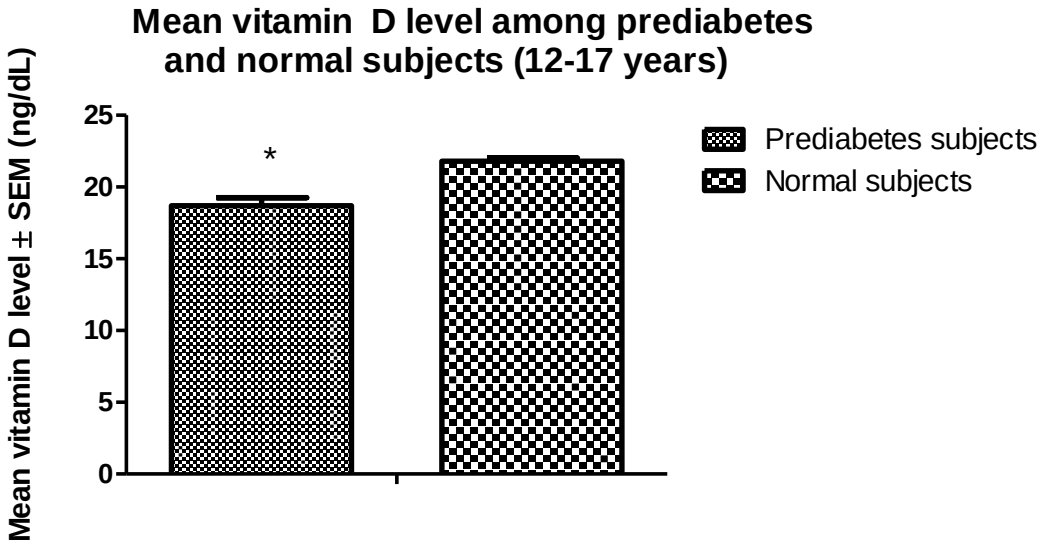

Figure 4.17 Mean level of vitamin D among prediabetes and normal subjects (12-17 years age group)

4.3.12.2 Association of vitamin D deficiency with prediabetes (18-35 years age group)

The prevalence of vitamin D deficiency among subjects of 18-35 years was 67.62% in present study.

The current study revealed that vitamin D deficiency among prediabetes subjects was 88.65% which was higher compared to non-prediabetes subjects (59.10%) in 18-35 years age group. Vitamin D deficiency was found significantly associated with prediabetes. (Table 4.59)

The prediabetic participants had lower mean vitamin D level (16.9±0.126 ng/dL) than non-prediabetic participants (21.6±0.183 ng/dL). Hence it can be inferred that vitamin D deficiency was significantly associated with incident prediabetes. (Table 4.60)

Table 4.59 Prevalence of vitamin-D deficiency among prediabetes and normal subjects (18-35 Years age group)

18 to 35 Years Age Group				
	Total (N = 1,010)	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	**P value**
Vitamin D Deficiency	683 (67.62%)	258 (88.65%)	425 (59.10%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.60 Mean level of vitamin D among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group			
	Prediabetes subjects (N = 291)	**Normal subjects (N = 719)**	**P value**
Mean Vitamin-D level ± SEM (ng/dL)	16.9±0.126	21.6±0.183	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

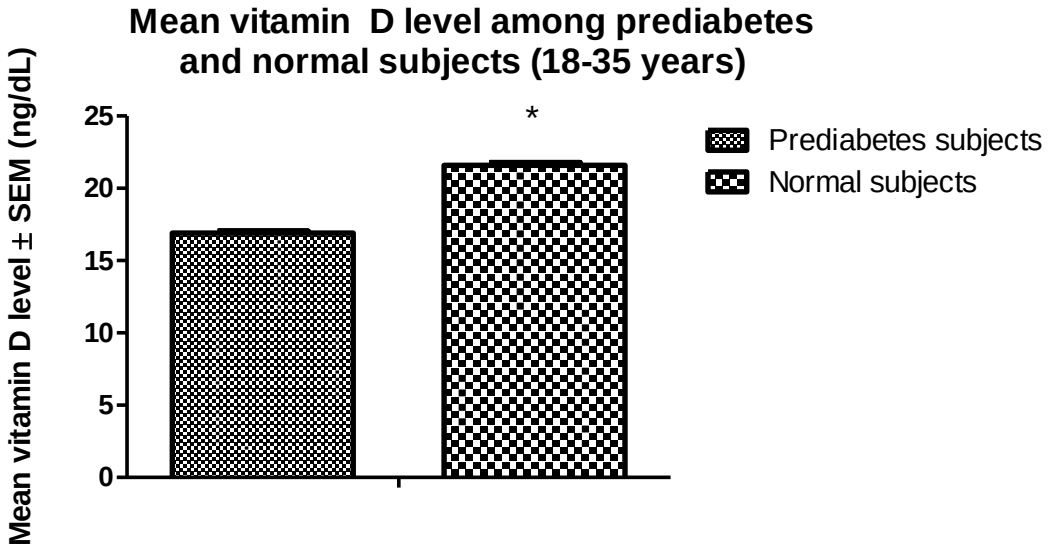

Figure 4.18 Mean level of vitamin D among prediabetes and normal subjects (18-35 years age group)

4.3.12.3 Association of vitamin D deficiency with prediabetes (36-55 years age group)

In current study the prevalence of vitamin D deficiency among subjects of 36-55 years old was 73.15%.

We found higher prevalence of vitamin D deficiency among prediabetes subjects (84.39%) than normal subjects (67.56%) among 36-55 years age group. We further found vitamin D deficiency was significantly associated with prediabetes in this age group. (Table 4.61)

The mean vitamin D level was higher among non-prediabetic (20.8±0.175 ng/dL) compared to prediabetics (16.9±0.186 ng/dL). These findings suggested that low mean level of vitamin D was considered as risk for developing prediabetes among 36-55 years age group and the association was found significant. (Table 4.62)

Table 4.61 Prevalence of vitamin-D deficiency among prediabetes and normal subjects (36-55 Years age group)

36 to 55 Years Age Group				
	Total (N = 946)	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Vitamin D Deficiency	692 (73.15%)	265 (84.39%)	427 (67.56%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.62 Mean level of vitamin D among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group			
	Prediabetes subjects (N = 314)	**Normal subjects (N = 632)**	**P value**
Mean Vitamin-D level ± SEM (ng/dL)	16.9±0.186	20.8±0.175	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

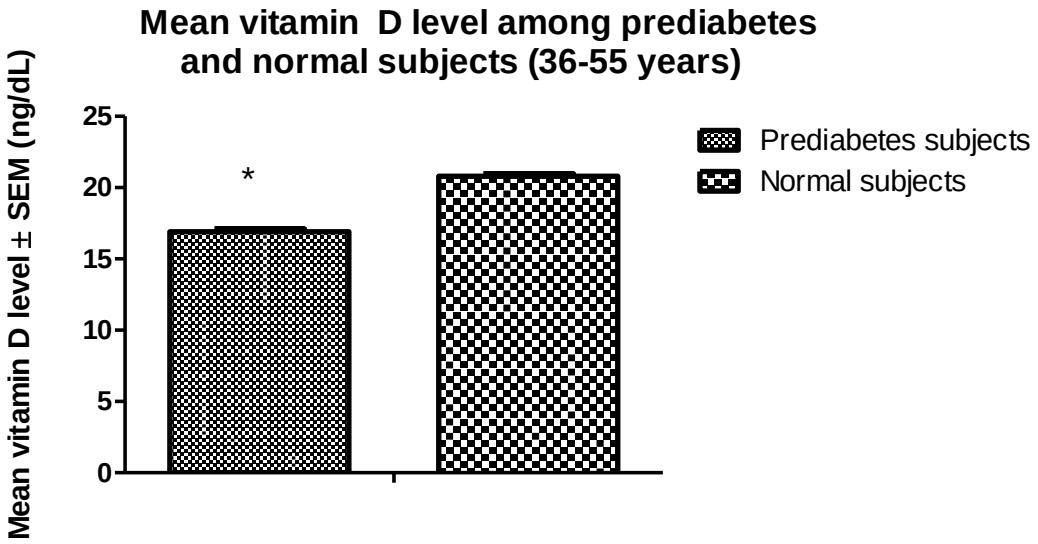

Figure 4.19 Mean level of vitamin D among prediabetes and normal subjects (36-55 years age group)

4.3.13 Association of hyperinsulinemia with prediabetes

4.3.13.1 Association of hyperinsulinemia with prediabetes (12-17 years age group)

The present study did not find any subjects of 12-17 years of age with abnormally higher level of insulin.

Though, the prediabetic participants had higher mean insulin level (17.4±0.605 mIU/L) than non-prediabetic participants (14.2±0.125 mIU/L). Hence it can be inferred that higher level of insulin was significantly associated with incident prediabetes. (Table 4.63)

Table 4.63 Mean insulin level among prediabetes and normal subjects (12-17 years age group)

12 to 17 Years Age Group			
	Prediabetes subjects (N = 23)	**Normal subjects (N = 433)**	**P value**
Mean Insulin level ± SEM (mIU/L)	17.4±0.605	14.2±0.125	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

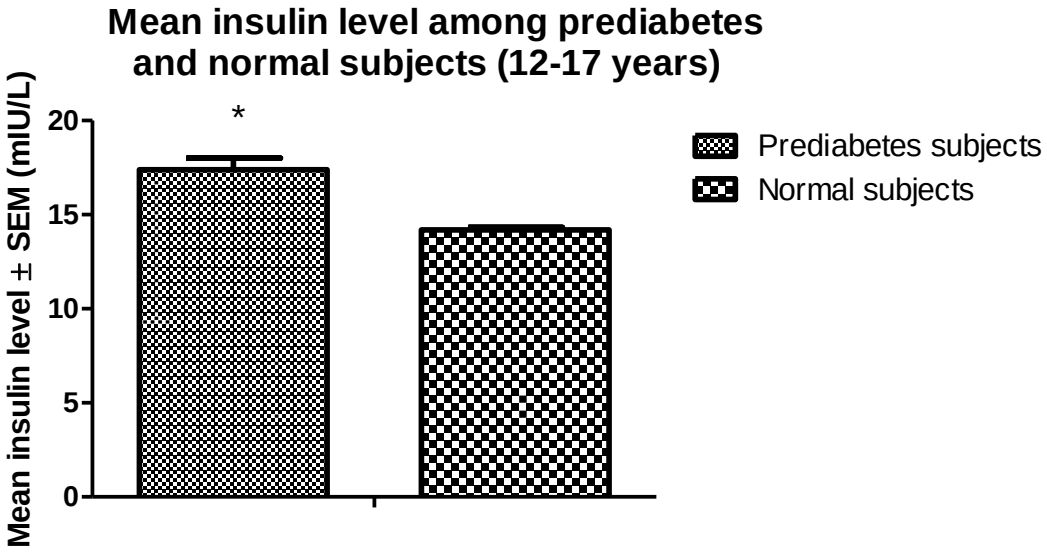

Figure 4.20 Mean insulin level among prediabetes and normal subjects (12-17 years age group)

4.3.13.2 Association of hyperinsulinemia with prediabetes (18-35 years age group)

In current study prevalence of hyperinsulinemia among subjects of 18-35 years age group was 16.73%.

Hyperinsulinemia among the subjects of same age group with prediabetes was 34.02% while in non-prediabetic subjects was 9.73%. Hyperinsulinemia was found significantly associated with prediabetes in this group. This finding implies higher risk of hyperinsulinemia among prediabetics than non-prediabetics. (Table 4.64)

The mean insulin level among prediabetes (23.2±0.305 mIU/L) was substantially higher among participants with prediabetes than prediabetes free participants (19.4±0.160 mIU/L). The mean level of insulin was found significantly associated with prediabetes. (Table 4.65)

Table 4.64 Prevalence of hyperinsulinemia in prediabetes and normal subjects (18-35 Years age group)

18 to 35 Years Age Group				
	Total (N = 1,010)	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	**P value**
Hyperinsulinemia	169 (16.73%)	99 (34.02%)	70 (9.73%)	0.026*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.65 Mean insulin level among subjects with prediabetes (18-35 years age group)

18 to 35 Years Age Group			
	Prediabetes subjects (N = 291)	**Normal subjects (N = 719)**	**P value**
Mean Insulin level ± SEM (mIU/L)	23.2±0.305	19.4±0.160	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

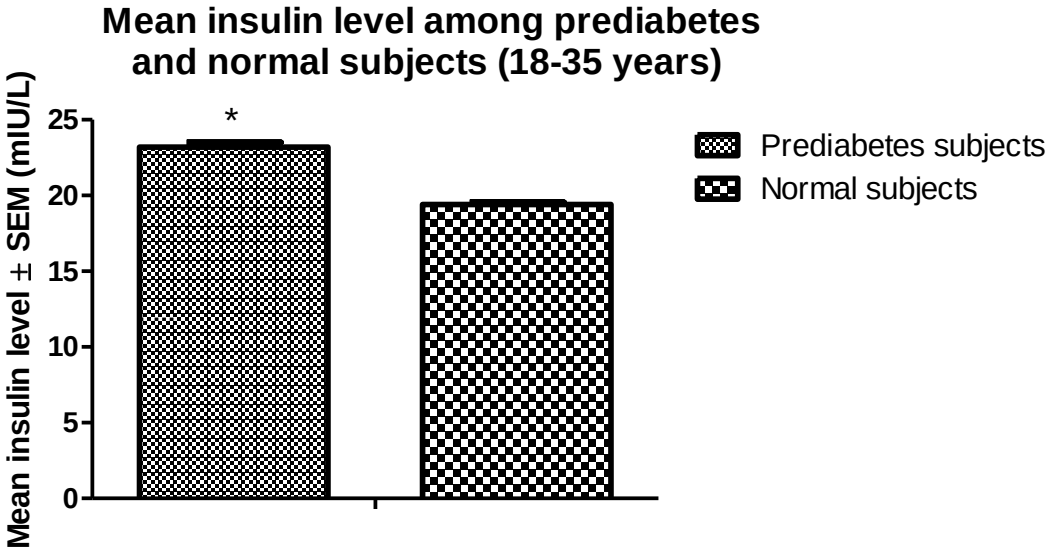

Figure 4.21 Mean insulin level among prediabetes and normal subjects (18-35 years age group)

4.3.13.3 Association of hyperinsulinemia with prediabetes (36-55 years age group)

The current study showed 31.18% prevalence of hyperinsulinemia among subjects of 36-55 years old.

In the same age group, we found greater prevalence of hyperinsulinemia among prediabetic subjects (49.36%) than subjects without prediabetes (22.15%). In this age group hyperinsulinemia was reported insignificant with prediabetes. (Table 4.66)

The mean insulin level among prediabetes (25.1±0.316 mIU/L) was greater among prediabetes subjects than subjects without prediabetes (20.2±0.151 mIU/L). The mean level of insulin was found significantly associated with prediabetes. (Table 4.67)

Table 4.66 Prevalence of hyperinsulinemia in prediabetes and normal subjects (36-55 Years age group)

36 to 55 Years Age Group				
	Total (N = 946)	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Hyperinsulinemia	295 (31.18%)	155 (49.36 %)	140 (22.15%)	0.763

Analysed by One-way chi-square test for goodness of fit.

Table 4.67 Mean insulin level among subjects with prediabetes (36-55 years age group)

36 to 55 Years Age Group			
	Prediabetes subjects (N = 314)	**Normal subjects (N = 632)**	**P value**
Mean Insulin level ± SEM (mIU/L)	25.1±0.316	20.2±0.151	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

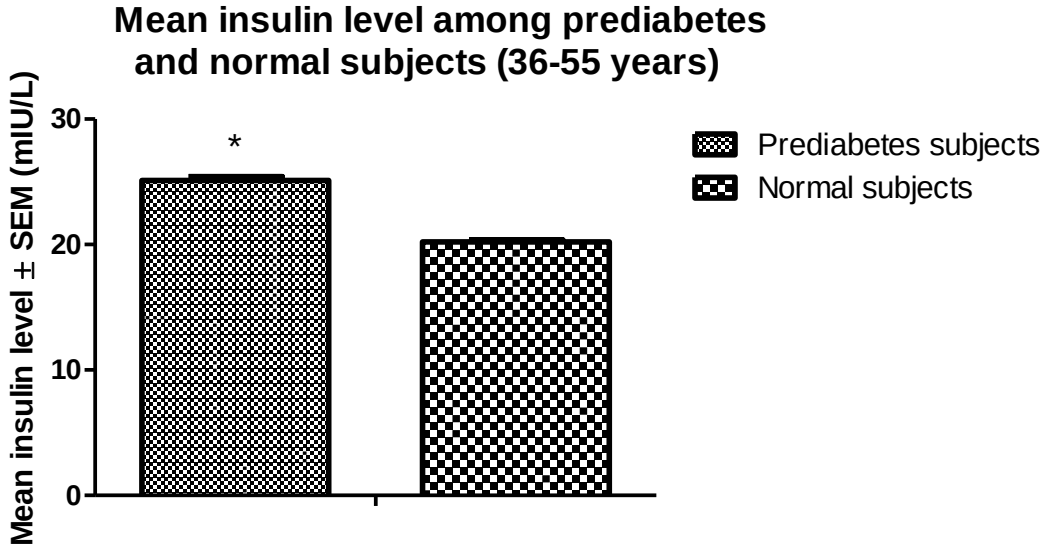

Figure 4.22 Mean insulin level among prediabetes and normal subjects (36-55 years age group)

4.3.14 Association of elevated level of C-reactive protein with prediabetes

4.3.14.1 Association of elevated level of C-reactive protein with prediabetes (12-17 years age group)

The present study reported 0.44% prevalence of elevated C-reactive protein among 456 subjects of age group 12-17 years.

In the age group of 12-17 years, out of 23 subjects with prediabetes, 2 subjects had elevated C-reactive protein (8.69%). Whilst none of the non-prediabetes subjects were found to have elevated C-reactive protein. Elevated C-reactive protein found to have no significant association with prediabetes at early age. (Table 5.68)

The mean C-reactive protein level in prediabetes subjects was 3.35±0.041 mg/L, which was found to be higher than non-prediabetics (0.84±0.019 mg/L). We further found elevated level of C-reactive protein were statistically associated with incident prediabetes. (Table 4.69)

Table 4.68 Prevalence of elevated C-reactive protein in prediabetes and normal subjects (12-17 Years age group)

12 to 17 Years Age Group				
	Total (N = 456)	**Prediabetes subjects (N = 23)**	**Normal subjects (N = 433)**	**P value**
Elevated C-reactive protein	2 (0.44%)	2 (8.69%)	0 (0.00%)	0.157

Analysed by One-way chi-square test for goodness of fit.

Table 4.69 Mean level of C-reactive protein among prediabetes and normal (12-17 years age group)

12 to 17 Years Age Group			
	Prediabetes subjects (N = 23)	**Normal subjects (N = 433)**	**P value**
Mean C-reactive protein level ± SEM (mg/L)	3.35±0.041	0.84±0.019	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

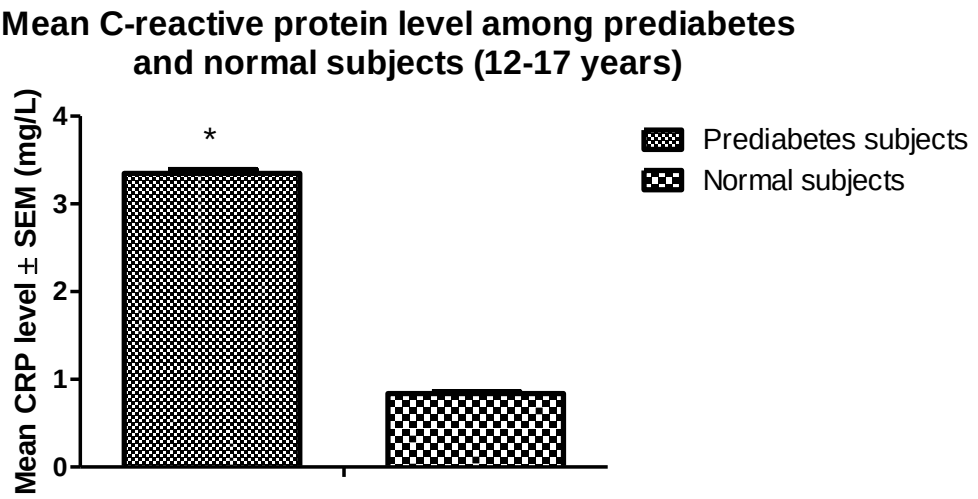

Figure 4.23 Mean level of C-reactive protein among prediabetes and normal (12-17 years age group)

4.3.14.2 Association of elevated level of C-reactive protein with prediabetes (18-35 years age group)

In present study we found 10.50% subjects of age group 18-35 years had elevated C-reactive protein.

Out of 291 prediabetes subjects, 28.86% subjects reported to have elevated C-reactive protein. The percentage prevalence of elevated C-reactive protein was higher among prediabetics than non-prediabetics (3.05%). We further found elevated C-reactive protein have significant association with prediabetes in this age group. (Table 4.70)

We found higher mean level of C-reactive protein in participants of prediabetes (2.12±0.059 mg/L) than prediabetes free participants (1.35±0.022 mg/L). The mean level of C-reactive protein was found significantly associated with incident prediabetes. (Table 4.71)

Table 4.70 Prevalence of elevated C-reactive protein in prediabetes and normal subjects (18-35 Years age group)

18 to 35 Years Age Group				
	Total (N = 1,010)	**Prediabetes subjects (N = 291)**	**Normal subjects (N = 719)**	**P value**
Elevated C-reactive protein	106 (10.50%)	84 (28.86%)	22 (3.05%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.71 Mean level of C-reactive protein among prediabetes and normal subjects (18-35 years age group)

18 to 35 Years Age Group			
	Prediabetes subjects (N = 291)	**Normal subjects (N = 719)**	**P value**
Mean C-reactive protein level ± SEM (mg/L)	2.12±0.059	1.35±0.022	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05

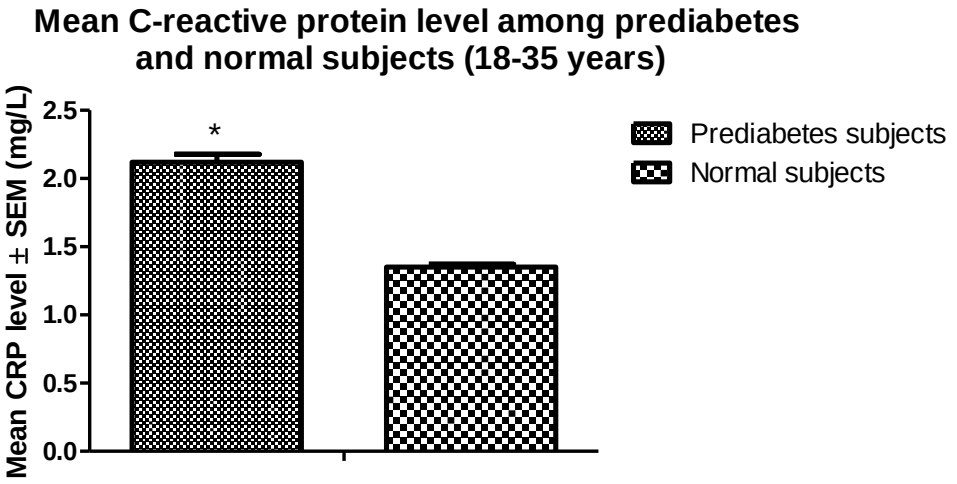

Figure 4.24 Mean level of C-reactive protein among prediabetes and normal (18-35 years age group)

4.3.14.3 Association of elevated level of C-reactive protein with prediabetes (36-55 years age group)

In the age group of 36-55 years, we noted out of 946 subjects 21.25% subjects had elevated C-reactive protein.

In the same age group, 41.71% subjects with prediabetes found to have elevated C-reactive protein which was remarkably higher than non-prediabetics (11.07%). Elevated C-reactive protein reported to have significant association with prediabetes in this age group showing increased risk of elevation of C-reactive protein and thus role of inflammation in prediabetes. (Table 4.72)

The mean C-reactive protein level was 2.55±0.056 mg/L among prediabetics following 1.7±0.036 mg/L in non-prediabetics. Therefore it can be re-established that mean level of C-reactive protein was significantly associated with prediabetes in this age group. (Table 4.73)

Table 4.72 Elevated C-reactive protein in prediabetes and normal subjects (36-55 Years age group)

36 to 55 Years Age Group				
	Total (N = 946)	**Prediabetes subjects (N = 314)**	**Normal subjects (N = 632)**	**P value**
Elevated C-reactive protein	201 (21.25%)	131 (41.71%)	70 (11.07%)	< 0.001*

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.73 Mean level of C-reactive protein among prediabetes and normal subjects (36-55 years age group)

36 to 55 Years Age Group			
	Prediabetes subjects (N = 314)	**Normal subjects (N = 632)**	**P value**
Mean C-reactive protein level ± SEM (mg/L)	2.55±0.056	1.7±0.036	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

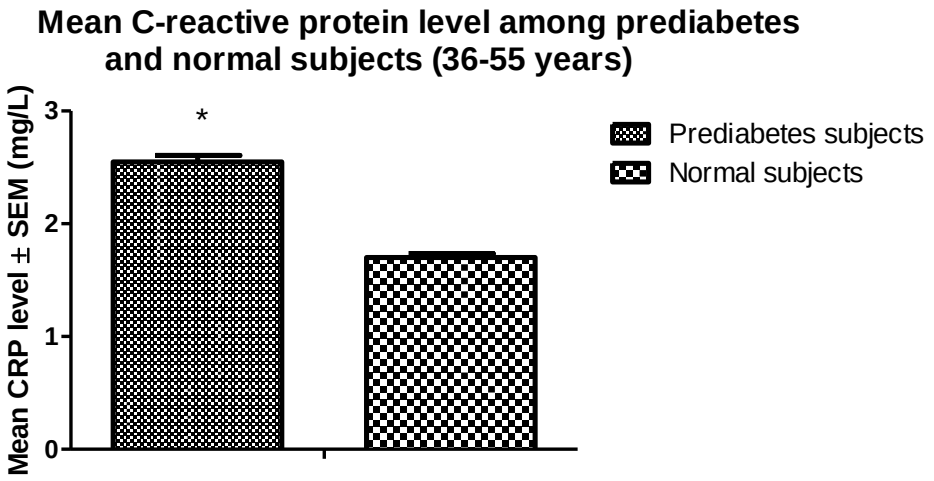

Figure 4.25 Mean level of C-reactive protein among prediabetes and normal (36-55 years age group)

4.4 Body mass index (BMI) and its associated condition

4.4.1 Association of pre-hypertension with BMI

4.4.1.1 Association of pre-hypertension with BMI (12-17 years age group)

In present study, prevalence of pre-hypertension among 78 underweight subjects was 5.12%, which was found to be lowest among all other group. Out of 279 healthy weight subjects, we found 11.82% prevalence of pre-hypertension. While prevalence of pre-hypertension among overweight and obese subjects was found 17.56% and 24.00% respectively. This study implies that the risk of pre-hypertension was increased as BMI increases among subjects of 12 – 17 years. Furthermore it was noted that pre-hypertension had significant association with BMI. (Table 4.74).

Table 4.74 Prevalence of pre-hypertension among subjects with different BMI (12-17 Years age group)

12 to 17 Years Age Group			P value
BMI Classification	**Total (N = 456)**	**Pre-hypertensive subjects**	
Underweight	78	4 (5.12%)	< 0.001*
Healthy Weight	279	33 (11.82%)	
Overweight	74	13 (17.56%)	
Obese	25	6 (24.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.1.2 Association of pre-hypertension with BMI (18-35 years age group)

In the age group of 18-35 years, the prevalence of pre-hypertension among 69 underweight and 702 healthy weight subjects was 15.94% and 26.53% respectively. Whereas, the prevalence of pre-hypertension among overweight subjects was 33.15% and obese subjects was 40.38%. We noted increasing prevalence of pre-hypertension among subjects as BMI increased and association was found significant. (Table 4.75)

Table 4.75 Prevalence of pre-hypertension among subjects with different BMI (18-35 Years age group)

18 Year to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Pre-hypertensive subjects**	
Underweight	69	11 (15.94%)	< 0.001*
Healthy Weight	702	185 (26.53%)	
Overweight	187	62 (33.15%)	
Obese	52	21 (40.38%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.1.3 Association of pre-hypertension with BMI (36-55 years age group)

In the age group of 36-55 years, we found 20% of pre-hypertension among underweight subjects, 27.24% among healthy weight subjects, 32.66% among overweight subjects and highest prevalence of pre-hypertension 45.09% among obese subjects. Overweight and obese subjects found to have high prevalence of pre-hypertension than other two in current study. We further found significant association between pre-hypertension and BMI in this age group. (Table 4.76)

Table 4.76 Prevalence of pre-hypertension among subjects with different BMI (36-55 Years age group)

36 Year to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Pre-hypertensive subjects**	
Underweight	45	9 (20.00%)	< 0.001*
Healthy Weight	602	164 (27.24%)	
Overweight	248	81 (32.66%)	
Obese	51	23 (45.09%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.2 Association of hypertension with BMI

4.4.2.1 Association of hypertension with BMI (12-17 years age group)

Prevalence of hypertension among 12-17 years old underweight and healthy weight subjects was 1.28% and 4.65% respectively. While 9.45% overweight and 8% obese subjects were identified to have hypertension. A significant increase in prevalence of hypertension was found among subjects as BMI increased. Hypertension and BMI was noted to have a significant association in this age group. (Table 4.77)

Table 4.77 Prevalence of hypertension among subjects with different BMI (12-17 Years age group)

12 to 17 Years Age Group			P value
BMI Classification	**Total (N = 456)**	**Hypertensive subjects**	
Underweight	78	1 (1.28%)	< 0.001*
Healthy Weight	279	13 (4.65%)	
Overweight	74	7 (9.45%)	
Obese	25	2 (8.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.2.2 Association of hypertension with BMI (18-35 years age group)

In the age group of 18-35 years, we found 11.59% underweight subjects had hypertension. Whereas 23.64% and 23.52% healthy weight and overweight subjects, respectively were noted to have hypertension. Prevalence of hypertension among obese subjects was 25%. The association of hypertension and BMI was noted significant. (Table 4.78).

Table 4.78 Prevalence of hypertension among subjects with different BMI (18-35 Years age group)

18 Year to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Hypertensive subjects**	
Underweight	69	8 (11.59%)	< 0.001*
Healthy Weight	702	166 (23.64%)	

Overweight	187	44 (23.52%)	
Obese	52	13 (25.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.2.3 Association of hypertension with BMI (36-55 years age group)

In present study, in the age group of 36-55 years, the prevalence of hypertension among underweight subjects was found to be 13.33%. While significantly increasing prevalence of hypertension was found among subjects with healthy weight (28.40%), overweight (39.51%) and obese subjects (52.94%). We noted that, subjects with high BMI had greater chances of risk of development of hypertension. (Table 4.79)

Table 4.79 Prevalence of hypertension among subjects with different BMI (36-55 Years age group)

36 to 55 Years Age Group			**P value**
BMI Classification	**Total (N = 946)**	**Hypertensive subjects**	
Underweight	45	6 (13.33%)	< 0.001*
Healthy Weight	602	171 (28.40%)	
Overweight	248	98 (39.51%)	
Obese	51	27 (52.94%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.3 Mean blood pressure among study participants according to their BMI

4.4.3.1 Mean blood pressure among study participants according to their BMI (12-17 years age group)

In the age group of 12-17 years, mean SBP among underweight subjects was 111.6±1.155 mmHg, while mean DBP was 63.7±0.883 mmHg. Similarly among healthy weight subjects, mean SBP was 116.8±0.569 mmHg and mean DBP was 69.1±0.335 mmHg. Whereas among overweight and obese subjects mean SBP was found to be 122.9±0.918 mmHg and 126.3±1.340 mmHg, respectively. Mean DBP among overweight and obese subjects was 74.2±0.453 mmHg and 78.4±1.080 mmHg, respectively. We noted that overweight and obese subjects had higher mean SBP and DBP, compared to underweight and healthy weight subjects. BMI was found to have positive statistically significant correlation with both SBP and DBP. (Table 4.80)

Table 4.80 Mean blood pressure of the study participants according to BMI categories (12-17 years age group)

12 to 17 Years Age Group				
BMI Classification	**Mean Systolic Blood Pressure ± SEM mmHg**	**P value**	**Mean Diastolic Blood Pressure ± SEM mmHg**	**P value**
Underweight	111.6±1.155	< 0.0001*	63.7±0.883	< 0.0001*
Healthy Weight	116.8±0.569		69.1±0.335	
Overweight	122.9±0.918		74.2±0.453	
Obese	126.3±1.340		78.4±1.080	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

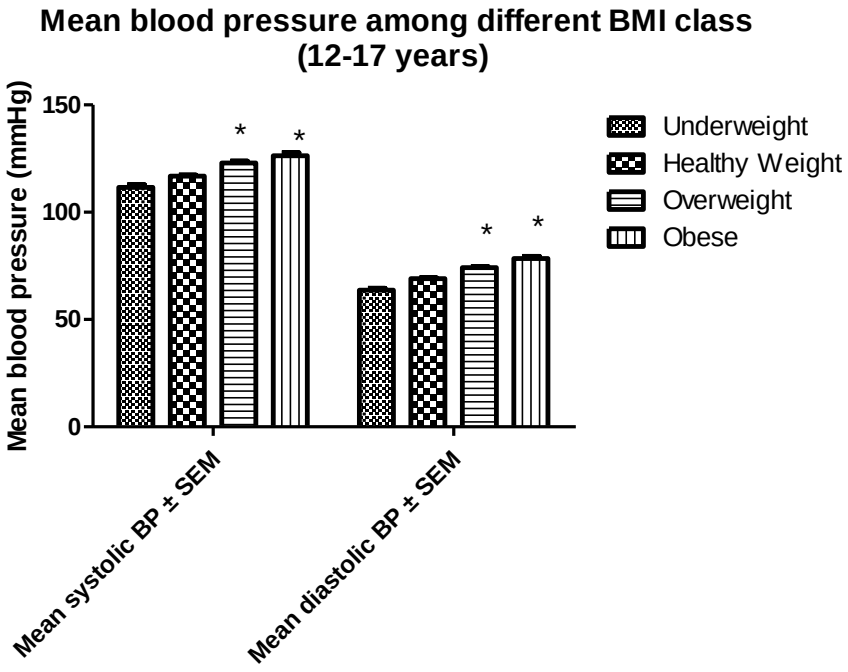

Figure 4.26 Mean blood pressure of the study participants according to BMI categories (12-17 years age group)

4.4.3.2 Mean blood pressure among study participants according to their BMI (18-35 years age group)

In the age group of 18-35 years, mean SBP among underweight subjects was 115.7±1.264 mmHg, while mean DBP was 69.7±0.783 mmHg. We found significant higher mean SBP among healthy weight, overweight and obese subjects which was respectively, 121.6±0.370 mmHg, 129.9±0.570 mmHg and 132.1±1.401 mmHg. Similarly mean DBP also found higher among healthy weight, overweight and obese subjects respectively (73.1±0.253 mmHg, 80.1±0.746 mmHg and 84.5±0.804 mmHg). We found mean SBP and DBP increased with increasing BMI. BMI was found to have positive statistically significant correlation with both SBP and DBP in current study. (Table 4.81)

Table 4.81 Mean blood pressure of the study participants according to BMI categories (18-35 years age group)

18 to 35 Years Age Group				
BMI Classification	**Mean Systolic Blood pressure ± SEM mmHg**	**P value**	**Mean Diastolic Blood pressure ± SEM mmHg**	**P value**
Underweight	115.7±1.264	< 0.0001*	69.7±0.783	< 0.0001*
Healthy Weight	121.6±0.370		73.1±0.253	
Overweight	129.9±0.570		80.1±0.746	
Obese	132.1±1.401		84.5±0.804	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

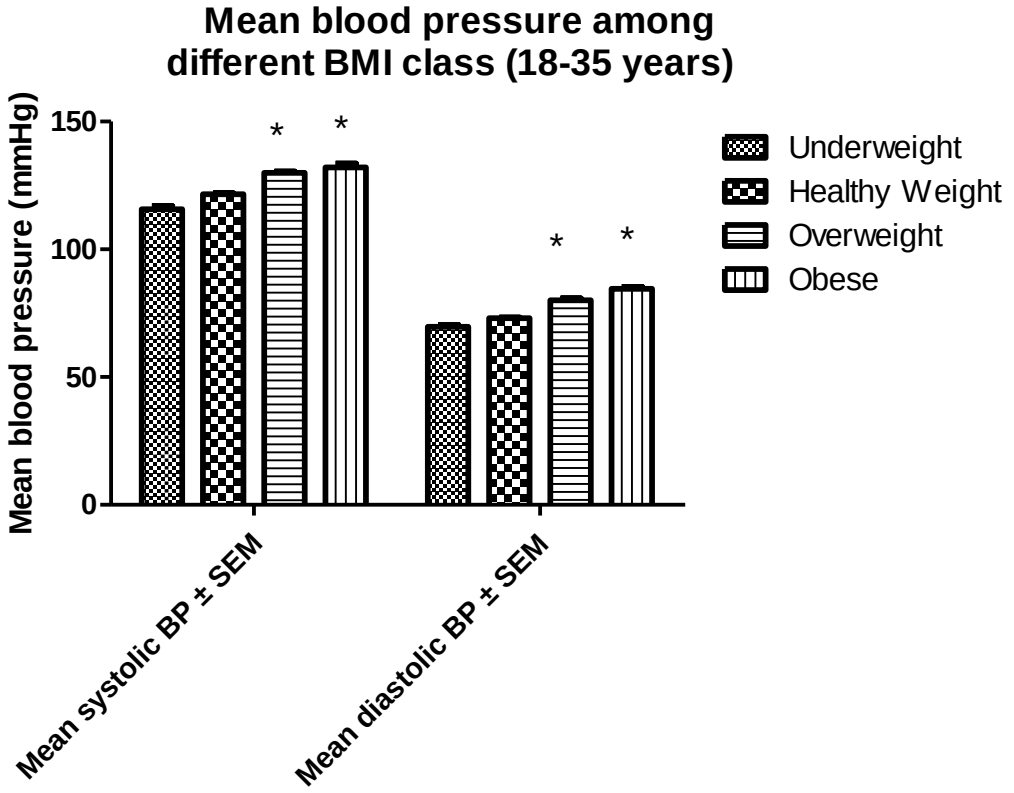

Figure 4.27 Mean blood pressure of the study participants according to BMI categories (18-35 years age group)

4.4.3.3 Mean blood pressure among study participants according to their BMI (36-55 years age group)

In current study, means SBP and DBP among underweight subject was 115.3±1.416 mmHg and 69.2±1.014 mmHg respectively. Mean SBP and DBP among healthy weight subject was found to have 123.9±0.444 mmHg and 72.3±0.375 mmHg respectively. Furthermore, among overweight subjects mean SBP was found 129.3±0.362 mmHg and mean DBP was found 81.6±0.267 mmHg. Among obese subjects mean SBP and DBP was found to be 134.5±0.672 mmHg and 84.1±0.938 mmHg, respectively. Both mean SBP and DBP were found to be significantly higher among subjects with higher BMI in present study. (Table 4.82)

Table 4.82 Mean blood pressure of the study participants according to BMI categories (36-55 years age group)

36 to 55 Years Age Group				
BMI Classification	**Mean Systolic Blood pressure ± SEM mmHg**	**P value**	**Mean Diastolic Blood pressure ± SEM mmHg**	**P value**
Underweight	115.3±1.416	< 0.0001*	69.2±1.014	< 0.0001*
Healthy Weight	123.9±0.444		72.3±0.375	
Overweight	129.3±0.362		81.6±0.267	
Obese	134.5±0.672		84.1±0.938	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

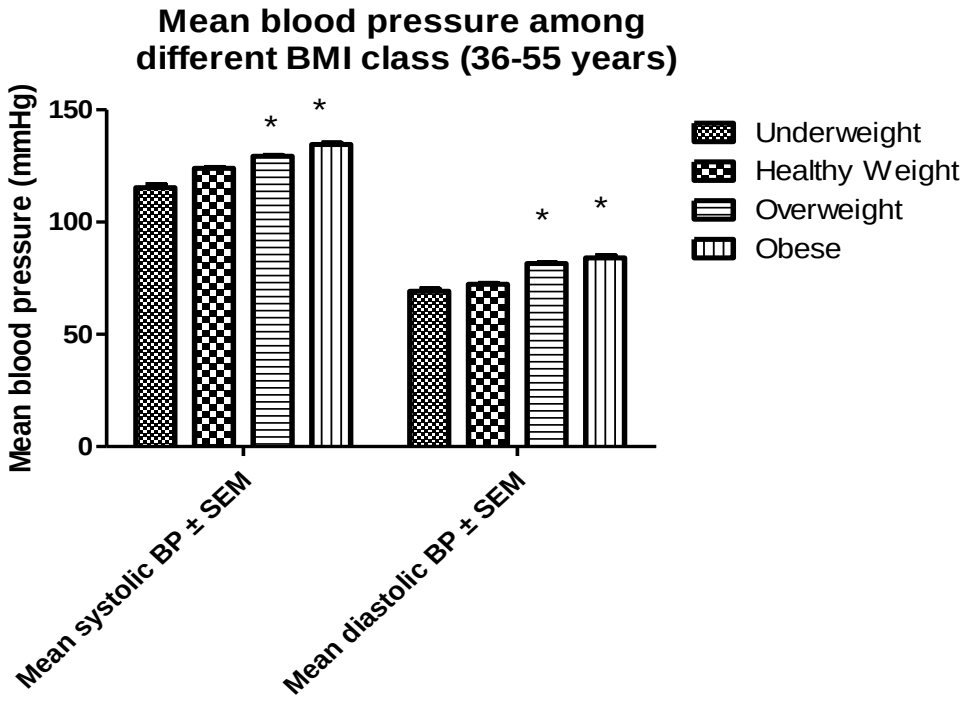

Figure 4.28 Mean blood pressure of the study participants according to BMI categories (36-55 years age group)

4.4.4 Mean BMI among subjects with pre-hypertension, hypertension and normal blood pressure

4.4.4.1 Mean BMI among subjects with pre-hypertension, hypertension and normal blood pressure (12-17 years age group)

In the age group of 12-17 years, mean BMI among subjects with normal blood pressure was 21.2±0.134 kg/m^2 while it was found higher among subjects with pre-hypertension and hypertension (24.6±0.481 kg/m^2 and 25.3±0.813 kg/m^2, respectively). In current study we found mean BMI among the subjects of 12-17 years had significant association with blood pressure. (Table 4.83, Table 4.84)

Table 4.83 Mean BMI among subjects with pre-hypertension and normal blood pressure (12-17 years age group)

12 to 17 Years Age Group			
	Normal Blood Pressure (N = 377)	**Pre-hypertension (N = 56)**	**P value**
Mean BMI ± SEM (kg/m^2)	21.2±0.134	24.6±0.481	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

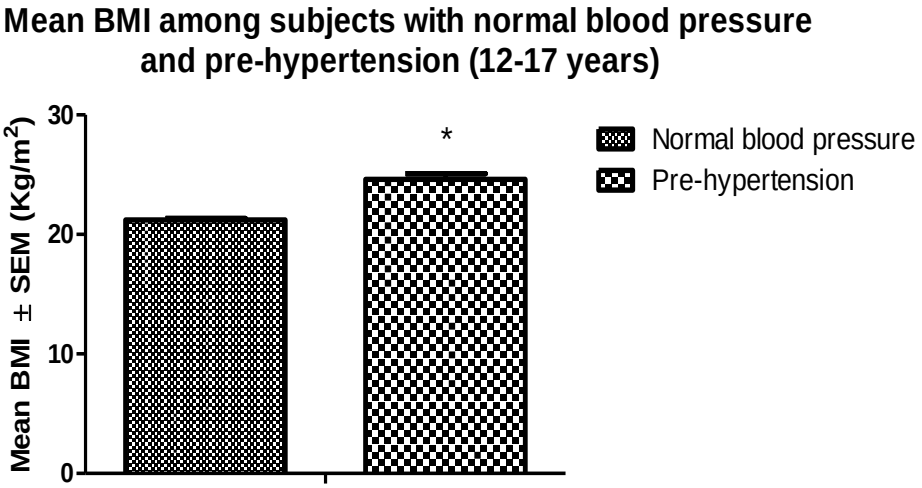

Figure 4.29 Mean BMI among subjects with pre-hypertension and normal blood pressure (12-17 years age group)

Table 4.84 Mean BMI among subjects with hypertension and normal blood pressure (12-17 years age group)

12 to 17 Years Age Group			
	Normal Blood Pressure (N = 377)	**Hypertension (N = 23)**	**P value**
Mean BMI ± SEM (kg/m^2)	21.2±0.134	25.3±0.813	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

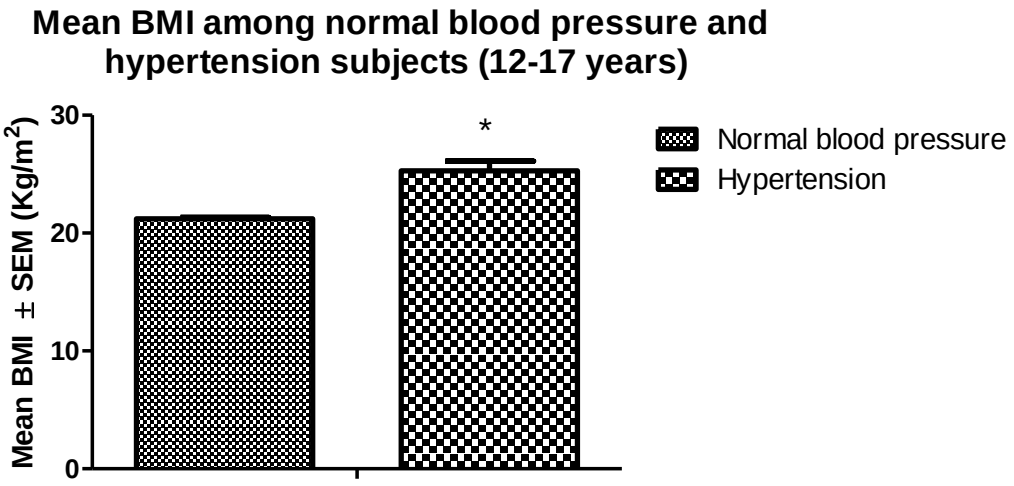

Figure 4.30 Mean BMI among subjects with hypertension and normal blood pressure (12-17 years age group)

4.4.4.2 Mean BMI among subjects with pre-hypertension, hypertension and normal blood pressure (18-35 years age group)

In the present study we found mean BMI among subjects with normal blood pressure was 22.7±0.130 kg/m^2, subjects with prehypertension 23.9±0.204 kg/m^2 and subjects with hypertension it was found high 24.6±0.243 kg/m^2 in 18-35 years age group. We further noted that mean BMI found to have significant association with blood pressure among subjects of 18-35 years. (Table 4.85, Table 4.86)

Table 4.85 Mean BMI among subjects with pre-hypertension and normal blood pressure (18-35 years age group)

18 to 35 Years Age Group			
	Normal Blood Pressure (N = 500)	**Pre-hypertension (N = 279)**	**P value**
Mean BMI ± SEM (kg/m^2)	22.7±0.130	23.9±0.204	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

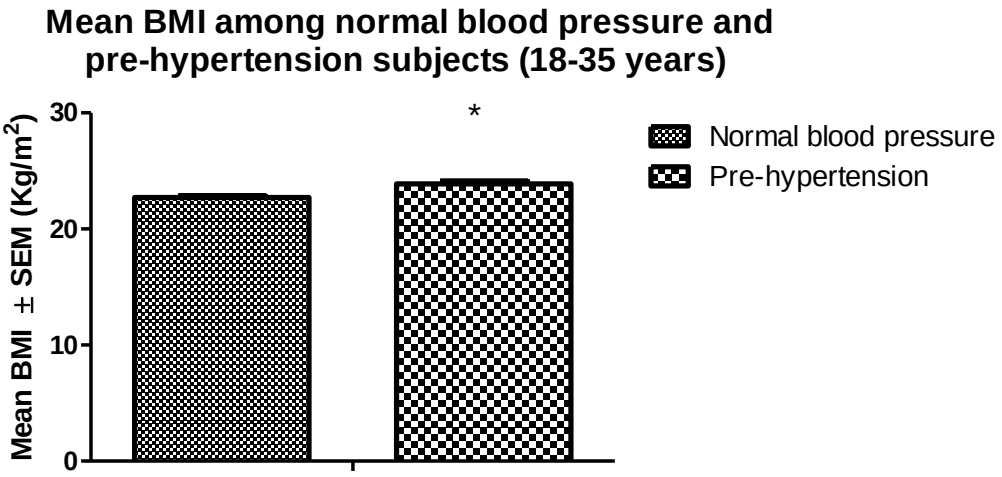

Figure 4.31 Mean BMI among subjects with pre-hypertension and normal blood pressure (18-35 years age group)

Table 4.86 Mean BMI among subjects with hypertension and normal blood pressure (18-35 years age group)

18 to 35 Years Age Group			
	Normal Blood Pressure (N = 500)	**Hypertension (N = 231)**	**P value**
Mean BMI ± SEM (kg/m^2)	22.7±0.130	24.6±0.243	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

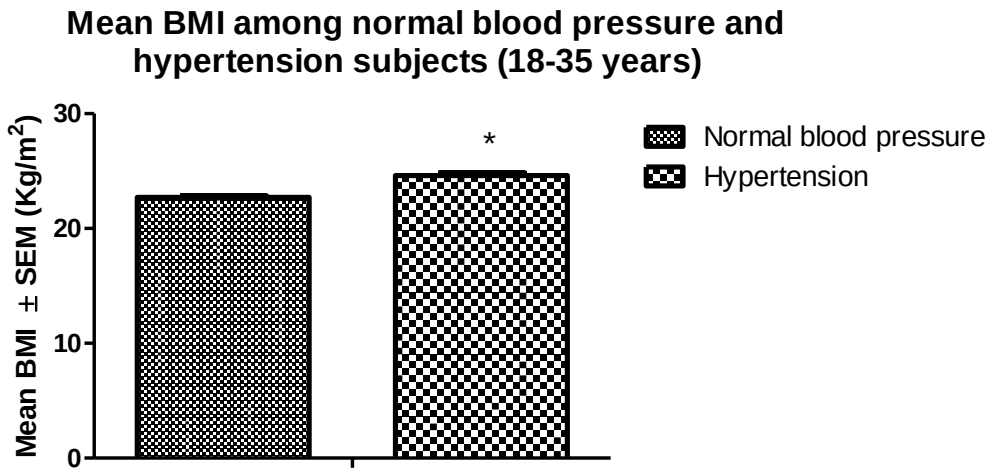

Figure 4.32 Mean BMI among subjects with hypertension and normal blood pressure (18-35 years age group)

4.4.4.3 Mean BMI among subjects with pre-hypertension, hypertension and normal blood pressure (36-55 years age group)

The mean BMI among subjects with normal blood pressure was 22.1±0.125 kg/m^2 following among pre-hypertension 24.4±0.204 kg/m^2 and hypertension 25.6±0.224 kg/m^2. This implies that mean BMI increases as blood pressure increases and the association of mean BMI was found in 36-55 years age group. (Table 4.87, Table 4.88)

Table 4.87 Mean BMI among subjects with pre-hypertension and normal blood pressure (36-55 years age group)

36 to 55 Years Age Group			
	Normal Blood Pressure (N = 367)	**Pre-hypertension (N = 277)**	**P value**
Mean BMI ± SEM (kg/m^2)	22.1±0.125	24.4±0.204	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

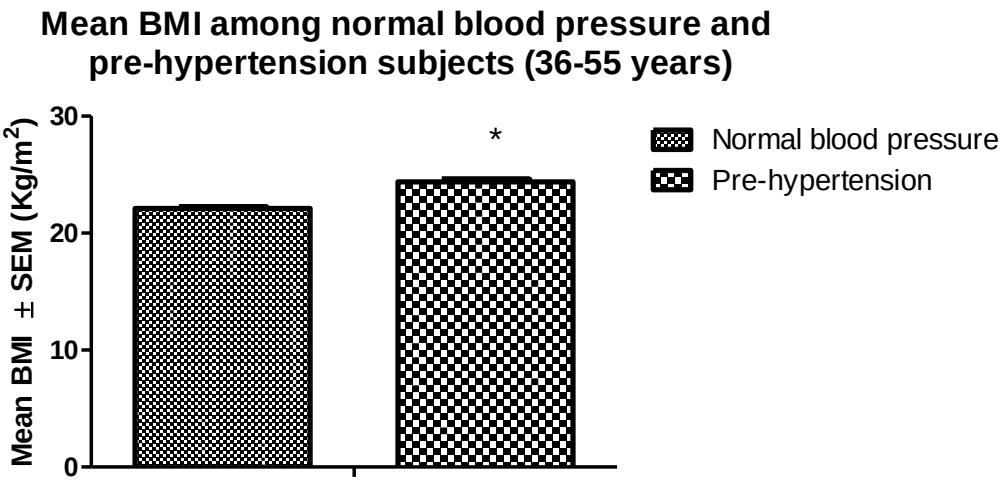

Figure 4.33 Mean BMI among subjects with pre-hypertension and normal blood pressure (36-55 years age group)

Table 4.88 Mean BMI among subjects with hypertension and normal blood pressure (36-55 years age group)

36 to 55 Years Age Group			
	Normal Blood Pressure (N = 367)	**Hypertension (N = 302)**	**P value**
BMI ± SEM (kg/m^2)	22.1±0.125	25.6±0.224	< 0.0001*

Analysed by unpaired t - test. * indicates statistical significance at p value less than 0.05.

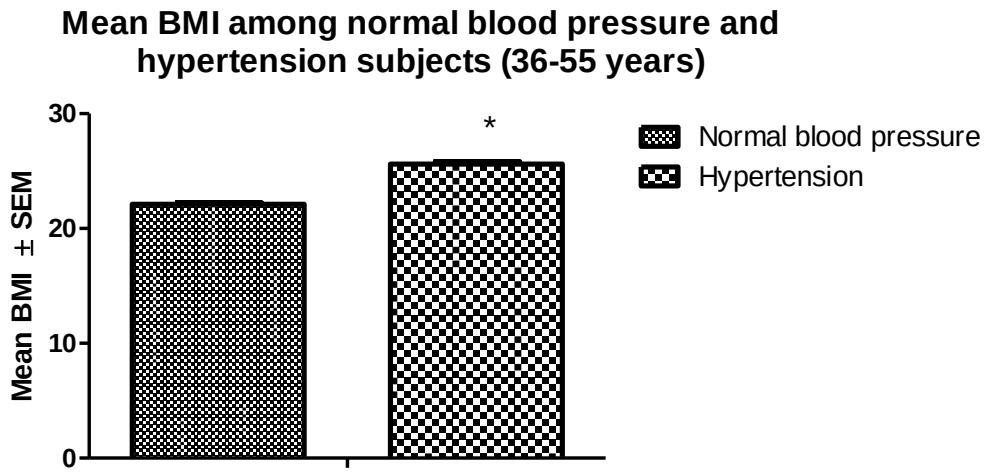

Figure 4.34 Mean BMI among subjects with hypertension and normal blood pressure (36-55 years age group)

4.4.5 Association of participation in exercise with BMI

4.4.5.1 Association of participation in exercise with BMI (12-17 years age group)

In the age group of 12-17 years, we found greater percentage of healthy weight subjects participate in exercise whereas greater percentage of underweight, overweight and obese subjects (19.73%, 23.02% and 7.23%, respectively) denied to take part in exercise. In this group, participation in exercise was found significantly associated with BMI demonstrating lacking of exercise leads to overweight and obesity. (Table 4.89)

Table 4.89 Prevalence of different weight category among subjects who participate in exercise (12-17 years age group)

12 to 17 Years Age Group					
Participation in exercise	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Yes (N = 304)	48 (15.78 %)	203 (66.77%)	39 (12.82%)	14 (4.60%)	0.003*
No (N = 152)	30 (19.73%)	76 (50.00%)	35 (23.02%)	11 (7.23%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.5.2 Association of participation in exercise with BMI (18-35 years age group)

In the age group of 18-35 years, the current study demonstrated greater percentage of underweight subjects (16.46%) participate in exercise whereas greater percentage of healthy weight, overweight and obese subjects denied their participation in exercise (70.82%, 21.26% and 5.71%, respectively). We found strong association of BMI with exercise in this age group stating exercise decreases risk of overweight and obesity. (Table 4.90)

Table 4.90 Prevalence of different weight category among subjects who participate in exercise (18-35 years age group)

18 to 35 Years Age Group					
Participation in exercise	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Yes (N = 328)	54 (16.46%)	219 (66.76%)	42 (12.80%)	13 (3.96%)	< 0.0001*
No (N = 682)	15 (2.19%)	483 (70.82%)	145 (21.26%)	39 (5.71%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.5.3 Association of participation in exercise with BMI (36-55 years age group)

The greater percentage of underweight and healthy weight subjects (17.79% and 66.79%, respectively) of 36-55 years of age reported they do participate in exercise whereas 29.34% overweight subjects denied their participation in any form of exercise. Among obese subjects though we found greater percentage for not participating in exercise, but the difference was not immense. In this group the association of BMI and exercise was found significant indicating risk of overweight and obesity decreases with exercise. (Table 4.91)

Table 4.91 Prevalence of different weight category among subjects who participate in exercise (36-55 years age group)

36 to 55 Years Age Group					
Participation in exercise	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Yes (N = 210)	30 (17.79%)	139 (66.19 %)	32 (15.23%)	9 (4.28%)	< 0.0001*
No (N = 736)	15 (2.03%)	463 (62.90%)	216 (29.34%)	42 (5.70 %)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.6 Association of participation in games and leisure activities with BMI

4.4.6.1 Association of participation in games and leisure activities with BMI (12-17 years age group)

The current study amongst subjects of 12-17 years old documented greater prevalence of indoor and outdoor activities amongst underweight (21.31% and 22.17%, respectively) and healthy weight (66.39% and 63.42%, respectively) subjects. Whereas greater percentage of overweight and obese subjects denied their participation in indoor and outdoor activities. We found indoor and outdoor activities have significant association with overweight and obesity. This shows any form of indoor or outdoor activities reduces risk of overweight and obesity. Irrespective of BMI categories, all the subjects reported to play on laptop/mobile and watch television. (Table 4.92)

Table 4.92 Prevalence of different weight category among subjects who participate in games and leisure activities (12-17 years age group)

12 to 17 Years Age Group						
Type of activity	**Variable**	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Indoor activity	**Yes (N = 122)**	26 (21.31 %)	81 (66.39%)	10 (8.19%)	5 (4.09%)	0.022*
	No (N = 334)	52 (15.56%)	198 (59.28%)	64 (19.16%)	20 (5.98%)	
Outdoor activity	**Yes (N = 257)**	57 (22.17%)	163 (63.42%)	28 (10.89%)	9 (3.50%)	0.000*
	No (N = 199)	21 (10.55%)	116 (58.29%)	46 (23.11%)	16 (8.04%)	
Playing on laptop/ mobile	**Yes (N = 456)**	74 (16.22%)	279 (61.18%)	78 (17.10%)	25 (5.48%)	-
	No (N = 0)	0	0	0	0	
Watching TV	**Yes (N = 456)**	74 (16.22%)	279 (61.18%)	78 (17.10%)	25 (5.48%)	-
	No (N = 0)	0	0	0	0	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.6.2 Association of participation in games and leisure activities with BMI (18-35 years age group)

Among the participants of 18-35 years old, we noted greater prevalence of participation in indoor and outdoor activities amongst underweight (8.87% and 9.94%, respectively) and healthy weight (82.25% and 78.36%, respectively) subjects. Whilst higher percentage of overweight and obese subjects denied their participation in indoor and outdoor activities in this group. We found indoor and outdoor activities found to have significant association with BMI demonstrating indoor or outdoor activities reduces risk of overweight and obesity. In this group also, irrespective of BMI class all the participants reported to participate in leisure activities like playing on laptop/mobile and television viewing. (Table 4.93)

Table 4.93 Prevalence of different weight category among subjects who participate in games and leisure activities (18-35 years age group)

18 to 35 Years Age Group						
Type of activity		**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Indoor activity	**Yes (N = 124)**	11 (8.87%)	102 (82.25%)	11 (7.25%)	2 (1.61%)	0.002*
	No (N = 886)	58 (6.54%)	600 (67.72%)	176 (19.86%)	50 (5.64%)	
Outdoor activity	**Yes (N = 171)**	17 (9.94%)	134 (78.36%)	13 (7.60%)	7 (4.09%)	0.0002*
	No (N = 839)	52 (6.19%)	568 (67.69%)	174 (20.73%)	45 (5.36%)	

Playing on laptop/ mobile	**Yes (N = 1,010)**	69 (6.83%)	702 (69.50%)	187 (18.51%)	52 (5.15%)	-
	No (N = 0)	0	0	0	0	
Watching TV	**Yes (N = 1,010)**	69 (6.83%)	702 (69.50%)	187 (18.51%)	52 (5.15%)	-
	No (N = 0)	0	0	0	0	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.6.3 Association of participation in games and leisure activities with BMI (36-55 years age group)

In present study we noticed greater prevalence of indoor and outdoor activities amongst underweight (18.64% and 19.23%, respectively) and healthy weight (66.10 % and 67.94%, respectively) subjects of age 36-55 years. In this age group, indoor and outdoor activities reported to have strong significant association with BMI. Similarly, higher percentage of underweight (14.45%), healthy weight (66.47%) and obese (9.24%) participants denied of playing games on laptop/mobile while greater percentage of overweight subjects reported to play games on laptop/mobile. We further found significant association showing increase risk of weight gain if more time is spend on playing game on laptop/mobile. All the participants irrespective of BMI class, reported to watch television. (Table 4.94)

Table 4.94 Prevalence of different weight category among subjects who participate in games and leisure activities (36-55 years age group)

36 to 55 Years Age Group						
Type of activity		**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Indoor activity	**Yes (N = 59)**	11 (18.64%)	39 (66.10%)	7 (11.86%)	2 (3.92%)	< 0.0001*
	No (N = 887)	34 (3.83%)	563 (63.47%)	241 (27.17%)	49 (5.52%)	
Outdoor activity	**Yes (N = 78)**	15 (19.23%)	53 (67.94%)	7 (8.97%)	3 (3.84%)	< 0.0001*
	No (N = 868)	30 (3.45%)	549 (63.24%)	241 (27.76%)	48 (5.52%)	
Playing on laptop/ mobile	**Yes (N = 773)**	20 (2.58%)	487 (63.00%)	231 (29.88%)	35 (4.52%)	< 0.0001*
	No (N = 173)	25 (14.45%)	115 (66.47%)	17 (9.82%)	16 (9.24%)	
Watching TV	**Yes (N = 946)**	45 (4.76%)	602 (63.64%)	248 (26.22%)	51 (5.39%)	-
	No (N = 0)	0	0	0	0	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

All in all, we have noticed that among all three-age groups, overweight and obese participants participating in indoor and outdoor activities was lower in percentage while they found to indulge more in playing on laptop/mobile and watching television compared to underweight and healthy weight subjects.

4.4.7 Association of dietary type with BMI

4.4.7.1 Association of dietary type with BMI (12-17 years age group)

In current study, we found higher number of underweight subjects had vegetarian (17.94%) and eggetarian (15.38%) dietary type while lowest prevalence of non-vegetarian dietary type (8.00%) was reported among underweight of age 12-17 years.

Among healthy weight subjects vegetarian and non-vegetarian dietary type was almost equally similar (62.00% and 60.00%, respectively). However, eggetarian dietary type in healthy weight subjects was 55.76%, which was lesser than other two.

On the contrary, overweight and obese subjects had greater prevalence of non-vegetarian and eggetarian dietary type in comparison to vegetarian.

Dietary type, whether vegetarian, non-vegetarian or eggetarian was not found significantly associated with BMI among 12-17 years age group in present study. (Table 4.95)

Table 4.95 Prevalence of different weight category among subjects with different dietary type (12-17 years age group)

12 to 17 Years Age Group					
Dietary Type	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Vegetarian (N = 379)	68 (17.94%)	235 (62.00%)	58 (15.30%)	18 (4.74%)	0.4550
Non-vegetarian (N = 25)	2 (8.00%)	15 (60.00%)	5 (20.00%)	3 (12.00%)	
Eggetarian (N = 52)	8 (15.38%)	29 (55.76%)	11 (21.15%)	4 (7.69%)	

Analysed by Pearson's chi-square test.

4.4.7.2 Association of dietary type with BMI (18-35 years age group)

In the age group of 18-35 years, there was the highest prevalence of vegetarian dietary form (8.03%) among underweight; but the difference was not immense in between non-vegetarian and eggetarian dietary forms since it was 4.10% and 5.39% respectively.

Similarly, healthy weight subjects were reported to have more vegetarian diet (71.96%) while non-vegetarian (67.12%) and eggetarian (65.90%) dietary form was nearly equal.

Our study documented higher prevalence of non-vegetarian and eggetarian dietary type among overweight and obese subjects than vegetarian dietary type. Additionally, the difference of non-vegetarian and eggetarian dietary type prevalence was not remarkable among both overweight and obese subjects.

We found that dietary type was not significantly associated with BMI among 18-35 years age group in present study. (Table 4.96)

Table 4.96 Prevalence of different weight category among subjects with different dietary type (18-35 Years age group)

18 to 35 Years Age Group					
Dietary Type	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Vegetarian (N = 585)	47 (8.03%)	421 (71.96%)	93 (15.89%)	24 (4.10%)	0.053
Non-vegetarian (N = 73)	3 (4.10%)	49 (67.12%)	16 (21.91%)	5 (6.84%)	
Eggetarian (N = 352)	19 (5.39%)	232 (65.90%)	78 (22.15%)	23 (6.53%)	

Analysed by Pearson's chi-square test.

4.4.7.3 Association of dietary type with BMI (36-55 years age group)

In the age group of 36-55 years, we found greater number of underweight and healthy weight subjects with vegetarian dietary type (5.06% and 64.53%, respectively) compared to non-vegetarian and eggetarian dietary type. However, the difference was not huge among all three dietary type in both underweight and healthy weight subjects.

We further found overweight and obese subjects had highest prevalence of non-vegetarian dietary form (28.57% and 10.71% respectively). The prevalence of eggetarian dietary form was almost as equal as vegetarian dietary form. In this age group dietary type was not found significantly associated with BMI. (Table 4.97)

Moreover, these all findings implies that BMI was not significantly associated with dietary type in any of the age group.

In all three age groups, we found high prevalence of variety dietary type among healthy weight subjects. This was due to the greater number of subjects were of healthy weight category in this study and underweight, overweight and obese subjects were less in number.

Table 4.97 Prevalence of different weight category among subjects with different dietary type (36-55 Years age group)

36 to 55 Years Age Group					
Dietary Type	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Vegetarian (N = 671)	34 (5.06%)	433 (64.53%)	173 (25.78%)	31 (4.61%)	0.507
Non-vegetarian (N = 56)	2 (3.57%)	32 (57.14%)	16 (28.57%)	6 (10.71%)	
Eggetarian (N = 219)	9 (4.10%)	137 (62.55%)	59 (26.94%)	14 (6.39%)	

Analysed by Pearson's chi-square test.

4.4.8 Association of frequency of junk food eating habit with BMI

4.4.8.1 Association of frequency of junk food eating habit with BMI (12-17 years age group)

We found that overweight and obese subjects had more everyday (20.63% and 11.11% respectively) and once in a week (17.39% and 5.65% respectively) frequency of junk food eating habit. Whereas underweight and healthy weight subjects had more once in fifteen day (20.22% and 64.04% respectively) and once in a month (20.27% and 63.51% respectively) frequency of eating junk food.

However, frequency of junk food eating habit found to have no significant association with BMI among age group of 12-17 years in present study. (Table 4.98)

Table 4.98 Association of BMI in subjects with different frequency of junk food habit (12-17 Years age group)

12 to 17 Years Age Group					
Frequency of Junk food eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 63)	8 (12.69%)	35 (55.55%)	13 (20.63%)	7 (11.11%)	0.3643
Once in a Week (N = 230)	37 (16.08%)	140 (60.86%)	40 (17.39%)	13 (5.65%)	
Once in 15 Days (N = 89)	18 (20.22%)	57 (64.04%)	11 (12.35%)	3 (3.37%)	
Once in a Month (N = 74)	15 (20.27%)	47 (63.51%)	10 (13.51%)	2 (2.70%)	

Analysed by Pearson's chi-square test.

4.4.8.2 Association of frequency of junk food eating habit with BMI (18-35 years age group)

Among subjects of 18-35 years, we found higher prevalence of everyday and once in a week frequency of junk food eating habit among overweight (22.30% and 20.06% respectively) and obese (7.91% and 5.73%) subjects. While once in fifteen days and once in a month junk food frequency was found higher among underweight (8.60% and 17.54% respectively) subjects. We noticed high number of healthy subjects had once in fifteen days (78.49%) junk food eating habit following once in a week (68.63%).

In present study we found frequency of junk food eating habit have significant association with overweight and obesity among age group of 18-35 years. (Table 4.99)

Table 4.99 Association of BMI in subjects with different frequency of junk food habit (18-35 Years age group)

18 to 35 Years Age Group					
Frequency of Junk food eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 139)	8 (5.75%)	89 (64.02%)	31 (22.30%)	11 (7.91%)	0.0004*
Once in a Week (N = 628)	35 (5.57%)	431 (68.63%)	126 (20.06%)	36 (5.73%)	
Once in 15 Days (N = 186)	16 (8.60%)	146 (78.49%)	21 (11.29%)	3 (1.61%)	
Once in a month (N = 57)	10 (17.54%)	36 (63.15%)	9 (16.66%)	2 (3.50%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.8.3 Association of frequency of junk food eating habit with BMI (36-55 years age group)

In current study, everyday and once in a week junk food eating habit was found to be high among overweight (36.27% and 31.23%, respectively) and obese (8.82% and 6.69%, respectively) than once in fifteen days (14.08% in overweight and 2.34% in obese) and

once in a month (19.56% in overweight and 2.89% in obese) junk food eating frequency. Furthermore, we also found that healthy weight and underweight subjects had more once in fifteen days (76.05% and 7.51%, respectively) and once in a month (72.46% and 5.07%, respectively) junk food eating frequency compared to everyday and once in month junk food frequency. Among the subjects of 36-55 years we found everyday frequency of junk food eating habit had significant association with BMI in current study. (Table 4.100)

Table 4.100 Association of BMI in subjects with different frequency of junk food habit (36-55 years age group)

36 to 55 Years Age Group					
Frequency of Junk food eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 102)	3 (2.94%)	53 (51.96%)	37 (36.27%)	9 (8.82%)	< 0.0001*
Once in a Week (N = 493)	19 (3.85%)	287 (58.21%)	154 (31.23%)	33 (6.69%)	
Once in 15 Days (N = 213)	16 (7.51%)	162 (76.05%)	30 (14.08%)	5 (2.34%)	
Once in a month (N = 138)	7 (5.07%)	100 (72.46%)	27 (19.56%)	4 (2.89%)	

Analysed by Pearson's chi-square test. * Significantly different from frequency of junk food eating habit in healthy weight group. It indicates statistical significance at p value less than 0.05

In all three age group it was noticed that overweight and obese subjects have greater frequency (everyday and once in a week) of junk food eating habit compared to underweight and healthy weight. It was also found that more number of underweight and healthy weight subjects ate junk food once in fifteen days and/or once in a month. We further found frequencies of junk food had no association with BMI in 12-17 years age group whilst it had positive association with BMI in 18-35 and 36-55 years age group.

4.4.9 Association of frequency of sweet eating habit with BMI

4.4.9.1 Association of frequency of sweet eating habit with BMI (12-17 years age group)

In present study, in the age group of 12-17 years, the highest number of underweight subjects had once in a week frequency of eating sweet (22.22%). The prevalence of underweight subjects who ate sweets everyday was 14.56%. While once in fifteen days (11.53%) and once in a month (10.16%) frequencies found to have small difference in prevalence among underweight subjects.

Greater number of healthy weight subjects had once in a month frequency of eating sweet (76.27%) followed by once in fifteen days (67.94%). Whereas, frequency of everyday and once in a week found to be nearly same (56.31% and 56.94%, respectively).

Everyday habit of eating sweet was 19.41% among overweight subjects which was documented the highest among all frequencies following once in a week (16.66%) and once in fifteen days (14.10%) habit of eating sweets. We found lowest prevalence for once in a month (11.86%) frequency of eating sweet among overweight subjects.

Obese subjects found to have higher prevalence of eating sweets everyday (9.70%) followed by once in fifteen days (6.41%), once in a week (4.16%) and once in a month (1.69%). The different frequencies of eating sweet found significantly associated with BMI among subject of 12-17 years. (Table 4.101)

Table 4.101 Association of BMI in subjects with different frequency of sweet eating habit (12-17 Years age group)

12 to 17 Years Age Group					
Frequency of sweet eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 103)	15 (14.56%)	58 (56.31%)	20 (19.41%)	10 (9.70%)	0.036*
Once in a Week (N = 216)	48 (22.22%)	123 (56.94%)	36 (16.66%)	9 (4.16%)	
Once in 15 Days (N = 78)	9 (11.53%)	53 (67.94%)	11 (14.10%)	5 (6.41%)	
Once in a Month (N = 59)	6 (10.16%)	45 (76.27%)	7 (11.86%)	1 (1.69%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.9.2 Association of frequency of sweet eating habit with BMI (18-35 years age group)

In the age group of 18-35 years, we found high number of underweight subjects had once in a week frequency of eating sweet (8.56%). While frequency of once in a month (5.79%), once in fifteen days (5.03%) and everyday (4.52%) of eating sweets among underweight subjects found insignificant.

Greater number of healthy weight subjects had once in a month habit of eating sweet (79.71%) followed by once in fifteen days (78.41%). Whereas, frequency of once in a week eating sweet was 68.15% and everyday habit of eating sweet was the lowest (64.90%) to other frequencies.

The prevalence of overweight subject was found was the greatest (21.88%) in everyday frequency of eating sweets following once in a week (19.18%), once in fifteen days (12.94%) and lowest in once in a month (11.59%).

We noted obese subjects had higher prevalence of eating sweets everyday (8.67%). However, the difference among the frequencies of eating sweets once in a week (4.09%), once in fifteen days (3.59%) and once in a month (2.89%) was negligible.

In this age group we identifies everyday sweet eating habit found significantly associated with BMI determines risk of overweight and obesity. (Table 4.102)

Table 4.102 Association of BMI in subjects with different frequency of sweet eating habit (18-35 Years age group)

18 to 35 Years Age Group					
Frequency of sweet eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 265)	12 (4.52%)	172 (64.90%)	58 (21.88%)	23 (8.67%)	0.0051*
Once in a Week (N = 537)	46 (8.56%)	366 (68.15%)	103 (19.18%)	22 (4.09%)	
Once in 15 Days (N = 139)	7 (5.03%)	109 (78.41%)	18 (12.94%)	5 (3.59%)	
Once in a Month (N = 69)	4 (5.79%)	55 (79.71%)	8 (11.59%)	2 (2.89%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05

4.4.9.3 Association of frequency of sweet eating habit with BMI (36-55 years age group)

In current study, the prevalence of once in a month frequency (16.66%) of sweet eating habit was the highest among underweight subjects, followed by once in fifteen days (5.37%). Whilst the prevalence of once in a week (3.48%) and everyday (3.05%) frequencies of eating sweet was similar and lower compared to other two.

Among healthy weight subjects once in a week and once in fifteen days frequency of eating sweet was significantly higher and equal (67.53% and 67.20%, respectively). Whereas, the once in a month frequency of sweet eating habit was 61.11%. Everyday junk food eating habit was the lowest (53.71%) among all frequencies in healthy subjects.

We documented the overweight subjects had more everyday sweets eating habit (33.62%). The prevalence of once in a week (24.61%) and once in fifteen days (23.65%) frequency of eating sweet were equally similar among overweight subjects. Nonetheless, only 19.44% overweight subjects found in frequency of once in a month eating sweet.

Greater number of obese subjects had everyday habit of eating sweet (9.60%). Other frequencies were found similar in this age group.

In current study in the age group of 36-55 years it was documented that frequencies of eating sweet has significant association with BMI. (Table 4.103)

Table 4.103 Association of BMI in subjects with different frequency of sweet eating habit (36-55 Years age group)

36 to 55 Years Age Group					
Frequency of sweet eating Habit	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Everyday (N = 229)	7 (3.05%)	123 (53.71%)	77 (33.62%)	22 (9.60%)	< 0.0001*

Once in a Week (N = 459)	16 (3.48%)	310 (67.53%)	113 (24.61%)	20 (4.35%)	
Once in 15 Days (N = 186)	10 (5.37%)	125 (67.20%)	44 (23.65%)	7 (3.76%)	
Once in a Month (N =72)	12 (16.66%)	44 (61.11%)	14 (19.44%)	2 (2.77%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

Results of frequencies of sweet eating habit among all three-age groups, it can be concluded that frequencies of eating sweet mainly everyday and once in a week had significant association with increased BMI.

4.4.10 Association of stress level with BMI

4.4.10.1 Association of stress level with BMI (12-17 years age group)

In present study, highest number of underweight (18.86%) and healthy weight (63.56%) subjects belonged to no stress level. The prevalence of low stress level among underweight subjects was 7.40%. Underweight subjects found to have lowest prevalence (6.66%) of medium level of stress. Whereas 50% of low level of stress and 40% of medium level of stress was found among healthy weight subjects.

Lowest number of overweight (13.69%) and obese (3.87%) subjects found to have no stress and the percentage prevalence was increased to the stress level. Likewise, 29.62% overweight subjects had low level of stress and 33.33% subjects had medium level of stress. Similarly, 12.96% obese found to have low level of stress and 20% subjects had medium level of stress.

This study demonstrated the low and medium level of stress was high among overweight and obese subjects. Conversely, more underweight and healthy weight subjects found under no stress level.

Present study among the subjects of 12-17 years found significant association of stress level of with BMI. (Table 4.104)

Table 4.104 Association of stress level with BMI classification (12-17 Years age group)

12 to 17 Years Age Group					
Stress level	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
No Stress (N = 387)	73 (18.86%)	246 (63.56%)	53 (13.69%)	15 (3.87%)	0.0003*
Low level of stress (N = 54)	4 (7.40%)	27 (50.00%)	16 (29.62%)	7 (12.96%)	
Medium level of stress (N = 15)	1 (6.66%)	6 (40.00%)	5 (33.33%)	3 (20.00%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.10.2 Association of stress level with BMI (18-35 years age group)

In this study we found there was no huge difference between the stress levels of no stress level and low stress level among underweight subjects as it was 7.07% and 6.70% respectively while lowest percentage (4.47%) found at medium level of stress.

The highest percentage among healthy weight subjects found at low level of stress (70.10%) followed by 69.69% at low level of stress and 65.67% at medium level of stress.

Among overweight subjects 18.42% had no stress and 18.04% had low level of stress and the difference was found negligible. The greater number of overweight subjects (20.89%) had medium level of stress.

Medium level of stress was the highest among obese subjects (8.95%) followed by less level of stress (5.15%) and no stress (4.80%). However, the prevalence of less level of stress and no stress was found nearly equal.

In present study we noticed level of stress had no significant association with BMI among subjects of 18-35 years age group. (Table 4.105)

Table 4.105 Association of stress level with BMI classification (18-35 Years age group)

18 to 35 Years Age Group					
Stress level	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
No Stress (N = 749)	53 (7.07%)	522 (69.69%)	138 (18.42%)	36 (4.80%)	0.8016
Low level of stress (N = 194)	13 (6.70%)	136 (70.10%)	35 (18.04%)	10 (5.15%)	
Medium level of Stress (N = 67)	3 (4.47%)	44 (65.67%)	14 (20.89%)	6 (8.95%)	

Analysed by Pearson’s chi-square test.

4.4.10.3 Association of stress level with BMI (36-55 years age group)

In the age group of 36-55 years, more underweight subjects had no stress (5.66%) while 3.80% subjects found to have low level of stress. We found none of the underweight subjects belonged to medium level of stress category.

Among healthy weight subjects, 64.16% found no stress whereas 63.58% had low level of stress. The prevalence of medium level of stress among healthy weight subjects was the lowest (54.83%).

On the flip side, medium level of stress among overweight (32.25%) and obese (12.90%) subjects was higher in prevalence. Whereas less number of overweight and obese subjects had low level of stress or no stress in comparison to medium level of stress.

In this age group we did not find level of stress had a significant association with BMI in present study. (Table 4.106)

Table 4.106 Association of BMI in subjects with stress level (36-55 Years age group)

36 to 55 Years Age Group					
Stress level	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
No Stress (N = 547)	31 (5.66%)	351 (64.16%)	141 (25.77%)	24 (4.38%)	0.2993
Low level of stress (N = 368)	14 (3.80%)	234 (63.58%)	97 (26.35%)	23 (6.25%)	
Medium level of stress (N = 15)	0 (0.00%)	17 (54.83%)	10 (32.25%)	4 (12.90%)	

Analysed by Pearson’s chi-square test.

From above findings of association of stress with BMI we noted in the age group of 12-17 years, stress level had significant association with BMI whilst we did not find any association of stress level with BMI among participants of 18-35 and 36-55 years age group.

4.4.11 Association of Socioeconomic class with BMI

4.4.11.1 Association of socioeconomic class with BMI (12-17 years age group)

In the age group of 12-17 years, more underweight subjects were belonging to upper lower socioeconomic class (44.06%) following lower middle (26.08%), lower (16.66%), upper middle (13.63%). While lowest percentage of underweight subjects belonged to upper class (7.31%).

Among healthy weight subjects, highest prevalence was found under upper middle (68.88%) and lower socioeconomic class (66.66%) following lower middle (56.52%). The prevalence of healthy weight subjects belonged to upper and upper lower class was nearly equal (47.56% and 47.45%, respectively).

On the contrary, greater percentage of overweight and obese subjects belonged to upper socioeconomic class (32.92% and 12.19%, respectively). We further noted second highest prevalence of overweight subjects (16.66%) were of lower socioeconomic class, although none of the obese subjects belonged to lower socioeconomic class. Equal prevalence of overweight and obese subjects were reported of upper middle and lower middle socioeconomic class.

In this age group we found socioeconomic class had a significant association with BMI in present study. This shows upper socioeconomic class is considered as predisposing factor for overweight and obesity in this age group. (Table 4.107)

Table 4.107 Association of socioeconomic class with BMI (12-17 years age group)

12 to 17 Years Age Group					
Socioeconomic class	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Upper (N = 82)	6 (7.31%)	39 (47.56%)	27 (32.92%)	10 (12.19%)	< 0.0001*
Upper middle (N = 286)	39 (13.63%)	195 (68.88%)	40 (13.98%)	12 (4.19 %)	
Lower Middle (N = 23)	6 (26.08%)	13 (56.52%)	3 (13.04%)	1 (4.34%)	
Upper Lower (N = 59)	26 (44.06%)	28 (47.45%)	3 (5.08%)	2 (3.38%)	
Lower (N = 6)	1 (16.66%)	4 (66.66%)	1 (16.66%)	0	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.11.2 Association of socioeconomic class with BMI (18-35 years age group)

Amongst participants of 18-35 years, we noted greater percentage of underweight subjects of lower (24.13%), upper lower (15.78%) and lower middle (5.29%) socioeconomic class.

Among healthy weight subjects, highest prevalence was found under upper middle (77.96%) following nearly similar percentage of healthy weight subjects belonged to lower and upper lower socioeconomic class (68.96% and 67.54%, respectively).

On the contrary to underweight and healthy weight subjects, greater percentage of overweight and obese subjects belonged to upper socioeconomic class (32.92% and 12.19%, respectively). We further found lowest percentage of overweight and obese subjects were belonged to lower socioeconomic class.

In this age group we found socioeconomic class had a significant association with BMI in present study indicating risk of overweight and obesity among subjects of upper socioeconomic class. (Table 4.108)

Table 4.108 Association of socioeconomic class with BMI (18-35 years age group)

18 to 35 Years Age Group					
Socioeconomic class	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Upper (N = 346)	13 (3.75%)	206 (59.53%)	103 (29.76 %)	24 (6.93%)	< 0.0001*
Upper middle (N = 472)	25 (5.29 %)	368 (77.96%)	57 (12.07%)	22 (4.66 %)	
Lower Middle (N = 49)	6 (12.24%)	31 (63.26%)	10 (20.40%)	2 (4.08%)	
Upper Lower (N = 114)	18 (15.78%)	77 (67.54%)	16 (14.03%)	3 (2.63 %)	
Lower (N = 29)	7 (24.13%)	20 (68.96%)	1 (3.44%)	1 (3.44%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.11.3 Association of socioeconomic class with BMI (36-55 years age group)

The current study amongst participants of 36-55 years reported highest percentage of underweight subjects of lower socioeconomic class (35.00%) following upper lower (23.37%) and lower middle (21.42%) socioeconomic class.

Among healthy weight subjects, greater prevalence was found under upper middle (69.75%) and upper (58.88%) socioeconomic class. We found nearly similar prevalence for upper lower and lower socioeconomic class.

On the other hand, greater percentage of overweight and obese subjects belonged to upper socioeconomic class (32.56% and 6.90%, respectively) and lowest percentage of overweight and obese subjects were of lower socioeconomic class

In current study in the same age group we noted upper socioeconomic class had a significant association with incident overweight and obesity. (Table 4.109)

Table 4.109 Association of socioeconomic class with BMI (36-55 years age group)

36 to 55 Years Age Group					
Socioeconomic class	**Underweight**	**Healthy Weight**	**Overweight**	**Obese**	**P value**
Upper (N = 304)	5 (1.64%)	179 (58.88%)	99 (32.56%)	21 (6.90%)	< 0.0001*
Upper middle (N = 517)	9 (1.74 %)	357 (69.05%)	124 (23.98%)	27 (5.22 %)	
Lower Middle (N = 28)	6 (21.42%)	15 (53.57%)	6 (21.42%)	1 (3.57%)	
Upper Lower (N = 77)	18 (23.37%)	41 (53.24%)	17 (22.07%)	1 (1.29%)	
Lower (N = 20)	7 (35.00%)	10 (50.00%)	2 (10.00%)	1 (5.00%)	

Analysed by Pearson's chi-square test. * indicates statistical significance at p value less than 0.05.

4.4.12 Association of hypercholesterolemia with BMI

4.4.12.1 Association of hypercholesterolemia with BMI (12-17 years age group)

In present study, we have not found any lipid abnormalities among subjects of 12-17 years.

Though, we found increasing mean cholesterol level with BMI. The mean cholesterol level among underweight was 138.6±2.163 mg/dL, healthy weight 148.2±1.239 mg/dL, overweight 160.3±2.139 mg/dL and obese was 169.6±4.140 mg/dL, which was found to be highest among all BMI classification. The significant correlation was found between BMI and mean cholesterol level showing risk of hypercholesterolemia among overweight and obese participants in future. (Table 4.110)

Table 4.110 Mean level of cholesterol level among different BMI subjects (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean Cholesterol Level ± SEM (mg/dL)**	**P value**
Underweight	138.6±2.163	0.000*
Healthy Weight	148.2±1.239	
Overweight	160.3±2.139	
Obese	169.6±4.140	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

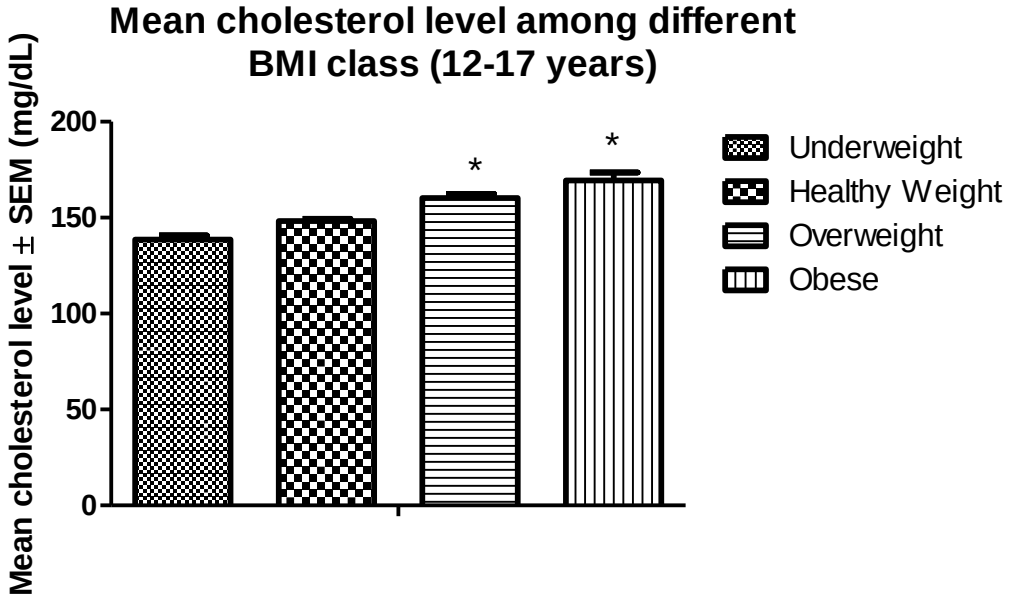

Figure 4.35 Mean level of cholesterol level among different BMI subjects (12-17 years age group)

4.4.12.2 Association of hypercholesterolemia with BMI (18-35 years age group)

In the age group of 18-35 years, none of the underweight subject found to have hypercholesterolemia; all had normal cholesterol level. Out of 702 healthy weight subjects 9.54% had hypercholesterolemia. Out of 187 overweight subjects 27.27% subject and out of 52 obese subjects 36.53% reported to have hypercholesterolemia. It can be inferred that risk of hypercholesterolemia increases with increased BMI and significant association was found between them in the age group of 18-35 years. (Table 4.111)

In the same age group, we found increasing mean cholesterol level with BMI. The mean cholesterol level among underweight was 146.9±2.335 mg/dL, healthy weight 167.7±0.751 mg/dL, overweight 177.6±1.784 mg/dL and obese was 188.2±3.398 mg/dL, which was found to be highest among all BMI classification. The significant correlation was found between BMI and mean cholesterol level which explains incident hypercholesterolemia among overweight and obese subjects. (Table 4.112)

Table 4.111 Association of hypercholesterolemia with BMI (18-35 Years age group)

18 to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Hypercholesterolemia**	
Underweight	69	0 (0.00%)	< 0.001*
Healthy Weight	702	67 (9.54 %)	
Overweight	187	51 (27.27%)	
Obese	52	19 (36.53%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.112 Mean level of cholesterol level among different BMI subjects (18-35 years age group)

18 to 35 Years Age Group		
BMI Classification	**Mean Cholesterol Level ± SEM (mg/dL)**	**P value**
Underweight	146.9±2.335	0.000*
Healthy Weight	167.7±0.751	
Overweight	177.6±1.784	
Obese	188.2±3.398	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

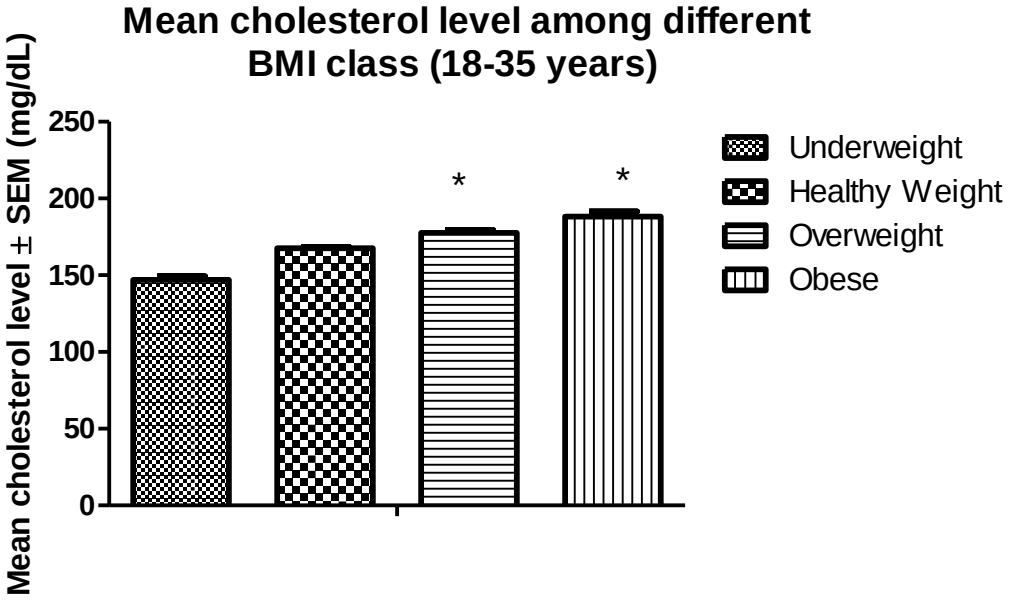

Figure 4.36 Mean level of cholesterol level among different BMI subjects (18-35 years age group)

4.4.12.3 Association of hypercholesterolemia with BMI (36-55 years age group)

In the age group of 36-55 years, the prevalence of hypercholesterolemia was the highest in obese subjects (60.78%) followed by overweight (46.37%) and the lowest among healthy weight subjects (21.76%). No underweight subjects were reported to have hypercholesterolemia. Hypercholesterolemia was found significantly associated with BMI demonstrating increased risk of hypercholesterolemia with higher BMI in this age group. (Table 4.113)

In the current study in same age group, mean cholesterol level among underweight, healthy weight, overweight and obese subjects was 147.8±3.011 mg/dL, 169.8±0.970 mg/dL, 183.2±1.721 mg/dL and 194.1±2.843 mg/dL respectively. This finding showed that mean cholesterol level was higher among overweight and obese subjects compared to underweight and heathy weight. Moreover, strong correlation of mean cholesterol level was found with BMI in this study. (Table 4.114)

Table 4.113 Association of hypercholesterolemia with BMI (36-55 Years age group)

36 to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Hypercholesterolemia**	
Underweight	45	0 (0.00%)	< 0.001*
Healthy Weight	602	131 (21.76 %)	
Overweight	248	115 (46.37%)	
Obese	51	31 (60.78%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.114 Mean level of cholesterol level among different BMI subjects (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean Cholesterol Level ± SEM (mg/dL)**	**P value**
Underweight	147.8±3.011	0.000*
Healthy Weight	169.8±0.970	
Overweight	183.2±1.721	
Obese	194.1±2.843	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

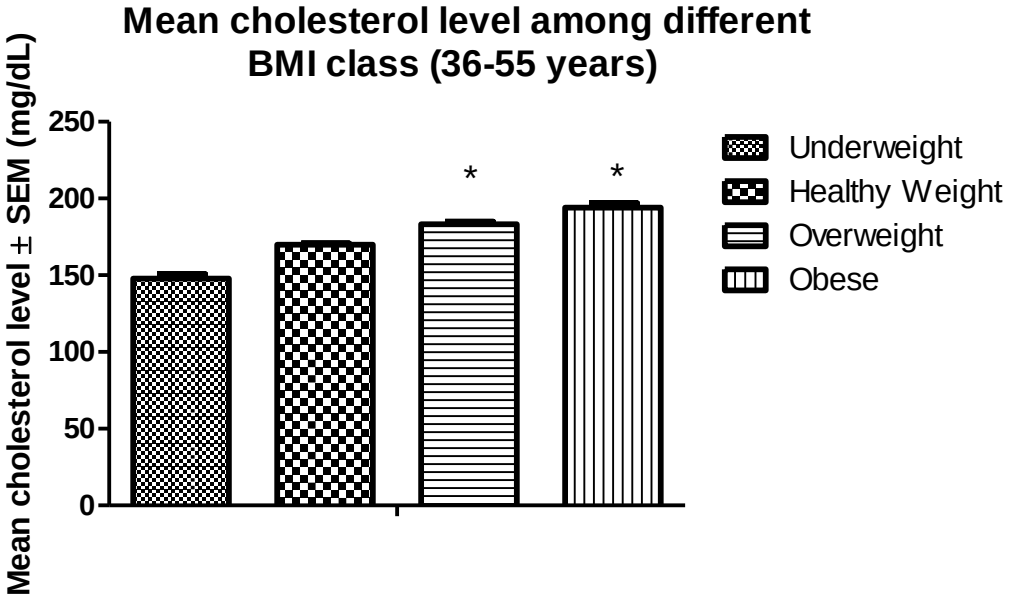

Figure 4.37 Mean level of cholesterol level among different BMI subjects (36-55 years age group)

4.4.13 Association of hypertriglyceridemia with BMI

4.4.13.1 Association of hypertriglyceridemia with BMI (12-17 years age group)

The current study have not reported abnormally elevated level of triglycerides among any participant 12-17 years of age.

However, mean triglyceride level was noted the highest among obese subjects; 137.1±2.100 mg/dL following overweight subjects (132.3±1.290 mg/dL), healthy weight subjects (120.2±0.635 mg/dL) and the lowest in underweight subjects (114.4±1.404 mg/dL). Furthermore, in this study we found mean triglyceride level was significantly associated with BMI. (Table 4.115)

Table 4.115 Mean level of triglyceride level among different BMI subjects (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean Triglyceride Level ± SEM (mg/dL)**	**P value**
Underweight	114.4±1.404	0.000*
Healthy Weight	120.2±0.635	
Overweight	132.3±1.290	
Obese	137.1±2.100	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

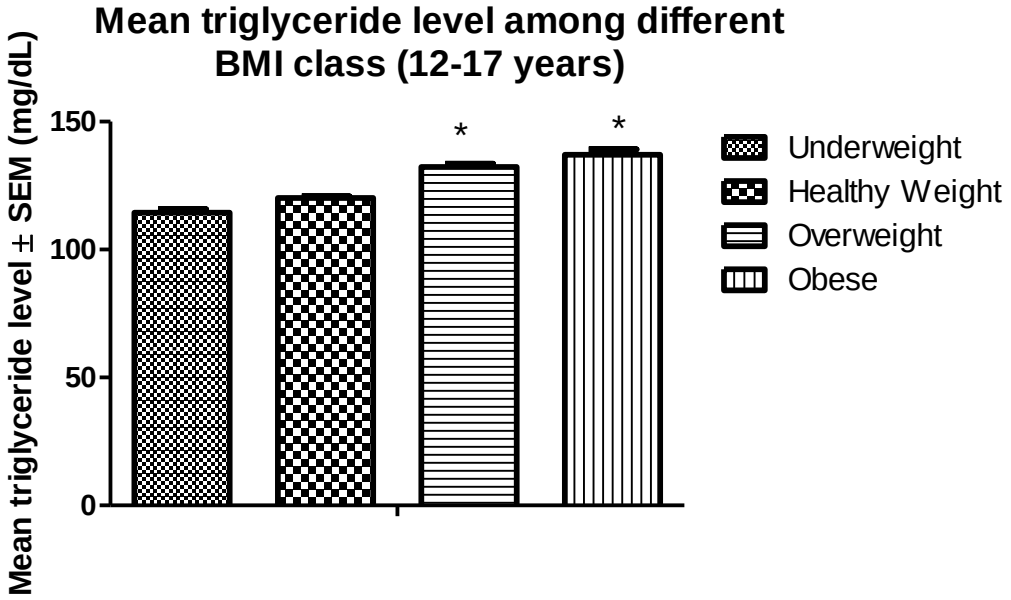

Figure 4.38 Mean level of triglyceride level among different BMI subjects (12-17 years age group)

4.4.13.2 Association of hypertriglyceridemia with BMI (18-35 years age group)

In the age group of 18-35 years, hypertriglyceridemia among obese subject was 36.53% which was the highest among overweight (28.34%) and healthy weight (12.96%). Here in this age group hypertriglyceridemia was absent among underweight subjects. (Table 4.116)

Among the participants of same age group, we noted mean triglyceride level was found the highest in obese group 143.1±2.316 mg/dL following overweight subjects (138.1±1.324 mg/dL), healthy weight subjects (126.2±0.615 mg/dL) and lowest in underweight subjects (116.5±1.529 mg/dL). Additionally, in this study mean triglyceride level was found significantly correlated with BMI demonstrating risk of hypertriglyceridemia is high among overweight and obese subjects than healthy weight subjects. (Table 4.117)

Table 4.116 Association of hypertriglyceridemia with BMI (18-35 Years age group)

18 to 35 Years Age Group			**P value**
BMI Classification	**Total (N = 1,010)**	**Hypertriglyceridemia**	
Underweight	69	0 (0.00%)	< 0.001*
Healthy Weight	702	91 (12.96%)	
Overweight	187	53 (28.34%)	
Obese	52	19 (36.53%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.117 Mean level of triglyceride level among different BMI subjects (18-35 years age group)

18 to 35 Years Age Group		
BMI Classification	**Mean Triglyceride Level ± SEM (mg/dL)**	**P value**
Underweight	116.5±1.529	0.000*
Healthy Weight	126.2±0.615	
Overweight	138.1±1.324	
Obese	143.1±2.316	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

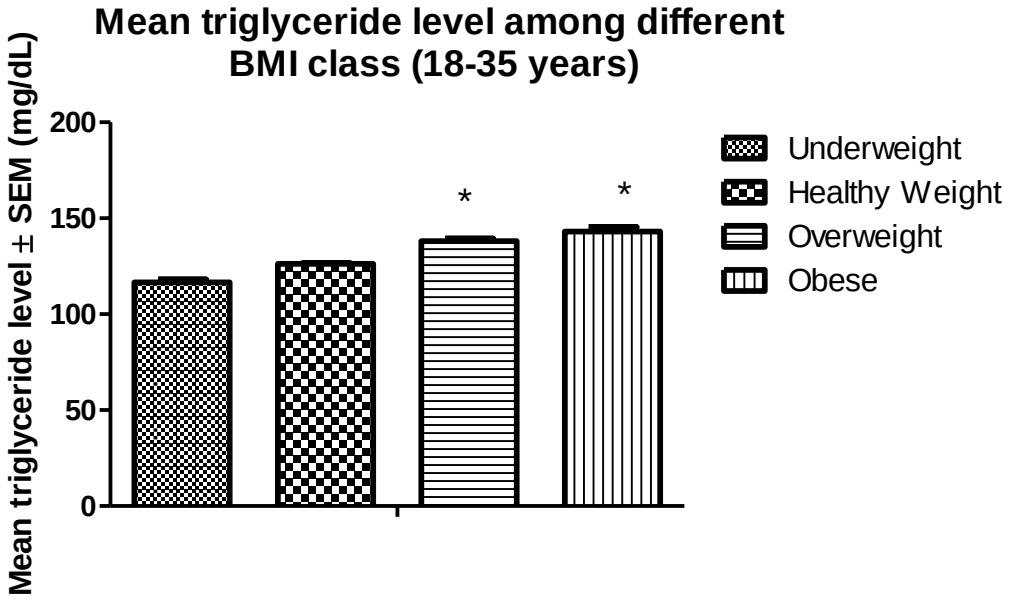

Figure 4.39 Mean level of triglyceride level among different BMI subjects (18-35 years age group)

4.4.13.3 Association of hypertriglyceridemia with BMI (36-55 years age group)

In the age group of 36-55 years, prevalence of hypertriglyceridemia in healthy weight was 30.23%, overweight was 59.27% and in obese was 64.70%. Likewise 18-35 years age group here in this group also we did not find hypertriglyceridemia among underweight participants. Hypertriglyceridemia was found significantly associated with BMI showing increased risk of elevated level of triglycerides with BMI. (Table 4.118)

In present study in the age group of 36-55 years, mean triglyceride level among underweight subjects was the lowest (116.1±1.848 mg/dL) and the highest among obese subjects (151.2±2.675 mg/dL). High mean triglyceride level was found significantly correlated with overweight and obesity among 36-55 years old subjects. (Table 4.119)

Table 4.118 Association of hypertriglyceridemia with BMI (36-55 Years age group)

36 to 55 Years Age Group			**P value**
BMI Classification	**Total (N = 946)**	**Hypertriglyceridemia**	
Underweight	45	0 (0.00%)	< 0.001*
Healthy Weight	602	182 (30.23 %)	
Overweight	248	147 (59.27%)	
Obese	51	33 (64.70 %)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.119 Mean level of triglyceride level among different BMI subjects (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean Triglyceride Level ± SEM (mg/dL)**	**P value**
Underweight	116.1±1.848	0.000*
Healthy Weight	135.2±0.827	
Overweight	144.8±1.365	
Obese	151.2±2.675	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

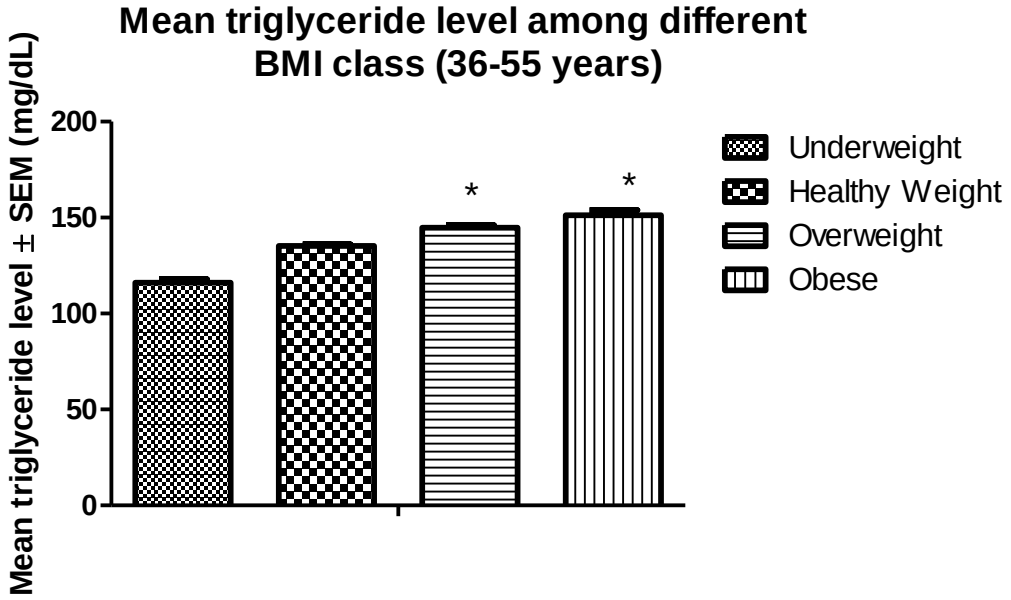

Figure 4.40 Mean level of triglyceride level among different BMI subjects (36-55 years age group)

4.4.14 Association of low HDL-C level with BMI

4.4.14.1 Association of low HDL-C level with BMI (12-17 years age group)

Though, none of the subjects of this age group reported to have abnormally low HDL-C level, mean level of HDL-C was found low among obese subjects (46.3±0.960 mg/dL) and overweight subjects (46.8±0.523 mg/dL) of the age group 12-17 years, which was found significantly increasing with decreasing BMI; healthy weight 51.8±0.323 mg/dL and underweight 50.2±0.577 mg/dL. Low level of HDL-C was found significantly associated with overweight and obesity in this age group stating risk of cardiovascular diseases with higher BMI. (Table 4.120)

Table 4.120 Mean level of HDL-C level among different BMI subjects (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean HDL-C Level ± SEM (mg/dL)**	**P value**
Underweight	50.2±0.577	0.000*
Healthy Weight	51.8±0.323	
Overweight	46.8±0.523	
Obese	46.3±0.960	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

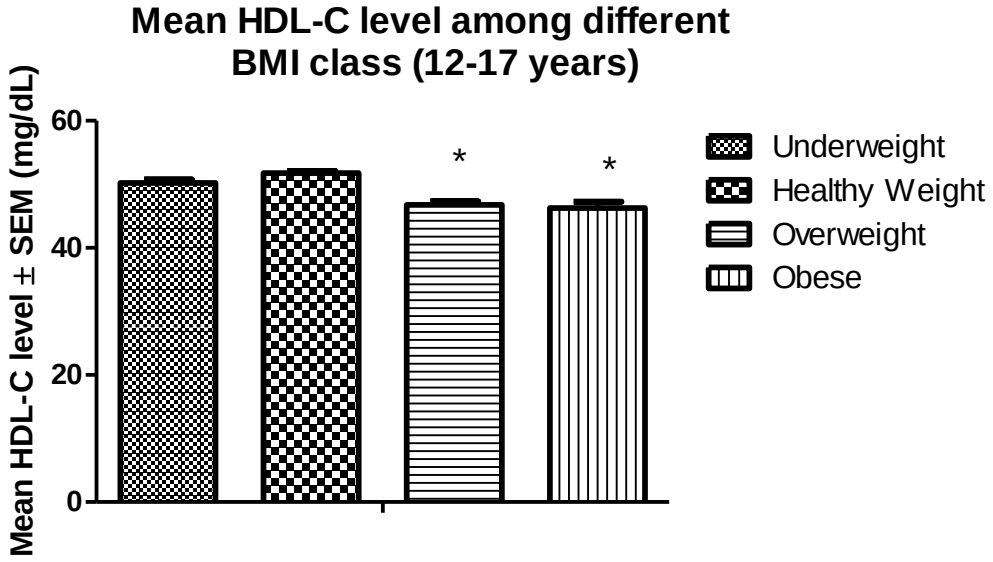

Figure 4.41 Mean level of HDL-C level among different BMI subjects (12-17 years age group)

4.4.14.2 Association of low HDL-C level with BMI (18-35 years age group)

In the age group of 18-35 years, the greater number of obese subjects had low level of HDL-C (65.38%) following overweight subjects (47.05%) and less number of healthy weight subjects (21.65%) reported to have low level of HDL-C. It implies, as BMI increases the level of HDL-C decreases and found significantly associated. (Table 4.121)

Mean HDL-C level was reported high among underweight subjects (48.6±0.626 mg/dL) following healthy weight subjects (46.5±0.287 mg/dL)and found immensely low among overweight and obese subjects (41.6±0.746 mg/dL and 38.5±1.428 mg/dL, respectively). This signifies that overweight and obese subjects had low level of HDL-C than underweight and healthy weight subjects and this correlation was found statistically significant among participants of 18-35 years age group. (Table 4.122)

Table 4.121 Association of low HDL-C with BMI (18-35 Years age group)

18 to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Low HDL-C**	
Underweight	69	0 (0.00%)	< 0.001*
Healthy Weight	702	152 (21.65 %)	
Overweight	187	88 (47.05%)	
Obese	52	34 (65.38%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.122 Mean level of HDL-C level among different BMI subjects (18-35 years age group)

18 to 35 Years Age Group		
BMI Classification	**Mean HDL-C Level ± SEM (mg/dL)**	**P value**
Underweight	48.6±0.626	0.000*
Healthy Weight	46.5±0.287	
Overweight	41.6±0.746	
Obese	38.5±1.428	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

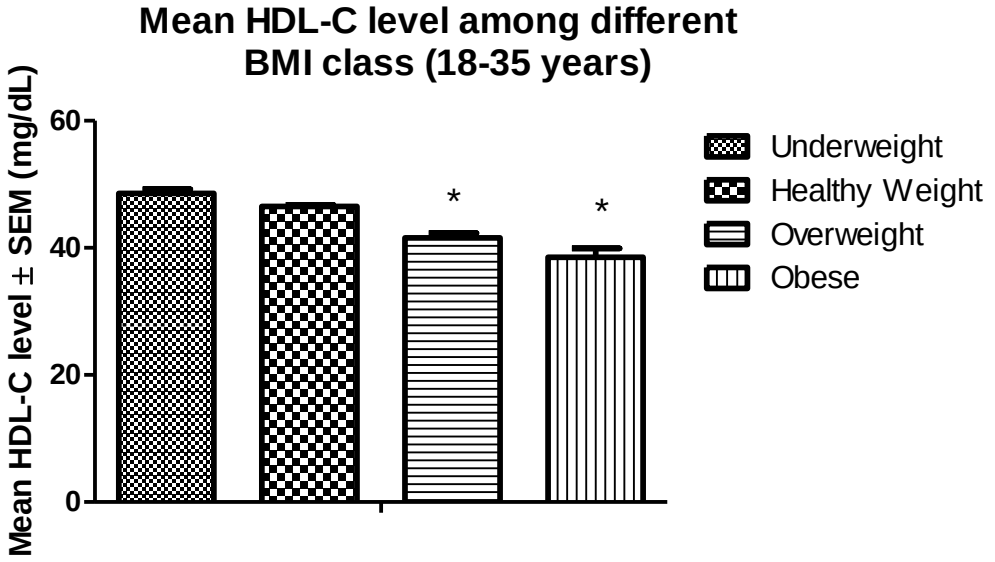

Figure 4.42 Mean level of HDL-C level among different BMI subjects (18-35 years age group)

4.4.14.3 Association of low HDL-C level with BMI (36-55 years age group)

In the age group of 36-55 years, the prevalence low HDL-C was the highest in percentage among obese subjects (80.39%) following overweight subjects (69.75%) and healthy weight subjects (35.21%). We found significant association between low HDL-C level and BMI which indicates risk of HDL-C level decreases with higher BMI. (Table 4.123)

In present study, underweight subjects had high mean level of HDL-C (49.2±0.760 mg/dL) which was decreasing substantially with BMI; likewise healthy weight reported to have 42.1±0.342 mg/dL, overweight had 37.9±0.711 mg/dL and obese were noted to have 36.6±1.330 mg/dL. The association of low HDL-C level was found significant with overweight and obesity. (Table 4.124)

Table 4.123 Association of low HDL-C with BMI (36-55 Years age group)

36 to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Low HDL-C**	
Underweight	45	0 (0.00%)	< 0.001*
Healthy Weight	602	212 (35.21%)	
Overweight	248	173 (69.75%)	
Obese	51	41 (80.39%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.124 Mean level of HDL-C level among different BMI subjects (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean HDL-C Level ± SEM (mg/dL)**	**P value**
Underweight	49.2±0.760	0.000*
Healthy Weight	42.1±0.342	
Overweight	37.9±0.711	
Obese	36.6±1.330	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

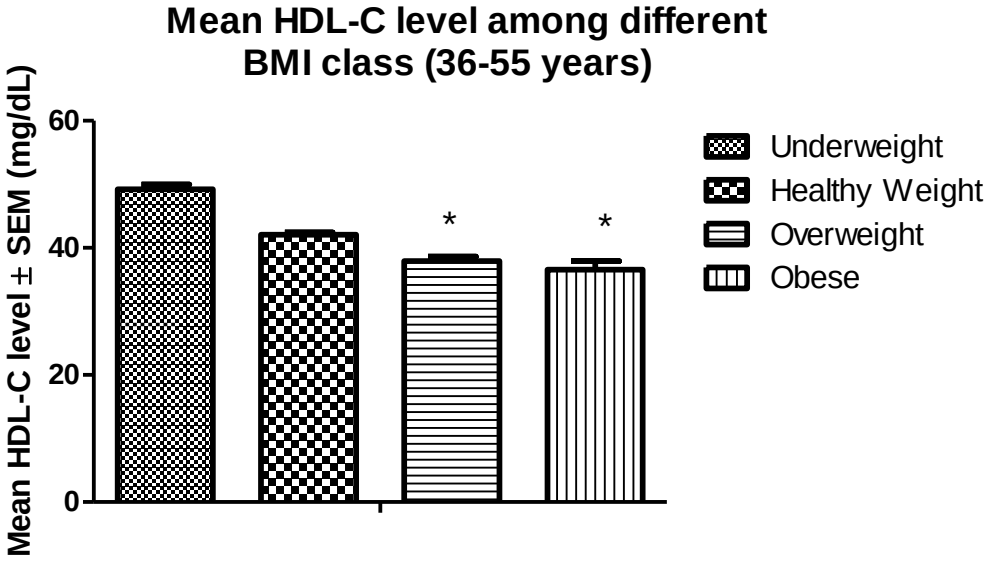

Figure 4.43 Mean level of HDL-C level among different BMI subjects (36-55 years age group)

4.4.15 Association of dyslipidemia with BMI

4.4.15.1 Association of dyslipidemia with BMI (12-17 years age group)

The present study have not reported dyslipidemia among any subjects of 12-17 years age group.

4.4.15.2 Association of dyslipidemia with BMI (18-35 years age group)

Dyslipidemia in the age group of 18-35 years was the highest among obese subjects (36.53%) following overweight (26.73%) and the lowest in healthy weight subjects (8.83%). None of the underweight subjects were reported to have dyslipidemia. We further found risk of dyslipidemia increased with higher BMI and the association was noted significant. (Table 4.125)

Table 4.125 Association of dyslipidemia with BMI (18-35 Years age group)

18 Year to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Dyslipidemia**	
Underweight	69	0 (0.00%)	< 0.001*
Healthy Weight	702	62 (8.83%)	
Overweight	187	50 (26.73%)	
Obese	52	19 (36.53%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

4.4.15.3 Association of dyslipidemia with BMI (36-55 years age group)

In current study, dyslipidemia was reported in 19.10% of healthy weight subjects, which was the lowest compared to overweight subjects (43.54%) and obese subjects (56.86%). In this group again we did not find any underweight subjects with dyslipidemia. (Table 4.126)

Table 4.126 Association of dyslipidemia with BMI (36-55 Years age group)

36 Year to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Dyslipidemia**	
Underweight	45	0 (0.00%)	< 0.001*
Healthy Weight	602	115 (19.10 %)	
Overweight	248	108 (43.54%)	
Obese	51	29 (56.86%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

4.4.16 Association of vitamin D deficiency with BMI

4.4.16.1 Association of vitamin D deficiency with BMI (12-17 years age group)

In the 12-17 years of age group, vitamin D deficiency was the highest in obese subjects (80.00%) among all other subjects. Likewise, vitamin D deficiency in overweight was found 70.27%, in healthy weight subjects it was 56.63% whereas the lowest in underweight subjects (28.20%). Vitamin D deficiency was found to have significant association with higher BMI. (Table 4.127)

Among the age group of 12-17 years, mean vitamin D level was found lowest among obese subjects (17.2±0.740 ng/dL) following overweight subjects (19.6±0.593 ng/dL) Healthy weight found to have vitamin D level 22.7±0.287 ng/dL. Whilst mean level was highest among underweight subjects (24.8±0.487 ng/dL). It was noted that risk of vitamin D deficiency increases with BMI and significant association was found between them. (Table 4.128)

Table 4.127 Association of vitamin D deficiency with BMI (12-17 Years age group)

12 to 17 Years Age Group			**P value**
BMI Classification	**Total (N = 456)**	**Vitamin D Deficiency**	
Underweight	78	22 (28.20%)	< 0.001*
Healthy Weight	279	158 (56.63%)	
Overweight	74	52 (70.27%)	
Obese	25	20 (80.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05.

Table 4.128 Mean level of vitamin D among different BMI subjects (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean Vitamin-D Level ± SEM (ng/dL)**	**P value**
Underweight	24.8±0.487	0.000*
Healthy Weight	22.7±0.287	
Overweight	19.6±0.593	
Obese	17.2±0.740	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

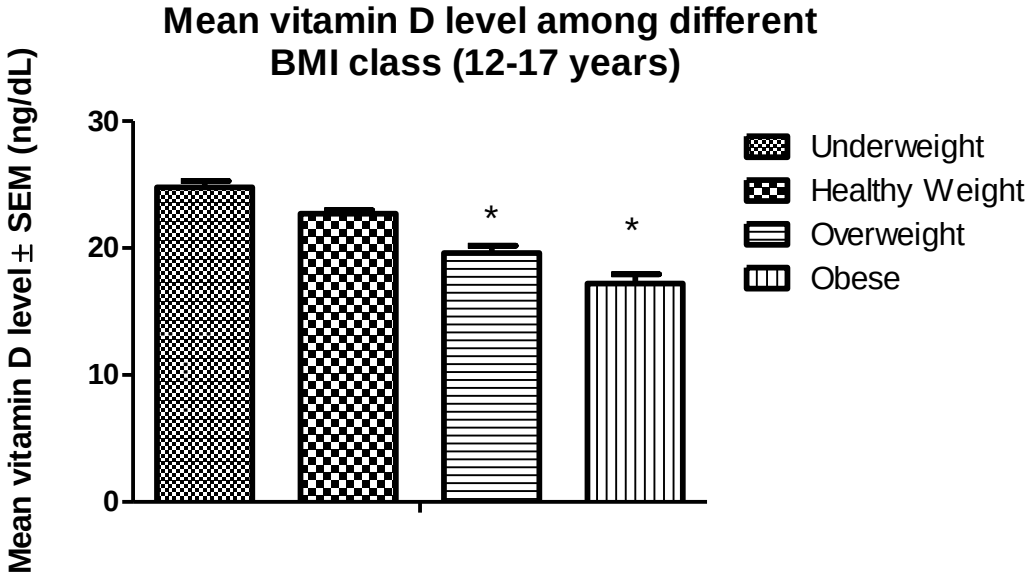

Figure 4.44 Mean level of vitamin D among different BMI subjects (12-17 years age group)

4.4.16.2 Association of vitamin D deficiency with BMI (18-35 years age group)

Prevalence of vitamin D deficiency, which was found the lowest in underweight subjects (59.42%) increasing with healthy weight subjects (63.39%), overweight (79.67%) and 92.30% in obese subjects. Vitamin D deficiency was significantly associated in the age group of 18-35 years. (Table 4.129)

In present study, mean vitamin D level among underweight (21.6±0.590 ng/dL) and healthy weight subjects (20.3±0.155 ng/dL) was found significantly higher compared to overweight (16.7±0.263 ng/dL) and obese (15.9±0.374 ng/dL) subjects. Among the age group of 18-35 years, strong significant association was found between BMI and vitamin D deficiency which explains risk of vitamin D deficiency is higher among overweight and obese participants. (Table 4.130)

Table 4.129 Association of vitamin D deficiency with BMI (18-35 Years age group)

18 to 35 Years Age Group			**P value**
BMI Classification	**Total (N = 1,010)**	**Vitamin D Deficiency**	
Underweight	69	41 (59.42%)	< 0.001*
Healthy Weight	702	445 (63.39%)	
Overweight	187	149 (79.67%)	
Obese	52	48 (92.30%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.130 Mean level of vitamin D among different BMI subjects (18-35 years age group)

18-35 years age group		
BMI Classification	**Mean Vitamin-D Level ± SEM (ng/dL)**	**P value**
Underweight	21.6±0.590	0.000*
Healthy Weight	20.3±0.155	
Overweight	16.7±0.263	
Obese	15.9±0.374	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

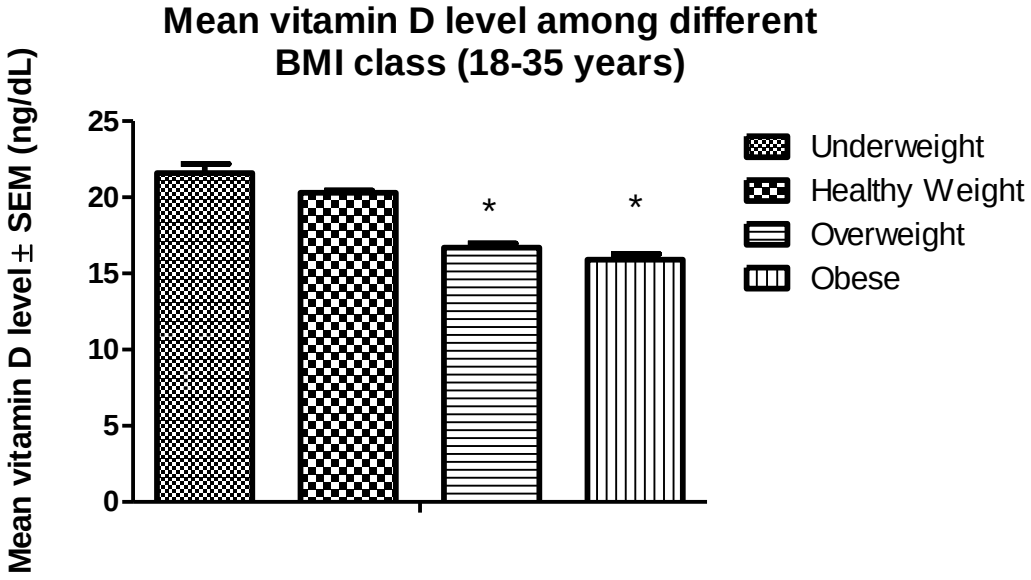

Figure 4.45 Mean level of vitamin D among different BMI subjects (18-35 years age group)

4.4.16.3 Association of vitamin D deficiency with BMI (36-55 years age group)

In the age group of 36-55 years, of all BMI categories, obese were reported to have more prevalence of vitamin D deficiency (92.15%), the deficiency was diminished in overweight (79.83%) followed by healthy weight (69.43%) and underweight (64.44%). Vitamin D deficiency was found significantly associated with higher BMI in this group. (Table 4.131)

The current study among the age group of 36-55 years noted risk of vitamin D deficiency increases with BMI since mean Vitamin D level was the highest among underweight subjects (21.1±0.730 ng/dL) and the lowest among obese subjects (14.9±0.476 ng/dL). Additionally, lower mean vitamin D level found to have strong significant association with overweight and obesity in present study. (Table 4.132)

Table 4.131 Association of vitamin D deficiency with BMI (36-55 Years age group)

36 to 55 Years Age Group			**P value**
BMI Classification	**Total (N = 946)**	**Vitamin D Deficiency**	
Underweight	45	29 (64.44%)	< 0.001*
Healthy Weight	602	418 (69.43%)	
Overweight	248	198 (79.83%)	
Obese	51	47 (92.15%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.132 Mean vitamin D level among subjects with different BMI (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean Vitamin-D Level ± SEM (ng/dL)**	**P value**
Underweight	21.1±0.730	0.000*
Healthy Weight	20.3±0.183	
Overweight	16.8±0.235	
Obese	14.9±0.476	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

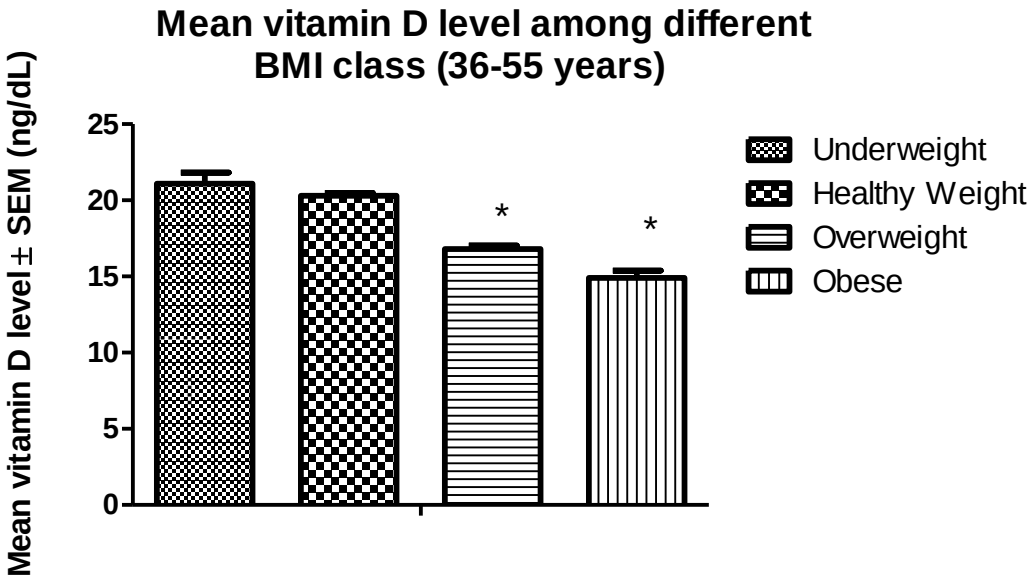

Figure 4.46 Mean vitamin D level among subjects with different BMI (36-55 years age group)

Above findings implies that in all three age groups, as BMI increases, the risk of vitamin D deficiency accelerates.

4.4.17 Association of hyperinsulinemia with BMI

4.4.17.1 Association of hyperinsulinemia with BMI (12-17 years age group)

In current study, we did not found hyperinsulinemia among any subjects of 12-17 years age group.

However, significantly increased level of insulin was noted with BMI. Mean insulin level was nearly similar among obese (21.5±0.540 mIU/L) and overweight (21.3±0.291 mIU/L) subjects. Whilst it was found lower among healthy weight (18.1±0.216 mIU/L) and underweight participants (13.7±0.294 mIU/L). In the age group of 12-17 years higher mean level of insulin was found significantly associated with obese and overweight category of BMI. (Table 4.133)

Table 4.133 Mean insulin level among subjects with different BMI (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean insulin Level ± SEM (mIU/L)**	**P value**
Underweight	13.7±0.294	0.000*
Healthy Weight	18.1±0.216	
Overweight	21.3±0.291	
Obese	21.5±0.540	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

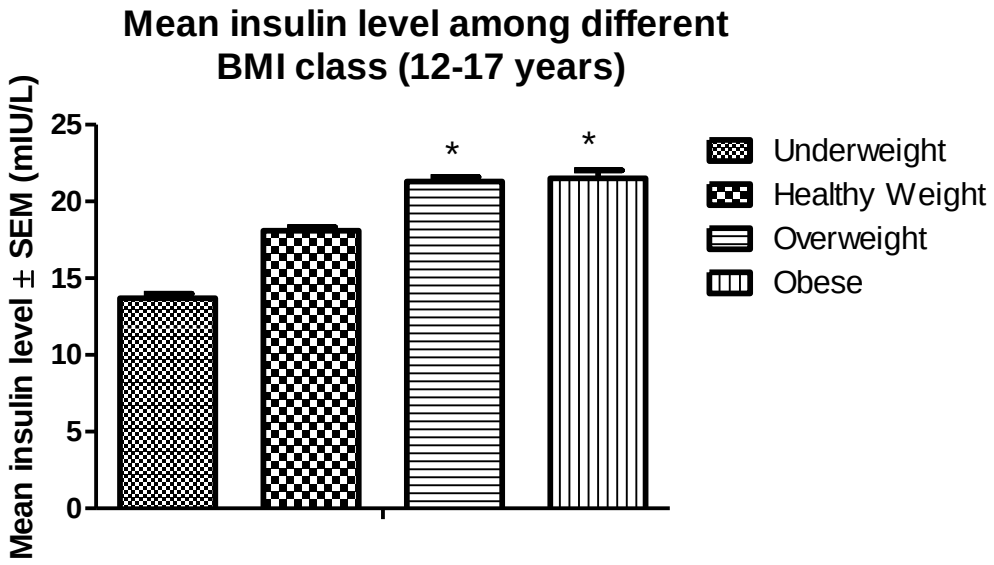

Figure 4.47 Mean insulin level among subjects with different BMI (12-17 years age group)

4.4.17.2 Association of hyperinsulinemia with BMI (18-35 years age group)

In the age group of 18-35 years, of all BMI categories, overweight and obese were reported to have more and nearly similar prevalence of hyperinsulinemia (35.82% and 36.53%, respectively). Prevalence of hyperinsulinemia was found decreasing with BMI; healthy weight subjects had 11.53% and underweight subjects had 2.89% prevalence of hyperinsulinemia. Elevated insulin level found to have significant association with higher BMI. (Table 4.134)

In the same age group, we found risk of hyperinsulinemia increases with BMI since mean insulin level was the lowest among underweight subjects (16.6±0.494 mIU/L) and the highest among obese subjects (25.1±0.652 mIU/L). Additionally, higher mean insulin level found to have strong significant association with incident overweight and obesity in present study. (Table 4.135)

Table 4.134 Association of hyperinsulinemia with BMI (18-35 Years age group)

18 to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Hyperinsulinemia**	
Underweight	69	2 (2.89 %)	< 0.001*
Healthy Weight	702	81 (11.53%)	
Overweight	187	67 (35.82%)	
Obese	52	19 (36.53%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.135 Mean insulin level among subjects with different BMI (18-35 years age group)

18 to 35 Years Age Group		
BMI Classification	**Mean insulin Level ± SEM (mIU/L)**	**P value**
Underweight	16.6±0.494	0.000*
Healthy Weight	19.2±0.155	
Overweight	22.4±0.424	
Obese	25.1±0.652	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

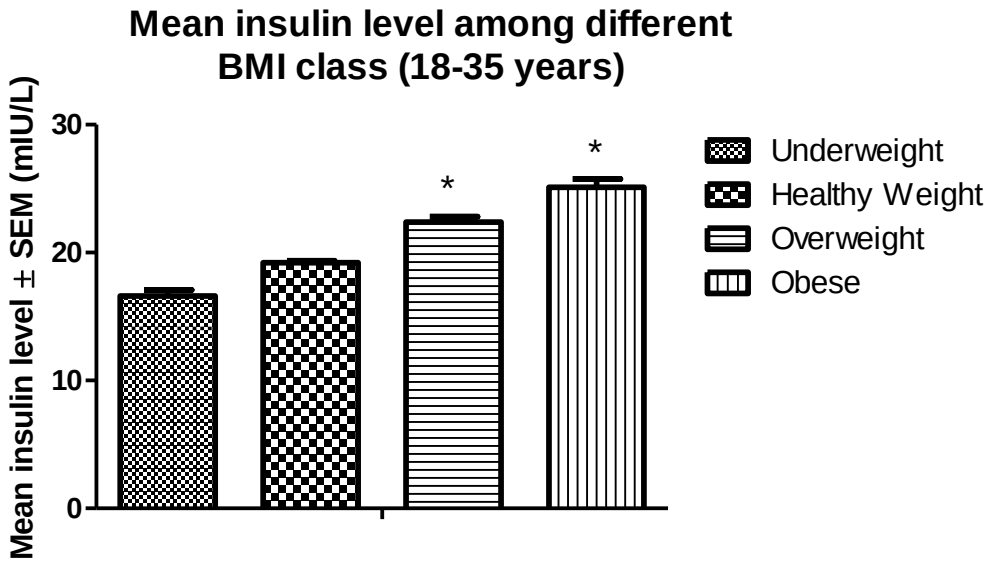

Figure 4.48 Mean insulin level among subjects with different BMI (18-35 years age group)

4.4.17.3 Association of hyperinsulinemia with BMI (36-55 years age group)

Hyperinsulinemia, which was found the lowest in underweight subjects (4.44%) increasing with healthy weight subjects (22.92%), overweight (50.80%) and 56.86% in obese subjects in the age group of 36-55 years. Hyperinsulinemia was found significantly associated with BMI in this group. (Table 4.136)

In present study, mean insulin level among underweight (18.3±0.507 mIU/L) and healthy weight subjects (21.2±0.200 mIU/L) was found significantly higher compared to overweight (24.7±0.330 mIU/L) and obese (26.6±0.672 mIU/L) subjects. Among the age group of 36-55 years, strong significant association was found between BMI and hyperinsulinemia. (Table 4.137)

Table 4.136 Association of hyperinsulinemia with BMI (36-55 Years age group)

36 to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Hyperinsulinemia**	
Underweight	45	2 (4.44%)	< 0.001*
Healthy Weight	602	138 (22.92%)	
Overweight	248	126 (50.80%)	
Obese	51	29 (56.86%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.137 Mean insulin level among subjects with different BMI (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean insulin Level ± SEM (mIU/L)**	**P value**
Underweight	18.3±0.507	0.000*
Healthy Weight	21.2±0.200	
Overweight	24.7±0.330	
Obese	26.6±0.672	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

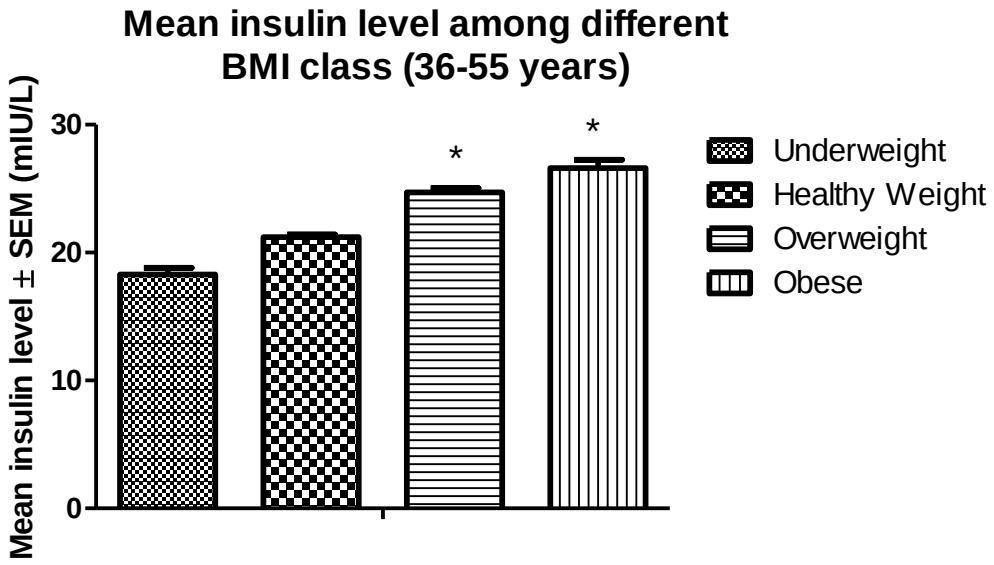

Figure 4.49 Mean insulin level among subjects with different BMI (36-55 years age group)

4.4.18 Association of elevated C-reactive protein with BMI

4.4.18.1 Association of elevated C-reactive protein with BMI (12-17 years age group)

The current study demonstrated, neither underweight nor healthy weight subjects of age group 12-17 years had elevated C-reactive protein level. The prevalence of elevated C-reactive protein level among overweight and obese of same age group was 1.35% and 4.00%, respectively. (Table 4.138)

Among the age group of 12-17 years, the mean C-reactive protein level was found increasing with BMI. Mean C-reactive protein level was found the highest among obese subjects (3.5 mg/L) following overweight subjects (3.2 mg/L). Healthy weight participants found to have C-reactive protein level 1.13±0.029 mg/L, whilst mean C-reactive protein level was lowest among underweight subjects (0.79±0.045 mg/L). It was noted that level of C-reactive protein increases with BMI and statistically significant association was found between them. (Table 4.139)

Table 4.138 Association of elevated C-reactive protein with BMI (12-17 Years age group)

12 to 17 Years Age Group			P value
BMI Classification	**Total (N = 456)**	**Elevated C-reactive protein**	
Underweight	78	0	-
Healthy Weight	279	0	
Overweight	74	1 (1.35 %)	
Obese	25	1 (4.00%)	

Table 4.139 Mean C-reactive protein level among subjects with different BMI (12-17 years age group)

12 to 17 Years Age Group		
BMI Classification	**Mean C-reactive protein Level ± SEM (mg/L)**	**P value**
Underweight	0.79±0.045	0.000*
Healthy Weight	1.13±0.029	
Overweight	3.2	
Obese	3.5	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

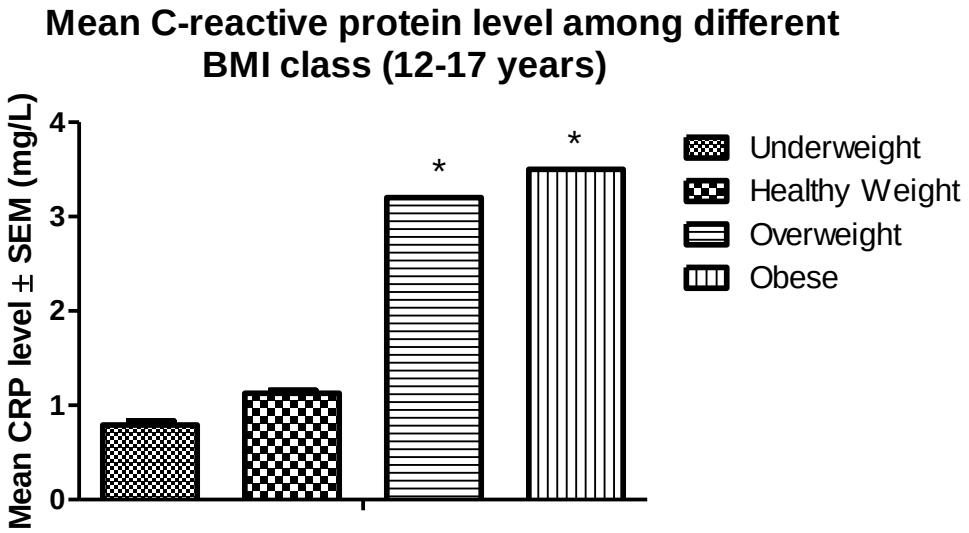

Figure 4.50 Mean C-reactive protein level among subjects with different BMI (12-17 years age group)

4.4.18.2 Association of elevated C-reactive protein with BMI (18-35 years age group)

Elevated C-reactive protein, which was found lowest in healthy weight subjects (9.11%) increasing with overweight subjects (15.50%) and 25.00% in obese subjects in the age group of 18-35 years. None of the underweight subjects noted to have elevated level of C-reactive protein. Elevated C-reactive protein noted to have significant association with BMI in this age group. (Table 4.140)

In present study, mean C-reactive protein level among underweight (1.03±0.060 mg/L) and healthy weight subjects (1.48±0.030 mg/L) was found significantly lower compared to overweight (2.11±0.058 mg/L) and obese (2.35±0.124 mg/L) subjects. Among the age group of 18-35 years, strong significant association was found between BMI and elevated C-reactive protein. (Table 4.141)

Table 4.140 Association of elevated C-reactive protein with BMI (18-35 Years age group)

18 to 35 Years Age Group			P value
BMI Classification	**Total (N = 1,010)**	**Elevated C-reactive protein**	
Underweight	69	0	< 0.001*
Healthy Weight	702	64 (9.11%)	
Overweight	187	29 (15.50 %)	
Obese	52	13 (25.00%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.141 Mean C-reactive protein level among subjects with different BMI (18-35 years age group)

18 to 35 Years Age Group		
BMI Classification	**Mean C-reactive protein Level ± SEM (mg/L)**	**P value**
Underweight	1.03±0.060	0.000*
Healthy Weight	1.48±0.030	
Overweight	2.11±0.058	
Obese	2.35±0.124	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

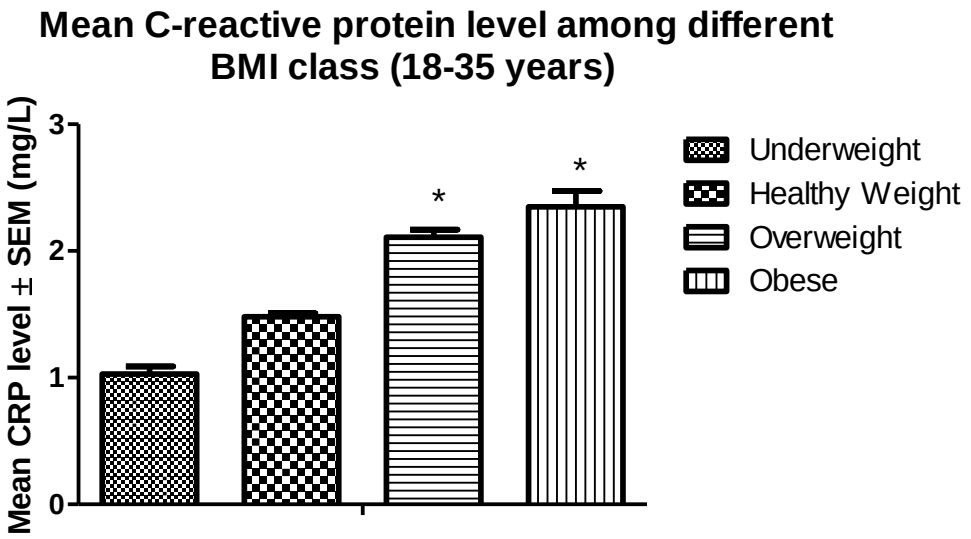

Figure 4.51 Mean C-reactive protein level among subjects with different BMI (18-35 years age group)

4.4.18.3 Association of elevated C-reactive protein with BMI (36-55 years age group)

In the age group of 36-55 years, of all BMI categories, obese were reported to have more prevalence of elevated C-reactive protein (35.29%), the prevalence of elevated level of C-reactive protein was diminished in overweight (29.43%) followed by healthy weight (17.94%) and underweight (4.44%). Elevated C-reactive protein found to have significant association with BMI. (Table 4.142)

In the same age group, we found risk of elevation of C-reactive protein increases with BMI since mean level of C-reactive protein was the lowest among underweight subjects (1.08±0.104 mg/L) and the highest among obese subjects (2.53±0.126 mg/L). Furthermore, in this age group also mean C-reactive protein level found to have strong significant association with BMI. (Table 4.143)

Table 4.142 Association of elevated C-reactive protein with BMI (36-55 Years age group)

36 to 55 Years Age Group			P value
BMI Classification	**Total (N = 946)**	**Elevated C-reactive protein**	
Underweight	45	2 (4.44%)	< 0.001*
Healthy Weight	602	108 (17.94%)	
Overweight	248	73 (29.43%)	
Obese	51	18 (35.29%)	

Analysed by One-way chi-square test for goodness of fit. * indicates statistical significance at p value less than 0.05

Table 4.143 Mean C-reactive protein level among subjects with different BMI (36-55 years age group)

36 to 55 Years Age Group		
BMI Classification	**Mean C-reactive protein Level ± SEM (mg/L)**	**P value**
Underweight	1.08±0.104	0.000*
Healthy Weight	1.79±0.036	
Overweight	2.37±0.050	
Obese	2.53±0.126	

Analysed by One-way ANOVA test. * indicates statistical significance at p value less than 0.05.

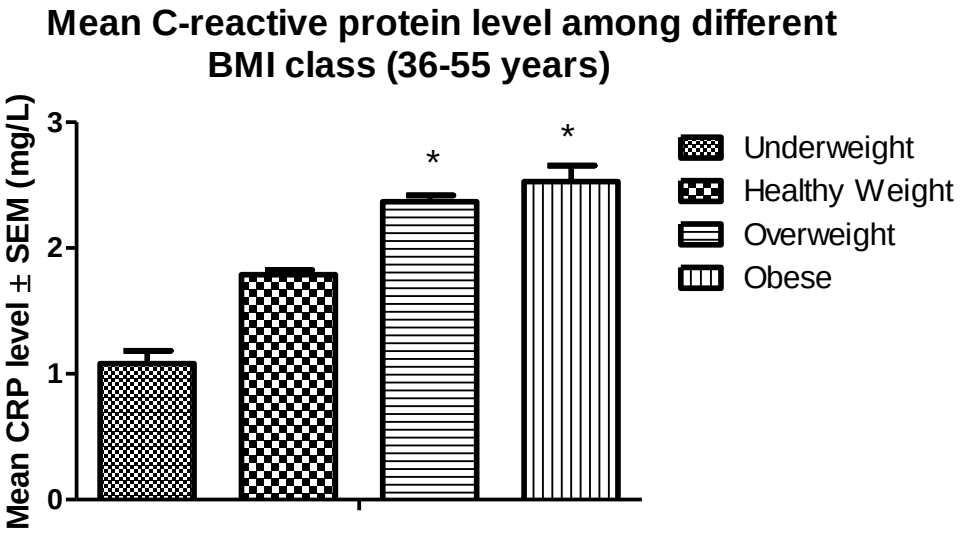

Figure 4.52 Mean C-reactive protein level among subjects with different BMI (36-55 years age group)

5. Discussion

Prediabetes is considered as an intermediate diabetes state or high risk-state of development of diabetes, which is defined by glycaemic variables that are elevated compared to normal but still lower than the diabetics (Tabak et al, 2012). Reports from expert panel of American Diabetes Association, 70% participants with prediabetes will eventually develop diabetes. Studies reported that roughly 5 – 10% prediabetes subjects converted to diabetes annually though the rate of conversion relies on characteristics of population and definition of prediabetes (Forouhi et al, 2007; Nathan et al, 2007). Additionally, researchers also documented, women with gestational diabetes have a 20%–60% risk of developing diabetes 5 to 10 years after pregnancy (Lauenborg et al, 2004; Bellamy et al, 2009). Thus, the early diagnosis of prediabetes provides an opportunity to healthcare professionals to recognize the patients who have greater risk for diabetes and to implement an intervention that helps such patients in delaying as well as to prevent diabetes and its associated complications.

Taranikanti et al in 2014 conducted study on South Indian rural adolescent school students and reported 7.1% prevalence of prediabetes, which in compared to our result was slightly higher, as our result shown 5.09% prevalence of prediabetes among 12-17 years old school going children. Narayanappa et al reported 3.7% prevalence of prediabetes in 5-10 years subjects of Mysore city (Narayanappa et al, 2011). Prevalence of prediabetes reported by Alam et al on subjects of more than 20 years age of Bangladesh was 6.6% (Alam et al, 2016). Muthunarayanan, et al in 2013 reported 8.5% prevalence of prediabetes and 10.1% prevalence of diabetes among rural adults of Tamil Nadu of age more than 20 years. Prevalence of prediabetes in our study among 18-35 years old participants was 28.81% and among 36-55 years old participants was 33.19%, demonstrating higher prevalence of prediabetes in our study when compared with Alam et al and Muthunarayanan et al. Whereas our results of prevalence of diabetes was found nearly similar with results of Muthunarayanan et al. (Muthunarayanan et al, 2015). Similarly, Dasappa et al in 2016 has reported 11.5% prevalence of prediabetes and 12.3% prevalence of diabetes among urban slum of Bangalore of more than 35 years age (Dasappa et al, 2015) and Gupta et al in 2016 reported 17.8% and 15.7% prevalence of prediabetes and diabetes respectively on adult population of age more than 20 years of middle-class family (Gupta et al, 2016). When our

results are compared with studies of Dasappa et al and Gupta et al, prediabetes prevalence was found higher in our study while diabetes prevalence was found nearly equal. ICMR study conducted on subjects of urban and rural area of four different parts of India and reported 8.3%, 12.8%, 8.1% and 14.6% prevalence of prediabetes of Tamil Nadu, Maharashtra, Jharkhand and Chandigarh respectively on overall subjects (Anjana RM, 2011).

The present study shown mean level of fasting blood sugar among 12-17 years, 18-35 years and 36-55 years was 83.4 ± 1.063 mg/dL, 94.7 ± 0.831 mg/dL and 107.3 ± 1.011 mg/dL, respectively. Furthermore, we found higher mean fasting blood sugar level among prediabetes participants than normal subjects. Hussain et al noted 4.75±0.04 mmol/L mean fasting blood sugar level of university students of Bangladesh (Hussain et al, 2017); markedly lower compared to our findings. Rare studies reported an association of mean fasting blood sugar level with age. The current study demonstrated risk of hyperglycaemia increases with age. Supporting our findings, Kutty et al in Kerala, India reported low levels of plasma glucose in the young age group (20-29 years) and higher plasma glucose levels in the old age group (>69 years) (Kutty et al, 2002).The difference in fasting blood sugar level between different age group could be multi-factorial; sex hormones, visceral adiposity, and muscle mass have been reported to regulate glucose metabolism (Grossmann et al, 2014; Otsuki et al, 2007; Kalyani et al, 2012; Fox et al, 2007).

In present study, overweight prevalence among participants of 12-17, 18-35 and 36-55 years age group was reported 16.23%, 18.51% and 26.22%, respectively whereas obesity prevalence was found 5.48%, 5.15% and 5.39%, respectively.

The prevalence of overweight and obesity among South Indian adolescent subjects was 14% (Prasad et al, 2016). Studies of Kotian et al (South Karnataka), Gamit et al (Surat) and Krutarth et al (Ahmedabad) on adolescents reported less prevalence of overweight than our results whilst prevalence of obesity was as consistent as our findings in these all three studies (Kotian et al, 2010; Gamit et al, 2015; Krutarth et al,2012). Our results for prevalence of overweight was lower than the findings reported by Baradol et al in Karnataka, Goyal et al and Chattwal et al (Baradol et al, 2014; Goyal et al, 2010; Chattwal et al, 2004). Researchers from Punjab, Jaipur and Chennai revealed that the prevalence of

overweight was 10-15%, which is nearer to our findings. While prevalence of obesity was as high as (5-11%) (Chattwal et al, 2004; Sidhu et al, 2005; Bansal et al, 2013; Thakur et al, 2009).

Studies on adults, performed by Sen et al and Gupta et al reported consistent results on prevalence of overweight with our data whereas findings on obesity prevalence was higher than current study (Sen et al, 2013; Gupta et al, 2016). Furthermore, the findings of Chauhan et al, Kumar et al and Sidhu et al on prevalence of both overweight and obesity documented higher prevalence in comparison to present study (Chauhan et al, 2015; Kumar et al, 2015; Sidhu et al, 2005). Study on North Indian subjects of 20-40 years reported overweight and obesity 16.3% and 5.1% respectively, which was found consistent with current study findings (Masoodi et al, 2010).

The present study documented high prevalence of prediabetes among overweight and obese subjects than healthy weight subjects in all three-age groups. We further noted that in all three age group the mean BMI was higher among subjects with prediabetes than non-prediabetics in current study. We also found no risk of prediabetes among underweight subjects with the prevalence of 0%. This finding explains the risk of prediabetes increases with BMI. Furthermore, among all three age groups, we noted BMI was significantly associated with incident prediabetes. Controlling excess weight is an important way to lower risk of onset of prediabetes and diabetes. Similarly, Dasappa et al documented that fasting glucose was positively associated with BMI only in women (Dasappa et al, 2015). Furthermore, other studies were in support of the present study by documenting the positive relationship between BMI and prediabetes (Pandeya et al., 2017; Anjana et al., 2011). Studies from the countries outside India also reported the association between BMI and prediabetes which was found consistent with present study (Bosi et al, 2009; Chen et al, 2010). Whereas, contrary reports from the studies performed by Lee et al (2011) and Gupta et al (2008) stated, there was no significant difference between prediabetes and BMI.

Previously it has been reported that family history of diabetes is associated with risk of diabetes (Valdez et al, 2007). However, present study documents negative association of family history of diabetes with risk of prediabetes with prevalence of 3.70% in 12-17 years. Reasons may explain the finding among 12-17 years age group that the overall prevalence

of prediabetes was very low in current study. Additionally, prediabetes is uncommon in adolescents. Whereas, in the age group of 18-35 years and 36-55 years, strong positive correlation between family history of diabetes with incident prediabetes was noted with prevalence of prediabetes among subjects with positive family history of diabetes 48.44% in 18-35 years and 73.78% in 36-55 years age group. These findings signify that family history of diabetes is an important predisposing factor for prediabetes. The current study findings were consistent with the findings of Wagner et al in 2013; author reported family history of diabetes is an inevitable risk factor for prediabetes, especially impaired fasting glucose and impaired glucose tolerance (Wagner et al, 2013). Additionally, studies form German and Sweden found 40% and 50% risk of prediabetes in participants with family history of diabetes (Wagne et al, 2013; Hilding et al, 2006). Whereas Dasappa et al on urban slums of Bangalore documented there is a strong association of diabetes with positive family history of diabetes, nonetheless an author has not reported association of prediabetes with positive family history of diabetes (Dasappa et al, 2015).

Very scanty studies have reported an association of family histories of obesity, thyroid and hypertension with risk of prediabetes. However, the findings of present study shown family history of obesity, thyroid and hypertension were considered as predisposing risk factor for development of prediabetes among participants of 18-35 and 36-55 years age group. Whilst family history of thyroid and hypertension found to have significant association with prediabetes in 12-17 years age group.

In current study we found 12.28%, 27.62% and 29.28% prevalence of pre-hypertension among 12-17, 18-35 and 36-55 years age group, respectively. Prevalence of hypertension was 5.04% in 12-17 years, 22.87% in 18-35 years and 31.92% in 36-55 years age group. We further noted that among subjects of all age groups the mean fasting blood sugar level was higher among pre-hypertensive and hypertensive subjects than subjects with normal blood pressure and shown significant association of elevated blood pressure with mean fasting blood sugar level. Narayanappa et al have reported 2.8% and 2.4% prevalence of pre-hypertension and hypertension on 10 to 16 years urban children of Mysore city (Narayanappa et al, 2012). Similarly, a study performed by Fallah et al in 2014 reported 3.3% of pre-hypertension and 6.8% of hypertension on Iranian children and adolescents (Fallah Z, 201). These findings of both the above studies regarding prevalence of pre-

hypertension was found lower than current study, whilst hypertension prevalence in study of Fallah et al was nearly similar with the present study. Ray et al have reported 79.8% prevalence of pre-hypertension in military adults including officers and other ranks of Southern India of age group 18 – 50 years (Ray et al, 2011). This study noted a significantly very high prevalence of pre-hypertension in an apparently healthy military population than current results. In 2013, one report was published by Srinivas et al on 18-35-year-old subjects of Andhra Pradesh and documented 30.15% prevalence of pre-hypertension and 7.75% prevalence of hypertension (Srinivas et al, 2013). The results of current study were consistent with the prevalence of pre-hypertension whilst the prevalence of hypertension was reported low compared to ours. Reddy et al reported 7.1% and 46.7% prevalence of pre-hypertension among college going students of age 18-21 years of urban and rural area of Udupi district, Karnataka (Reddy et al, 2015). Wang et al conducted study on subjects of non-Hispanic white, non-Hispanic black, Mexican American and other American adults of aged 18 years and above and reported 31.2%, 30.4%, 30.9% and 31.0% of pre-hypertension and 27.7%, 32.6%, 18.1% and 23.7% of hypertension respectively (Wang et al, 2004). This result on pre-hypertension and hypertension prevalence among young aged group warns about possible cardiovascular risks and forewarn us to be alert and need to take precautions before it becomes too late.

Prehypertension and prediabetes are the paramount risk factors of cardiovascular disease. Coexisting prehypertension and prediabetes might have more solemn consequences regarding cardiovascular diseases than expected with either prehypertension or prediabetes alone (Wu et al, 2011). Furthermore, development of hypertension is likelihood in diabetes patients (ADA, 2002; Channanath et al, 2013; Lago et al, 2007). In current study, the prevalence of coexisting prehypertension or hypertension and prediabetes in the age group was significantly higher, pointing towards the close association between the two important cardiovascular risk factors. Wu et al documented 11% prevalence of coexisting pre-hypertension and prediabetes among subjects of 18 years and older of Northern and North-eastern China (Wu et al, 2011); the evidences of this study were lower than ours. Corresponding study was conducted by Muthunarayanan et al in 2015 on subjects of age 20 years and above of Tamil Nadu and found prevalence of prediabetes and diabetes was higher among subjects with systolic hypertension (> 140 mmHg) and the association of systolic hypertension and prediabetes was found statistically significant (Muthunarayanan

et al, 2015). Anjana et al in ICMR-INDIAB study supporting the findings of current study and Muthunarayanan et al's study by reporting prediabetic and diabetic conditions were significantly associated with hypertension (Anjana et al, 2011). Likewise, hypertension is one of the risk factors for the development of prediabetes, reported by Sushma et al (2011). Balagopal et al. had reported, increase in systolic and diastolic BP is significantly associated with the increase in respondents' blood glucose levels (Balagopal et al, 2008) which was consistent with our findings.

Unhealthy lifestyle such as physical inactivity and improper diet puts individual at risk of chronic non-communicable disease including type 2 diabetes and cardiovascular diseases. According to ICMR studies in 2014 reported 66.8%, 60.0%, 55.2% and 34.9% inactivity amongst population of Chandigarh, Tamilnadu, Maharashtra and Jharkhand, respectively. According to this study, the estimated number of inactive individuals from above prevalence in India would be 392 million which showed less than 10% population of India was inactive. This study also found 50% subjects of rural and 65% subjects of urban area were physically inactive which was lesser than our findings of urban subjects (Anjana et al, 2014).

In present study we noted that prevalence of participation in exercise and physical activity declines with age. The important role of physical activity in promoting functional health, delaying or preventing non-communicable disease such as osteoporosis, coronary artery disease, non-insulin-dependent diabetes mellitus and disability, and reducing mortality has been established throughout years (Haley et al, 2010; Hubert et al, 2002; Jonker et al, 2006). Kotian et al documented risk of overweight was 21 times higher among those participating < two hour/weeks in any type of physical activity (Kotian et al, 2010).

It is ubiquitously accepted that physical activity in any forms is considered beneficial to the prevention of many chronic diseases. A study conducted in a rural Demographic Surveillance Site in eastern Uganda noted that person who met the WHO minimum recommended physical activity level had a significantly lower risk of abnormal glucose regulation (Mayega et al, 2013). A report of meta-analysis published by Aune et al in 2015 said that various types of physical activity were beneficial to the prevention of diabetes and decrease the risk of diabetes (Aune et al, 2015). Furthermore, several studies reported that

physical activity could improve insulin sensitivity and glucose tolerance and then delay the onset of diabetes in prediabetes subjects (Knowler et al, 2002; Lindstrom et al, 2003; Malin et al, 2012; Mensink et al, 2003). These all findings of above stated researchers were consistent with present study as study found significant association between physical activity and incidence prediabetes which claimed that physical activity had protective effect on prediabetes in all age groups. Furthermore, we noted that greater number of subjects were inactive to participate in exercise. This was due to in India physical activity levels in the occupational domain decline; majority of the people must spend extra time for their leisure physical activity. To combat this scourge, the Government should provide the facilities for individuals in both urban and rural areas to engage in recreational physical activities. This could be done by doing robust mass awareness among the citizens of India.

It is generally said that lacking physical leisure activities like playing indoor and outdoor games would encourage to have sedentary lifestyle, since such activities play a pivotal role to have both physical and mental healthy life. The current study among 18-35 and 36-55 years age group showed increased prevalence of prediabetes among subjects who did not participate in physical leisure activities such as indoor and outdoor games. The present study also find significant association between sedentary leisure activities (playing on laptop/mobile or watching television) and prediabetes prevalence as all the participant whether prediabetics or non-prediabetics noted to indulge in these activities. Studies show negative effect of watching television and playing games on mobile or laptop to an increase in obesity on children as well as adults both (Rosiek et al, 2015). One of the major reasons for childhood obesity was watching television or using computers as shown by another studies (Laxmaiah et al, 2007; Eisenmann et al, 1999).

In present study we observed substantially higher prevalence of vegetarian (67.79%) dietary form than other two; non-vegetarian (6.38%) and eggetarian (25.83%). In contrast to current study Banjade et al conducted his study in the state of Karnataka reporting 42.7% prevalence of vegetarian and 57.3% prevalence of non-vegetarian subjects (Banjade et al, 2014). In support of Banjade's study, Khanna et al also reported 61.9% non-vegetarians, 22.2% lacto vegetarians (Vegetarians) and 15.9% ovo-lacto vegetarians (eggetarians) among healthy athletes of New Delhi (Khanna et al, 2006). Granted, the findings of these

two studies were disparate to current study; one of the reasons might be the cultural diversity.

In current study, among the participants of 18-35 and 36-55 years, we found significant association of dietary form with risk of developing prediabetes, whilst in 12-17 years old participants, dietary type was found to have no significant association in risk of prediabetes. We further noted greater percentage of prediabetes among non-vegetarian dietary form following eggetarian in all age group. This suggests that saturated fats and high amount of animal proteins present in non-vegetarian food increases the risk of developing prediabetes. As per our knowledge, none of the studies found showing direct correlation of different dietary forms with incident prediabetes. However, supporting findings of current study, a case-cohort study among European adults reported high protein intake from animal source increases incidence of type 2 diabetes (Nielen et al, 2014). Muthunarayanan et al reported participants who consumed vegetable <3 days was higher in prediabetics and the difference was not statistically significant (Muthunarayan et al, 2015). Similarly, Zhao et al in China found that high-fat diet was not associated with developing prediabetes (Zhao et al, 2016). Study conducted on middle-aged US population documented total, low-fat, and high-fat dairy consumptions were associated with a 39%, 32%, and 25% lower risk of incident prediabetes whereas low-fat skimmed milk, whole-milk, yogurt, cheese and cream were nonlinearly associated with incident prediabetes (Hruby A,2017).

Junk foods consumption in all age has become almost a global phenomenon. India ranks 10^{th} in the fast food with 2.1% of expenditure in annual total spending. Junk food is characterized by having a higher content of fat and a lower content of starchy and fibre-rich food, together with a higher intake of sugar-rich beverages; this overdependence on junk food culminates in overweight/obesity, type 2 diabetes and coronary artery disease. Studies have found that if healthy dietary habits are not well formed in adolescents and unpleasant lifestyle patterns persisted during the transition from adolescents to adulthood, these behaviours may carry out for a lifetime, which would increase the risk for chronic non-communicable diseases (Larsen et al, 2004; Kelder et al, 1994; Niemeier et al, 2006).

Joseph et al conducted study on high school boys of Mangalore city and reported 97.3% children eats junk food while the result of present study said 100% children eat junk food

with different frequency. Furthermore, in the same study author reported 10.5% and 15.4% children eat junk food every day and once in a week respectively which was found to be very low especially in once in a week frequency of eating junk food compared to present study's result (Joesph et al, 2015).

Astrup et al reported that individuals who had more than two fast-food meals per week gained more weight and had greater increase in insulin resistance than individuals who ate less than one fast-food meal per week (Astrup et al,2008). Few studies have been reported till date describing association of junk food habit and risk of developing prediabetes. In India, we have not found any literature on risk of prediabetes due to junk food habit. The results of current study are in support with result published by Astrup et al in the age group of 18-35 years and 36-55 years. No interaction was existed between incident prediabetes with junk food habit in 12-17 years age group. The reason could explain this discrepancy was study participants of all three-age group especially school going subjects have different lifestyles and environment.

One of the studies stated that 4.4% children of aged 5 to 11 years do not eat sweets at all while 95.6% children accepted to eat sweets mainly in the forms of candy (Viswanath et al, 2004); however, these findings were almost equivalent to our data in all other age group reporting 100% eating of sweet.

In present study we noted that risk of prevalence of prediabetes was decreased as frequency of eating sweet decreased. Additionally, sweet frequency was associated with developing prediabetes in all three age groups: 12-17, 18-35 and 36-55 years with greater percentage prevalence of prediabetes with everyday sweet eating frequency. No data has been published till today demonstrating sweet frequency found interlinked with risk of developing prediabetes.

With the advent of the 21st century, an overwhelming majority has become the victim of stress and anxiety due to the fast-paced lifestyle and inundated pressure at work place. Because of heavy school schedule and unrealistic expectations and demands from parent and teachers, this predicament has been increasing among adolescents and children. Nonetheless, in present study we found the stress level was not grievous among all three

age groups. Sandal et al stated that depression and stress level was 40% whereas stress and anxiety was 50% among students of 9th to 12th standard of Chandigarh city (Sandal et al, 2017). One study was performed on the medical students of Puducherry categorized stress level as denial (25.7%), normal (25.9%), mild (33.6%), moderate (13.5%), severe (0.7%) and profound (0.7%) (Kumar et al, 2017). Similar study was conducted by Gobbur et al on post graduate doctors and documented 69.51% normal subjects while stress level was present in 30.49% (Gobbur et al, 2016). Additionally, the study on critical care doctors reported 40% of moderate to severe level of stress claiming two vital reasons; too many responsibilities at a time and treating VIP patients (Amte et al, 2015). The greater number of the IT employees of Bangalore were reported to have 67.11 of moderate level of Perceived stress and 91.27% subjects had no professional stress (Ramesh et al, 2016).

Research has indicated that stressful conditions have an adverse impact on diabetes (Lloyd et al, 2005). Bjorntop explains an association between stress and the onset of diabetes. The author claims that psychological reaction to stress either to fight or flight leads to the activation of hypothalamus-pituitary-adrenal axis that causes various endocrine abnormalities which antagonize the action of insulin (Bjorntop, 1997). There was no report on association of stress and prediabetes till date. In current study, it was apparent that, level of stress was interlinked with risk of prediabetes and significant association of stress level with incident prediabetes was noted among participants of 12-17, 18-35 and 36-55 years age group. In support of current findings in all three-age groups, other studies showed positive associations between stress and diabetes (Gul et al, 2016; Eriksson et al, 2013; Bener et al, 2011). Bhandary et al revealed that perceived stress level was found to be high among diabetic than non-diabetic subjects (Bhandary et al, 2013). A similar study conducted by Takehiro et al. proved the relationship between psycho-social factors and the glycaemic control of patients with Type 2 Diabetes (Takehiro et al, 2009).

In all three-age group, the study findings demonstrated that participants with upper, upper middle and lower middle class have a high prevalence of prediabetes than upper lower and lower socioeconomic class. Moreover, we found significant association of socioeconomic class with incident prediabetes among subjects of all three age groups. The possible reasons for this could be high income, sedentary lifestyle and westernization of developing country like India. Till date no studies have been reported on association of prediabetes and

socioeconomic class. However, strong association of diabetes with socioeconomic class was reported by many studies. Mudhaliar et al (Mudhaliar et al, 2017) in rural setting of India and Kim et al (Kim et al, 2015) in Korea, reported inverse association of socioeconomic status and prevalence of diabetes. This was consistent with ICMR-INDIAB population-based study demonstrated high prevalence of diabetes in low socioeconomic class of urban area while high prevalence of diabetes in high socioeconomic class in rural area (Anjana et al, 2017); reasons could explain this diversity are poorer awareness of health care and diseases as well as cost of screening and treatment.

The current study found 13.56%, 16.14%, 27.13% and 12.97% prevalence of hypercholesterolemia, hypertriglyceridemia, low HDL-C level and dyslipidemia, respectively among 18-35 years old subjects. Whereas increasing prevalence of lipid abnormalities were reported in participants of 36-55 years age group; hypercholesterolemia 29.28%, hypertriglyceridemia 32.98%, low HDL-C level 40.80 and dyslipidemia 26.64%.

Thomas et al reported 14% prevalence of hypercholesterolemia and 7.2% prevalence of hypertriglyceridemia on young adults of Chennai (Thomas et al, 2015). Joshi et al in subjects of more than 20 years reported 18.3% of hypercholesterolemia and 68.9% of subjects with low HDL-C level of Tamilnadu. In Jharkhand author has reported 4.9% and 76.8% of hypercholesterolemia and low HDL-C level respectively (Joshi et al, 2015). Joshi et al has also reported 38.6% and 22.8% of hypertriglyceridemia among subjects of Chandigarh and Maharashtra respectively of age more than 20 years (Joshi et al, 2015). Al-Sabah et al has conducted study on young adults of age 20-40 years at Baghdad hospital, Iraq in 2014 and reported 32.5% of hypercholesterolemia, 29.5% of hypertriglyceridemia and 38.5% of low HDL-C level. The results of Al-Sabah's study found to be very high in prevalence compared to results of current study which may be due to a smaller number of subjects in study conducted at Baghdad hospital.

In present study, in the age group of 12-17 years, none of the subjects was reported with any of the lipid abnormalities, but mean level of cholesterol, triglyceride and HDL-C was significantly associated with prediabetes. In the age group of 18-35 and 36-55 years, hypercholesterolemia, hypertriglyceridemia, low HDL-C level and dyslipidemia was noticeably higher among prediabetic subjects than the normal subjects. With regards in the

studies of Kansal et al, 2016 (Wardha) and Balgi et al, 2017 (Mysore), mean cholesterol and triglycerides level was higher among subjects with prediabetes than control group and mean level of HDL-C was lower among prediabetics. The mean cholesterol level in present study among12-17, 18-35 years and 36-55 years old subjects was 177.9±3.503, 191.1±1.512 and 203.3±1.479, respectively. These findings were nearly equal to the results of Balgi et al, 2017 whereas the mean cholesterol level found higher in the study of Kansal et al, 2016 and lower in the study of Shankar et al, 2011. We found mean triglyceride level among prediabetic subjects of 12-17 years age group was 128.5±2523, 18-35 years age group was 147.1±1.155 and among 36-55 years age group was 155.6±1.665 which was remarkably higher than the reports of Balgi et al, 2017 and Kansal et al, 2016. However, the present study's finding was lower than the context of Dutta et al, 2013. In current study, the mean HDL-C level was 47.7±0.897, 41.8±0.592 and 37.9±0.547, respectively among 12-17 years, 18-35 years and 36-55 years old prediabetics. This data was indistinguishable with the report of Balgi et al, 2017 whilst the mean HDL-C level was reported higher by Shankar et al, 2011; Kansal et al, 2016 and Dutta et al, 2013than the current study. The mean cholesterol level reported by Poorsoltan et al was likely to the present study for age group 36-55 years; however, mean triglycerides and mean HDL-C level was significantly higher than the findings of the current study for both the age groups (18-35 and 36 to 55 years) (Poorsoltan et al, 2013). The finding of these all studies establishes the fact that lipids plays a pivotal role in pathogenesis of prediabetes.

It can be inferred from above results that prediabetics are also prone to develop more macrovascular complication of diabetes, which is alarming. The link between diabetes and atherosclerosis is, however, not completely understood (Goldberg et al, 2010). Nonetheless, many studies have clearly explained that diabetic complications are linked with several mechanisms such as; dyslipidemia, platelet activation, and altered endothelial metabolism (Brownlee M, 2001; Jokl et al, 1997; Taskinen MR, 2003). Additionally, cardiovascular disease is the leading cause of death among adult diabetic patients (Preis et al, 2009). This study recommend, early screening of prediabetics for lipid abnormalities are mandatory to avoid cardiovascular complications.

It is irrefutable that vitamin D deficiency has become the biggest health threat in the healthcare sector globally. Prevalence of vitamin D deficiency in current study among

participants of 12-17 years age group was 55.26% whilst it was higher in 18-35 years age group (67.62%) and 36-55 years age group (73.15%). Marwaha et al conducted study on older children, 93.7% children aged 6-17 years have shown to be deficient in vitamin D (Marwaha et al, 2010). Puri et al reported similar prevalence of vitamin D deficiency in children from both upper (91.9%) and lower (89.6%) socio economic strata (Puri et al, 2008). We found nearby similar results of vitamin D deficiency among school children. In one study from Kashmir, 58.5% adults have been shown to suffer from vitamin D deficiency (Daga et al, 2012). Sahu et al reported 88.6% prevalence of vitamin D deficiency among 10-20 years of aged girls while 32% prevalence of vitamin D deficiency among pregnant women (Sahu et al, 2009). Similar kind of studies have been conducted by International Osteoporosis Foundation in 2009 and reported 91%, 78% and 84% prevalence of vitamin D deficiency among healthy school girls, healthy hospital staff and pregnant women respectively (Mithal et al, 2009). The probable of vitamin D deficiency among Indian could be low dietary intake of vitamin D, high fiber and phytate intake which decreases vitamin D level (Khadilkar et al, 2010), reduced exposure to sunlight (Ekbote et al, 2010), pollution (Agarwal et al, 2002) or reduced exposure of skin to sun light because of cultural and traditional habits like “burkha” or “parda” (Sanwalka et al, 2016).

Vitamin D deficiency affects either insulin sensitivity and beta cell function or both which contributes as an important risk factor for pathogenesis of type 2 diabetes mellitus (Efendic et al, 2012). Recent epidemiological evidence demonstrated association of vitamin D insufficiency with adverse metabolic risk and in the pathogenesis of cancer, cardiovascular diseases, type 2 diabetes and other diseases (Chiu et al, 2004; Hypponen et al, 2007; Broucher et al, 1995; Scragg et al, 2004; Pittas et al, 2007). In association of this the current study demonstrated vitamin D deficiency among subjects of 12-17 years was 86.95% with mean vitamin D level 18.7±0.563 in prediabetics. While in 18-35 years and 36-55 years old subject vitamin D deficiency among prediabetes subjects was 88.65% and 84.39% respectively along with mean level 16.9±0126 and 16.9±0.186. We noted that the mean vitamin D level was higher among normal subjects than prediabetics irrespective of age group. Subsequently, we also found vitamin D deficiency as well mean vitamin D level were significantly associated with prediabetes which signifies insulin insensitivity and beta cell dysfunction due to vitamin D deficiency contributes to incident prediabetes. The present study posed higher prevalence of vitamin D deficiency among prediabetes subjects

compared to the findings estimated by Srinath et al (South India) (Srinath et al, 2016) and Zhang et al (Zhang et al, 2016) (Kuwaiti adults) representing the prevalence 72.5% and 53.9% respectively. The findings of the studies of Dutta et al and Shankar et al were consonant with the reports of present study indicating positive association between lower serum vitamin D level and prediabetes ((Dutta et al, 2013; Shankar et al, 2011).

Insulin resistance is a condition in which the body produces insulin but when muscle, fat, and liver cells do not respond effectively to insulin that makes glucose difficult to get absorbed easily from the bloodstream. Apparently, the demand of insulin in the body would increase to assist glucose to enter cells. To the limit beta cells in the pancreas stands with this increased demand for insulin by producing more but with over time, beta cells fail to satisfy body's increased demand for insulin. Hence with inadequate insulin, excess glucose builds up in the bloodstream, leading to diabetes, prediabetes, and other solemn health disorders. Impaired glucose tolerance and type 2 diabetes mellitus are the outcome of depleted insulin secretion and insulin resistance. According to some researcher's insulin resistance and hyperinsulinemia already exists before blood glucose abnormalities apparent in diabetic or prediabetic patients (Groop et al, 2000; Praveen et al, 2012). Hence, researches have suggested that the type 2 diabetes mellitus process should be classified into three stages: hyperinsulinemia stage, prediabetes stage, and diabetes stage (Groop et al, 2000).

Hyperinsulinemia among 18-35 years and 36-55 years old subjects was respectively 16.73% and 31.18%, whilst none of the participants of 12-17 years were reported to have hyperinsulinemia in current study. We further evaluated association of hyperinsulinemia with prediabetes and found significantly associated in 18-35 years age group but not in 36-55 years age group. However, we found a significant correlation between mean level of insulin and prediabetes in all three age groups. To the best of my knowledge no account of the study reporting association of hyperinsulinemia and prediabetes has been published till date. However, Salazar et al and Haffner et al demonstrated that individuals with prediabetes were more insulin resistant than those with normal fasting glucose (Salazar et al, 2016; Haffner et al, 2003). Whilst Yang et al suggested insulin resistance existed in subjects with hyperinsulinemia with normal glucose, impaired glucose tolerance and type 2 diabetes mellitus which was consistent in the three groups (Yang et al, 2016).

C-reactive protein, a major acute phase protein act inevitably as a marker of inflammation. It is linked with increased risk of diabetes (Chase et al, 2004; Doi et al, 2005; Hu et al, 2009). Studies have reported inflammation in glucose imbalance which was demonstrated by elevated level of inflammatory biomarkers like C-reactive protein (Shoelson et al, 2006). Inflammation is broadly considered as a pivotal risk factor in pathophysiology of coronary heart disease (Shrivastava et al, 2015). C-reactive protein, being a prototype of inflammatory marker plays a vital role in prediction of coronary heart disease (Calabro et al, 2012). Several studies on middle aged men (Ridker et al, 1997; Koenig et al, 1999), post-menopausal women (Ridker et al, 1998) and elderly men and women (Tracy et al, 1997) have shown C-reactive protein a strong and independent risk factor for coronary heart disease.

The current study shown prevalence of elevated C-reactive protein among 12-17, 18-35 and 36-55 years age group was 0.44%, 10.50% and 21.25%, respectively. We further found C-reactive protein was elevated among subjects with prediabetes than control among all three age groups. These findings were consistent with the studies of Lin et al, 2009 and Sabanayagam et al, 2011. The mean level of C-reactive protein among prediabetic subjects of 12-17, 18-35 and 36-55 years was respectively, 3.35±0.041 mg/L, 2.12±0.059 mg/Land 2.55±0.056 mg/Lin current study. Furthermore, the mean C-reactive protein level in the study of Lin et al was almost indistinguishable with the findings of current study (Lin J, 2009). Nonetheless, the mean of C-reactive protein (4.2 ± 9.0) of Sabanayagam's study was not remarkably higher than our study (Sabanayagamet al, 2011). Our study provides evidence for a positive association of elevated C-reactive protein level with prediabetes suggesting role of inflammation and the risk of cardiovascular disease even at prediabetes stage. This finding of current study was supported by Jaiswal et al (Jaiswal et al, 2012).

Obesity acts as a foremost predisposing factor in the aetiology of hypertension (Davy et al, 2004; He et al, 2009). Our study showed prevalence of pre-hypertension and hypertension was significantly high among overweight and obese subjects of all three age groups. These finding was supported by Israeli et al., 2006 (Israel), whose results showed pre-hypertension prevalence increased with increasing age and was significantly higher in the overweight and obese adolescent subjects (Israeli et al,2006). Another report published by Greenlund et al in 2004 among subjects of United States concluded that overweight and

obesity was the most prevalent risk factor for pre-hypertension (Greenlund et al, 2006). According to our data, prevalence of prehypertension among overweight and obese subjects of 12-17 years was respectively 17.56% and 24%. These findings were similar to studies done on Lithanian adolescents (Dulskiene et al, 2014) and school going children of Egypt (Allam et al, 2016). A study conducted among adolescents of Tripura, India reported higher prevalence of pre-hypertension and hypertension among overweight and obese subjects in compared to our results (Sutradhar et al, 2017). On the contrary, study reported by Ujunwas et al showed very less prevalence of prehypertension among overweight (3.6%) and obese (0.6%) subjects in comparison to our findings (Ujunwas et al, 2013). Widjaja et al reported overweight and obesity in 36.8% of hypertensive, in 28.9% of pre-hypertensive subjects (Widjaja et al, 2013).

Prevalence of prehypertension and hypertension among overweight subjects of 18-35 years age group was 33.15% and 23.52% respectively while among obese subjects, prehypertension was 40.38% and hypertension was 25%. However, 45.09% pre-hypertension and 52.94% hypertension was reported among 36-55 years old obese subjects while prevalence of pre-hypertension and hypertension among overweight subjects of 36-55 years was 32.66% and 39.51% respectively. Our findings were consistent with reports published by Dua et al on 18-50 years adults of Delhi stated prehypertension and hypertension was highest among overweight subjects (Dua et al, 2014). A study conducted by Yadav et al reported overweight/obese subjects had a high prevalence of both hypertension (41%) as well as pre-hypertension (30%) in an affluent north Indian population (Yadav et al, 2008) in comparison to our findings. Study outside of India reported body mass index found to be a significant feature in both the pre-hypertensive and hypertensive groups (Al-Maqbaliet al, 2013). Rahmanian et al observed higher number of pre-hypertensive subjects were of overweight and further reported pre-hypertensive subjects had 1.77% chances of becoming overweight. All these findings imply relationship between pre-hypertension, hypertension and overweight, obesity as observed in current study. The risk of hypertension among overweight and obese participants increases because as mass increases it causes an inadequate vasodilatation in the presence of increased blood volume and cardiac output (Doll et al, 2002).

Rahmanian et al reported mean BMI in subjects with normotension and prehypertension 25.3 ± 4.3 and 26.8 ± 4.3 among subjects of age more than 30 years. (Rahmanian et al, 2012). Other studies documented very low mean BMI among normotensive, pre-hypertensive and hypertensive adolescents in comparison to our findings (Arora et al, 2017; Sutradhar et al, 2017; Dulskieneet al, 2014). A report published by Gupta et al on healthy disease-free obese subjects of 35-70 years reported mean systolic blood pressure 121 ± 13 and mean diastolic blood pressure 77 ± 7 (Gupta et al, 2010). On the other hand, our study documents 134.5 ± 4.8 mean systolic and 84.1 ± 6.7 mean diastolic blood pressure which was found detectably higher in comparison to findings reported by Gupta et al (Gupta et al, 2010).

It is widely accepted that any kind of physical activity performed regularly induces numerous changes in skeletal muscles and cardiorespiratory system which contributes in prevention and treatment of many metabolic diseases (Pedersen BK, 2006; Hawley JA, 2004). The present study revealed that in all the age group the prevalence of higher BMI was noted among subjects who did not participate in any form of exercise. We found physical inactivity found to have significant association with higher BMI in all three age groups. Studies performed by Hussain el al in 2016 among 10-15 years old school children of Karnataka, Goyal et al in 2010 on adolescent school children of Ahmedabad and Shylesh et al in 2011 among 11-15 years old children of Coimbatore revealed that subjects who did not participate in physical activities have risk of overweight and obesity (Hussain et al, 2016; Goyal et al, 2010; Shylesh et al, 2007). Daley et al in 2007 reported large proportion of women of menopausal age who were sedentary or not active were overweight and obese (Daley et al, 2007). Similar report was published by Pereko et al demonstrating significant association between physical activity and BMI among 18-91-year-old subjects of Ghana (Perekoet KK, 2013). The relationship between physical inactivity and overweight/obesity may exist due to busy schedule of job and inflexibility in work timings, may find people difficult to engage them in physical activity. This shows need for a nationwide study demonstrating role impact of physical activity on body mass index as countries like India is undergoing urbanization. Increasing physical activity among subjects of all the age is widely considered as important intervention for problem of unhealthy weight.

The current study noted subjects who denied their participation in any form of indoor and outdoor activities were of overweight and obese group. Studies from Hussain et al and Goyal et al also demonstrated remarkable effect of outdoor sports on BMI (Hussain et al, 2016; Goyal et al, 2010). Bharti et al among school going children of Wardha city reported playing outdoor games less than 30 minutes a day was an important co-relates of overweight and obesity (Bharti et al, 2008). All the subjects of three age group were reported to watch television daily in our study. Bhattacharya et al in Guwahati reported higher prevalence of obesity among school going children who viewed television more than 5 hours/day, who played video games every day 3-4 hours and in those who played indoor and outdoor games only 5 hours/week (Bhattacharya et al, 2016). Shaukat et al in the Lahore reported an association between BMI groups and physical activity. Author further revealed that majority in the overweight group (48.9%) watch TV for more than 2 hours (Shaukat et al, 2013). Our findings demonstrated all the participants of 12-17 years and 18-35 years play games on laptop/mobile. Whilst higher prevalence of overweight and obese respondents of 36-55 years play games on laptop/mobile. Globally participation in any forms of games have been decreased due to sedentary work forms such as playing games on mobile/laptop, watching television for longer period of times, changes in mode of transformation and urbanization.

Diet is considered as one of the important cognitive factor of body weight. Diet plays a crucial role in management as well as control of many lifestyle disorders. In present study we found dietary type was not significantly linked with BMI in all three age groups. Though, we found prevalence of overweight and obesity was higher among subjects with non-vegetarian and eggetarian diet. These findings were supported by Wang et al reported eating more meat are risk factors for being overweight and obese (Wang et al, 2016). Other findings published by Thaddanee et al among school children of Ahmedabad reported more prevalence of overweight and obesity among mix dietary type subjects compared to vegetarian subjects, which strongly supports findings of present study (Thaddanee et al, 2016). Whilst Vadera et al reported nearly similar prevalence such as 21.3% and 25.1% of overweight/obesity among vegetarian and mixed dietary type respectively among adults of Jamnagar city (Vadera et al, 2010). On contrary to current study, more prevalence of overweight and obesity was reported among adults with vegetarian diet than mix dietary type by Ghosh et al, 2015. It has long been known that animal-based foods contain more

fat than others. Regular consumption of such high fat foods may lead to overweight and obesity.

Admittedly, excessive consumption of junk food has a detrimental effect leading the deficiency of essential nutrients and gradually increases weight since junk foods are deprived of nutrients and contains empty calories. Goyal et al documented more than two servings outside home increases the risk of overweight and obesity. The author also reported consumption of junk food more than once daily doubles the risk of overweight and obesity (Goyal et al, 2011). Children who consumed fast foods once or more a day had higher chances of being overweight and obese compared to less frequent consumers of junk food (Joesph et al, 2015). Thaddanee et al reported same findings concluding subjects with more than two frequency of junk food were overweight and obese than subjects with less than two frequency in a week (Thaddanee et al, 2015). The current study's findings were in support of results documenting everyday and once in a week frequency of junk food leads to overweight and obesity. However, present study found frequencies of junk food had no significant association with BMI in 12-17 years age group whereas it was found significantly associated with BMI among participants of 18-35 and 36-55 years age group. Pereira et al found significant association of fast food with increased BMI and insulin resistance (Pereira et al, 2005). This shown fast food consumption has linked with obesity and type 2 diabetes. Similarly, Anderson et al also found a strong association between fast-food consumption and weight gain (Anderson et al, 2011). Young generation gets easily influenced by attractive advertisements displays in social media and television. Easy access to shopping malls, restaurants, free home deliveries of food from restaurants and supermarkets play crucial role for developing unhealthy and high-calorie food habits.

In present study we found obese subjects noted to have everyday consumption of sweet in all three age groups. Additionally, we found significant association of frequency sweet consumption with BMI among all three age groups. Unfortunately, none of the studies reported association of consumption of sweets with BMI till today. To our mind, some constructive researches should be performed demonstrating association of frequencies of sweet eating habit with incident overweight/obesity.

According to Sinha et al, stress is linked with obesity. The neurobiology of stress is associated with hunger and energy (Sinha et al, 2013). Jaaskelainen et al in his study observed an association stress-related eating behaviour and obesity (Jaaskelainen et al, 2014). Under stressful situation, human body releases glucocorticoids (cortisol) and catecholamine (adrenaline and noradrenaline) through activation of hypothalamic-pituitary-adrenocortical axis and sympathetic nervous system, respectively (Tsatsoulis et al, 2006; Holmes et al, 2010). Hypercortisolemia following an elevated insulin becomes persistent in chronic stress (Tsatsoulis et al, 2006; Holmes et al, 2010, Dallman MF, 2010). Together, an action of elevated cortisol and insulin increases the intake of more liking foods which contributes to body fat gain (Dallman MF, 2010). Furthermore, study have shown an elevated level of cortisol causes leptin resistance which is correlated with an elevated release of neuropeptide γ (Tsatsoulis et al, 2006). Both, cortisol and neuropeptide γ known to increase hunger (Cameron et al, 2007). Khushboo et al claimed stress found to have association with increased consumption of food which leads to overweight and obesity among hosteller girls (Khushboo et al, 2012). The findings of present study claimed that with the advent of stress, BMI increases. We found greater number of overweight and obese subjects had medium level of stress compared to underweight and healthy weight subjects with no stress. However, we did not find significant association of level of stress with BMI in 18-35 and 36-55 years age group whilst it was found significantly associated with BMI among 12-17 years old participants. Gupta et al conducted study on undergraduate medical students and reported 11.8% variation of BMI was contributed by stress in males while no significant correlation of stress with BMI was found in female due to a smaller number of female participants in study (Gupta et al, 2009). Another study on medical students of Jorhat Medical College, Assam conducted by Goswami et al reported 3.6% prevalence of stress among obese and 15.9% prevalence among overweight (Goswami et al, 2017). Whilst in current study (in 18-35 years age group) medium level of stress among overweight subjects was 20.89% and among obese subjects was 8.95%. As compared to Goswami's study we found the prevalence of stress was almost identical in both studies; additionally, there was a strong correlation between psychological stress and body weight. Similarly Block et al reported high baseline body mass index, weight gain was associated with increasing levels of psychosocial stress (Block et al, 2009).

As economy of India increases, urbanization is taking place at a faster rate. Socioeconomic class is a core part of mental as well as physical health. Income levels directly affects dietary habits and behaviour which includes physical activity and dietary habit that can lead to obesity. In the present study most of the overweight and obese subjects were of upper and upper middle socioeconomic class and among all three group we found positive significant association of socioeconomic class with BMI. Studies of Tharkar et al in urban India, Goyal et al in Ahmedabad, Akinyemiju et al in Ghana and Nematy et al in Iran shown higher socioeconomic class has a significant association with increasing BMI (Tharkar et al, 2009; Goyal et al, 2010; Akinyemiju et al, 2016; Nematy et al, 2009). A study on women in Chandigarh found higher number of overweight subjects from middle socioeconomic class. The reason could explain this diversity was majority of women were of middle socioeconomic class. (Dewan et al, 2016). Increasing BMI has positive association with wealth index and residency status of New Delhi, Andhra Pradesh and Tamil Nadu reported by Patel et al (Patel et al, 2017). However, a report from Fezeu et al (2006) revealed that the positive relationship between socioeconomic class and BMI is not true for all developing countries. Inverse relationship between BMI and socioeconomic class was reported by Mbada et al among Nigerian adults and Fokeena et al among adolescents of Mauritius (Mbada et al, 2009; Fokeena et al, 2012). Low level of education and poverty among individuals in lower socioeconomic class could be the reasons for negative relationship between socioeconomic class and BMI.

It has long been known that obesity is defined as accumulation of abdominal, subcutaneous and visceral fat which found to have strong association with metabolic syndrome and cardiovascular risks (Abate et al, 1995). Obesity attributes abnormalities in lipid metabolism that results in elevation of lipid stores (Nicholas SB, 1999). The stage of insulin resistance in peripheral tissues reported to have a pivotal link between an obesity, metabolic syndrome and dyslipidemia (Klob et al, 2013). Insulin resistance leads to increase hepatic flux of fatty acids from dietary sources and intravascular lipolysis which contributes to lipid abnormalities (Klob et al, 2013). The metabolic lipid abnormalities associated with obesity enhances the risk of coronary heart disease. The excess body fat, obesity and sedentary lifestyle constitute risk factors for cardiovascular disease (Rexrode et al, 1996).

Minakshi et al conducted study on 100 Indian children above 6 years of age and reported 15% subjects found to have high triglyceride level and 69% subjects found to have low HDL-C level (Minakshi B, 2016). One cross-sectional survey done on 1083 school-going Indian children (12–17 years) reported hyperglycemia 28.3%, hypertriglyceridemia 40.0%, low HDL-C 61.7%, and elevated blood pressure 31.6% in overweight subjects and demonstrated a trend for increase in prevalence as BMI increased (Singh R, 2007). Conversely, regarding to these studies in the age group of 12-17 years we did not find any subjects having lipid abnormalities, but we found mean level of cholesterol, triglyceride and HDL-C were significantly linked with BMI.

The current study among participants of 18-35 and 36-55 years age group noted greater prevalence of hypercholesterolemia, hypertriglyceridemia, low level of HDL-C and dyslipidemia among overweight and obese than healthy weight and underweight subjects. Moreover, the association of lipid abnormalities was noted significant with higher BMI. We further found mean values of total cholesterol and total triglycerides were significantly higher whilst mean values of HDL-C was lower among overweight and obese than other groups. Gayathri et al in 2017 conducted study on 270 participants of both gender in the age of 18- 55 years of SRM medical college hospital and research centre, Kattankulathur and the author reported statistically significant higher value of various lipid parameters like total cholesterol, triglycerides and LDL-C in overweight, obese individuals (Gayathri et al, 2007). These findings are consonant with present study demonstrating elevated cholesterol, triglyceride, low level of HDL-C and dyslipidemia were significantly associated with BMI. The result of Sharma et al and Chadha et al was not supporting the present findings since in this context HDL-C showed no statistically significant association with BMI (Sharma et al, 2015; Chadha et al, 2006). The evidences of mean cholesterol levels in study of Funderburk et al were almost similar with the present study whilst the present context had claimed that mean triglyceride level was significantly higher along with lesser HDL-C in comparison to the study of Funderburk et al (Funderburk et al, 2013).

Vitamin D plays vital role in the development and maintenance of good bone health, through regulation of calcium and phosphorus homeostasis (Washington, D.C. National Academic Press, 2011). Wortsman et al have explained several possibilities of vitamin D deficiency among obese subjects which includes decreased sun exposure due to sedentary

lifestyle, poor dietary habit and increased clearance of vitamin D due to storage in adipose tissue (Wortsman et al, 2000). Although the role of vitamin D in many metabolic diseases is still not clear and under investigation, researchers reported that inadequate vitamin D has been significantly associated with overweight and/or obesity and as metabolic syndrome including raised plasma glucose concentration and insulin resistance (Pacifico et al, 2011). In present study we found that more subjects living in Ahmedabad had vitamin D deficiency.

A study from United States documented, the prevalence of vitamin D deficiency in healthy weight, overweight, obese, and severely obese children between 6 to 18 years of age has been found to be 21%, 29%, 34%, and 49%, respectively (Turer et al, 2012). In contrast to these findings the prevalence of vitamin D deficiency among 12-17 years old subjects of all BMI classification was substantially higher. Children of Saudi Arabia of aged 4-13 years with vitamin D deficiency had a higher BMI than those with normal vitamin D level reported by Al-Agha et al (Al-Agha et al, 2016). Kannan et al supported these results by documenting risk for vitamin D deficiency increases in children with severe obesity (Kannan et al, 2016). Another study performed by Kumaratne et al on 13-19 years of Hispanic American Adolescents and reported mean vitamin D level among underweight (26.5 ng/mL), healthy weight (26 ng/mL), overweight (24 ng/mL) and obese (22.5 ng/mL). Present study supported findings of Kumaratne et al regarding underweight and healthy weight subjects whereas the mean value of vitamin D among overweight and obese were lesser than his study (Kumaratne et al, 2017).

In present study among the age group of 12-17, 18-35 years and 36-55 years, prevalence of vitamin D deficiency was found higher among overweight and obese subjects than others. Additionally, we found strong significant correlation of vitamin D deficiency and mean low level of vitamin D with BMI in all three age groups. The findings of present study were consonant with the studies of Vimaleswaran et al and Lagunova et al on adults claiming that higher BMI leads to lower vitamin D status, providing evidence for the role of obesity as a causal risk factor for the development of vitamin D deficiency (Vimaleswaran et al, 2013; Lagunova et al, 2009). On the flipside, findings of present study were variant with the studies of Baradaran et al stating there was no significant association between vitamin D level and BMI (Baradaran et al, 2012).

In explanation of relationship between hyperinsulinemia and obesity, one of the hypothesis was proposed by Lele et al stating, obese type 2 diabetes mellitus patient have dysfunction of muscle UCP2 and UCP3 that promotes fat deposition. However, the leptin level among those subjects was high but they reported to have leptin resistance due to hyperinsulinemia (Lele et al, 2007). Shrinivasan et al conducted study in three age groups of the population enrolled in the Bogalusa Heart Study; children (5-7 years), adolescents (12-14 years) and young adults (20-24 years). The author showed temporal relationship between obesity with hyperinsulinemia and supported role of obesity in hyperinsulinemia (Shrinivasan et al, 1999). The present study also documented increasing mean level of insulin with BMI and found statistically significant relation between BMI and hyperinsulinemia in18-35 and 36-55 years age group. Though, none of the participants of 12-17 years age group had abnormally elevated insulin level, mean insulin level was found significantly associated with BMI.

Inflammatory cytokine; interleukin-6 originating from adipose tissue induce insulin resistance by direct action on the insulin-signaling cascade (Hotamisligil et al, 1993; Hotamisligil et al, 1994) and regulate production of C-reactive protein by the liver (Heinrich et al, 1990; Mackiewicz et al, 1991). Other inflammatory marker, tumor necrosis factor-α does not stimulate directly production of C-reactive protein but it induces its action via interleukin-6 (Mackiewicz et al, 1991).

Present study showed prevalence of elevated C-reactive protein was high amongst overweight and obese participants of all age group compared to underweight and healthy weight subjects. We further found mean level of C-reactive protein increases with weight. This indicates presence of inflammation associated with unhealthy weight. Some studies are in support to our analysis demonstrating positive association between BMI and elevated C-reactive protein (Pearson et al, 2003; Mattsson et al, 2008; Hiura et al, 2003; Ford et al, 2005; Guran et al, 2007; Roh et al, 2007; Kitsios et al, 2013; Shin et al, 2008). Similarly, elevated levels of inflammatory markers were reported by Giannini et al among obese prepubertal children compared to normal-weight (Giannini et al, 2008) demonstrating role of inflammation in overweight and obesity.

6. Summary and Conclusion

The outcome of current study confirmed that prevalence of prediabetes was increasing with age as it was noted 5.09%, 28.81% and 33.19% among 12-17, 18-35 and 36-55 years age group, respectively. It was also found that mean fasting blood sugar level was higher among 36-55 years old participants compared to 12-17 and 18-35 years. Prevalence of prediabetes was remarkably increasing with BMI in all three age groups and noted the highest prevalence among obese participants. It was also found that mean BMI was significantly higher among prediabetic participants than normal subjects. Family history of diabetes, thyroid and hypertension was significantly associated with incident prediabetes whereas family history of obesity was found to have no significant association with prediabetes in 12-17 years old participants. Family history of diabetes, obesity, thyroid and hypertension was established as predisposing factor for prediabetes in 18-35 and 36-55 years age group. In all three age groups, we found prevalence of prediabetes was significantly higher among subjects with pre-hypertension and hypertension compared to normal blood pressure showing prediabetes is likelihood in pre-hypertensive and hypertensive subjects. The present study also asserted that the mean fasting blood sugar level was reported significantly higher among pre-hypertensive and hypertensive participants than normal subjects. The study postulate that incidence of prediabetes was more among subjects who did not participate in physical activity compared to subjects who participates claiming physical activity in any form had protective effect on prediabetes in all age groups. Participation in either indoor or outdoor games had no significant association with prediabetes in 12-17 years old participants, whereas playing indoor and outdoor games showed positive significance in decreasing the risk of incident prediabetes in 18-35 and 36-55 years old subjects. The current study found all the participant whether prediabetics or normal subjects of 12-17 and 18-35 years play on laptop/mobile. In all three age groups, playing on laptop or mobile had significant association with incidence prediabetes. Irrespective of age groups and prediabetes, all the subjects reported to watch television. Prevalence of prediabetes was higher among subjects with non-vegetarian dietary form following eggetarian and vegetarian dietary form in all three age groups. However, dietary type was not significantly associated with risk of prediabetes in 12-17 years old participants. In 12-17 years, frequency of junk food found not significantly associated with

prediabetes whereas in 18-35 years, we noted once in a week and in 36-55 years every day and once in a week frequency were the highest. Frequencies of eating sweet was significantly associated with incidence prediabetes in all three age groups with highest prevalence of prediabetes among everyday sweet consumers. In all three age groups, prediabetes prevalence was the highest among subjects with medium level of stress which was significantly associated with prediabetes. With respect to socioeconomic status, prediabetes was significant in all three age groups with remarkably higher prevalence of prediabetes among upper socioeconomic class. Lipid abnormalities such as hypercholesterolemia, hypertriglyceridemia, low HDL-C and dyslipidemia were noted higher in prevalence among prediabetics than normal subjects. We further noted that mean lipid levels were strongly associated with incident prediabetes. Similarly, vitamin D deficiency, hyperinsulinemia and elevated C-reactive protein also found associated with prediabetes in all the age groups.

The current study noted 16.23%, 18.51% and 26.22% prevalence of overweight while 5.48%, 5.15% and 5.39% prevalence of obesity among 12-17, 18-35 and 36-55 years participants, respectively. In all three age groups we noted that prevalence of pre-hypertension and hypertension was higher among overweight and obese participants than healthy weights. Further, mean systolic and diastolic blood pressure was significantly higher among overweight and obese subjects. The current study stated BMI increases with physical inactivity in all three age groups. The prevalence of overweight and obesity was found significantly higher among participants of all age groups who did not play indoor and outdoor games. We found greater percentage of participants with non-vegetarian dietary type were of overweight and obese category in all three age groups, though the association of dietary type and BMI was not significant. The frequency of junk food eating had no significant association with overweight and obesity in 12-17 years old participants whilst everyday junk food eating habit postulated to increase BMI among participants of other two groups. Habit of eating sweets everyday associated with obesity in all three-age groups. Level of stress had an association with obesity in school going children but no significant association with obesity was noted among 18-35 and 36-55 years old participants. The study reported greater prevalence of overweight and obesity among upper socioeconomic class subjects in all three-age groups and the association was found significant. The study revealed that overweight and obese subjects were at increased risk

of abnormal lipid profile including hypercholesterolemia, hypertriglyceridemia, low HDL-C level and dyslipidemia compared to healthy weight participants. Vitamin D deficiency, hyperinsulinemia and elevated C-reactive protein had significant association with BMI in current study.

An increasingly prevalence of overweight and obesity due to unhealthy lifestyle increases risk of prediabetes, metabolic syndrome and cardiovascular complications. Early identification of prediabetes and lifestyle modification including proper diet, weight loss and physical activity delays or prevents progression to diabetes.

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Questionnaire

Name: _____ Today's Date: _____

Address: _____

City, State, Pincode: _____

Telephone: Home (_____) _____ Birth Date: _____

Mobile: _____ Sex: □ Male / □ Female

Background

1. How old are you? _____ years old.

2. How tall are you? _____feet _____ inches **BMI:** _____

3. How much do you weight? _____ kgs.

4. Please circle the highest year of school completed:

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23+

(primary) (high school) (college/university) (graduate school)

5. Are you currently (check √ only one):

□ married □ separated □ widowed □single □divorced

6. Please indicate below which chronic condition(s) you have:

□ Diabetes type 2 □ Diabetes type 1 □ High cholesterol

□ High blood pressure □ Hypothyroid

□ Heart disease Type of heart disease: _____

□ Lung disease Type of lung disease: _____

□ Other chronic condition Specify: _____

□ Don't know:

7. Have any of the members of your immediate family or other relatives been diagnosed with diabetes (type 1 or type 2)?

□ No

□ Yes: grandparent, aunt, uncle or first cousin (but no own parent, brother, sister or child)

□ Yes: parent, brother, sister or own child

8. Have any of the members of your immediate family or other relatives been diagnosed with obesity?

□ No

□ Yes: grandparent, aunt, uncle or first cousin (but no own parent, brother, sister or child)

□ Yes: parent, brother, sister or own child

9. Have any of the members of your immediate family or other relatives been diagnosed with hypothyroid?

□ No

□ Yes: grandparent, aunt, uncle or first cousin (but no own parent, brother, sister or child)

□ Yes: parent, brother, sister or own child

10. Have any of the members of your immediate family or other relatives been diagnosed with hypertension?

□ No

□ Yes: grandparent, aunt, uncle or first cousin (but no own parent, brother, sister or child)

□ Yes: parent, brother, sister or own child

General Health

1. In general, would you say your health is: (check √ only one)

□ Excellent ..... 1 □ Very good..... 2

□ Good..... 3 □ Fair..... 4

□ Poor..... 5

Physical activity

1. Do you perform physical exercise in any form at least 1 – 3 hours per week?

□ Yes □ No

2. Do you play any of the indoor games at least 1-3 hours per week?

□ Yes □ No

3. Do you play any of the outdoor games at least 1-3 hours per week?

□ Yes □ No

4. Do you play on laptop/mobile more than 1 hour per day?

□ Yes □ No

5. Do you watch television more than 1 hour per day?

□ Yes □ No

Diet

1. Are you □ Vegetarian □ Non vegetarian □ Eggetarian

2. Do you eat junk food? YES / NO

□ Everyday □ Once in a week □ Once in a fifteen days □ Once in a month

3. Do you eat chocolates/sweets? YES / NO

□ Everyday □ Once in a week □ Once in a fifteen days □ Once in a month

Medication history

1. Do you take any medication regularly?

□ Yes □ No

If yes, then what kind of medication?

□ Allopathic □ Homeopathic □ Ayurvedic

1a. Name the medication you take on regular basis.

Stress related questionnaire

Questions	Never	Rarely	Sometimes	Often	Very often
1. I have no friends/ I feel lonely.	0	1	2	3	4
2. I feel insecure because of too much competition in getting good grades and a good job.	0	1	2	3	4
3. I feel sad/depressed with my family life.	0	1	2	3	4
4. I feel nobody cares for me.	0	1	2	3	4
5. I feel I have too much pressure because of my studies and Career.	0	1	2	3	4

Interpretation of score:

0 - 5: well control over stress

6 – 10: less level of stress

11 – 15: medium level of stress: Should reconsider means of coping with stress

16 – 20: very high level of stress: counselling necessary

Socioeconomic Status Scale

Kuppuswami's Socioeconomic scale

(A)	**Education Score**	
1	Professional or honors	7
2	Graduate or Post graduate	6
3	Intermediate or post high school dip	5
4	High school certificated	4
5	Middle school certificate	3

6	Primary school certificate	2
7	Illiterate	1
(B)	**Occupation Score**	
1	Profession	10
2	Semi-profession	6
3	Clerical, shop owner	5
4	Skilled worker	4
5	Semi-skilled worker	3
6	Unskilled workers	2
7	Unemployed	1

(c)	**Monthly family income in Rs. (1976)**	**Score**	**Modified for 2014 in Rs.**
1	≥ 2000	12	≥36, 017
2	1,000 - 1,999	10	18,000 - 36,016
3	750 – 999	6	13,495 - 17,999
4	500 – 749	4	8,989 - 13,494
5	300 – 499	3	5,387 - 8,988
6	101 – 299	2	1,803 - 5,386
7	≤ 100	1	≤ 1,802

Total Score	**Socioeconomic Class**
26 – 29	Upper (I)
16 – 25	Upper middle (II)
11 – 15	Middle / Lower middle (III)
5 – 10	Lower / Upper lower (IV)
< 5	Lower (V)

Biochemical Analysis

Name of the Test	Findings	Reference limit
Fasting Blood Sugar		< 100 mg/dL: Normal 100-125 mg/dL: Prediabetes ≥ 126 mg/dL: Diabetes
Total Cholesterol		< 200 mg/dL: Normal ≥ 200 mg/dL: Hypercholesterolemia
HDL cholesterol		Low HDL-C: < 40 mg/dL in men < 50 mg/dL in women
Triglycerides		< 150 mg/dL: Normal ≥ 150 mg/dL: Hypertriglyceridemia
Vitamin D		< 20 ng/dL: Deficiency 20 – 30 ng/dL: Insufficiency > 30 ng/dL: Sufficiency
Insulin		< 25 mIU/L: Normal ≥ 25 mIU/L: Hyperinsulinemia
Hs-CRP		< 1 mg/L: Low risk of cardiovascular disease 1 – 3 mg/L: Medium risk of cardiovascular disease > 3 mg/L: High risk of cardiovascular disease