TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES.

A Dissertation Submitted to

Department of physiotherapy

In partial fulfillment of the requirements for the

Award of the degree of

Master of physiotherapy in Obstetrics and Gynecology.

Supervised by :

Dr. Manpreet Kaur Kang

MSPT (Assistant Professor)

Submitted by:

Preeti Madaan

MPT IInd Y(Obs &Gynae)

(Registration No-11300114)

Phagwara, Punjab.

May,2015.

CERTIFICATE

This is to certify that the dissertation work entitled **"TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES. "**was carried out by **Ms.Preeti Madaan, Register No. 11300114,** Department of Physiotherapy, Lovely Professional University, towards partial fulfillment of the requirements of Master of Physiotherapy (Obstetrics & Gynecology) degree programme.

Dr. Jasobanta Sethi,
MPT, Ph.D, FIAP
Professor & Head,
Department of Physiotherapy,
Lovely Professional University
Phagwara - 144402

Date:

Place:

CERTIFICATE

This is to certify that **Ms.Preeti Madaan, Registration No- 11300114** has partially completed MPT PTY-608 dissertation –I titled **"TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES.**" under my guidance and supervision. To the best of my knowledge, the present work is the result of her original investigation and study. No part of this dissertation has been submitted for any other degree or diploma.

The dissertation is fit for the submission and partial fulfillment of the conditions for the award of **MPT (Obstetrics and Gynecology).**

Signature of Supervisor

Dr. Manpreet Kaur Kang
MPT (Sports)
Assistant Professor
Lovely Professional University
Phagwara, Punjab. 144402

Date:

Place:

DECLARATION

I hereby declare that the dissertation entitled, "**TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES.**" submitted for the MPT degree is entirely my original work and all ideas and references have been duly acknowledged. It does not contain any work for the award of any other degree or diploma.

Ms.Preeti Madaan

Date: Investigator

Place: Registration No-11300114

ACKNOWLEDGEMENT

First I would like to thank almighty God and my Parents for giving me the courage to complete my dissertation work.

I express my heartful thanks to **Mr.Ashok Mittal** ,Honorable Chancellor, LPU,**Mrs.Rashmi Mittal**, Worthy Pro Chancellor,LPU, **Dr.Ramesh Kanwar**, Worthy Vice Chancellor ,LPU, **Dr.Monica Gulati** , Senior Dean,LFAMS for providing all the facilities to carry out the project work.

I express the whole hearted thanks to **Dr.Jasobanta Sethi**, Professor, Head & COS of the Department, Department of Physiotherapy, Lovely Professional University, Phagwara , Punjab for giving me the opportunity to work on this project.

With my extreme gratitude, I wish to express my acknowledgement to my guide **Dr.Manpreet Kaur Kang (PT)** , Assistant Professor , Department of Physiotherapy, Lovely Professional University, Phagwara, Punjab, for her valuable guidance and tremendous efforts in carrying out this project from its inception to completion.

I am also thankful to all my faculty members **Dr.Michael Raj** (MPT Neurology),**Dr.Sarvanan** (MPT Cardiology), **Dr.Ajay Parsad Gautam** (MPT, PhD Cardiology),**Dr.Sridhar** (MPT Neurology), **Dr.Rati** (MPT Orthopaedics), **Dr.Harpreet Kaur** (MPT Orthopaedics), **Dr.Gayatri** (MPT Orthopaedics), **Dr.Himani** (MPT Neurology), **Dr.Priyanka** (MPT Neurology),**Dr.Bhanu** (MPT Paediatrics), **Dr.Ruchika** (MPT Orthopaedics), **Dr.Biswajeet Kanungo** (MPT Orthopaedics) for their valuable advice and help regarding this thesis.

I wish to express my thanks to all the subjects who volunteered to participate in the study. Last but not the least I would like to thank my sister and my friends for their all time and support.

Preeti Madaan

DEDICATION

I would like to dedicate this thesis to Almighty God who gave me all the strength and insight to carry on my life in good form. My parents who have been supportive throughout my life that ways acted as a source of inspiration, motivation and encouragement. To my teachers, who made me learn at every required occasion and without whom it was never possible for me to reach this junction. To my all friends, who have always been helpful and stimulating in my personal and professional growth.

TABLE OF CONTENTS

INTRODUCTION

Sleep disorders or snoring is very common problem of pregnancy, associated with worsening of sleep quality, increased day time laps , night wakening and progressive snoring. As a consequence of snoring in pregnancy symptoms of increased daytime sleepiness which results in fatigue are present especially when occurs in combination with obstructive sleep apnea (OSA). Currently .It is thought to estimated to affect approximately 6.5% of females of childbearing age.[1]

There is less chances of snoring in nonpregnant females (17%)[8] as compared to females who are pregnant (41%)[9] and snoring comes back to its original levels in postnatal period. (18%).[9] There is significant reduction in dimensions of upper airways in pregnant female when compared to nonpregnant females while in sitting position and there is decreased mean pharyngeal areas in sitting , supine lying and side lying postures in pregnancy as compared to the state of nonpregnant females. The females who are pregnant had much smaller mean pharyngeal areas when compared with females in post-partum period.[10]

The high levels of BMI before pregnancy has considered to be a major risk rather than weight gain during pregnancy..[11]The high pre-pregnancy BMI along with excessive weight gain during pregnancy both are the major risk factors for development of complications in pregnancy. The amount of total weight gain in pregnancy is thought to be determined by various factors like dietary habits of mother and her physical activity during pregnancy which can be modified easily according to the will of the female.[12].High amount of total energy intake along with increased proportion of protein and lipid intake and a lower percentage of carbohydrates are concerned with increase in gestational weight [13] The actual diet of a pregnant female who is already obese and gaining more weight in her pregnancy contains high amount of fat and lower proportion of carbohydrates as the source of energy when compared to the diet of pregnant femal who attined suboptimal weight..[14]

Obesity is considered to an epidemic not even in the developed countries rather in the whole world. It is expected that if there is increase in the rate of both adult as well as adolescent obesity, the prevalence of child bearing age obesity (aged 15–44 y) should also be increased. Various population and cohort studies determined obesity to be concerned with an higher risk of

development of co-morbid conditions during pregnancy. [15] Moreover there are implications of obesity over the fetus which may result in short term consequences of medical issues like pre-mature birth, still birth or macrosomia etc., and may also leads to long-term complications associated with fetus in utero programming. [16]

The maternal complications due to obesity in pregnancy is generally considered to be associated with pregravid weight as compared to gestational weight or postpartum weight. Actually during pregnancy increased accretion of subcutaneous fat is distributed rather than in lean pregnant females in the accrual of adipose mass in pregnancy is more peripheral [17]

The obese pregnant female is at the greater risk for potential medical as well as obstetric complications during gestational period of her life, which might got associated with short term adverse effects on fetus. Furthermore obese pregnant women is at high risk of getting miscarriage either she got conception naturally or with the help of assistive reproductive aids. In late pregnancy the obese women is also having at risk of development of pregnancy associated hypertension, pre-eclampsia or gestational diabetes mellitus.[18] As consequences of the above mentioned obstetric maternal complications, there is significantly high risk of having preterm delivery in morbidly obese pregnant females having less than 37 weeks of period of gestation [19].

Maternal obesity is a significant risk factor for neural tube defects in neonates of obese women. Although the risk of other congenital anomalies are less clear, the increased risk of neural tube defects in obese women appears to be independent of the risk of maternal diabetes and despite folic acid fortification in the flour in some countries. Factors such as decreased maternal smoking, an increased incidence of diabetes and increasing maternal BMI have all been implicated. The increase in maternal weight at delivery was the factor most strongly correlated with the increase in birth weight.[20-22]

The mechanisms underlying the increase snoring during pregnancy are unclear. Some studies reported that snorers with or without obstructive sleep apnea have narrower upper airways (UAs) than nonsnorers even when awake. [23]In pregnancy, the physiological changes potentially predisposing to increased resistance and reduced cross-sectional area of the upper airways

include the following: weight gain and decreased functional residual capacity (FRC) due to mass displacement of the diaphragm, pharyngeal edema of pregnancy; and, possibly, the effect of sleep deprivation or fragmentation on pharyngeal dilator muscle activity and upper airway collapsibility.[24]

There is one evidence which explained the protective response of elevated levels of circulating peripheral progesterone against the development of snoring which is associated with increased ventilator drive. Moreover snoring is considered to be responsible for higher incidence of disorders of breathing occurred due to sleep disturbances, and the rate of prevalence of snoring becomes higher in ongoing pregnancy[25]. Decreases in both duration and quality of sleep are common in pregnant women as a result of hormonal and physical factors. The most probable cause of snoring during pregnancy is a narrowing of the upper airways due to edema. Some studies reported smaller upper airway dimensions among pregnant women compared to non-pregnant controls. High estradiol levels are the main cause of edemas in the airway mucosa and other tissues.[26]

Breathing disorders due to sleep problems are commonly associated with worsening of cardiovascular problems in females. Generally snoring is linked with greater risk of increased glycosylated hemoglobin levels in females of premenopausal period. Similarly, obstructive sleep apnea (OSA) is thought to be associated with reduced sensitivity of insulin. In females who were suffering from obstructive sleep apnea, linkage with metabolic syndromes has been explained. [27]. In the one latest study the risk of development of hypertension in patients who were suffering from obstructive sleep apnea were higher than the control group(apnoea/hypopnoea index <15) .[28-29]

GDM (gestational diabetes mellitus), which is defined as “glucose intolerance with onset or first recognition during pregnancy is a state of temporary insulin resistance.” [30] Insulin resistance is linked to obesity, inflammation, cardiovascular disease and secretion of adipocytokines.[31] A variety of polypeptides secreted from adipose tissue play a major role in the metabolic homoeostasis and the process of Type II diabetes..[32-34] These days, adipocytokinevisfatin, which was abbreviated in the traditional time as PBEF (pre-B-cell colony-enhancing factor) , was

thought to be seen in visceral fat.[35] Furthermore it is identified in skeletal muscle, liver, bone marrow as well as lymphocytes. Visfatin affects the insulin signal transduction pathway by inducing phosphorylation of the receptors of insulin receptor and IRS1 and 2 (insulin receptor substrate 1 and 2) in the hepatocytes.[36]

Furthermore, modes of action for modulating the sensitivity of insulin is considered to be an autocrine / paracrine function on adipocytes of viscera along with endocrine function. There was elevation of concentration of in patients who were suffering from type II diabetes ; however, data on visfatin in clinical settings are rare up until now. [37]The expression of the adipocytokineleptin, which reduces the production of glucose from hepatocytes as well as the ability of uptake of glucose in muscles is influenced by alterations in glucose metabolism. As visfatin is considered to contribute to the regulation of glucose levels, it could be presumed that changes in leptin might be related to visfatin.[38]

The exact physiology of mechanism through which there is increased risk of development of gestational diabetes mellitus as a consequence of snoring or sleep is the stimulation of metabolic process which targets on the action of insulin and the regulation of glucose. More precisely, there is an obstruction upper respiratory tract as a consequence of snoring or sleep apnea which causes desaturation of oxygen levels, hence elevating the levels of catecholamine and cortisol , ultimately leading to increase in resistance to insulin.[33]

According to an agreement which was accepted internationally regarding gestational diabetes mellitus definition is as “carbohydrate intolerance that begins or is first recognized during pregnancy”. Although the adverse impact of gestational diabetes mellitus on the outcomes of pregnancies have many controversies still there is requirement for screening as well as management of pregnant females who are suffering from gestational diabetes mellitus.[34] The reports of various studies of randomized controlled trials as well as cohort multicenter studies, signified the actual requirement for management of hyperglycemia in pregnant females who have GDM to improve the quality of outcomes of pregnancy .[35]

Gestational diabetes mellitus is generally comprised of and leads to adverse outcomes of pregnancy like macrosomia and low section cesserean surgery. GDM is also thought to cause long term ill-health effects on the mother as well as their children like higher risk of developing type 2 diabetes mellitus, obesity linked to mother as well as child and cardiovascular complications .[36] There were many studies which investigated the association between GDM and maternal obesity and determined increased prevalence of gestational diabetes mellitus in pregnant females who were obese rather than those who were having normal weight. Additionally, insulin was required in pregnant obese females who were having gestational diabetes mellitus to control the blood glucose level where as in pregnant females who were having normal weight glucose level was maintained with diet only. When obesity occurs in addition with gestational diabetes mellitus it results in worse perinatal outcomes..[37] Various studies determined the effect of obesity on pregnancy outcomes and maternal hyperglycemia on the outcomes of delivery individually. Some studies examined the individual effect of obesity on fetal weight as well as and the effect of GDM on the weight of the fetus, type of delivery whether C-section or normal vaginal and pregnancy associated hypertension The results of these studies indicated greater independent effect of obesity the adverse pregnancy outcomes rather than that of gestational diabetes mellitus. .[38]

1.1 NEED FOR THE STUDY:

Snoring, obesity and gestational diabetes mellitus all of these parameters independently or combination of these are associated with increased rate of morbidity as well as mortality. Till date there is paucity of data regarding correlation of snoring, BMI and gestational diabetes mellitus in pregnancy, so there is need to determine the correlation between snoring, BMI and gestational diabetes mellitus in pregnancy.

1.2 SIGNIFICANCE OF THE STUDY:

As snoring remains unnoticed unless until sleep of the partner gets disturbed and normal population does not take snoring into consideration seriously and it also shows adverse impact on fetal outcomes. Evaluation of the above parameters will also help in improving the quality of maternal as well as fetal life.

1.3 OBJECTIVE:

1. To determine the correlation between snoring and BMI in pregnancy.
2. To determine the relationship between snoring and gestational diabetes mellitus in pregnancy.
3. To determine whether there is any association between BMI and gestational diabetes mellitus in pregnancy.

1.4 HYPOTHESIS:

Alternate Hypothesis (HI): There is significant relationship between Snoring, BMI and Gestational Diabetes Mellitus in pregnancy.

Null Hypothesis (HO): There is no significant relationship between snoring, BMI and gestational diabetes mellitus in pregnancy.

1.5 OPERATIONAL DEFINITIONS:

Snoring is “the vibration of respiratory structures and the resulting sound, due to obstructed air movement during breathing while sleeping.”

The body mass index (BMI), or Quetelet index, is “a measure of relative weight based on an individual's mass and height.”

Gestational diabetes mellitus (GDM) is defined as “carbohydrate intolerance of variable severity with onset or first recognition during pregnancy and often diagnosed at 24–28 weeks of gestation.”

REVIEW OF LITERATURE

Spiegel K et al (1999) did a research on Impact of sleep debt on metabolic and endocrine function and found that Glucose tolerance was lower in the sleep-debt condition than in the fully rested condition ($p<0.02$), as were thyrotropin concentrations ($p<0.01$). Evening cortisol concentrations were raised ($p=0.0001$) and activity of the sympathetic nervous system was increased in the sleep-debt condition ($p<0.02$).

Franklin et al (2000) did retrospective cross sectional study to see the relationship of Snoring, Pregnancy induced hypertension and Growth retardation of fetus and found that Snoring is common in pregnancy and is a sign of pregnancy-induced hypertension. Snoring indicates a risk of growth retardation of the fetus.

Paul E. Peppard et al (2000) conducted a study on Prospective Study of the Association between Sleep-Disordered Breathing and Hypertension and found a dose–response association between sleep-disordered breathing at base line and the presence of hypertension four years later that was independent of known confounding factors. The findings suggest that sleep-disordered breathing is likely to be a risk factor for hypertension and consequent cardiovascular morbidity in the general population.

Izci B et al (2004) did a research on Sleep complaints: snoring and daytime sleepiness in pregnant and pre-eclamptic women and concluded that Snoring and sleepiness increased in the third trimester of pregnancy, particularly in patients with pre-eclampsia. However, the study suggests that sleepiness in pregnancy is largely due to factors other than snoring or breathing pauses.

Edwards et al (2005) did a study Severity of sleep disordered breathing improves following parturition. In his longitudinal study of sleep disordered breathing during pregnancy and post-partum,10 women of third trimester of pregnancy who were suspected for sleep disordered breathing were taken. Full overnight polysomnography and continuous systemic blood pressure were measured during the third trimester of pregnancy and 3 months following delivery and

concluded that late pregnancy may be associated with increased severity of sleep-disordered breathing and associated blood-pressure responses.

Malin Svenssen et al (2006) conducted a study on Risk Factors Associated With Snoring in Women With Special Emphasis on Body Mass Index and found that the prevalence of self-reported habitual snoring in women was strongly dependent on age and BMI. The importance of other risk factors differed depending on BMI, with alcohol dependence being associated with self-reported snoring in lean women, whereas physical inactivity was a risk factor for self-reported snoring in women with a high BMI.

Perez-Chada D et al (2007) conducted a study on Snoring, witnessed sleep apneas and pregnancy-induced hypertension and concluded that Snoring does increase during pregnancy, and this increase is associated with sleepiness, higher BMI at the start of pregnancy and higher prevalence of edema, but not with weight gain.

Kristen L. Knutson and Eve Van Cauter (2008) did a research on Associations between Sleep Loss and Increased Risk of Obesity and Diabetes and found that experimental sleep restriction is associated with an adverse impact on glucose homeostasis. Insulin sensitivity decreases rapidly and markedly without adequate compensation in beta cell function, resulting in an elevated risk of diabetes. Sleep curtailment is also associated with a dysregulation of the neuroendocrine control of appetite, with a reduction of the satiety factor, leptin, and an increase in the hunger-promoting hormone, ghrelin. Thus, sleep loss may alter the ability of leptin and ghrelin to accurately signal caloric need, acting in concert to produce an internal misperception of insufficient energy availability. The adverse impact of sleep deprivation on appetite regulation is likely to be driven by increased activity in neuronal populations expressing the excitatory peptides orexins that promote both waking and feeding.

Tasali E et al (2008) conducted a study on Slow-wave sleep and the risk of type 2 diabetes in humans and found that a clear role for SWS in the maintenance of normal glucose homeostasis. Furthermore, the data suggest that reduced sleep quality with low levels of SWS, as occurs in aging and in many obese individuals, may contribute to increase the risk of type 2 diabetes.

Foster GD et al (2009) did research on Obstructive sleep apnea among obese patients with type 2 diabetes and found that there is likelihood of OSA in obese patients with type 2 diabetes, especially among individuals with higher waist circumference and BMI.

Louis JM et al (2010) conducted a study on Maternal and neonatal morbidities associated with obstructive sleep apnea complicating pregnancy and found that pregnancies complicated by OSA are at risk for preeclampsia, medical complications and Preterm birth.

Hutchison BL et al (2012) conducted a postal survey of maternal sleep in late pregnancy and concluded that sleep problems are common in women in late pregnancy, and increase markedly compared with before pregnancy.

Louis et al (2012) did a research on perinatal outcomes associated with obstructive sleep apnea in obese pregnant women.In his prospective observational study,175 women underwent an overnight sleep study using a portable home monitor. Studies were manually scored by a central masked Sleep Reading Center using American Academy of Sleep Medicine diagnostic criteria. An apnea hypopnea index of 5 or greater was considered diagnostic of OSA. Perinatal outcomes were compared between women with and without OSA and concluded that OSA among obese pregnant women is associated with more frequent preeclampsia, neonatal intensive care unit admissions, and cesarean delivery.

O'Brien LM et al (2012) conducted a prospective cohort study on Pregnancy-onset habitual snoring, gestational hypertension, and preeclampsia and concluded that New-onset snoring during pregnancy is a strong risk factor for gestational hypertension and preeclampsia.

Fung AM et al (2013) conducted a study on effects of maternal obstructive sleep apnoea on fetal growth and found that OSA may be associated with reduced fetal growth in late pregnancy. Further evaluation is warranted to establish whether OSA may be an important contributor to adverse perinatal outcome, including stillbirth.

MATERIALS AND METHODOLOGY

3.1 STUDY DESIGN: Cross sectional study.

3.2 STUDY SETTING: Goodwill Hospital, Jalandhar

Shri Baldev Raj Mittal Hospital, Phagwara.

Deepak Hospital, Ludhiana.

Chabbra Hospital Ludhiana.

Seerat Hospital Ludhiana.

3.3 POPULATION AND SAMPLING:

Type of Sampling: Convenient Sampling.

Sample Size (Female) : 40

3.4 CRITERIA:

INCLUSION CRIETERIA:

1. Age > 18 Years.
2. Pregnancy: Nulliparous & Multiparous
3. Gestational Age > 28 Weeks.
4. BMI > 18

EXCLUSION CRITERIA:

1. Age > 35 Years.
2. Non-pregnant Female.
3. Gestational Age < 28 Weeks
4. Any Systemic illness.
5. Any psychological problem.

3.4 PARAMETERS:

1. ESS (Epworth Sleepiness Scale)
2. Glucose Tolerance Test.
3. BMI

3.6 INSTRUMENTAL TOOL:

1. Epworth Sleepiness Scale (ESS)
2. Weighing machine
3. Wall mounted Stadiometer

3.7 PROCEDURE:

Pregnant women in second trimester of gestation who visited Deepak ,Chabbra and Seerat hospitals were invited to take part in the present study. Informed consent in the written form had been got signed from 40 participants. Age, pre-pregnancy BMI, current weight, height and Blood sugar level had been recorded.

The subjects were given to fill questionnaire with their partner. The questionnaire contained the Epworth Sleepiness Scale, which is a standardized instrument used for measuring sleepiness in patients who suffers from snoring or obstructive sleep apnea. In this scale the female was ranked according to their sleeping probability for 8 different situations on a scale of increasing probability from 0 to 3.Subjects were tested for Glucose Tolerance Test (GTT) and four readings had been obtained.

3.8 STATISTICAL ANALYSIS:

Data obtained will be analyzed by Karl Person's Correlation.

$$r = \frac{\sum d_x d_y - \frac{\left(\sum d_x\right) \times \left(\sum d_y\right)}{N}}{\sqrt{\sum {d_x}^2 - \frac{\left(\sum d_x\right)^2}{N}} \times \sqrt{\sum {d_y}^2 - \frac{\left(\sum d_y\right)^2}{N}}}$$

where,

d_x = Deviation of X series from assumed mean

d_y = Deviation of Y series from assumed mean

$\sum d_x d_y$ = Sum of multiples of d_x and d_y

$\sum {d_x}^2$ = Sum of squares of d_x

$\sum {d_y}^2$ = Sum of squares of d_y

$\sum d_x$ = Sum of deviations of X series

$\sum d_y$ = Sum of deviations of Y series

N = Total numbers of observations

DATA ANALYSIS AND RESULT:

40 females of third trimester pregnancy were taken.

Table:4.1—Categorization of females based on their pre-pregnancy BMI.

S.No.	Pre-Pregnancy BMI	No. of females	N
1.	Underweight	0	40
2.	Normal Weight	9	40
3.	Overweight	22	40
4.	Obesity	9	40

Table 4.1 shows categorization of females according to their pre-pregnancy BMI.BMI includes 4 subclasses – Underweight having BMI below 18.5, Normal weight having BMI between 18.5-24.9, Overweight having BMI between 24.9-29.5 and Obese females who were ving BMI more than 30.

Table: 4.2 – Categorization of females based on their gestational age.

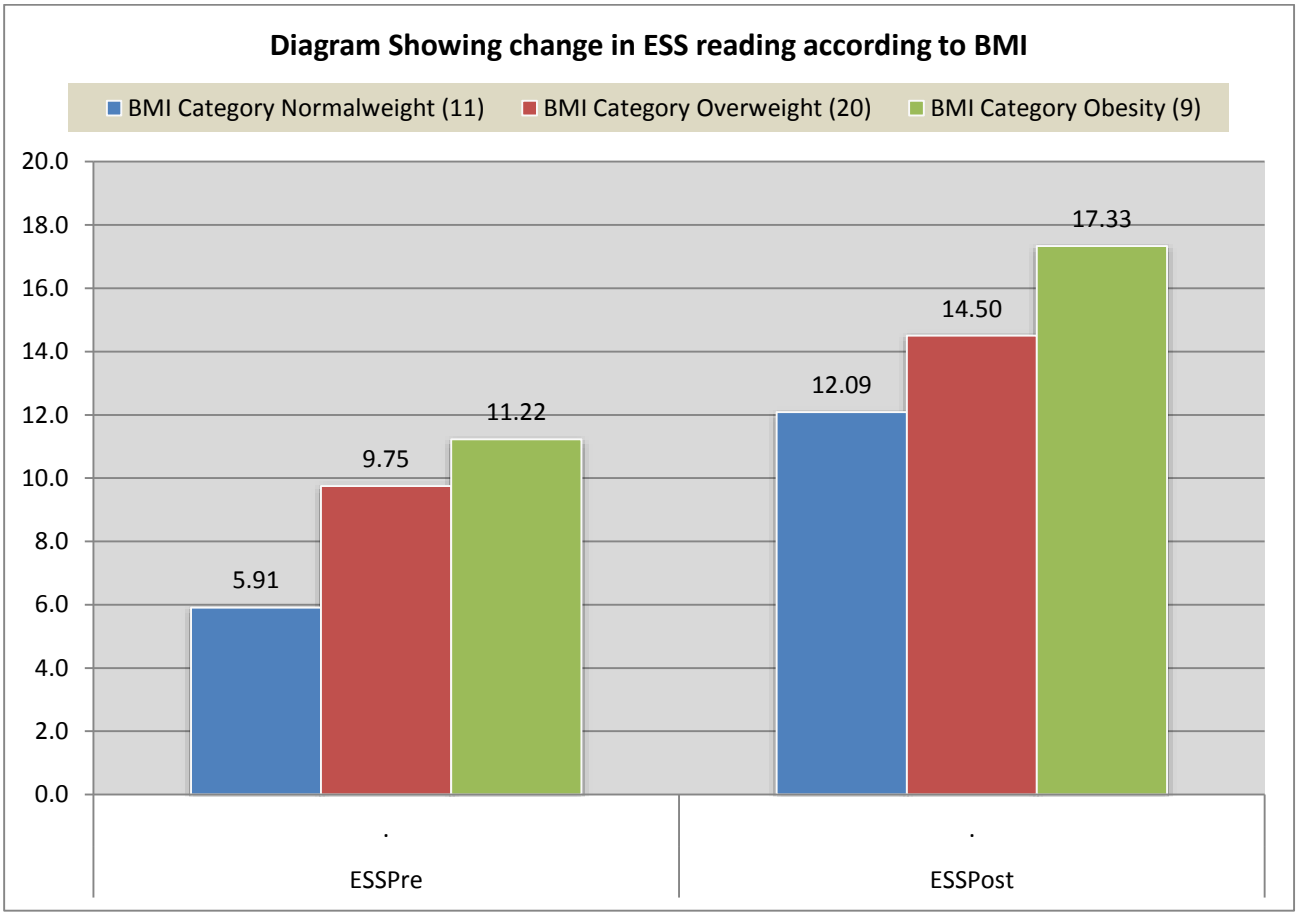
S.No.	Gestational age of pregnancy	No. of females	N
1.	27 Wks- 30 Wks	14	40
2	31 Wks- 35 Wks	19	40
3	36 Wks- 40 Wks	7	40

Table 4.2 shows the categorization of females according to their gestational age in pregnancy. All the females taken were of third trimester. Third trimester is further divided into 3 subclasses—Class 1 having gestational age between 27weeks – 30 weeks, Class II is having gestational age between 31weeks-35 weeks and Class III having gestational age between 36 weeks- 40 weeks.

Table 4.3 – Change in ESS reading according to BMI

	BMI Category			
	Underweight (0)	Normalweight (11)	Overweight (20)	Obesity (9)
ESSPre	.	5.91	9.75	11.22
ESSPost	.	12.09	14.50	17.33

Table 4.3 shows changes in ESS (Epworth Sleepiness Scale) reading that represents snoring from pre-pregnancy state to during pregnancy according to the categories of BMI.

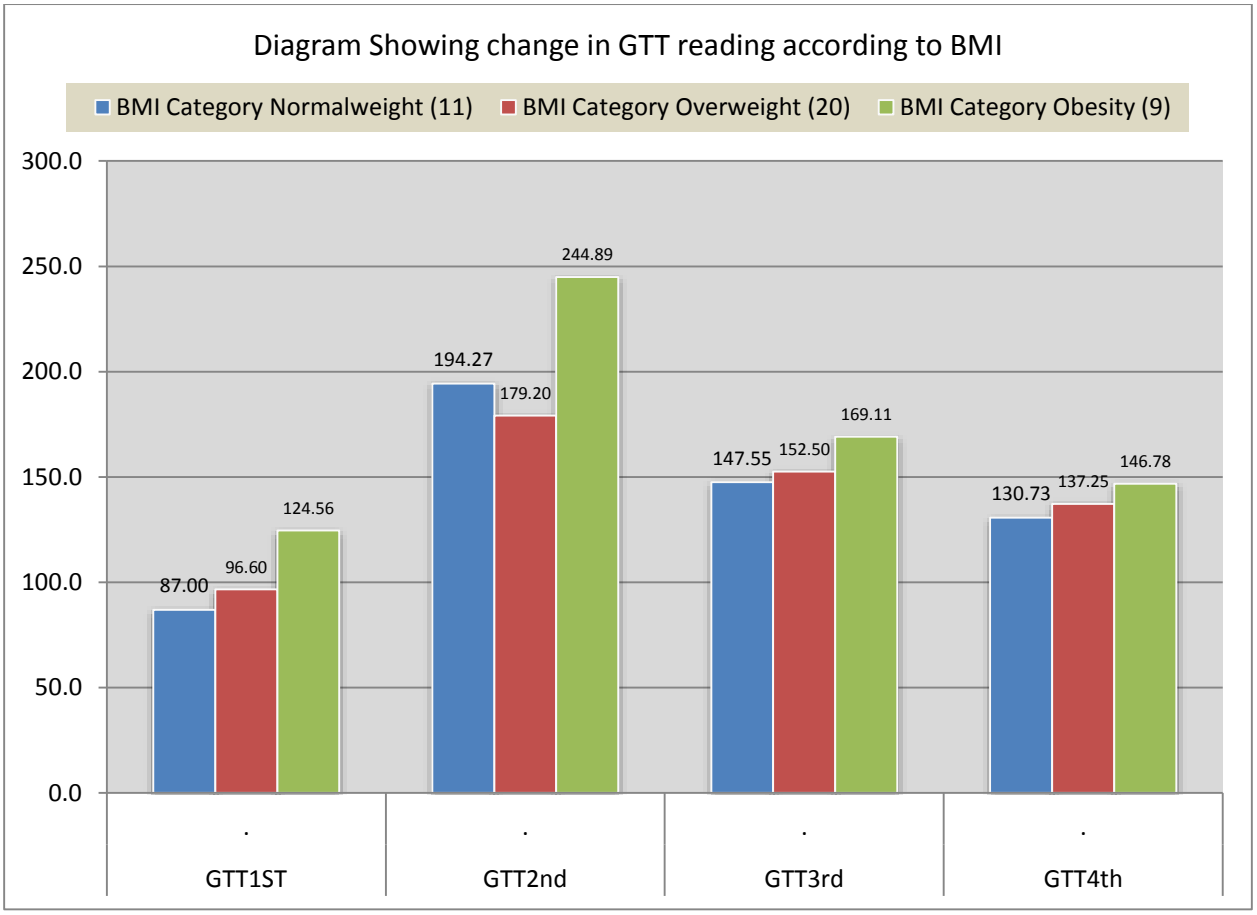


Graph 4.1: shows change in ESS reading according to BMI

Table 4.4—shows glucose tolerance test (GTT) readings based on categories of BMI

Mean	BMI Category			
	Underweight (0)	Normalweight (11)	Overweight (20)	Obesity (9)
GTT1ST	.	87.00	96.60	124.56
GTT2nd	.	194.27	179.20	244.89
GTT3rd	.	147.55	152.50	169.11
GTT4th	.	130.73	137.25	146.78

Table 4.4 shows glucose tolerance test readings which was taken every hourly and categorized on basis of BMI.



Graph 4.2—Shows changes of glucose tolerance test reading according to BMI

Table 4.5: Correlation Matrix of BMI, ESS and GTT Readings

Correlations									
		BMIPre	BMIPost	ESSPre	ESSPost	GTT1ST	GTT2nd	GTT3rd	GTT4th
BMIPre	Pearson Correlation								
	P value								
	N								
BMIPost	Pearson Correlation	.953**							
	P value	0.000							
	N	40							
ESSPre	Pearson Correlation	.513**	.567**						
	P value	0.001	0.000						
	N	40	40						
ESSPost	Pearson Correlation	.441**	.498**	.765**					
	P value	0.004	0.001	0.000					
	N	40	40	40					
GTT1ST	Pearson Correlation	.700**	.678**	0.203	0.144				
	P value	0.000	0.000	0.210	0.376				
	N	40	40	40	40				
GTT2nd	Pearson Correlation	.358*	.409**	0.129	0.292	.543**			
	P value	0.023	0.009	0.427	0.067	0.000			
	N	40	40	40	40	40			
GTT3rd	Pearson Correlation	.469**	.488**	0.147	0.172	.742**	.664**		
	P value	0.002	0.001	0.365	0.289	0.000	0.000		
	N	40	40	40	40	40	40		
GTT4th	Pearson Correlation	0.303	.313*	0.113	0.243	.590**	.567**	.756**	
	P value	0.057	0.049	0.487	0.130	0.000	0.000	0.000	
	N	40	40	40	40	40	40	40	

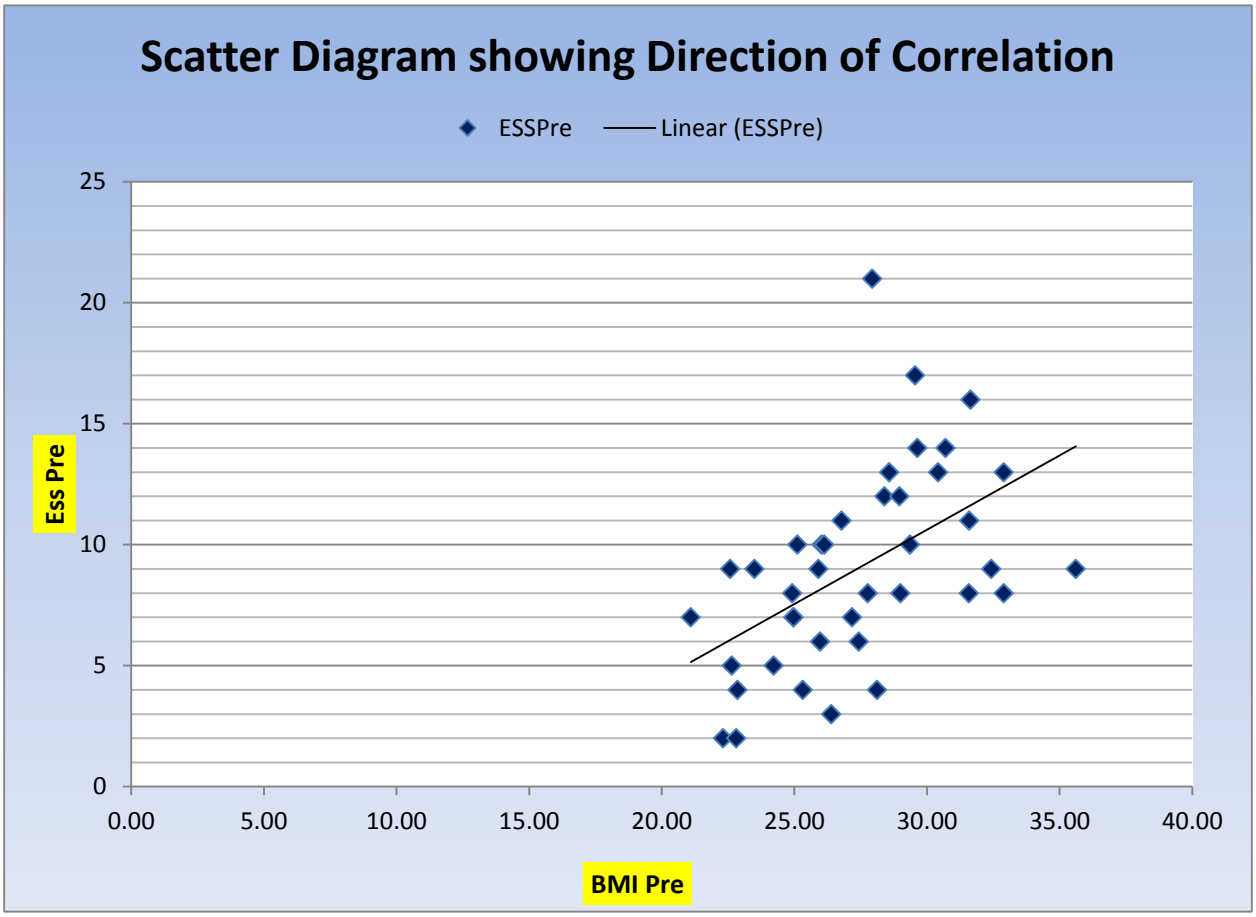
**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed). Table Value at 5% df 38=0.312

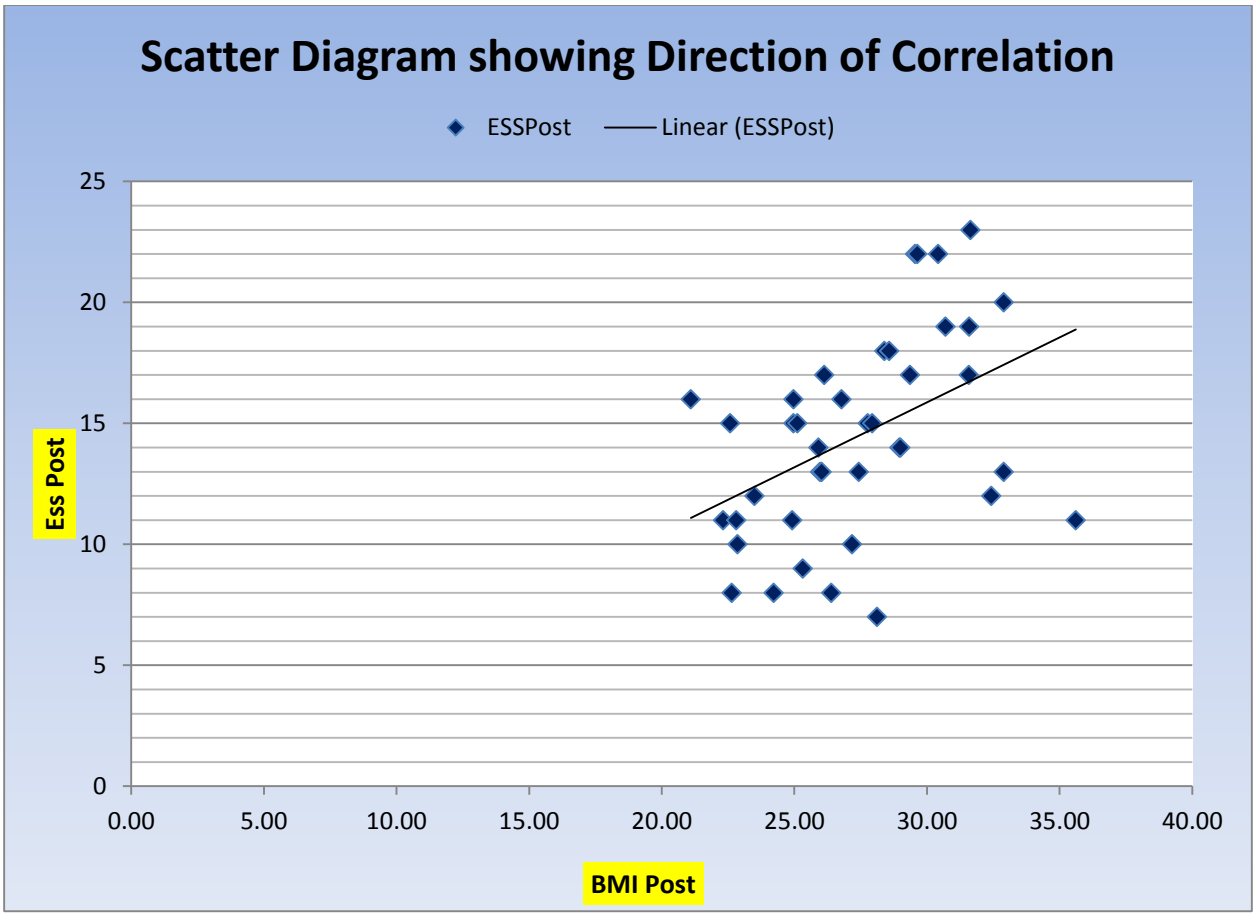
Table 5 shows correlation matrix which was based on the basis of Karl Person's correlation. The correlation coefficient between BMI Pre and BMI Post, (r=0.953) , between BMI Pre and ESS

Pre,(r=0.513) , between ESS Pre and BMI Post ,(r= 0.567) ,between ESS Pre and ESS Post (r=0.765),

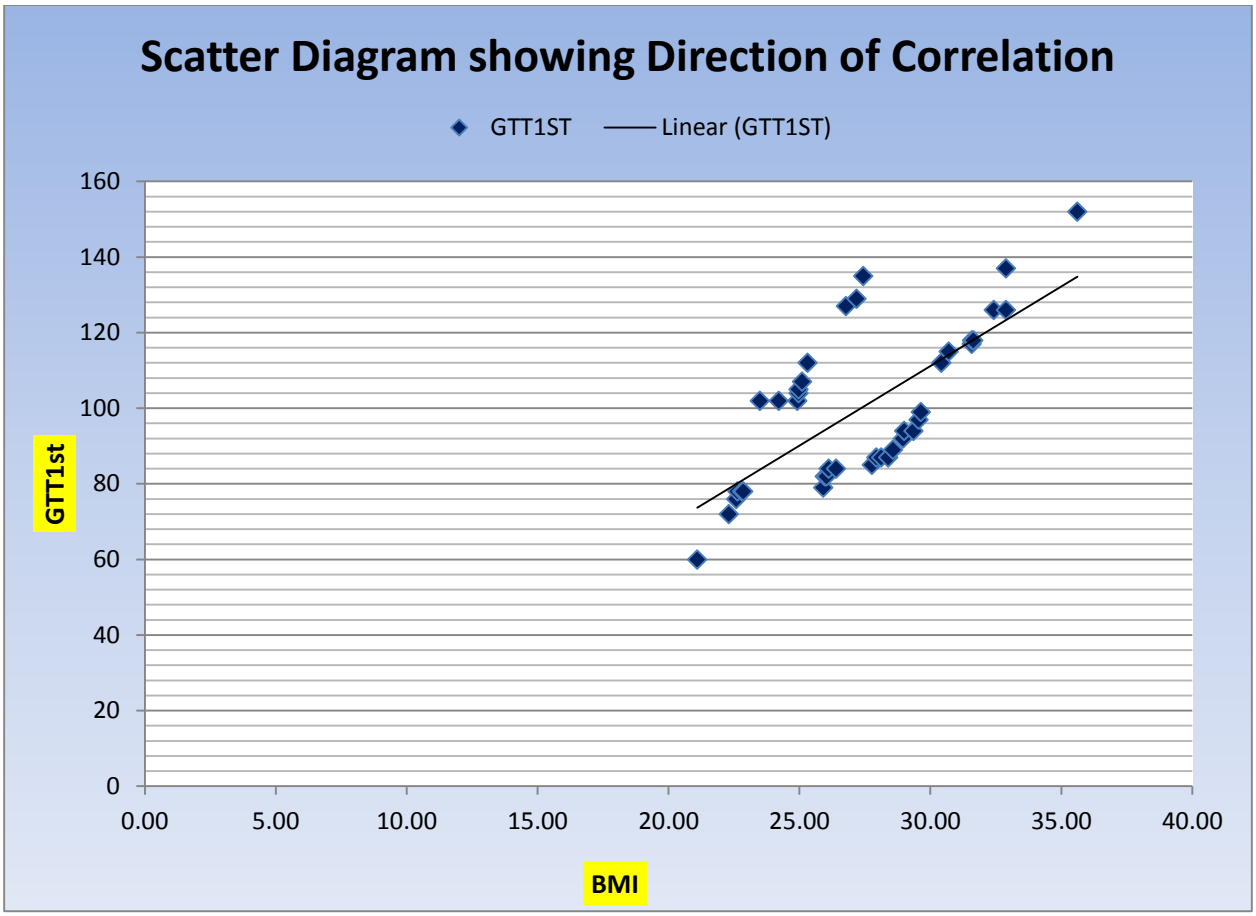
between ESS Post and BMI Pre , (r= 0.441) ,between ESS Post and BMI Post (r=0.498) , between GTT 1^{st} and BMI Pre ,(r=0.700) ,between GTT 1^{st} and BMI Post (r=0.678) ,between GTT 1^{st} and ESS Pre ,(r= 0.203) , between GTT 1^{st} and ESS post ,(r=0.144) ,between GTT 2^{nd} and BMI Pre ,(r=0.358) , between GTT 2^{nd} and BMI Post ,(r=0.409) ,between GTT 2^{nd} and ESS Pre , (r=0.129), between GTT 2^{nd} and ESS Post ,(r=0.292), between GTT 3^{rd} and BMI Pre ,(r=0.469) , between GTT 3^{rd} and BMI Post ,(r=0.488) ,between GTT 3^{rd} and ESS Pre , (r=0.147), between GTT 3^{rd} and ESS Post ,(r=0.172), between GTT 4^{th} and BMI Pre ,(r=0.303) , between GTT 4^{th} and BMI Post ,(r=0.313) ,between GTT 4^{th} and ESS Pre , (r=0.113), between GTT 4^{th} and ESS Post ,(r=0.243)



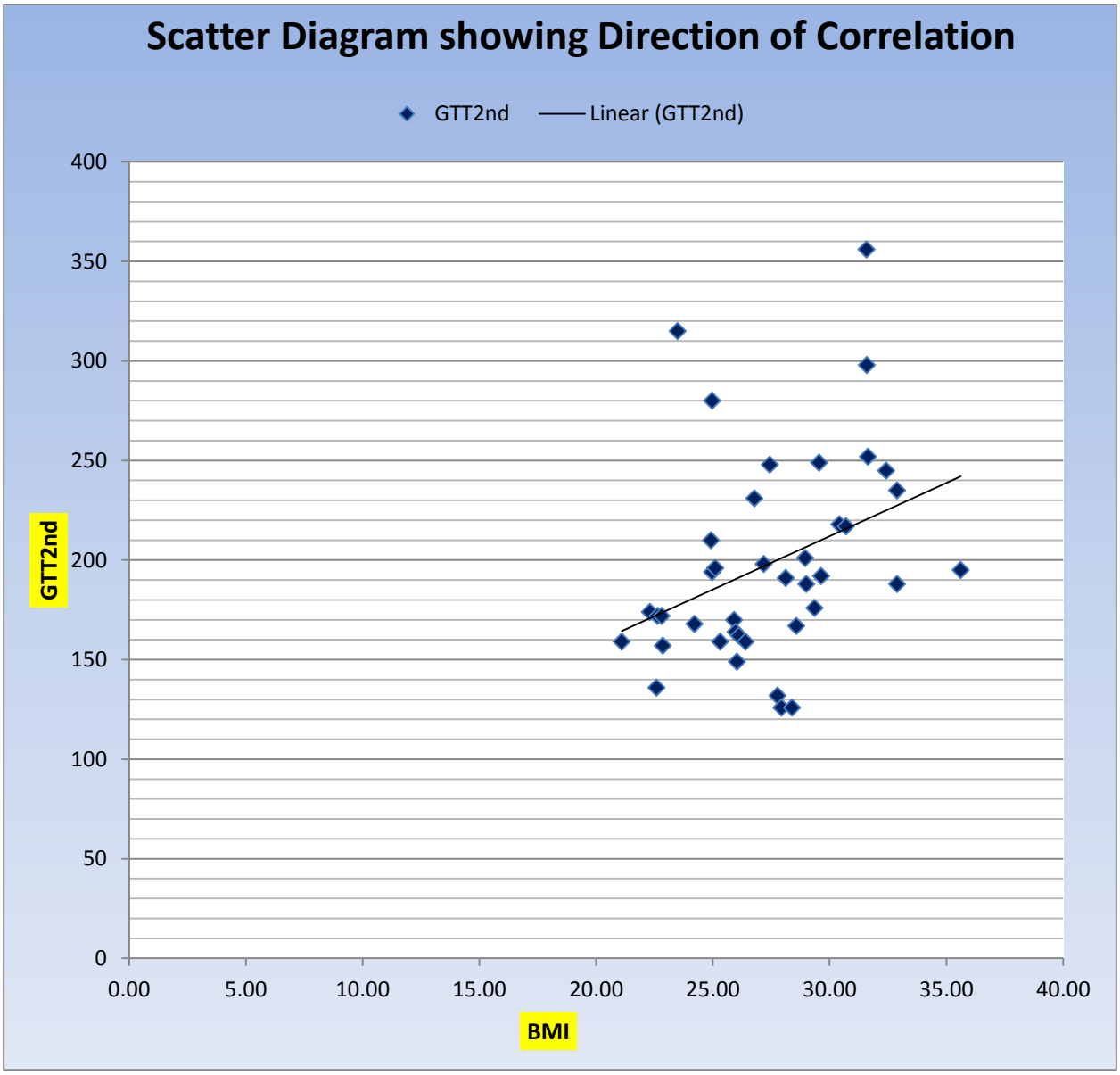
Graph 4.3: Represents scatter diagram of correlation between pre-pregnancy ESS reading and pre-pregnancy BMI.



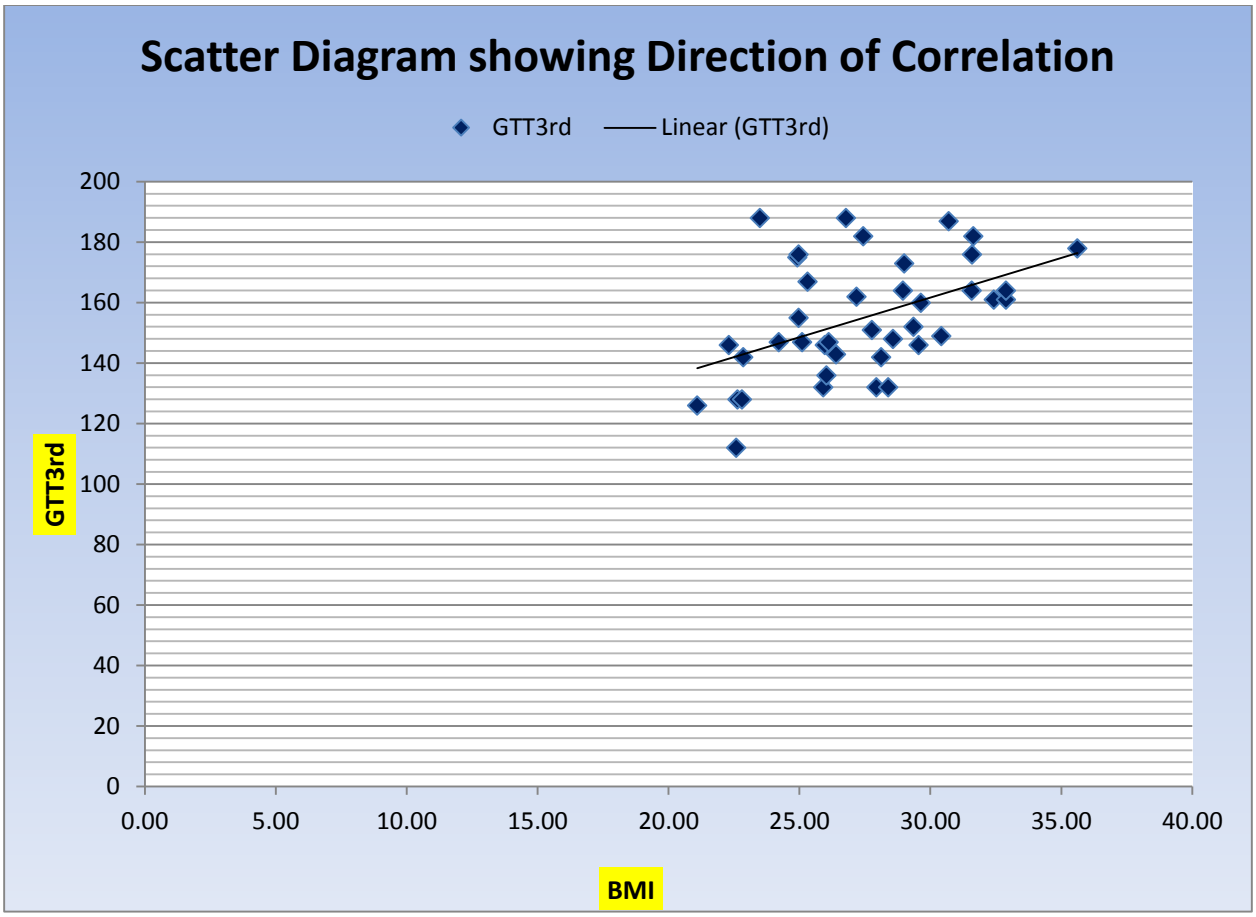
Graph 4.4: Represents scatter diagram showing correlation between ESS reading during pregnancy and BMI during pregnancy.



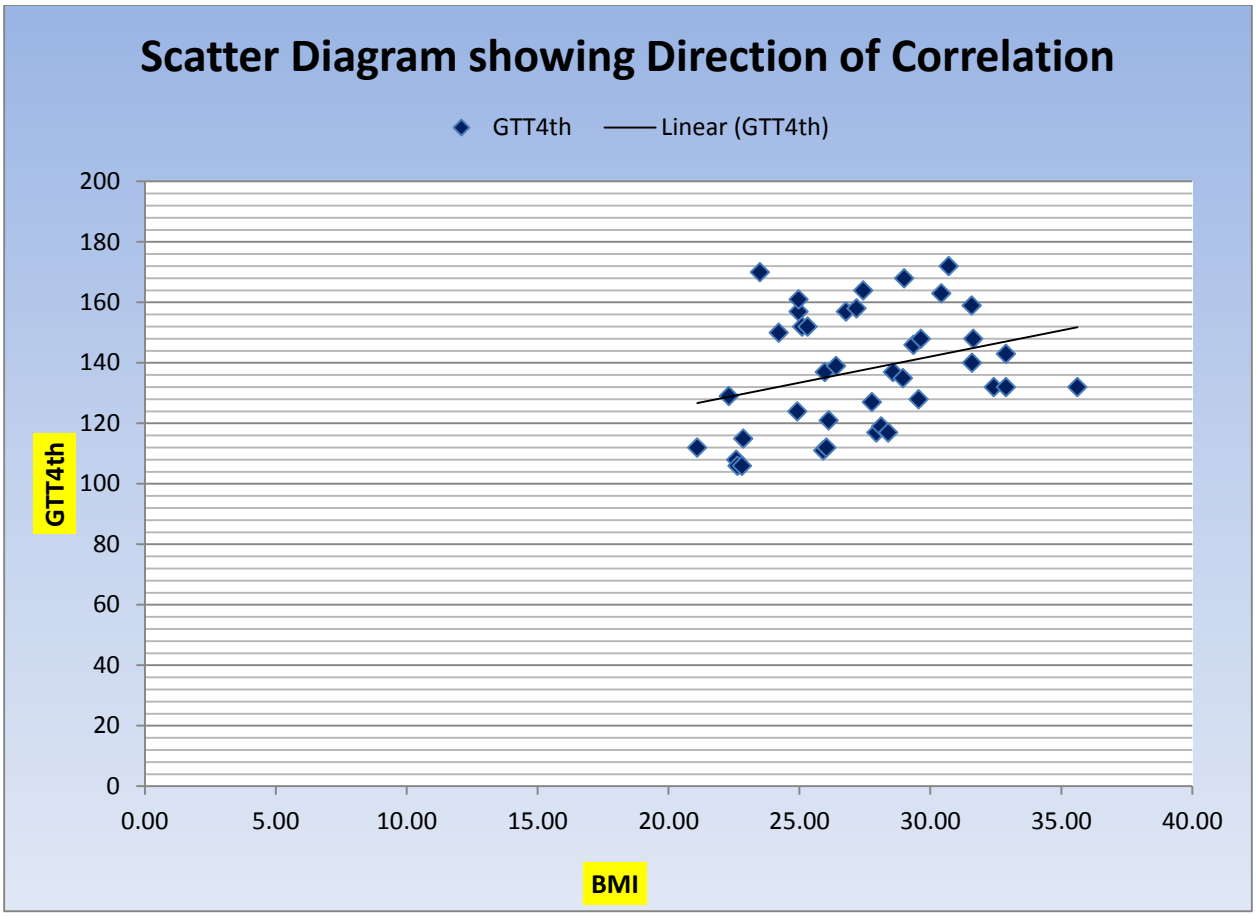
Graph 4.5: Represents scatter diagram of correlation between GTT 1st reading and BMI reading.



Graph 4.6: Represents scatter diagram of correlation between GTT 2nd reading and BMI reading.



Graph 4.7: Represents scatter diagram of correlation between GTT 3rd reading and BMI reading.

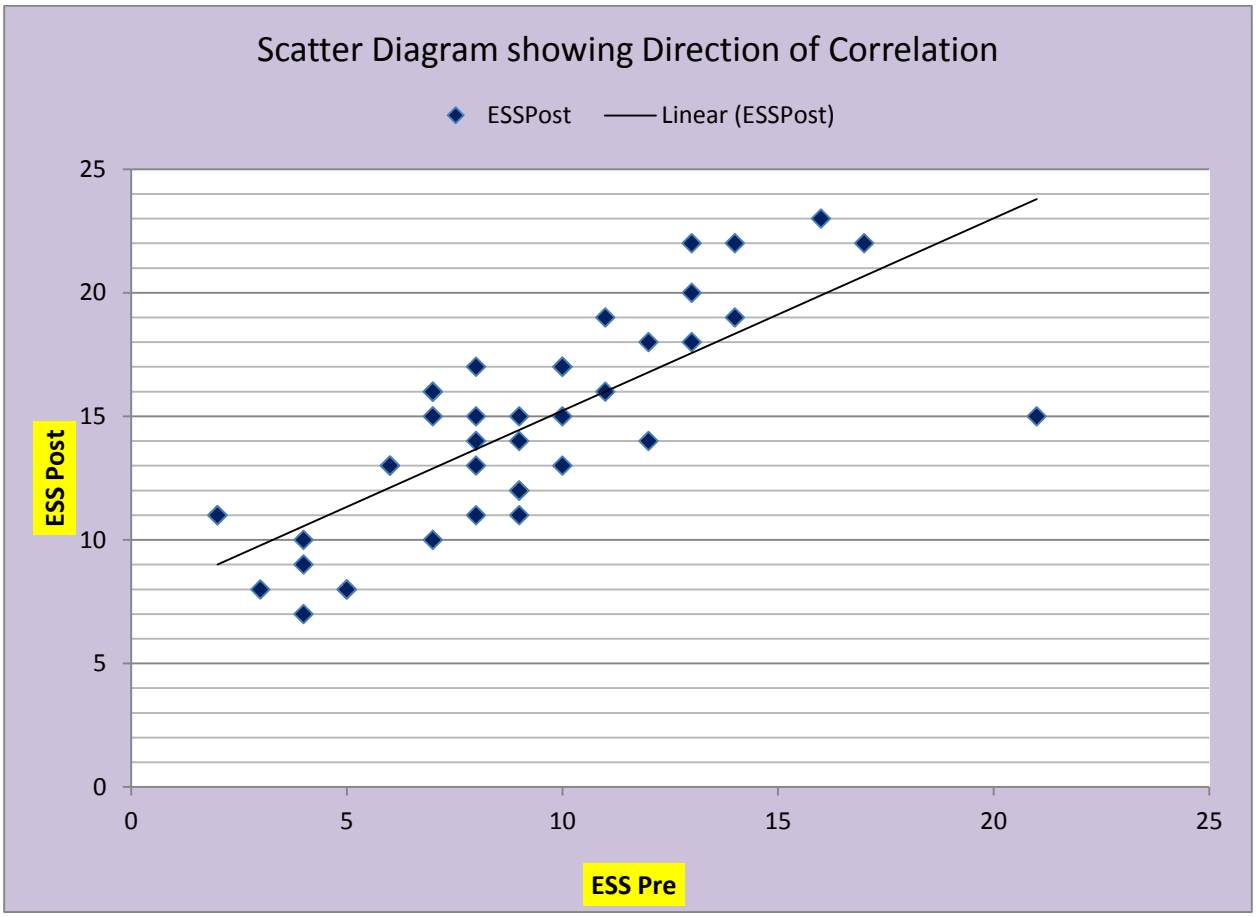


Graph 4.8: Represents scatter diagram of correlation between GTT 4th reading and BMI reading.

Table 4.6: Correlation Matrix of ESS and GTT Readings.

Correlation between ESS Pre and……		ESSPre
ESSPost	Pearson Correlation	.765**
	P value	0.00
	N	40
GTT1ST	Pearson Correlation	.679**
	P value	0.00
	N	40
GTT2nd	Pearson Correlation	.355*
	P value	0.02
	N	40
GTT3rd	Pearson Correlation	.557**
	P value	0.00
	N	40
GTT4th	Pearson Correlation	.489**
	P value	0.00
	N	40

Table 4.6 shows correlation matrix which was made based on the basis of Karl Person's correlation. The correlation coefficient between ESS Pre and ESS Post (r=0.765) ,between ESS Pre and GTT 1st ,(r= 0.679) ,between ESS Pre and GTT 2nd , (r=0.355), between ESS Pre and GTT 3rd , (r=0.557), between ESS Pre and GTT 4th , (r=0.489)

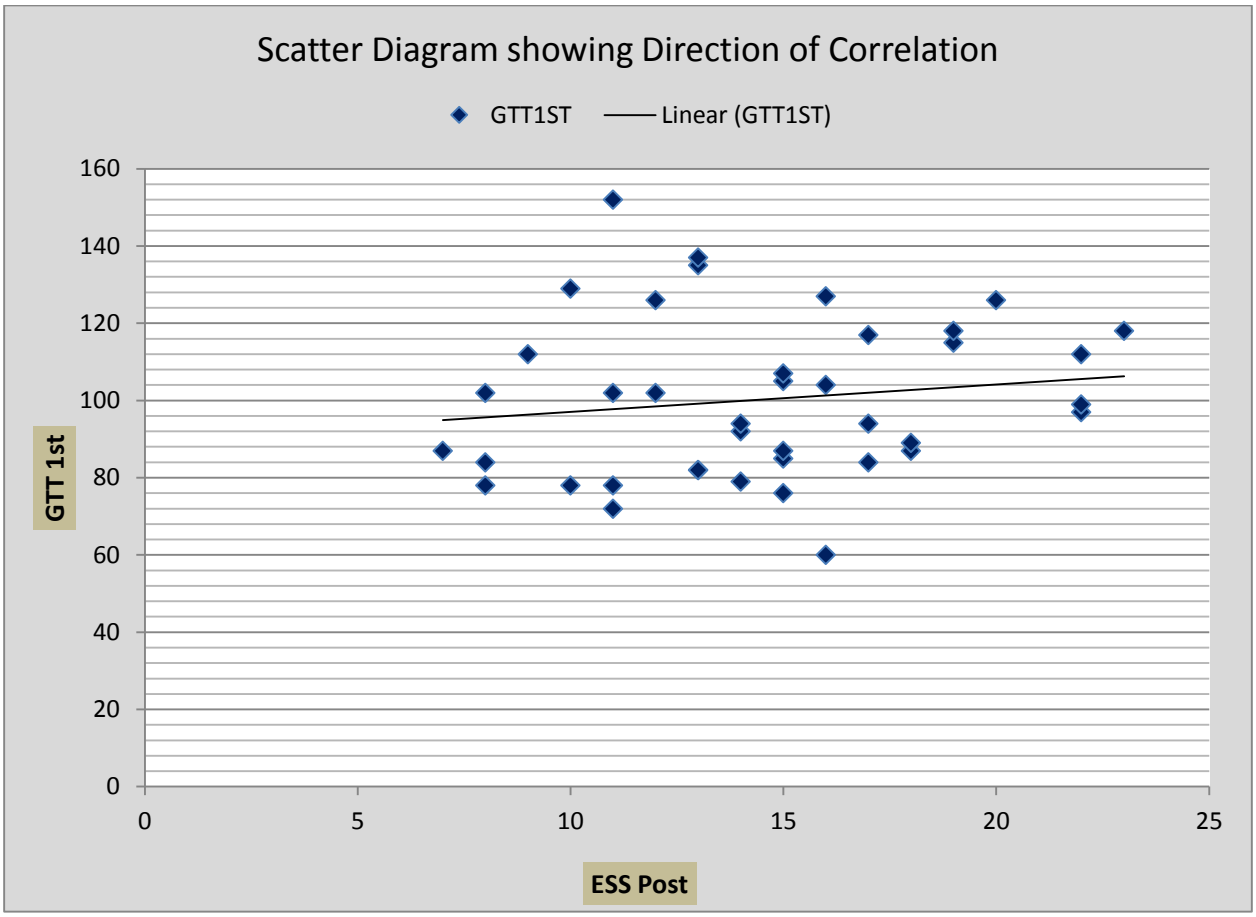


Graph 4.9: Represents scatter diagram of correlation between ESS Pre reading and ESS Post reading.

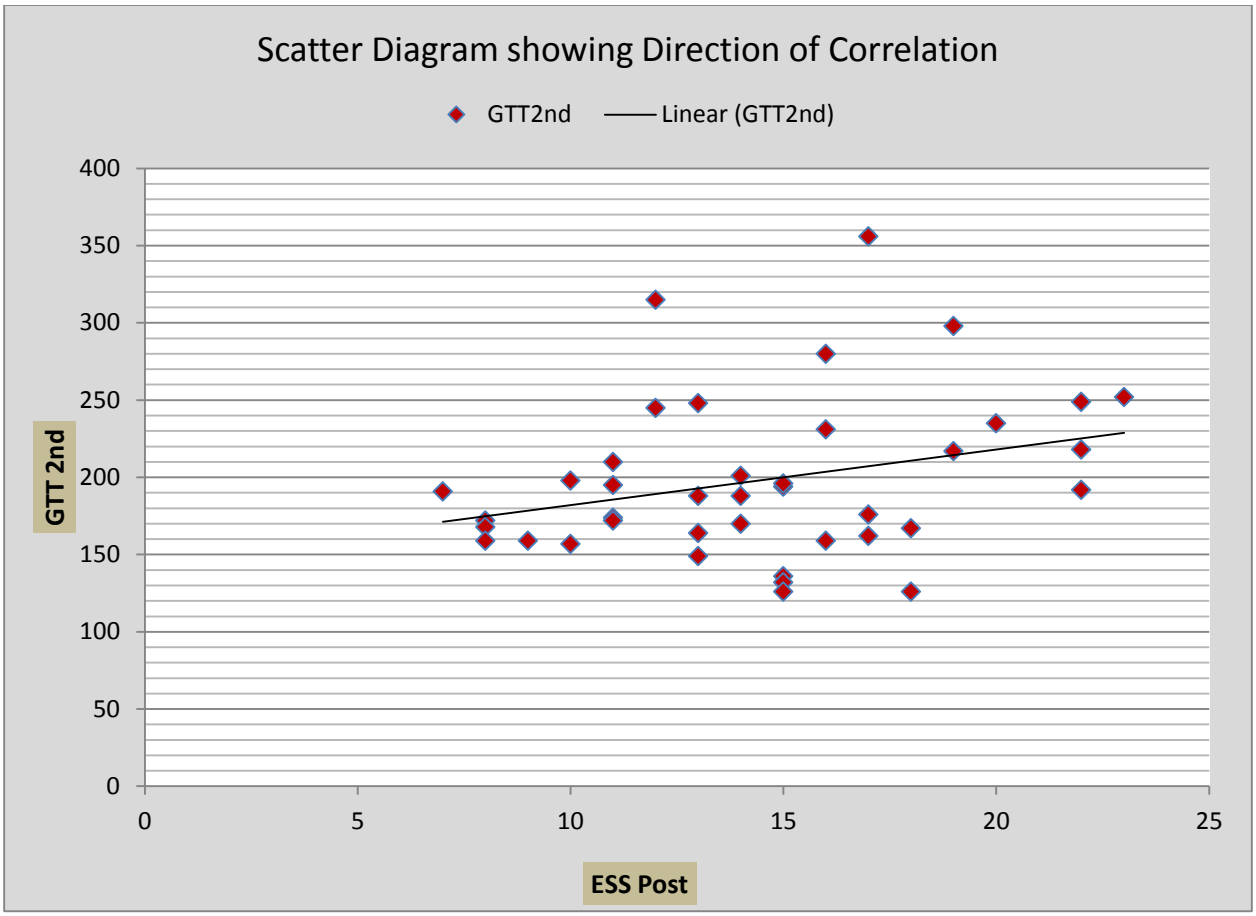
Table 4.7: Correlation matrix of ESS post and GTT readings.

Correlation between ESS Post and……		ESSPost
GTT1ST	Pearson Correlation	.626**
	P value	0.00
	N	40
GTT2nd	Pearson Correlation	.315*
	P value	0.05
	N	40
GTT3rd	Pearson Correlation	.433**
	P value	0.01
	N	40
GTT4th	Pearson Correlation	.432**
	P value	0.01
	N	40

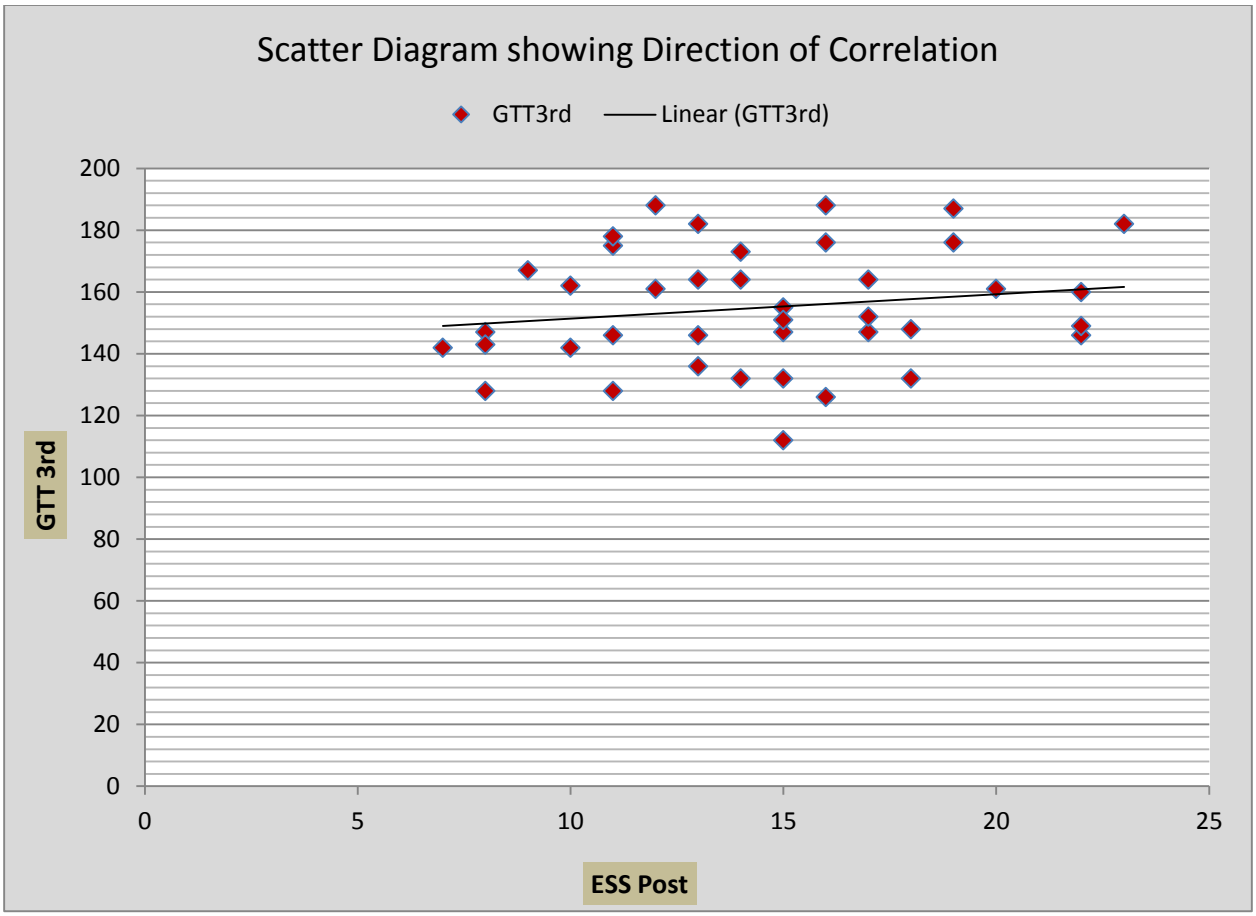
Table 4.7 shows correlation matrix which was based on the basis of Karl Person's correlation. The correlation coefficient between ESS Post and GTT 1st ,(r= 0.626) ,between ESS Post and GTT 2nd , (r=0.315), between ESS Post and GTT 3rd , (r=0.433), between ESS Post and GTT 4th , (r=0.432)



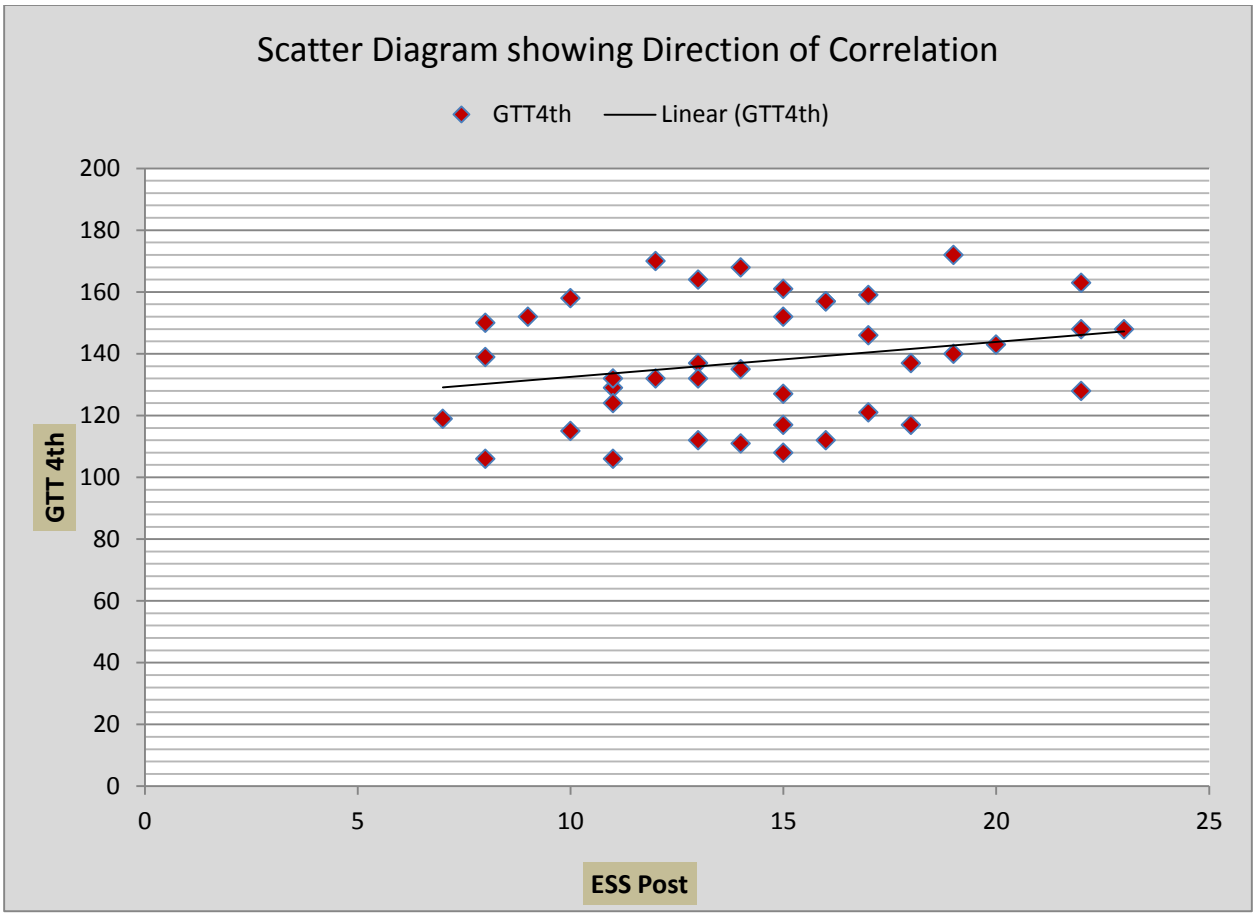
Graph 4.10: Represents scatter diagram of correlation between GTT 1st reading and ESS Post reading.



Graph 4.11: Represents scatter diagram of correlation between GTT 2nd reading and ESS Post reading.



Graph 4.12: Represents scatter diagram of correlation between GTT 3rd reading and ESS Post reading.



Graph 4.13: Represents scatter diagram of correlation between GTT 4th reading and ESS Post reading.

DISCUSSION

Snoring is very common problem of pregnancy characterized with obstruction of upper airways ,hypoventilation and nocturnal hypoxia. Physiological and hormonal changes that occur in pregnancy leads to changes in architecture of sleep. Hence pregnant women is at the risk of the development or worsening of preexisting apnea. As apneic episodes are commonly associated with oxy-hemoglobin desaturation, that means obstructive sleep apnea along with pregnancy can lead to maternal as well as fetal complications due to low oxygen reserves during pregnancy.(50) Long-term complications associated with snoring and sleep apnea especially OSA include cardiovascular and non-cardiovascular morbidities and mortality. With progression of pregnancy there occurs hyperglycemia which is associated with increasing resistance to insulin since mid-gestation period of pregnancy. Hyperglycemia is thought to play very important role in fetal development by providing sufficient quantity of glucose.(51)

The results of our present study showed significant positive linear and direct relationship between snoring and pregnancy. The exact mechanism underlying snoring during pregnancy is not well known but the most possible cause of development of snoring while pregnancy is reduction of the upper pharyngeal passage as a consequence of oro-pharyngeal oedema. Narrowing of upper airways are secondary to hormonal changes of pregnancy especially estrogen which mediates the mechanisms of induction of hyperemia, oedema of nasopharyngeal mucosa, and rhinitis with increased resistance to the airflow. Physiological hypervolemia during the course of pregnancy leads to eventually increment in weight of the mother and displacement of the diaphragm in upward direction may further increase the risk of snoring or sleep apnea in pregnancy. There is decrease in lumen size of passage of upper airways, which is major risk factor for snoring, had been identified in third trimester females of pregnancy. There is increased drive for respiration in pregnancy as diaphragm has to put extra effort which results in increase in pressure of suction at oropharyngeal junction, which further can lead to collapse of pharyngeal passage.(53)

The results identified in the research by Izci et al in 2003 were in agreement with the results of present study which reported that there is increase in circumference of lumen of upper respiratory tract in late pregnancy while in sitting position but the pregnant women had a much lesser lumen of upper airway passage as compared to non-pregnant controls in lying position.

This study failed to answer the if there is no difference in diameters and circumferences of neck then how it leads to narrowing of pharyngeal passage. Moreover, there is loss of defense by upper airways and dilatation of pharyngeal muscles eventually results in more reduction of dimensions of upper airways in pregnant female. The cross sectional design and biasness in selection criteria are the major limitations of this study. Joel-Cohen et al in 1978 first reported the enhancing collapsibility of sleep apnea during pregnancy. (54)

The results from our study indicate positive correlation between obesity and snoring. The exact cause of snoring in obese females is not clear but there is gain in weight and increased congestion of nasal mucosa in ongoing pregnancy are the predisposing factors which lead to development of or worsening of sleep apnea. In addition to above factors in obese people the upper airway results in reduced lumen circumference and approximation of pharyngeal walls leads to collapsibility of orophayngeal junction, which is predisposing factor for development of snoring or apnea. Moreover, there is excessive fat depots around the thorax leads to reduction of compliance of thoracic cavity and decrease in functional residual capacity, eventually increases demand of oxygen by body.(55)

During sleep apnea or snoring there is alteration of hormones which are linked to obesity and weight maintenance. In obesity there is increased fat mass leads to have higher levels of leptin. This hyperleptinemia is associated with decreased responses at cellular level to leptin hence resulting in inability to achieve the effect of leptin and responsible for implication of abnormal breathing patterns in obesity.(56) There are increased levels of various other adipokines, such as tumor necrosis factor a (TNFa) and interleukin-6 (IL-6), in obesity which is responsible for depression of activity of central nervous system and neuromuscular control of upper airways. Hence enhancing the severity of obstructive sleep apnea or snoring , which further stimulates the production of proinflammatory substances, developing a vicious cycle. Association of Sleep apnea with elevations of plasma levels of TNFa and IL-6 elevations is besides from occurrence of obesity. Visceral fat is thought to be more responsible the development of sleep apnea, and sleep apnea rather than subcutaneous or total body fat which further contributes to the development of gestational diabetes mellitus irrespective of presence of obesity. Protein adiponectin is secreted by adipose tissue. There is reduction in levels of circulating adiponectin

along with parallel elevation in visceral fat .There is decrease in adiponectin level in obesity as well as in sleep apnea or snoring..[57-58]

The present study showed the direct positive correlation between Gestational Diabetes Mellitus and Snoring. The exact mechanism of development of diabetes mellitus as a consequence of snoring is not known but heavy snoring is thought to be a predictor of obstructive sleep apnea which results in causing mechanical obstruction of the upper airway passage. There is increased resistance to insulin in pregnant females who are also suffering from snoring Mechanical obstruction of upper airway leads to desaturation of oxygen which further leads to increase in levels of catecholamine via enhancing the activity of sympathetic activity which results in increased insulin resistance and increased levels of peripheral circulating insulin by enhancing the process of glycogenolysis, gluconeogenesis, and glucose intolerance. The research by Strohl et al justifies the result of our study that episodes of obstructive sleep apnea and oxygen desaturation eventually results in resistance to insulin recptors because of elevation in catecholamine or cortisol levels.[59]The study by Brooks et al (1996) of 10 obese females who are having diabetes and also suffering from obstructive sleep apnea was also in agreement with results of our study in the manner that recordings of response of insulin prior to intervention and after intervention of 4 months that is pre and post readings had been taken. The intervention involved treatment of continuous positive airway pressure to reduce snoring and obstructive sleep apnea. There was significant improvement to insulin responsiveness after the treatment of CPAP. [60] Wilcox et al in his study also justified our explanation that Snoring is having impact on glucose metabolism eventually resulting in insulin resistance except the occurrence of central obesity. According to them the possible mechanism was increased activity of sympathetic nervous system which had been evidenced to occur in sleep apneas. .[61]

The results of our study determined a direct and positive correlation between gestational diabetes mellitus and obesity. There is disturbance in the breathing pattern as a consequence of obesity such as snoring or obstructive sleep apnea. Insulin resistance and diabetes mellitus are also caused by obesity. These above mentioned independent interactions of obesity to snoring and gestational diabetes mellitus has led to a speculation whether there is any direct relationship pf snoring and gestational diabetes mellitus which might be the reflection of obesity. There is an important role of obesity in the development of gestational diabetes mellitus via different

mechanisms that is chronic subclinical inflammation, low-grade acute phase response, and abnormal regulation of adipokines. There is direct and adverse impact of abnormal metabolism of adipose tissues on lipid and glucose homeostasis. The dysfunction of adipose tissues metabolism in obesity leads to abnormal secretion of cytokines levels which are connected to insulin resistance which further contributes to elevate the levels of fatty acids in circulation and there will be excessive load of fatty acids in skeletal muscles or liver. Elevated levels of fatty acids causes reduction of response of insulin in adipose tissues in conditions like obesity and insulin resistance which further leads to development of gestational diabetes mellitus.(63)

Mohammed Quatanani and Mitchell A.Lazar (2007) carried out a review on mechanisms and suggested that it is not only factor which primarily describes any interaction between obesity and insulin resistance. There are multiple pathways including neuroendocrine , immune system and inflammation which got disturbed and responsible for deviating the cell intrinsic function in different metablic tissues like fat or muscle.When there is problem in one pathway its chains are also get disturbed. The limitation of this study is that they could not determine the elements AT cellular level which leads to development of state of insulin-resistance.(64)

Results of our study revealed direct positive correlation between gestational diabetes mellitus and pregnancy. Basically there are two main pathways which causes gestational diabetes mellitus; insulin resistance and chronic subclinical inflammation. Either of the two factors to are responsible for development of resistance to insulin, first, the tissues are not able to respond to insulin and second beta cells from pancreas are not able to secrete insulin in sufficient amount. Besides of all the above factors, hormones which are produced by the placenta during pregnancy and increased fatty tissue of mother are the major factors which results in development of gestational diabetes mellitus. With increase of visceral fat there is definite reduction of adiponectin level like in insulin resistance during pregnancy. Decreased levels of adiponectin are associated with subclinical inflammation with subsequent development of gestational diabetes mellitus.(65) The concentration of adipocyte leptin increases as the pregnancy progresses because of secretion of leptin by placenta and returns to normal level after delivery. There is increased levels of leptin in mother which enhances the mobilization of fat to fetus.(66)

The adipokine RBP-4, which is synthesized in hepatocytes and adipose tissues, carrier for retinol and plays an important role in maintaining the metabolism of glucose and insulin sensitivity. In diseased glucose tolerance states like obesity or insulin resistance concentration of RBP – 4 becomes higher. In normal ongoing pregnancy, there is increased concentration of RBP-4 between early and late pregnancy with simultaneously reduction in insulin sensitivity. [67)]

Inflammatory mediators which are produced by monocytes and macrophages in the adipose tissue are TNF –α and IL-6 which are increased during obesity. These inflammatory mediators have various effects on insulin sensitivity resulting in insulin resistance. During pregnancy , placenta secretes these cytokines and chronic inflammation in the adipose tissue is responsible for pregnancy-induced insulin resistance. TNF is produced at the maximal rate by placenta during late pregnancy and reduces rapidly after delivery. Reversely TNF-α is responsible for inhibition of secretion of insulin and glucose uptake which is being regulated by insulin in gestational diabetes mellitus.[(68)]There is one in vitro experiment which investigated that placenta of females who are having gestational diabetes mellitus secretes more TNF-α when glucose stimulus is given as compared to placenta who is having normal glucose tolerance. An inflammatory agent named C- reactive protein (CRP), which is concerned with abnormal states of metabolism like insulin resistance. During first trimester of pregnancy, concentration of CRP becomes elevated and is responsible for development of gestational diabetes mellitus. [(69)]

CONCLUSION

It is the first study conducted which explores the relationship between snoring, BMI (Body Mass Index) and gestational diabetes mellitus in pregnancy. The results of our study concluded that there is strong and positive linear correlation between Snoring , BMI (Body Mass Index) and gestational diabetes mellitus in pregnancy.

LIMITATIONS OF THE STUDY

1. Sample size was small.
2. Study was restricted to smaller geographical area.
3. Subjects were not classified trimester wise.
4. Follow up was not taken.

FUTURE SCOPE OF THE STUDY

1. Study can be done with large sample size.
2. Larger geographical area can be taken for future study.
3. All trimesters of females can be taken to participate in the study.
4. Follow-up should be taken.

REFERENCES

1. Santiago JR, Nolledo MS, Kinzler W, Santiago TV. Sleep and sleep disorders in pregnancy. *Ann Intern Med*. 2001;14(5):396–408. doi: 10.7326/0003-4819-134-5-200103060-00012.
2. Hutchison BL, Stone PR, McCowan LM, Stewart AW, Thompson JM, Mitchell EA. A postal survey of maternal sleep in late pregnancy. *BMC Pregnancy Childbirth*. 2012;14:144. doi: 10.1186/1471-2393-12-144.
3. Pien GW, Fife D, Pack AI, Nkwuo JE, Schwab RJ. Changes in symptoms of sleep-disordered breathing during pregnancy. *Sleep*. 2005;14(10):1299–1305.
4. Ursavas A, Karadag M, Nalci N, Ercan I, Gozu RO. Self-reported snoring, maternal obesity and neck circumference as risk factors for pregnancy-induced hypertension and preeclampsia. *Respiration*.2008;14(1):33–39. doi: 10.1159/000107735.
5. Izci B, Martin SE, Dundas KC, Liston WA, Calder AA, Douglas NJ. Sleep complaints: snoring and daytime sleepiness in pregnant and pre-eclamptic women. *Sleep Med*. 2005;14(2):163–169.
6. O'Brien LM, Bullough AS, Owusu JT, Tremblay KA, Brincat CA, Chames MC, Kalbfleisch JD, Chervin RD. Pregnancy-onset habitual snoring, gestational hypertension, and preeclampsia: prospective cohort study. *Am J Obstet Gynecol*. 2012;14(6):487. e481-489.
7. Franklin KA, Holmgren PA, Jonsson F, Poromaa N, Stenlund H, Svanborg E. Snoring, pregnancy-induced hypertension, and growth retardation of the fetus. *Chest*. 2000;14(1):137–141. doi: 10.1378/chest.117.1.137..
8. Connolly G, Razak ARA, Hayanga A, Russell A, McKenna P,McNicholas WT. Inspiratory flow limitation during sleep in preeclampsia:comparison with normal pregnant and nonpregnant women. *Eur Respir J* 2001;18:1-5.
9. Edwards N, Blyton DM, Kirjavainen T, Kesby GJ Sullivan CE. Nasal continuous positive airway pressure reduces sleep-induced blood pressure increments in preeclampsia. *Am J Respir Crit Care Med* 2000;162:252-7.

10. Izci B, Vennelle M, Liston WA, Dundas KC, Calder AA, Douglas NJ. Sleep-disordered breathing and upper airway size in pregnancy and post-partum. *Eur Respir J*. 2006;14(2):321–327doi:10.1183/09031936.06.00148204.
11. Lefcourt LA Rodis JF. Obstructive sleep apnea in pregnancy.*Obstet Gynecol Surv* 1996;51:503-6.
12. Schoenfeld A, Ovadia Y, Neri A Freedman S. Obstructive sleep apnea(OSA)-implications in maternal-fetal medicine. *A hypothesis.Med Hypotheses* 1989;30:51-4.
13. Bourjeily G, Raker CA, Chalhoub M,Miller MA. Pregnancy and fetal outcomes of symptoms of sleep-disordered breathing. *Eur Respir J* 2010;36:849–855
14. Facco FL, Grobman WA, Kramer J, HoKH, Zee PC. Self-reported short sleep duration and frequent snoring in pregnancy:impact on glucose metabolism.*Am J Obstet Gynecol* 2010;203:142, e1–e5
15. Qiu C, Enquobahrie D, Frederick IO,Abetew D, Williams MA. Glucose intolerance and gestational diabetes risk in relation to sleep duration and snoring during pregnancy: a pilot study. *BMC Womens Health* 2010;10:17
16. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540–545
17. Izci-Balserak B, Pien GW. Sleep-disordered breathing and pregnancy: potential mechanisms and evidence for maternal and fetal morbidity. *Curr Opin Pulm Med*. 2010;14(6):574–582. doi: 10.1097/MCP.0b013e32833f0d55.
18. August EM, Salihu HM, Biroscak BJ, Rahman S, Bruder K, Whiteman VE. Systematic review on sleep disorders and obstetric outcomes: scope of current knowledge. *Am J Perinatol.* 2013;14(4):323–334.
19. Pamidi S, Pinto LM, Marc I, Benedetti A, Schwartzman K, Kimoff RJ. Maternal sleep-disordered breathing and adverse pregnancy outcomes - a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2013. p. 435-438.
20. Fung AM, Wilson DL, Lappas M, Howard M, Barnes M, O'Donoghue F, Tong S, Esdale H, Fleming G, Walker SP. Effects of maternal obstructive sleep apnoea on fetal growth: a prospective cohort study.*PloS one*. 2013;14(7):e68057. doi: 10.1371/journal.pone.0068057.

21. Cedergren MI. Maternal morbid obesity and the risk of adverse pregnancy outcome. *Obstet Gynecol*.2004;14(2):219–224. doi: 10.1097/01.AOG.0000107291.46159.00.
22. Olafsdottir AS, Skuladottir GV, Thorsdottir I, Hauksson A, Steingrimsdottir L. Maternal diet in early and late pregnancy in relation to weight gain. *Int J Obes* 2006;30:492–9.
23. Ovesen P, Rasmussen S, Kesmodel U. Effect of Prepregnancy Maternal Overweight and Obesity on Pregnancy Outcome. *Obstetrics & Gynecology*. 2011; 118(2 Part 1):305–12.10.1097/AOG.0b013e3182245d49.
24. 35.Vricella LK, Louis JM, Mercer BM, Bolden N. Impact of morbid obesity on epidural anesthesia complications in labor. *American Journal of Obstetrics and Gynecology*. 2011; 205(4):370.e1–e6.
25. Goffman D, Madden RC, Harrison EA, Merkatz IR, Chazotte C. Predictors of maternal mortality and near-miss maternal morbidity. *J Perinatol*. 2007; 27(10):597–601.
26. Catalano PM, Ehrenberg HM. Review article: The short- and long-term implications of maternal obesity on the mother and her offspring. BJOG: *An International Journal of Obstetrics & Gynaecology*. 2006; 113(10):1126–33.
27. Ovesen P, Rasmussen S, Kesmodel U. Effect of Prepregnancy Maternal Overweight and Obesity on Pregnancy Outcome. *Obstetrics & Gynecology*. 2011; 118(2 Part 1):305–12.10.1097/AOG. 0b013e3182245d49
28. Louis JM, Auckley D, Sokol RJ, Merce BM. Maternal and neonatal morbidities associated with obstructive sleep apnea complicating pregnancy. *Am J Obstet Gynecol* 2010;202:261, e1–e5
29. Loube DI, Poceta JS, Morales MC, Peacock MD Mitler MM. Selfreported snoring in pregnancy. Association with fetal outcome. *Chest* 1996;109:885-9.
30. Henry, O. A. and Beischer, N. A. (1991) Long-term implications of gestational diabetes for the mother.*Baillieres Clin. Obstet. Gynaecol*. 5, 461–483
31. Schernthaner, G. H. and Schernthaner, G. (2005) Insulin resistance and inflammation in the early phase of type 2 diabetes: potential for therapeutic intervention. *Scand. J. Clin. Lab. Invest. Suppl*. 240, 30–40.
32. Steppan, C. M., Bailey, S. T., Bhat, S. et al. (2001) The hormone resistin links obesity to diabetes.*Nature (London)* 409, 307–312

33. Axelsson, J., Heimburger, O., Lindholm, B. and Stenvinkel, P. (2005) Adipose tissue and its relation to inflammation: the role of adipokines. *J. Renal Nutr*. 15, 131–136
34. Hotamisligil, G. S. (1999) The role of TNFα and TNF receptors in obesity and insulin resistance. *J. Intern. Med*. 245, 621–625
35. Fukuhara, A., Matsuda, M., Nishizawa, M. et al. (2005) Visfatin: a protein secreted by visceral fat that mimics the effects of insulin. *Science* 307, 426–430
36. Sethi, J. K. and Vidal-Puig, A. (2005) Visfatin: the missing link between intra-abdominal obesity and diabetes?*Trends Mol. Med*. 11, 344–347
37. Berndt, J., Kloting, N., Kralisch, S. et al. (2005) Plasma visfatin concentrations and fat depot-specific mRNA expression in humans. *Diabetes* 54, 2911–2916
38. Chen, M. P., Chung, F. M., Chang, D. M. et al. (2006) Elevated plasma level of visfatin/pre-B cell Colonyenhancing factor in patients with type 2 diabetes mellitus.*J. Clin. Endocrinol. Metab*. 91, 295–299
39. Wellhoener, P., Fruehwald-Schultes, B., Kern, W. et al (2000) Glucose metabolism rather than insulin is a main determinant of leptin secretion in humans. *J. Clin. Endocrinol. Metab*. 85, 1267–1271
40. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354:1435–1439.
41. Peppard PE, Young T, Palta M, Skatrud J. Prospective study of the association between sleep-disordered breathing and hypertension. *N Engl J Med.* 2000;14(19):1378–1384. doi: 10.1056/NEJM200005113421901.
42. Svensson M, Lindberg E, Naessen T, Janson C. Risk factors associated with snoring in women with special emphasis on body mass index: a population-based study. *Chest.* 2006;14(4):933–941. doi: 10.1378/chest.129.4.933.
43. Izci B, Riha RL, Martin SE, et al. The upper airway in pregnancy and pre-eclampsia. *Am J Respir Crit Care Med* 2004;167:137-40.
44. Perez-Chada D, Videla AJ, O'Flaherty ME, Majul C, Catalini AM, Caballer CA, Franklin KA. Snoring, witnessed sleep apnoeas and pregnancy-induced hypertension. *Acta Obstet Gynecol Scand*.2007;14(7):788–792. doi: 10.1080/00016340701281919.
45. Knutson KL, Van Cauter E. Associations between sleep loss and increased risk of obesity and diabetes. *Ann N Y Acad Sci* 2008;1129:287–304

46. Tasali E, Leproult R, Ehrmann DA, Van Cauter E. Slow-wave sleep and the risk of type 2 diabetes in humans. *Proc Natl Acad Sci USA* 2008;105:1044–1049.
47. Foster GD, Sanders MH, Millman R, et al.;Sleep AHEAD Research Group. Obstructive sleep apnea among obese patients with type 2 diabetes. *Diabetes Care* 2009; 32:1017–1019.
48. Louis JM, Auckley D, Sokol RJ, Mercer BM. Maternal and neonatal morbidities associated with obstructive sleep apnea complicating pregnancy. *Am J Obstet Gynecol* 2010;202:261, e1–e5
49. Hutchison BL, Stone PR, McCowan LM, Stewart AW, Thompson JM, Mitchell EA. A postal survey of maternal sleep in late pregnancy. *BMC Pregnancy Childbirth*. 2012;14:144. doi: 10.1186/1471-2393-12-144.
50. O'Brien LM, Bullough AS, Owusu JT, Tremblay KA, Brincat CA, Chames MC, Kalbfleisch JD, Chervin RD. Pregnancy-onset habitual snoring, gestational hypertension, and preeclampsia: prospective cohort study. *Am J Obstet Gynecol*. 2012;14(6):487. e481-489.
51. Fung AM, Wilson DL, Lappas M, Howard M, Barnes M, O'Donoghue F, Tong S, Esdale H, Fleming G, Walker SP. Effects of maternal obstructive sleep apnoea on fetal growth: a prospective cohort study.*PloS one.* 2013;14(7):e68057. doi: 10.1371/journal.pone.0068057.
52. Holdcroft A, Bevan DR, O'Sullivan EF, et al. Airway closure during pregnancy. *Anesthesia* 1977; 32:517–523
53. Colditz GA, Willett WC, Rotnitzky A, Manson JE 1995 Weight gain as a risk factor for clinical diabetes mellitus in women. *Ann Intern Med* 122:481–486
54. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC 1994 Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in women. *Diabetes Care* 17:961–969
55. Kopelman PG. Sleep apnoea and hypoventilation in obesity. *Int J Obes* 1992; 16:S37–S42
56. Ang CK, Tan TH, Walters WAA, et al. Postural influence on maternal capillary oxygen and carbon dioxide tension. *BMJ 1969;* 4:201–203

57. Bradley TD, Brown IG, Grossman RF, Zamel N, Martinez D, Phillipson EA, Hoffstein V. Pharyngeal size in snorers, nonsnorers , and patients with obstructive sleep apnea. *N Engl J Med* 1986; 315:1327–1331.
58. Hotamisligil GS, Shargill NS, Spiegelman BM. Adipose expression of tumor necrosis factor-alpha: direct role in obesity-linked insulin resistance. *Science* 1993; 259: 87.
59. Fried SK, Bunkin DA, Greenberg AS. Omental and subcutaneous adipose tissues of obese subjects release interleukin-6: depot difference and regulation by glucocorticoid. *J Clin Endocrinol Metab* 1998; 83: 847.
60. Kelley DE, Mokan M, Simoneau JA, Mandarino LJ. Interaction between glucose and free fatty acid metabolism in human skeletal muscle. *J Clin Invest* 1993;92:91–98.
61. Ryan AS, Nicklas BJ. Reductions in plasma cytokine levels with weight loss improve insulin sensitivity in overweight and obese postmenopausal women. *Diabetes Care* 2004; 27: 1699.
62. Kraegen EW, Cooney GJ, Ye JM, Thompson AL, Furler SM. The role of lipids in the pathogenesis of muscle insulin resistance and β cell failure in type II diabetes and obesity. *Exp Clin Endocrinol Diabetes* 2001;109 (Suppl 2):S189–S201.
63. Yang D, Liu Z, Yang H, Luo Q. Effects of continuous positive airway pressure on glycemic control and insulin resistance in patients with obstructive sleep apnea: a meta-analysis. *Sleep Breath.* 2013;17:33–8.
64. Dawson A, Abel SL, Loving RT, et al. CPAP therapy of obstructive sleep apnea in type 2 diabetics improves glycemic control during sleep. *J Clin Sleep Med*. 2008;4:538–42.
65. Stephens JM, Lee J, Pilch PF. Tumor necrosis factor-α-induced insulin resistance in 3T3-L1 adipocytes is accompanied by a loss of insulin receptor substrate-1 and GLUT4 expression without a loss of insulin receptor-mediated signal transduction. *J Biol Chem* 1997;272:971–976.
66. The relation of markers of inflammation to the development of glucose disorders in the elderly: the cardiovascular health study. *Diabetes*. 2001;50(10):2384–2389.
67. Greenberg AS, McDaniel ML. Identifying the links between obesity, insulin resistance and *β*-cell function: potential role of adipocyte-derived cytokines in the pathogenesis of type 2 diabetes. *European Journal of Clinical Investigation*. 2002;32, supplement 3:24–34.

68. Yudkin JS, Stehouwer CDA, Emeis JJ, Coppack SW. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? *Arteriosclerosis, Thrombosis, and Vascular Biology*. 1999;19(4):972–978.
69. Di Benedetto A, Russo GT, Corrado F, et al. Inflammatory markers in women with a recent history of gestational diabetes mellitus. *Journal of Endocrinological Investigation*. 2005;28(1):34–38.

VI. APPENDICES

6.1 APPENDIX

INFORMED CONSENT

TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES.

I have been informed by Ms.Preeti Madaan ,student in lovely Professional University,Phagwara that study entitled " To explore the correlation between snoring, BMI and Gestational diabetes mellitus in pregnant females." is being conducted. The purpose of the study is to determine the correlation between snoring, BMI and Gestational diabetes mellitus in pregnant females. As a part of the study subject will have to fill the questionnaire.

I understand that, there is no risk involved in this study. All the information regarding me will be kept confidentially; only Ms.Preeti Madaan and her guide Dr.Manpreet Kaur (MSPT) will have access to the names of the subjects participating in this study and will not be shared by any other person. I understand that my assent is voluntary and I have any right to withdraw or discontinue the participation at any stage of the study without assigning any reason to it.

I ,PREETI MADAAN, have explained to Mrs._____. The purpose of the research ,the procedure ,required in the language she could understand to the best of my ability.

I _____ voluntary agree to participate in her study. My entire questions have been answered satisfactorily. I reserve my right to withdraw at any instant and I have the contact address of Ms.Preeti Madaan, if required any further information.

Signature of Participant Signature of Researcher.

6.2 APPENDIX

ASSESSMENT FORM

TO EXPLORE THE CORRELATION BETWEEN SNORING, BMI AND GESTATIONAL DIABETES MELLITUS IN PREGNANT FEMALES.

Name : **Age/Gender:** **Date:**

Occupation: **Height:** **Contact No:**

Weight : **Gestational Age:** **Marital Status:**

Address:

CHIEF COMPLAINT:

HISTORY OF PRESENT ILLNESS:

1. Do you snore?	
Does your snoring disturb your room partner?	
Is snoring becoming progressively worse?	
Do you snore when you are on your back?	
Has your snoring caused you to wake up suddenly?	
2.Are you excessively tired during the day no matter how much you got the night before?	
Do you fall asleep while driving or at stop lights?	
Do you fall asleep watching television?	
3.Do you awaken with headaches?	
4.Do you nap : If so ,are the naps refreshing or do you awaken from naps still tired?	
5. Have you noticed your heart pounding or beating irregularly during night?	
6.Have you noticed that parts of your body jerk during sleep?	
7.Have you been told that you kick at night?	
8.Do you have difficulty initiating sleep?	
9.Do you have difficulty maintaining sleep?	
10.Do you awaken earlier in the morning than you would like to?	

PAST MEDICAL HISTORY:

H/O Diabetes Mellitus Yes/ No

H/O Systemic Hypertension Yes/No

H/O Sleep Apnea Yes/No

H/O Thyroid Disease Yes/No

Any Other……

PAST SURGICAL HISTORY (Include Dates of Surgery):

Tonsillectomy ……

Tracheotomy ……

Adenoidectomy ……

Other Surgeries ……

OBSTETRIC HISTORY:

G_P_L_A_

G- Gravid

P-Parity

L-Living Child

A-Abortion

MENSTRUAL HISTORY:

Menarche:

Cycle: Regular/Irregular

Duration of cycle:

Flow: Heavy/Normal

SLEEP HISTORY:

Sleep Hours –

Frequency of Naps:

Snoring:

Sleep Disturbance:

PSCHYCOLOGICAL HISTORY:

H/O Depression:

Mood Fluctuations:

Behavioral Changes:

ON OBSERVATION:

BUILT: Ectomorphic / Mesomorphic /Endomorphic

ON EXAMINATION:

BMI (Body Mass Index)

BMI	**Weight Status:**
Below 18.5	**Underweight**
18.5-24.9	**Normal**
25.0-29.9	**Overweight**
30.0 And above	**Obese**

DIAGNOSIS:

6.3 APPENDIX MASTERCHART

S.No	Age	Height	Weight Pre	Weight Post	BMI Pre	BMI Post	ESSPre	ESSPost	GTT1ST	GTT2nd	GTT3rd	GTT4th
CS1	22	158	65	79	26.04	31.65	10	13	82	149	136	112
CS2	25	161.5	65	76	24.92	29.14	8	11	102	210	175	124
CS3	21	160.5	68	81	26.40	31.44	3	8	84	159	143	139
CS4	27	145.5	65	79	30.70	37.32	14	19	115	217	187	172
CS5	24	153	55	75	23.50	32.04	9	12	102	315	188	170
CS6	22	156	68	84	27.94	34.52	21	15	87	126	132	117
CS7	26	149	58	72	26.12	32.43	10	17	84	162	147	121
CS8	29	151	75	88	32.89	38.59	13	20	126	235	161	143
CS9	28	157	70	84	28.40	34.08	12	18	87	126	132	117
CS10	22	162	60	72	22.86	27.43	4	10	78	157	142	115
CS11	21	155	60	75	24.97	31.22	7	16	104	280	176	157
CS12	22	162	68	80	25.91	30.48	9	14	79	170	132	111
CS13	24	157	75	90	30.43	36.51	13	22	112	218	149	163
CS14	25	163	60	78	22.58	29.36	9	15	76	136	112	108
CS15	29	160	62	75	24.22	29.30	5	8	102	168	147	150
CS16	30	155.5	68	83	28.12	34.33	4	7	87	191	142	119
CS17	32	151	52	66	22.81	28.95	2	11	78	172	128	106
CS18	31	157	67	80	27.18	32.46	7	10	129	198	162	158
CS19	29	162	76	92	28.96	35.06	12	14	92	201	164	135
CS20	27	157	55	69	22.31	27.99	2	11	72	174	146	129
CS21	25	152	60	69	25.97	29.86	6	13	82	164	146	137
CS22	23	155	71	89	29.55	37.04	17	22	97	249	146	128
CS23	22	149	72	84	32.43	37.84	9	12	126	245	161	132
CS24	20	151	75	88	32.89	38.59	8	13	137	188	164	132
CS25	27	157	66	84	26.78	34.08	11	16	127	231	188	157
CS26	21	155	60	77	24.97	32.05	7	15	105	194	155	161
CS27	23	162	72	85	27.43	32.39	6	13	135	248	182	164
CS28	26	152	67	80	29.00	34.63	8	14	94	188	173	168
CS29	30	163	78	90	29.36	33.87	10	17	94	176	152	146
CS30	22	147	60	75	27.77	34.71	8	15	85	132	151	127
CS31	24	159	64	73	25.32	28.88	4	9	112	159	167	152
CS32	28	152	73	85	31.60	36.79	11	19	118	298	176	140
CS33	25	153	53	64	22.64	27.34	5	8	78	172	128	106
CS34	20	148	78	95	35.61	43.37	9	11	152	195	178	132
CS35	22	162	75	84	28.58	32.01	13	18	89	167	148	137
CS36	24	158	74	92	29.64	36.85	14	22	99	192	160	148
CS37	27	151	72	86	31.58	37.72	8	17	117	356	164	159
CS38	21	148	55	65	25.11	29.67	10	15	107	196	147	152
CS39	29	157	52	68	21.10	27.59	7	16	60	159	126	112
CS40	26	159	80	98	31.64	38.76	16	23	118	252	182	148

6.4 APPENDIX ASSESSMENT TOOLS

EPWORTH SLEEPLINESS SCALE

Patient Questionnaire for Snoring :

Name _____ Occupation

Age Height ft inches Weight pounds

Marital Status: □□Single □□Engaged □□Married □□Separated □□Divorced □□Widowed

The following questionnaire will help you measure your general level of daytime sleepiness. Answers are rated on a reliable scale called the Epworth Sleepiness Scale (ESS) - the same assessment tool used by sleep experts worldwide. This test was developed by Dr. Murray Johns at Epworth Hospital in Melbourne, Australia in 1991. Each item describes a routine daytime situation. Use the scale below to rate the likelihood that you would doze off or fall asleep (in contrast to just feeling tired) during that activity. If you haven't done some of these things recently, consider how you think they would affect you. Please note that this scale should not be used to make your own diagnosis. It is intended as a tool to help you identify your own level of daytime sleepiness, which can be a symptom of a sleep disorder.

Use the following scale to answer the questions:
0 = would never doze 1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

Task :	0	1	2	3
Sitting and Reading				
Watching Television				
Sitting inactive in public place e.g Theatre				
As a passenger in car for a hr. without break.				
Lying down to rest in afternoon.				

Sitting and talking to someone.				
Sitting quite after lunch when took no alcohol.				
In a car while stopped in a traffic.				

_____TOTAL SCORE

Sleep is a health issue; without proper sleep, the human body cannot function well. Good sleep helps ward off illnesses, helps maintain healthy skin, bones, and muscles and helps to balance hormones and other important brain and body chemicals. **If your total score is 10 or higher, review it with your doctor or other health care provider.**