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Statistical and Real life Significance of Obesity as an Etiopathogenesis of Type 2 Diabetes Mellitus

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ABSTRACT: The exponential growth of Type 2, non-insulin-dependent diabetes over the past decade demands a comprehensive study. In the Indian context, Diabetes is fast gaining the status of a potential epidemic with an alarming number of more than 62 million diabetic individuals. Etiopathogenesis of type 2 diabetes is intricate in its patterns and the studies on the same still remain incomprehensive. Its etiology is determined by the interaction of genetic and environmental factors. Though the contribution of genetic components is imperative it has a polygenic origin. The two dominating factors in this regard are - insulin resistance (which is mainly due to obesity and physical inactivity) and deficient insulin production. Although approximately 80% of type 2 diabetics are obese, 2/3 of overweight or obese persons show normal glucose metabolism. The diabetogenic effect of obesity is due to the capacity of excessive fat mass to induce or aggravate insulin resistance. The recent increase in the number of people who abstain from physical activity is also a contributing factor as lack of exertion increases insulin resistance. The study aims to statistically analyze how obesity can be perceived as the main risk factor that is associated with type 2 diabetes mellitus (T2DM). It also examines various other significant factors and obtains a general statistical model through a real dataset. The generalized results and concepts that have been incorporated are seen to have ample statistical validity and real-life significance.

Keywords: Coefficient of determinant, Diabetes Mellitus, Etiopathogenesis, polygenic, Regression model, Risk factor.

Abbreviations: T2DM (type 2 diabetes mellitus), BMI (Body mass index), WC (waist circumference), ANOVA (Analysis of variance).

I. INTRODUCTION

Diabetes mellitus is seen to be one of the greatest challenges of the contemporary age that has affected a considerable percentage of population across the globe. There are multiple and wide-ranging factors- in isolation or in combination- like genetic factors, obesity, rising living standards and changes in lifestyle. Type 2 diabetes is a chronic condition that affects the way your body metabolizes sugar (glucose) which is an important source of fuel for your body. With type 2 diabetes, your body either resists the effects of insulin or does not produce enough insulin to maintain normal glucose levels. Type2 diabetes (formerly known as adult-onset diabetes) was deemed to be exclusive to adults alone but recent studies show that more children are being diagnosed with this disorder, in all probability due to the rise in childhood obesity. There is no absolute cure for type2 diabetes except weight loss, proper diet and routine exercise. If diet and exercise are not adequate to manage your blood sugar, you may also require further medications or insulin therapy.

Factors that may increase risk of type 2 diabetes include: weight, Fat distribution, Inactivity, Family history, Race, Age, Pre diabetes, Gestational diabetes, polycystic ovarian syndrome and mental stress. In this study we are dealing with only the effect of obesity and excessive eating.

Obesity is the "accumulation of adipose tissue to excess and to extent that impairs physical and psychosocial health and well-being". Obesity is a ubiquitous disease recently recognized as a major health problem in all age groups and is quite a heterogeneous condition not only for the different related medical issues but also for the associated psychological and psychiatric conditions. Epidemiological studies have shown that obesity is an important risk factor for the development of T2DM. It was reported that, about 80% of all Type 2 diabetics are overweight. It is often stated that obesity is the most important risk factor for T2DM.

Baseline information for this study was obtained *via* a questionnaire on general information, physical examination (height, weight, and blood pressure), laboratory tests (triglycerides, total cholesterol, blood glucose, aspartate aminotransferase (AST) or serum glutamic-oxaloacetic transaminase (*SGOT)* and alanine aminotransferase (ALT) or Serum glutamic pyruvic transaminase (SGPT)), Body mass index (BMI) and waist circumference **(**WC). Prevalence of T2DM patients is high and increases with multiple components of metabolic syndrome (Metabolic syndrome is a cluster of conditions that occurs together, increasing risk of heart disease, stroke and type2 diabetes. These conditions include increased blood pressure, high blood sugar, excess body fat around the waist, and abnormal cholesterol or triglyceride levels).

BMI is obtained by dividing body weight in kilograms by height in meters squared. In developed countries, subjects with a BMI ≥ 25 kg/m^2 are defined as overweight and those with a BMI ≥ 30 kg/m^2 are defined as obese, and there are good associations and positive predictive effects between BMI and obesity-related chronic diseases. Obesity and T2DM are closely related. This study aims to summarize the evidence of the association between them.

II. REVIEW OF LITERATURE

Abdullah S Al-Goblan *et al*., (2014) [1] conducted a study on Mechanism linking diabetes mellitus and obesity and concluded that Diabetes and obesity are chronic disorders that are on the rise worldwide. BMI has a strong relationship to diabetes and insulin resistance. Aydan Ercan *et al*., (2013) [2] conducted a study on Obesity-related abnormal eating behaviours in Type 2 diabetic patients to determine the obesity-related abnormal eating behaviours in Type 2 diabetic patients and showed that Type 2 diabetes and obesity were like conjuncted-twins and causes poor glycemic control and complications. The frequency of obesity-related abnormal eating behaviours, such as binge eating and night eating, were very common in Type 2 diabetic patients.

Daousi *et al*., (2017) [3] conducted a studyPrevalence of obesity in type 2 diabetes in secondary care: association with cardiovascular risk factors. The findings indicated that Obesity is the rule among patients attending this hospital diabetes clinic, with 86% of those with type 2 diabetes overweight or obese. Obesity is associated with significantly worse cardiovascular risk factors in this patient group, suggesting that more active interventions to control weight gain would be appropriate.Eugenio Cersosimo *et al*.,(2018) [4] conducted a study on Pathogenesis of T2DM and showed that At least eight distinct pathophysiologic abnormalities have been associated with T2DM. It is well established that decreased peripheral glucose uptake (mainly muscle) combined with augmented endogenous glucose production are characteristic features of insulin resistance.

Fery *et al*., (2005) [5] studied Etiopathogenesis and pathophysiology of type 2 diabetes . The findings indicated that Type 2 diabetes is unlikely to induce relevant eating disturbances in obese patients, apart from an increase in restraint. Abnormalities of eating attitudes and behaviour are associated with an impairment of metabolic control. Guillausseau *et al*., (2003) [6] conducted a study on Pathogenesis of T2DM. The study concluded that Simple changes in lifestyle, such regular moderate physical activity, and control of body weight, should permit to avoid the explosion in prevalence of type 2 diabetes.

Herpertz *et al*., (2006) [7] studied Eating disorders and diabetes mellitus suggested that Even if a binge eating disorder in persons with a type 2 diabetes does not occur more frequently than in those metabolically healthy persons, it does depict a risk factor for an accelerated weight gain which as rule involves an increase of insulin resistance. Mannucci *et al*., (2002) [8] studied Eating behaviour in obese patients with and without T2DM. The findings indicated that Type 2 diabetes is unlikely to induce relevant eating disturbances in obese patients, apart from an increase in restraint. Abnormalities of eating attitudes and behaviour are associated with an impairment of metabolic control.

Marcelo Papelbaum *et al., (*2019) [9] conducted study on Does binge-eating matter for glycemic control in type 2 diabetes patients? Findings demonstrate that the pool of evidence regarding the association between ED and T2DM seems to justify screening diabetic patients for abnormal eating behaviours. In addition, when obesity is present, eating psychopathology investigation is even more recommended, since it may disrupt obesity treatment and indirectly affects diabetes control. Nieto-Martínez *et al*., (2017) [10] conducted a study onAre Eating Disorders Risk Factors for Type 2 Diabetes? A Systematic Review and Meta-analysisconcluded that Eating disorders (ED) affect energy intake modifying body fat depots. Prior evidence suggests that binge eating disorder (BED) and bulimia nervosa (BN) could increase the risk for type 2 diabetes (T2D), while anorexia nervosa (AN) could reduce it.

Prasanth *et al.,* (2020) [11] studied about the Significance of Diabetes Mellitus in the Risk Factor Analysis of Non Alcoholic Steatohepatitis and Cardiovascular Disease. Here the baseline characteristics were age, obesity, diabetic level, hypertension, cholesterol level etc. and derived a statistical model for estimating the association of many such components with blood sugar. So here also the significance of obesity with type 2 diabetes is noticed.

Sally Abbott *et al.,* (2018) [12] studied Binge eating disorder and night eating syndrome in adults with type 2 diabetes: a systematic review. Findings demonstrate that a considerable proportion of adults with pre-existing T2DM have clinical BED and NES.The effect of newer anti-diabetes therapies that reduce appetite, and the increasing use of very low calorie diets and bariatric surgery in the management of T2DM, make screening and early diagnosis of these eating disorders even more important.

Bassuk *et al*., (2005) [13] studied Epidemiological evidence for the role of physical activity in reducing risk of type 2 diabetes and cardiovascular disease and suggest that physically active individuals have a 30-50% lower risk of developing type 2 diabetes than do sedentary persons and that physical activity confers a similar risk reduction for coronary heart disease. Risk reductions are observed with as little as 30 min of moderate-intensity activity per day. Sindelka *et al.,* (1996) [14] conducted a study on the effect of body weight on insulin activity. The findings indicated that the BMI has a decisive role in the action insulin.

Waqas Sam *et al*., (2017) [15] conducted a study on Effect of diet on T2DM: A review. The study suggests that T2DM patients require reinforcement of DM education including dietary management through stakeholders (health-care providers, health facilities, etc.) to encourage them to understand the disease management better, for more appropriate self-care and better quality of life.

III. METHODOLOGY AND DATA ANALYSIS

The proposed study aims to summarize the evidence of the association between obesity and T2DM. We studied the interaction between BMI and waist circumference with respect to the risk of developing T2DM in childbearing diabetic women. 410 subjects were randomly selected from various hospitals in the 2 districts, Palakkad and Malappuram - at Kerala to make a real data analysis. Obesity is the leading factor in the pathogenesis of health disorders such as hypertension and type-2 diabetes mellitus. Since BMI is the associated with obesity, the present study has been carried out to evaluate the association between Blood sugar level and BMI.

BMI is a measurement of a person's leanness or corpulence based on their height and weight, and is intended to quantify tissue mass. It is widely used as a general indicator of whether a person has a healthy body weight for their height. Specifically, the value obtained from the calculation of BMI is used to categorize whether a person is underweight, normal weight, overweight, or obese depending on what range the value falls between. Being overweight or obese can cause many serious health problems. Two methods that are commonly used to estimate whether a person is with a healthy weight or not are by checking BMI and WC. Waist circumference is considered as a good estimate of body fat, especially your internal fat deposits, and your likelihood of developing weight-related disease. Health professionals often use BMI and waist circumference together to assess whether someone is overweight or obese and to assess their risk of diabetes. Although waist circumference and BMI are interrelated, waist circumference provides an independent prediction of risk over and above that of BMI. Waist circumference measurement is particularly useful in patients who are categorized as normal or overweight on the BMI scale. At BMIs ≥ 35, waist circumference has little added predictive power of disease risk beyond that of BMI. It is therefore not necessary to measure waist circumference in individuals with BMIs ≥ 35. BMI table for adults- This (Table 1) is the World Health Organization's (WHO) recommended categorized body weight based on BMI values for adults. It is used for both men and women, age 18 or older. The frequency- from a sample of 410 T2DM childbearing diabetic women - data collected from Palakkad and Malappuram district - of each category - also noted in the said format.

Table 1: Frequency table - BMI of diabetic persons.

Category	BMI range - kg/m^2	Frequency
Under weight	<18.5	0
Normal	18.5 - 25	44
Overweight	25 - 30	294
Obese Class I	30 - 35	62
Obese Class II	35 - 40	8
Obese Class III	> 40	2
Total		410

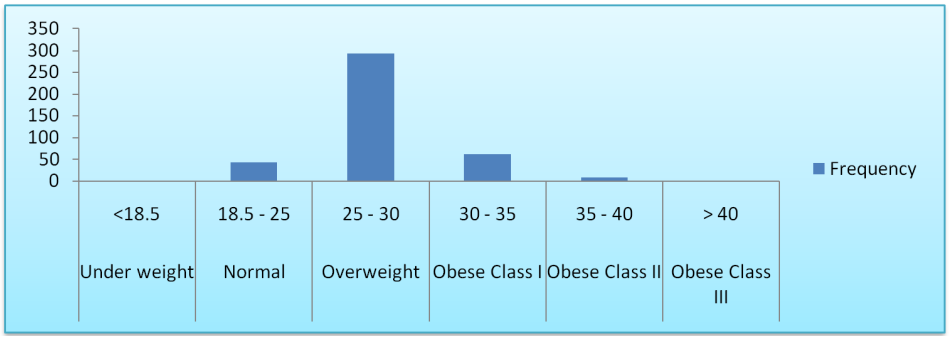

Fig. 1. BMI - Frequency – Bar diagram.

From Table 1 and Fig. 1, it is clear that 366 persons are with abnormal weight limits.294 persons are of them are overweight and 74 are at obese class. Two assumptions are testing herewith, initially the ***null* hypothesis H_0: *percentage of diabetic patients with weight abnormality is 85, against the alternative H_1: percentage of diabetic patients is with weight abnormality is greater than85.***

The test statistic Z = ($\bar{p}$– P)/ square root of (PQ/n). Here the sample proportion $\bar{p}$= 366/410 = 0.89 and population proportion under H_0 is P = 0.85& sample size n = 410. Then the calculated Z =2.42 and from the standard normal table Z_α = 1.64at α = 0.05, the p-value is p = 0.0078 and less than α = 0.05, it is concluded that the null hypothesis is rejected and H_1 is accepted, ie., the Percentage of abnormality is greater than 85 is accepted.

Then the null hypothesis

***H_0: percentage of diabetic patients is with* Obesity *are* 15*, against the alternative H_1: percentage of diabetic patients is with* Obesity *is not equal to* 15.**

The test statistic Z = ($\bar{p}$– P)/ square root of (PQ/n). Here the sample proportion $\bar{p}$= 72/410 = 0.176 and population proportion under H_0 is P = 0.15 & sample size n = 410. Then the calculated Z =1.452 and from the standard normal table Z_α = 1.96at α = 0.05, the p-value is p = 0.1464 and not less than α = 0.05, it is concluded that the null hypothesis is not rejected and $H_0$is accepted, ie, **the Percentage of *diabetic patients are with* Obesity is 15 is accepted**.

Table 2 and Fig. 2 indicates maximum frequency belongs to PP in between 180-230& 230-280 at Overweight category. That is out of 294 in Overweight category 220 of them belongs to PP in 180 to 280. Although 44 normal weight & 60 less than 180 PP cases are there existing but only 10 in both. This is a clear implication of the significance of weight in PP.

Table 3, Explains, only 8.7% (36 out of 410) of Type2 diabetic in Disease Risk range w.r.to normal WC(Women <88cm (35in)) and abnormal BMI (greater than 25) where 89.3% (366 out of 410) of them are in Disease Risk range w.r.to abnormal WC(Women > 88cm(35in)) and abnormal BMI (greater than 25).

Table 2: Type2 diabetic - Frequency – BMI & PP.

Category	BMI range - kg/m^2	Blood sugar				Total
		<180	180-230	230-280	280above	
Underweight	<18.5	0	0	0	0	0
Normal	18.5 - 25	10	34	0	0	44
Overweight	25 - 30	40	120	100	34	294
Obese Class I	30 - 35	10	10	30	12	62
Obese Class II	35 - 40	0	0	2	6	8
Obese Class III	> 40	0	0	0	2	2
Total		60	164	132	54	410

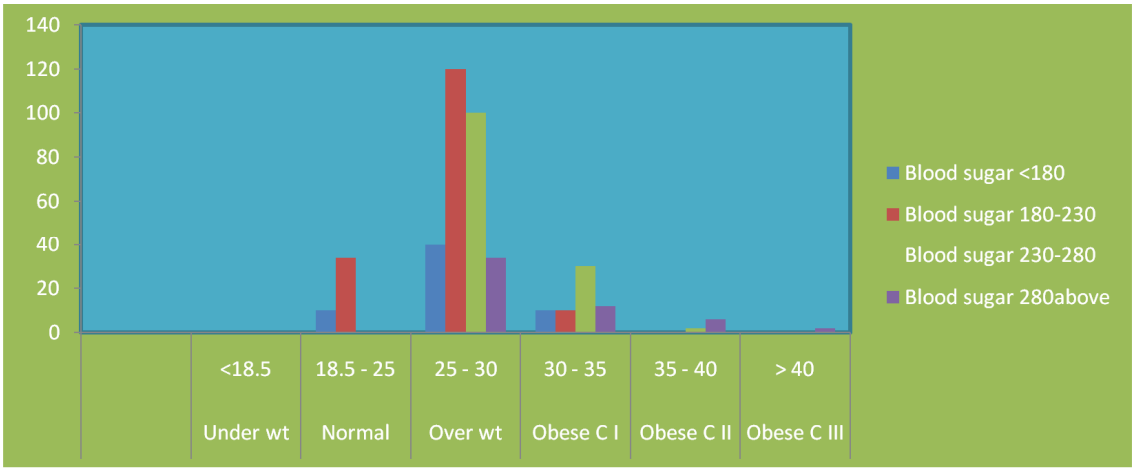

Fig. 2. Type2 diabetic - Frequency – bar chart – BMI & PP.

Table 3: Type2 diabetic - Disease Risk w.r.to BMI& WC.

Category	BMI range - kg/m^2	WC Women <88cm(35in)	Frequency	WC Women >88cm(35in)	Frequency	Total
Underweight	<18.5	—	0	—	0	0
Normal	18.5 - 25	—	16	—	28	44
Overweight	25 - 30	Increased risk	34	High	260	294
Obese Class I	30 - 35	High	2	Higher	60	62
Obese Class II	35 - 40	Higher	0	Higher	8	8
Obese Class III	> 40	Extremely high	0	Extremely high	2	2
Total			52		358	410

Table 4: Descriptive Statistics - related components of Type2 diabetic.

Components	Mean	Std. Deviation
Bloodsugar(PP)	228.48	37.995
Waist Circumference WC	37.30	3.095
Body Mass Index (BMI)	27.79	2.651
Cholestrol (Ch)	226.51	41.938
Serum Glutamic-Oxaloacetic Transaminase (SGOT)	19.11	11.881
Serum Glutamic Pyruvic Transaminase (SGPT)	35.75	8.646
Try Glycerin (TG)	225.02	65.469
Systolic Pressure (SP)	128.54	15.150
Diastolic Pressure (DP)	83.74	7.954
Age	29.17	4.613

Table 5: Pearson Correlation coefficient - related components of Type2 diabetic.

	WC	BMI	Ch	SGOT	SGPT	TG	SP	DP	Age
Blood Sugar (PP)	0.648	0.890	0.156	0.248	0.333	0.213	0.025	0.069	0.105

Table 6: Linear Regression: Model Summary.

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	0.907	0.823	0.819	16.206	0.823	205.520	9	398	.000

a. Predictors: (Constant), Age, SGOT, TG, SP, BMI, SGPT, Ch, WC, DP. **b**. Dependent Variable: Bloodsugar(PP).

Table 7: ANOVA.

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	485776.333	9	53975.148	205.52	.000
	Residual	104525.468	398	262.627		
	Total	590301.801	407			

a. Predictors: (Constant), Age, SGOT, TG, S P, BMI, SGPT, Ch, WC, DP
b. Dependent Variable: Blood sugar (PP).

Table 8: Model Coefficients.

	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
	B	Std. Error	Beta			Tolerance	VIF
(Constant)	-144.979	14.290		-10.145	**.000**		
WC	.980	.400	.079	2.448	**.015**	.425	2.356
BMI	12.233	.430	.853	28.451	**.000**	.495	2.022
Ch	-.031	.027	-.034	-1.137	**.256**	.491	2.037
SGOT	.086	.073	.027	1.179	**.239**	.864	1.157
SGPT	.005	.126	.001	.036	**.971**	.545	1.835
TG	.043	.017	.075	2.513	**.012**	.503	1.988
SP	.533	.085	.212	6.240	**.000**	.384	2.602
DP	-.994	.163	-.208	-6.084	**.000**	.381	2.622
Age	.242	.176	.029	1.369	**.172**	.970	1.031

Linear Regression Model : 1
Blood sugar (PP) = 0.980WC + 12.233 BMI -0.031 Ch +0.086SGOT + 0.005 SGPT + 0.043 TG + 0.533 SP - 0.994 DP +0.242Age + (Constant)
Blood sugar (PP) = 0.980WC + 12.233 BMI -0.031 Ch +0.086SGOT + 0.005 SGPT + 0.043 TG + 0.533 SP - 0.994 DP +0.242Age - 144.979
Blood sugar (PP),

$$\mathbf{Y= 0.98X_1 + 12.23\ X_2 - 0.03X_3 +0.086\ X_4 + 0.005\ X_5+ 0.043\ X_6 + 0.53\ X_7-0.994\ X_8+0.24X_9 - 144.98}$$

Since the calculated R square = .823 (table 6) implies 82.3 % of the variation can be explainable by this model & **ANOVA indicates the model is good for the data since p < 0.05.** Collinearity Statistics implies the multi-collinearity is not significant since all VIF are less than 10 & there are mainly 5 components (WC, BMI, TG, SP & DP) which is significant (all p < 0.05) for the model and the effect of all other 4 components (Ch, SGOT, SGPT & Age) are not much significant (all p > 0.05). Hence consider another improvised model with all significant components (WC, BMI, TG, SP & DP).

Table 9: Model Summary.

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	0.906	0.821	0.819	16.217	0.821	368.510	5	402	.000

a. Predictors: (Constant), DP, BMI, TG, WC, SP. **b**. Dependent Variable: Blood sugar (PP)

Table 10: ANOVA.

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	484578.476	5	96915.695	368.510	.000[b]
	Residual	105723.325	402	262.993		
	Total	590301.801	407			

a. Dependent Variable: Blood Sugar (PP). b. Predictors: (Constant), DP, BMI, TG, WC, SP

Table 11: Coefficients.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
	B	Std. Error	Beta			Tolerance	VIF
(Constant)	-143.797	12.552		-11.456	.000		
WC	.863	.377	.070	2.286	.023	.478	2.091
BMI	12.44	.417	.868	29.865	.000	.528	1.895
TG	.032	.014	.055	2.373	.018	.816	1.225
SP	.554	.084	.221	6.611	.000	.400	2.502
DP	-1.005	.162	-.210	-6.219	.000	.390	2.563

a. Dependent Variable: Blood Sugar (PP). **b**. Predictors: (Constant), DP, BMI, TG, WC, SP

Linear Regression Model: 2
Bloodsugar (PP) = 0. .863WC + 12.44 BMI + 0.032 TG + 0.554 SP - 1.005 DP + (Constant)
Bloodsugar (PP) = 0. .863WC + 12.44 BMI + 0.032 TG + 0.554 SP - 1.005 DP -143.797
Bloodsugar (PP), **$Y= 0. .863 X_1 + 12.44 X_2 + 0.032 X_3 +0.554 X_4 - 1.005 X_5 -143.797$**
Since the calculated R square = .821 (Table 9) implies 82.1 % of the variation can be explainable by this model and is just 0.2% less than that of 9 components &**ANOVA indicates the model is also good for the data since p < 0.05. But while considering the correlation coefficients (**Table 5**), the maximum correlation with PP is for MBI &WC, hence consider a modified model with these two independent factors since our aim is to identify the affect of these two to PP.**

Table 12: Model Summary.

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate	Change Statistics				
					R Square Change	F Change	df1	df2	Sig. F Change
1	0.893	0.798	0.797	17.107	0.798	805.240	2	407	.000

a. Predictors: (Constant), BMI, WAIST CIRC. **b**. Dependent Variable: BLOODSUGAR (PP)

Table 13: ANOVA.

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	471331.613	2	235665.806	805.240	.000[b]
	Residual	119114.731	407	292.665		
	Total	590446.344	409			

a. Dependent Variable: Blood sugar (PP). b. Predictors: (Constant), BMI, WAIST CIRC

Table 14: Coefficients.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	Collinearity Statistics	
	B	Std. Error	Beta			Tolerance	VIF
Constant	-145.120	10.611		-13.677	.000		
WC	1.194	.365	.097	3.267	.001	.559	1.788
BMI	11.840	.427	.826	27.746	.000	.559	1.788

a. Predictors: (Constant), BMI, WC. **b.** Dependent Variable: Blood sugar (PP)

Linear Regression Model :3
Bloodsugar (PP) = 1.194+ 11.84 BMI + (Constant)
Bloodsugar (PP) = 1.194 WC + 11.84 BMI - 1.005 - 145.120
Bloodsugar (PP), **$Y=$** 1.194 **X_1 +** 11.84 **X_2**-145.120
calculated R square = 0.798 (Table 12) implies 79.8 % of the variation can be explainable by this model and is just 2.5% less than that of 9 components &ANOVA indicates the model is also good for the data since p < 0.05.

IV. CONCLUSION

The correlation of nine independent variables with the dependent variable blood sugar is observed. ANOVA tests were conducted. We found that the R- square value regarding the individual significance of nine components is 0.823. As our study is related with obesity, we further analyzed using the two components BMI and WC. Then the R square value is found to be 0.798 which implies 79.8% of the variation can be explained by this model and is just 2.5% less than that of by nine components. It also implies the other independent variables negligibly effects on the depended variable blood sugar. These regression models can be concluded as the best fit model and is generalized the study. The result & concepts incorporated with both statistical & real life significance. The impact of the two components BMI and WC on blood sugar indicates the Significance of Obesity as an Etiopathogenesis of T2DM.

V. FUTURE SCOPE

Instead of - high insulin usage & chemical medicines – an innovative indication to consider the impact of the two components BMI and WC on blood sugar and as it indicates the Significance of Obesity as an Etiopathogenesis of T2DM . The empirical analysis,

results and concepts incorporated with both statistical & real life significance.

Conflict of Interest. No.

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