

Study on Obesity in Women- An Epidemiological Approach

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Abstract- Obesity is a nutritional disorder spanning all ages and is one of the most severe problems of the 21st century. The current study was conducted to understand and analyze obesity, factors influenced by obesity [like body mass index (BMI), body fat percent (B.F %), blood pressure (B.P), fasting glucose, fasting insulin & lipid profile] and the role of environment & genetics in causing obesity using pedigree analysis and bio statistical methods on a study population.

Index Terms- Body mass index, Lipid profiles, Multifactorial inheritance, Null hypothesis, Obesity.

I. INTRODUCTION

Obesity is a major contributor to morbidity and mortality, and its prevalence has increased markedly in the last 30 years. This is thought to have occurred due to the increased availability of energy-dense foods and the reduced requirements for physical exertion during work and domestic life. It has also been demonstrated that obesity is partially genetically regulated. Obesity has become a colossal metabolic impairment causing serious public health concern with dramatic increase in health care costs. Hence there is a need to study the various aspects of obesity to arrive at a solution.

Obesity is defined as abnormal or excessive fat accumulation that may impair health. It is viewed as a syndrome rather than a single disease. It results in morbidity and mortality as it is associated with other diseases like diabetes mellitus, cardio vascular disease, sleep apnea, endometrial cancer, colon cancer etc. In obese women, the relative risk of uterus, cervix and breast cancer is higher than non-obese women. Genetic conditions known to be associated with predilection for obesity includes Prader-willi syndrome, Bardet-Biedl syndrome & Chon syndrome.

In the past decade calculations of body mass index (BMI) and body fat percent (B.F %) have evolved as standard measurements used to co-relate weight with morbidity and mortality. Obesity with its complications is costly to society. An estimated \$52 billion is the direct medical cost spent each year for treating obesity and related diseases. The above amount is expected to rise as the prevalence of obesity continues to rise. Thus, worldwide obesity prevalence is a cause for concern in the developed and the developing nations.

The aim of the current study was to establish the co-relation between obesity and different factors like BMI, BF%, blood pressure (BP), fasting glucose, fasting insulin and lipid profiles; to estimate the influence of external environment like food habits and physical activity on obesity and to analyze the inheritance of obesity in families.

II. MATERIALS AND METHODS

a. Study population:

All subjects were female & from St. Ann's college for women, Hyderabad, India. A survey was conducted & a total of 60 obese & 60 non obese subjects aged from 17-19 years volunteered for our research. For pedigree analysis 300 nuclear families were identified. All the subjects received information about the research and signed a consent form.

b. Clinical evaluation:

The age, body height, weight and tape measurements of all the 120 subjects were recorded and the body mass index (BMI) was calculated as the weight in kilograms divided by the square of the height in meters. Total body fat percent (BF %) was estimated from standard body composition analyzer available in internet (www.lowcarbdiets.about.com).

After overnight fasting, blood samples of 30 subjects (18 obese & 12 non-obese) were collected & blood pressure (B.P), fasting glucose, fasting insulin & lipid profile [total cholesterol (T. Ch.), triglycerides, cholesterol high density lipoprotein (Ch. HDL), cholesterol low density lipoprotein (Ch. LDL), cholesterol very low density lipoprotein (Ch. VLDL), T. Ch. /Ch. HDL ratio] were estimated by hiring services from United Hospitals, Hyderabad, India.

c. Effect of external environment:

To evaluate the role of external environment in obesity, the 120 subjects were inquired about their food habits & amount of physical activity in their daily life .To analyze the food habits of the subjects, they were asked to choose among three categories namely nutritious, moderate & junk. For physical activity, they were asked to answer in a yes or no pattern.

d. Pedigree analysis :

To analyze the inheritance of obesity, pedigrees of 300 nuclear families (two generation) with a total of 1184 individuals were made. All the individuals diagnosed with obesity were considered as affected & the rest as normal. The pedigrees were divided into three categories based on the mating type (father x mother of propositus) namely, normal x normal (N x N), normal x affected (N x A) or affected x normal (A x N) & affected x affected (A x A). Percentage of progeny affected for each category was calculated using segregation analysis & the results were interpreted on the basis of Mendelian laws.

e. Statistical analysis:

All the results were interpreted using statistical methods. Results for BMI, B.F %, B.P, fasting glucose, fasting insulin and lipid profile were estimated using ‘Student’s t test’ by calculating mean, standard error and t value for each character. Analysis of the effect of food habits and physical activity on obesity was done by using ‘Pearson’s chi-square analysis’ and ‘Yate’s chi-square analysis’ respectively.

A t-test is any statistical hypothesis test in which the test statistic follows a student’s t distribution if the null hypothesis is supported. It can be used to determine if two sets of data are significantly different from each other. A chi-square test is any statistical hypothesis test in which the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true.

In statistical inference of observed data of a scientific experiment, the null hypothesis refers to a general or default position that there is no relationship between two measured phenomena or that a potential influencing factor has no effect. Rejecting or disproving the null hypothesis and thus concluding that there is a relationship or a measurable effect is pivotal in the modern practice of science.

f. List of formulas:

1. Mean-

$$\bar{X} = \frac{\sum X}{N}$$

2. Variance-

$$s^2 = \frac{\sum X^2 - \frac{(\sum X)^2}{N}}{N - 1}$$

3. Standard deviation-

$$s = \sqrt{\frac{\sum X^2 - \frac{(\sum X)^2}{N}}{N - 1}}$$

4. Standard error-

$$s_{\bar{X}} = \frac{s}{\sqrt{N}}$$

5. t value-

$$t = \frac{|\bar{x}_1 - \bar{x}_2|}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}$$

6. Pearson’s chi-square-

$$\chi^2 = \sum \frac{(O - E)^2}{E}$$

7. Yate's chi-square-

$$\chi^2_{Yates} = \frac{N(|ad - bc| - N/2)^2}{N_S N_F N_A N_B}$$

III. RESULTS AND FINDINGS

Table 1: BMI & B.F% of obese and non-obese subjects

S.No.	Character	Obese		Non obese		't' value
		Mean	Standard error	Mean	Standard error	
1	BMI	24.605	0.245	20.088	0.099	4.719
2	B.F%	38.907	0.582	23.766	0.354	12.788

Degrees of freedom =N1+N2-2

= 60+60-2 =118

t value for 118 degrees of freedom at 5% loss of significance =1.658

Null Hypothesis: No correlation between obesity & the above factors

If t value is <1.658 –Accept the null hypothesis

If t value is >1.658 –Reject the null hypothesis

Since the t value for BMI & B.F% was found to be >1.658, null hypothesis was rejected.

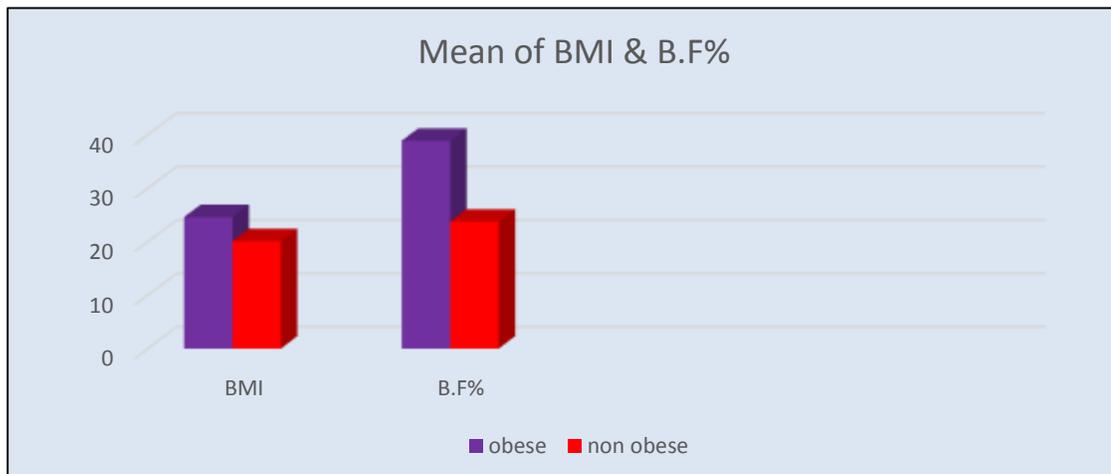


Figure 1: Mean of BMI & BF% of obese & non obese subjects

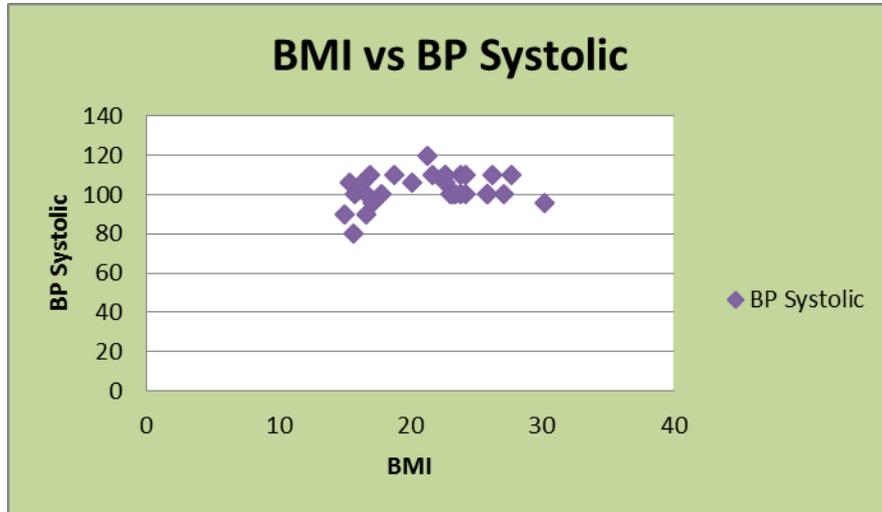


Figure 2: BMI Vs BP Systolic

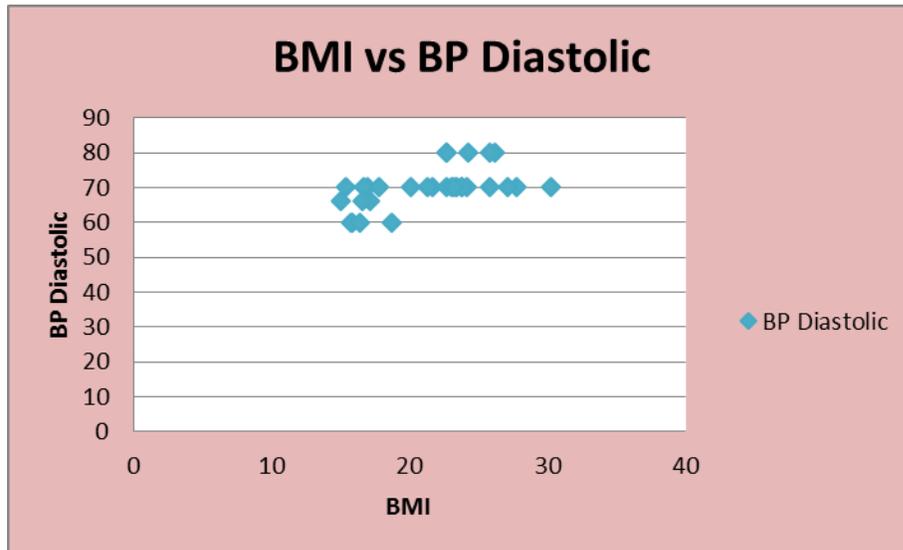


Figure 3: BMI Vs BP Diastolic

Table 2: BP, fasting glucose, fasting insulin & lipid profiles of obese & non obese subjects

S.No.	Character	Obese		Non obese		't' value
		Mean	Standard error	Mean	Standard error	
1	Blood pressure	4.406	0.025	3.461	0.048	17.871
2	Fasting glucose	155.37	2.127	143.943	3.542	2.766
3	Fasting insulin	24.63	1.267	12.329	1.058	7.443
4	Total cholesterol	288.03	6.520	280.429	5.619	0.883
5	Triglycerides	153.04	10.119	113.571	6.854	3.229
6	Ch.HDL	83.7	6.108	83.571	2.125	0.019
7	Ch.LDL	184.5	5.126	175	5.868	1.219
8	Ch.VLDL	31.4	2.015	23.571	1.388	3.199
9	T.Ch/HDL Ch. Ratio	8.19	0.225	6.671	0.195	5.098

Degrees of freedom =N1+N2-2

= 18+12-2 =28

t value for 28 degrees of freedom at 5% loss of significance =1.701

Null Hypothesis: No correlation between obesity & the above factors

If t value is <1.701–Accept the null hypothesis

If t value is >1.701–Reject the null hypothesis

For the following factors, null hypothesis was accepted since the t value was found to be <1.701

1. Total Cholesterol
2. Ch. HDL
3. Ch. LDL

For the following factors, null hypothesis was rejected since the t value was found to be >1.701

1. B.P
2. F.G
3. F.I
4. Triglycerides
5. Ch. VLDL
6. T. Ch./Ch. HDL Ratio

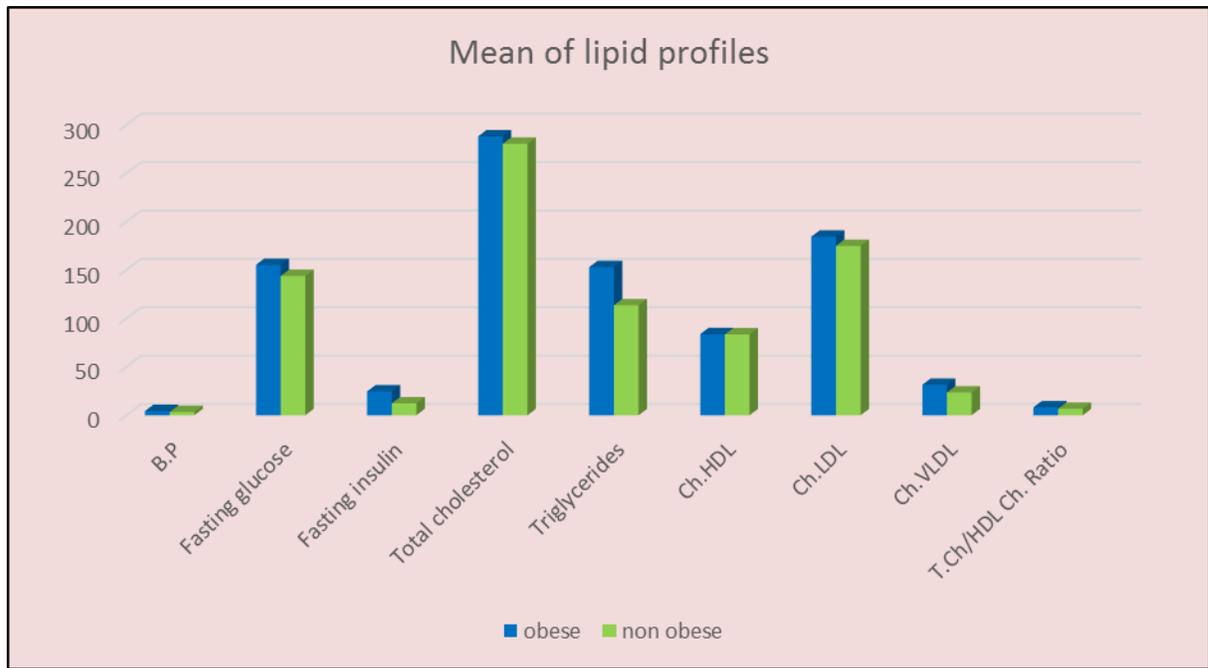


Figure 4: Mean of Lipid Profiles of obese & non-obese subjects

Table 3: Effect of Food Habits

Type of food	Obese	Non obese	Row Total
Nutritious	0	16	16
Moderate	38	17	55
Junk	48	1	49
Column Total	86	34	Grand Total=120
	χ^2	57.36384	

Degrees of freedom = (Row total-1) (Column total-1)
 = (3-1) (2-1) = 2

χ^2 value for 2 degrees of freedom at 5% loss of significance = 5.991

Null Hypothesis: No effect of food habits on obesity

If χ^2 value is <5.991–Accept the null hypothesis

If χ^2 value is >5.991–Reject the null hypothesis

Since the χ^2 value was >5.991, null hypothesis was rejected.

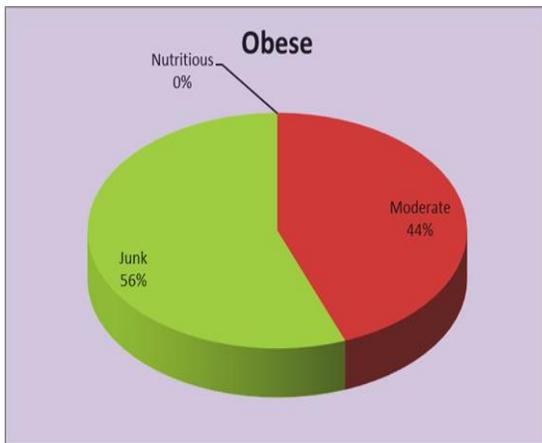


Figure 5: Food habits of obese subjects

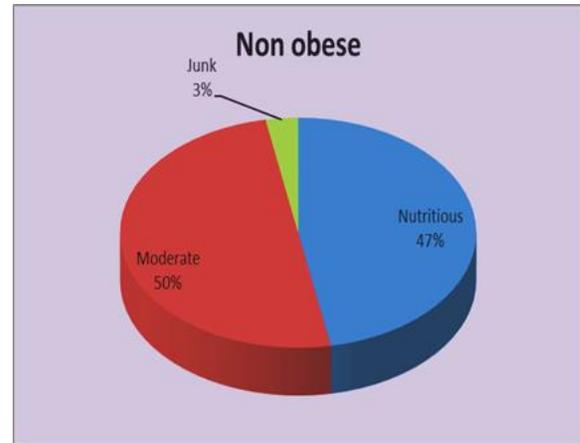


Figure 6: Food habits of non-obese subjects

Table 4: Effect of Physical activity

Exercise	Obese	Non obese	Row Total
Yes	10	30	40
No	76	4	80
Column Total	86	34	Grand Total=120

χ^2	67.84
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Degrees of freedom = (Row total-1) (Column total-1)
 = (2-1) (2-1) = 1

χ^2 value for 1 degrees of freedom at 5% loss of significance = 3.841

Null Hypothesis: No effect physical activity on obesity

If χ^2 value is <3.841–Accept the null hypothesis

If χ^2 value is >3.841–Reject the null hypothesis

Since the χ^2 value was found to be >3.841, null hypothesis was rejected.

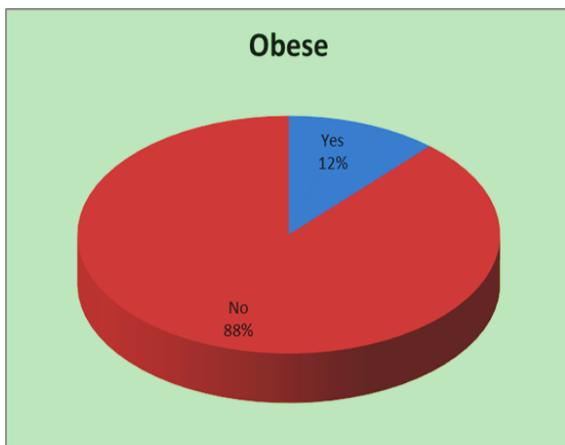


Figure 7: Answers of obese subjects about physical activity

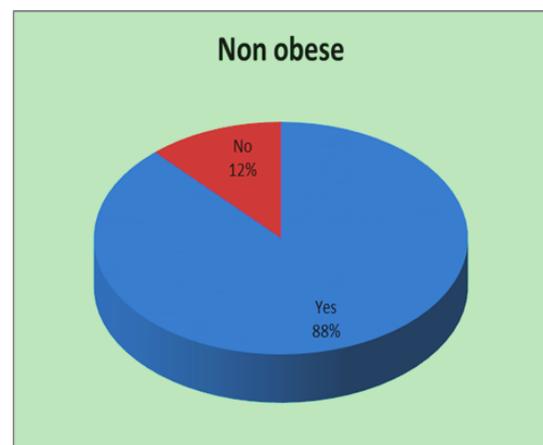
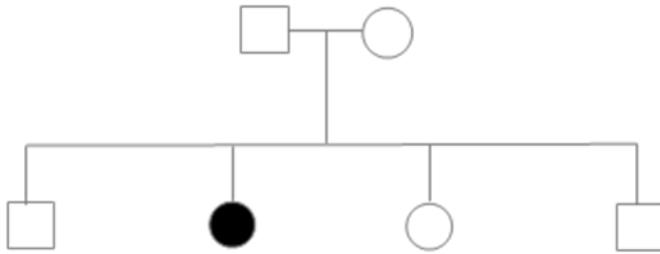
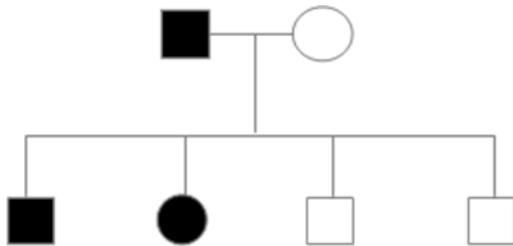


Figure 8: Answers of non-obese subjects about physical activity

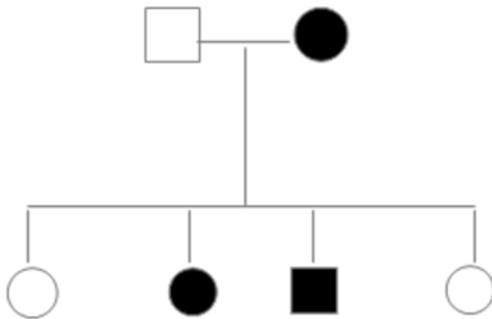
1. NxN



2. AxN



3. NxA



4. AxA

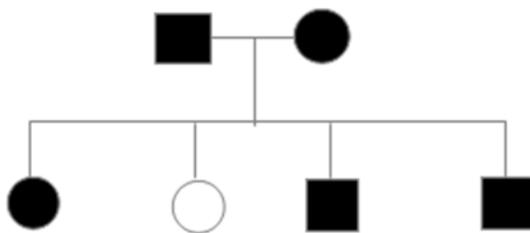


Figure 9: Sample pedigrees

Table 5: Pedigree Analysis

S.No.	Mating type (M x F)	Total frequency	No. of progeny affected	Total progeny	% of progeny affected
1	N x N	100	62	241	25.70%
2	N x A & A x N	100	66	187	35.30%
4	A x A	100	87	156	55.80%

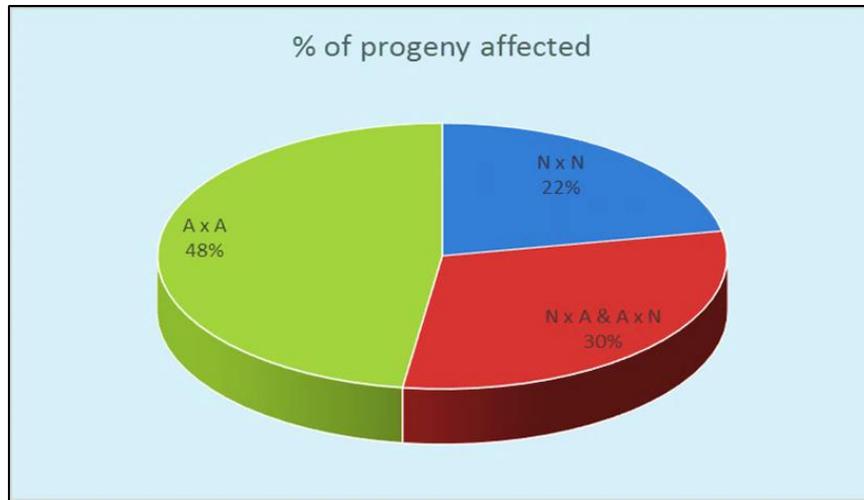


Figure 10: Percentage of progeny affected

IV. DISCUSSION

Obesity is a leading preventable cause of death worldwide, with increase in prevalence in adults, especially women and children, and authorities view it as one of the most serious public health problems of the 21st century.

The BMI is used in a wide variety of contexts as a simple method to assess how much an individual's body weight departs from what is normal or desirable for a person of his or her height. Obesity leads to excess fat deposition due to which a person's bodyweight is at least 20% higher than it should be. This results in an increase in BMI value. However, the BMI measurement can sometimes be misleading as it does not measure the % of body fat. Hence BF% is a more accurate measure of fitness level since it calculates a person's relative body composition without regard to height or weight. The 't' values of BMI and B.F % were found to be much higher than the table value (4.719>1.658, 12.788>1.658 respectively). Thus it can be concluded that a strong correlation exists between obesity, BMI and B.F%. It has been observed that obese subjects show higher BMI & B.F% values. This is in accordance with the already established method of using BMI & B.F% to identify obese subjects.

Obesity is known to increase the risk of elevated blood pressure. The 't' value of B.P was found to be much higher than the table value (17.871>>1.701). This shows that obesity leads to high B.P. However our present study could not support the fact that a linear relationship exists between BP & BMI. The fasting blood glucose & insulin levels of obese subjects were observed to be more than non-obese subjects (t value for blood glucose =2.765 & for insulin =7.443). Hence the current study supports the fact that obesity elevates blood glucose & insulin levels. This is the cause for diabetes in severely obese people.

A lipid profile is a measurement of various lipids that are found in the blood. It is a known fact that obese patients suffer from high cholesterol levels in their blood which in turn leads to cardiovascular disorders. In the current study, values of triglycerides, Ch. VLDL & T. Ch./Ch.HDL ratio were found to be higher in obese subjects ('t' values 3.229, 3.199 & 5.097 respectively) which indicates that a direct relationship exists between them & obesity. Such elevated lipid levels in blood might result in diabetes & coronary diseases. However any definite relationship between obesity and total Cholesterol, Ch. HDL & Ch. LDL could not be observed. High Total Ch. Levels in obese subjects were not observed may be due to the fact that the sample population contained subjects belonging to teenage & these obese subjects might develop high Total. Ch. levels in future.

The origin of obesity is complex and is it thought to be affected by the behavior and lifestyle of the individual. The current study reveals that food habits & physical activities influence obesity to a great extent (χ^2 values 57.364 & 67.84 respectively). Eating food

rich in fat content & having high calorie value is one of the major causes of obesity. The excess fat gets deposited in adipose tissue resulting in increase in body weight. Thus obesity is caused due to lack of energy balance i.e. more energy input than energy output. Leading a sedentary lifestyle also leads to obesity as the excess calories are not burned up. The study also showed that obese subjects lead a very inactive lifestyle as they get tired soon. Apart from food habits & physical activities, metabolism, surrounding environment, health conditions and medicines are also known to influence obesity.

Like many other medical conditions obesity is the result of an interplay between genetic and environmental factors. Although genetic deficiencies are currently considered rare, variations in these genes may predispose one to common obesity. Recent research has revealed that several genes contribute to obesity. From the current study, it can be concluded that obesity follows multifactorial inheritance in which inheritance of the phenotypic characteristic is attributable to two or more genes. Unlike monogenic traits, these traits were not found to follow a particular pattern of Mendelian inheritance.

V. CONCLUSION

The current study shows considerable evidence that obesity causes a significant increase in BMI, B.F %, B.P, fasting glucose, fasting insulin, triglycerides, Ch. VLDL & T.Ch./HDL Ch. ratio. Obesity could be a multifactorial trait, influenced by more than one gene and triggered by internal and external environmental factors like food habits & physical activity.

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VII. REFERENCES

- [1] Ana Elisa Madalena Rinaldi, Erick Prado de Oliveira. Dietary intake and blood lipid profile in overweight and obese children and adults. Centre for Physical and Nutritional Metabolism, Sao Paulo State University, Department Of Public Health, Botucatu City, Sao Paulo State, Brazil. Rinaldi et al. BMC Research Notes 2012, 5:598
- [2] Asviandri, Lita Farlina, Eka Agustia Rini, Association between lipid profile and blood pressure in obese adolescents in Padang Endocrinology Division Department of Child Health, Medicine Faculty, Andalas University. International Journal of Paediatric Endocrinology 2013, 2013(Suppl1):P88
- [3] Beevers G, Lip GYH, O'Brien E. ABC of hypertension, 4th Ed London: BMJ Books; 2001.
- [4] Chandrasekharan Nair Kesavachandran, Vipin Bihari & Neeraj Mathur, The normal range of body mass index with high body fat percentage among residents of Lucknow city in north India Epidemiology Division, Indian Institute of Toxicology Research (CSIR), Lucknow, India, Indian J Med Res 135
- [5] Dietz WH, Robinson TN: Overweight children and adolescents. N English J Med 2005, 352:2100-2109.
- [6] E. A. Francischetti, V. A. Genelhu. Obesity-hypertension: an ongoing pandemic Hypertension Clinic, Laboratory of Clinical and Experimental Pathophysiology, CLINEX, Rio de Janeiro State University, Rio de Janeiro, Brazil International Journal of Clinical practice.
- [7] Feinleib M. (1985): Epidemiology of obesity in relation to health hazards. Annals of Internal Medicine 103:1019-1024.
- [8] Han JC, Lawlor DA, Kimm SYS: Childhood obesity. Lancet 2010, 375:1737-1748.
- [9] H. M. Whyte. Blood Pressure and Obesity. American Heart Association. Circulation 1959; 19:511-516
- [10] Jaswant Singh Thakur and Sujay Bisht. BLOOD LIPID PROFILE OF OBESE AND NON-OBESE SEDENTARY COLLEGE STUDENTS. VSRD TECHNICAL & NON -TECHNICAL JOURNAL IN TERNATI ONA L JOUR NAL .VSRD-TNTJ, Vol. I (1), 2010, 26-29.
- [11] Kearney PM, Whelton M, Reynolds K et al. Global burden of hypertension: analysis of worldwide data. Lancet 2005; 365: 217-23.
- [12] .Kidambi S, Kotchen J, Grim C, Raff H, Mao J, Singh R, Kotchen T. Association of adrenal steroids with hypertension and the metabolic syndrome in blacks. Hypertension 2007; 49: 704-711.
- [13] Krebs NF, Himes JH, Jacobson D, Nicklas TA, Guilday P, Styne D: Assessment of Child and Adolescent Overweight and Obesity. Paediatrics 2007, 120: S193-S228.
- [14] Massie`ra F, Bloch-Faure M, Ceiler D, Murakami K, Fukamizu A, Gasc JM, Quignard-Boulange´ A, Neyrel R. Adipose angiotensinogen is involved in adipose tissue growth and blood pressure regulation. FASEB J 2001; 15: 2727-2729.
- [15] Paolo Palatinia and Gianfranco Parati. Blood pressure measurement in very obese patients: a challenging problem. Department of Clinical and Experimental Medicine, University of Padua, Padua. Journal of Hypertension 2011, 29:425-429.
- [16] Prentice AM: The emerging epidemic of obesity in developing countries. Int J Epidemiology 2006, 35:93-99.
- [17] Reilly JJ: Tackling the obesity epidemic: new approaches. Arch Dis Child 2006, 91:724-726.
- [18] Swinburn BA, Caterson I, Seidell JC, James WP: Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr 2004, 7(1A):123-146.
- [19] Vasilios Kotsis1, Stella Stabouli, Sofia Papakatsika, Zoe Rizos. Mechanisms of obesity-induced hypertension. Department of Medicine, Hypertension Center, Papageorgiou Hospital, Aristotle University of Thessaloniki, Thessaloniki, Greece. Hypertension Research (2010) 33, 386-393; doi:10.1038/hr.2010.9.
- [20] Whelton PK, He J, Muntner P. Prevalence, awareness, treatment and control of hypertension in North America, North Africa and Asia. J Hum Hypertens 2004; 18: 545-51.

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