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Predication of Biochemical Changes in Obese Women

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Abstract: **Obesity is a chronic disorder of multifactorial causes and is now classified as a serious public health problem. The aim of the present study is to assess biochemical changes and to predict the significant factors that contribute to metabolic influence in obese women. Two hundred twelve women, aged between 20-40 years, living in Jeddah, Saudi Arabia were conducted with this study. Studied subjects visited the clinic of the Center of Excellence for Osteoporosis Research (CEOR), during November 2013 to September 2014 and provided their blood samples to measure fasting serum glucose, total cholesterol (TC), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), triglyceride (TG), calcium, phosphorous and magnesium. The results of the present study showed there were significant differences in age (P< 0.002) and serum TG (P< 0.0001) among control, overweight and obese women. The importance predictors that effect on BMI were revealed by using Automatic Linear Regression, along with the effect direction and the P-value. The significant variables arranged in descending order of importance were as follows: serum TG (45%, positive effect, P< 0.0001), age (39%, positive effect, P< 0.0001) and serum HDL-C (16%, negative effect, P< 0.009). It was concluded that age, serum TG and serum HDL-C are predictive factors of obesity.**

Keywords: **Obesity- Biochemical Changes- Age- Women.**

I. INTRODUCTION

Obesity is a disorder of body weight regulatory systems distinguished by an accumulation of excess body fat [1] and is recently known as a chronic, heterogeneous and multifactorial disease [2]. The cause of obesity is complex and multi-factorial as environmental, social and genetic factors [3]. The main cause of obesity results from positive energy balance where energy intake exceeds the energy expenditure [4] through excessive food intake and lack of physical activity [3]. The metabolic changes initiated from augmented adipocytes mass; most effects of obesity comprise dyslipidemias, glucose intolerance, insulin resistance, triglyceride [1,5] increased arterial blood pressure; elevated circulating concentrations of triacylglycerol, lowered of high density lipoprotein cholesterol [6]. Some physical and mental illnesses can increase risk of obesity as hypothyroidism, Cushing's syndrome, growth hormone deficiency and the eating disorders [7]. Certain medications like steroids, atypical antipsychotics and some fertility drugs may lead to gain the weight [8].

Pathophysiology of obesity is involved in appetite and body regulation [9-11] through 1. Hormonal signals secreted according to amount of fat 2. Adipocytokines released response to inflammatory condition 3. Acute-phase released response to metabolic syndrome 4. Appetite (Leptin and resistin decrees appetite while adiponectin and adipocytokines increase appetite) 5. Gut peptide, Glucostatic and thermostatic hypothesizes responsible for appetite and body weight regulation.

Obesity is considered a major public health problem in developed countries due to the increase of its prevalence and the fact that it is associated with several diseases [2]. Medical consequences of obesity include cardiovascular disease,

coronary heart disease ,hypertension, congestive heart failure, arrhythmias, sudden death, diabetes, gallbladder disease, arthritis, infertility, sleep disorders, stroke and peripheral vascular disease, metabolic syndrome as high blood cholesterol and triglyceride levels [12-13]. Obesity causes of death worldwide; in the United States obesity is estimated to cause 111,909 to 365,000 deaths per year while 1 million (7.7%) of deaths in Europe are attributed to excess weight [7].

The aim of the present study is assess the biochemical changes, predict the factors that contribute to obesity and build a predictive model that can be used to estimate the BMI for the Saudi women based on the significant metabolic factors.

II. SUBJECTS AND METHODS

Two hundred twelve women, aged between 20-40 years, living in Jeddah, Saudi Arabia were contacted by phone to explain the study importance and to arrange an appointment in Center of Excellence for Osteoporosis Research (CEOR) clinic in King Abdulaziz University (KAU). Women who agreed to participate in the study were signed a written informed consent and answered a questionnaire concerning to medical history and drug intake. The subjects should be free of all diseases that interfere with obesity such as polycystic ovary syndrome, diabetes mellitus, hyperthyroidism, hyperprolactinaemia, menstrual disturbance, hypertension, psychiatric disorder. The study was conducted from November 2013 to September 2014. The study was approved by the Human Research Ethics Committee of CEOR, KAU and was in agreement with ethical standards of the Helsinki Declaration of 1975. Studied subjects were medically examined at the CEOR clinic and provided blood samples.

The body mass index (BMI) was calculated as weight in kilograms (kg) per square height in meter (m^2); the weight in Kg (women were wearing light clothes and no shoes) and height in centimeter (cm) were measured by a balance (Detecto, MO, USA; range of weight 0.1-180 kg and range of height 0-200 cm). World Health organization (WHO) classifies normal weight between 18.5 - 24.9 kg/m^2, overweight between 25 - 29 kg/m^2, obesity $\geq$ 30 kg/m^2 [7,12].

BIOCHEMICAL ANALYSIS:

Venous blood was collected in the morning after fasting for 12 hrs. Serum samples were centrifuged at 3,000 g/ 15 min by (Clay – Adams, USA) centrifuge then stored at -80°C until examined the following parameters; fasting serum glucose, total cholesterol (TC), high density lipoprotein (HDL-C), low density lipoprotein (LDL-C), triglyceride (TG), calcium, phosphorous and magnesium. Biochemical kit for the determination of glucose, TC, HDL-C, LDL-C, TG, calcium, phosphorous and magnesium were measured by using VITROS 250 Chemistry System Autoanalyzer supplied by Ortho-Clinical Diagnostics, Jonson & Jonson Co., USA; results are expressed as mmol/L. As per the Clinical Diagnostics Laboratory at CEOR, intra- and inter- assay coefficients of variance (CVs) were glucose (5.5% and 15.5%, respectively), TC (4.5% and 6%, respectively), HDL-C (3% and 4.3%, respectively), LDL-C (4.5% and 9%, respectively), TG (1.5% and 3%, respectively), calcium (2% and 4%, respectively), and for phosphorous and magnesium were (1.5% and 2.5%, respectively).

STATISTICAL ANALYSIS:

Statistical analysis was performed using SPSS program (version 22). One-Way ANOVA test was used to examine differences among the groups for different variables. Automatic Linear Regression was used to predict the factor of obesity. Differences were considered significant at $P < 0.05$.

III. RESULTS

The present study was performed to assess the biochemical changes and to predict the factors that contribute to obesity. The age and metabolic parameters were exhibited in table 1; there were significant differences in age of the studied women ($P < 0.002$) and serum TG ($P < 0.0001$). The boxplots for each variable were illustrated in Fig. 1.

This study was designed to build a predictive model that can be used to estimate the BMI for the Saudi women based on the significant metabolic factors. The predictive model was performed by using Automatic Linear Regression to show the importance predictors that effect on BMI. Table 2 reflects the importance of each of the significant variables, along with the effect direction and the P-value. The accuracy of the model was estimated by (17%). The significant variables arranged in descending order of importance were as follows: serum TG (45%, positive direction, $P < 0.0001$), age (39%, positive direction, $P < 0.0001$) and serum HDL-C (16%, negative direction, $P < 0.009$), Fig. 2 and 3 reflect the results discussed.

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Table 1. Age and Metabolic Parameters between Different Groups; Control, Overweight and Obese Women

Variables	Control (n= 97)	Overweight (n= 66)	Obese (n= 49)	P-value
Age (year)	27.37±5	29.73±5.6	30.51±6.2	0.002***
Serum Fasting Glucose (mmol/L)	3.5 ± 2	3.7 ± 1.3	3.6 ± 1.2	0.822
Serum TC (mmol/L)	4.0 ± 1.1	4.3 ± 1.2	4.3 ± 1.2	0.200
Serum TG (mmol/L)	0.73 ± 0.3	0.82 ± 0.34	1.11 ± 0.6	0.0001***
Serum HDL-C (mmol/L)	1.2 ± 0.3	1.2 ± 0.4	1.2 ± 0.4	0.852
Serum LDL-C (mmol/L)	2.5 ± 0.8	2.7 ± 0.8	2.7 ± 0.88	0.269
Serum Calcium (mmol/L)	2.27 ± 0.4	2.37 ± 0.42	2.35 ± 0.42	0.378
Serum Phosphorous (mmol/L)	1.81±1.2	1.80±1.2	1.74±1.2	0.940
Serum Magnesium (mmol/L)	0.707±0.1	0.76±0.1	0.72±1.2	0.152

Values are presented as means±SD; TC: total cholesterol; TG: triglycerides; HDL-C: high density lipoprotein cholesterol; LDL: low density lipoprotein cholesterol; ***highly significant P< 0.0001.

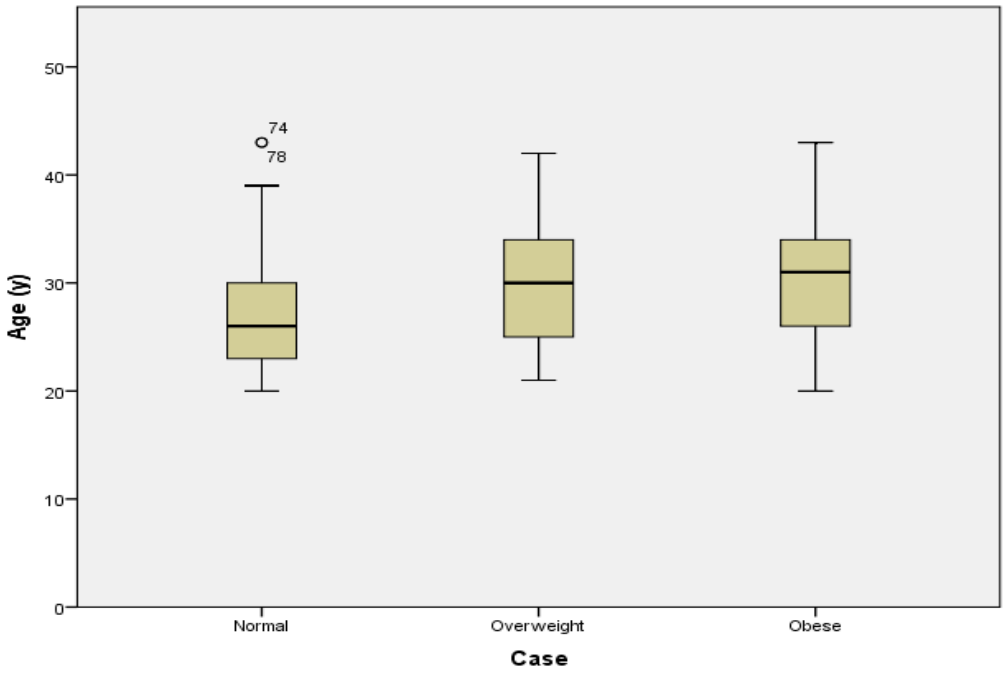
Table 2. Importance and Significant of Variables Affecting BMI

Predictor	Importance	Coefficient Estimation	P-value
Serum Triglycerides (mmol/L)	45 %	Positive	0.001***
Age (year)	39 %	Positive	0.001***
Serum HDL-C (g)	16 %	Negative	0.009**

HDL-C: high density lipoprotein cholesterol; **significant P< 0.01; ***highly significant P< 0.0001.

To apply and check the accuracy of the predictive model, a new volunteer (out of the studied women who used to obtain the model) was used to predict the BMI. The measurements of this volunteer was as follows: (serum TG= 2.6 mmol/L, age= 28 years, and serum HDL-C= 0.8 mmol/L). By the help of this model, the predicted value for the BMI was found to be 31.41 kg/m^2. The true value for the BMI for this volunteer was equal to 32.44 kg/m^2. This means that the error for estimating equals to 1.03 kg/m^2 only, which gives an idea about the accuracy of the model and how we can use it to predict the BMI.

Note that the line in the middle of each boxplot indicates the value of the median value and the small circles or stars (if exist) reflect the outliers in the data)



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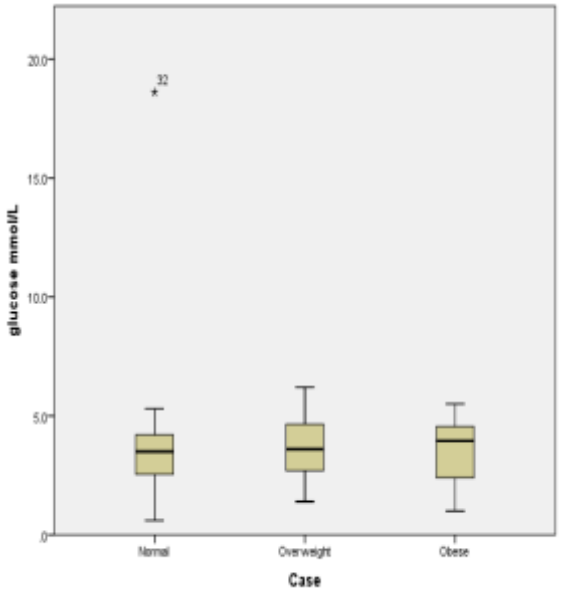

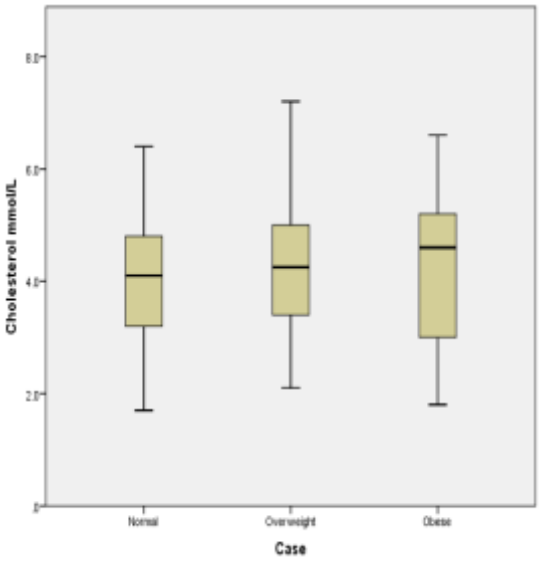

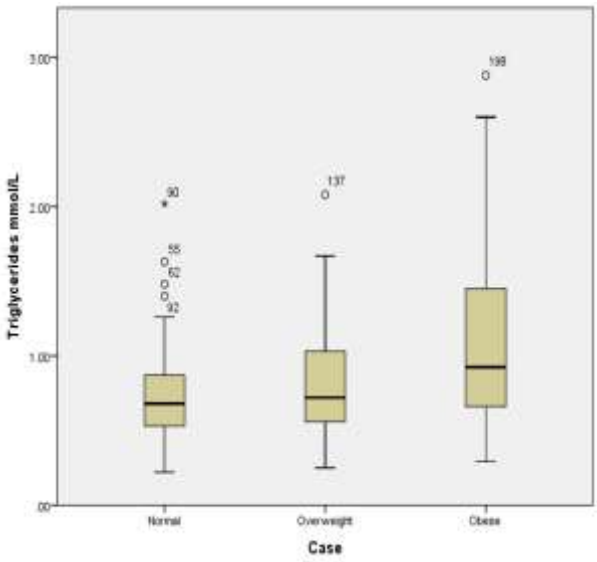

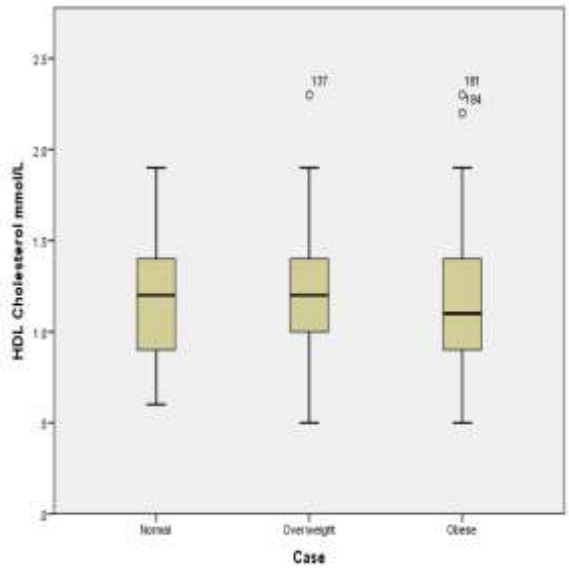

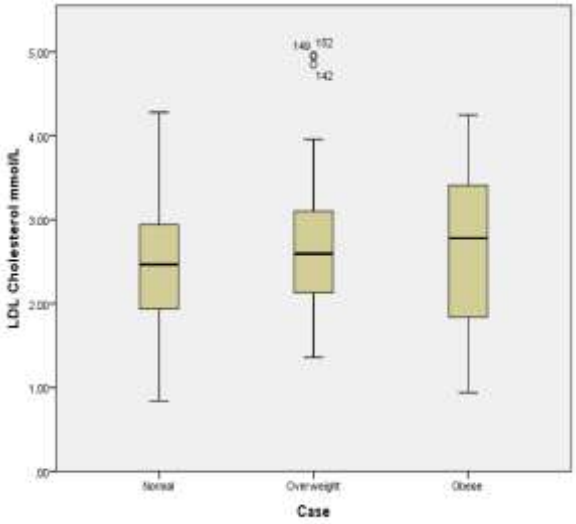

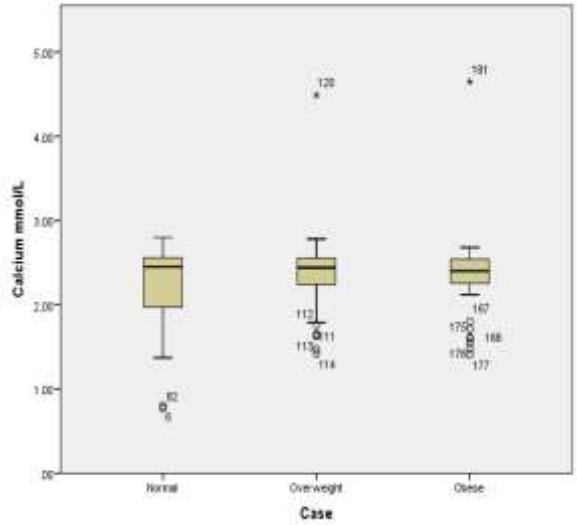

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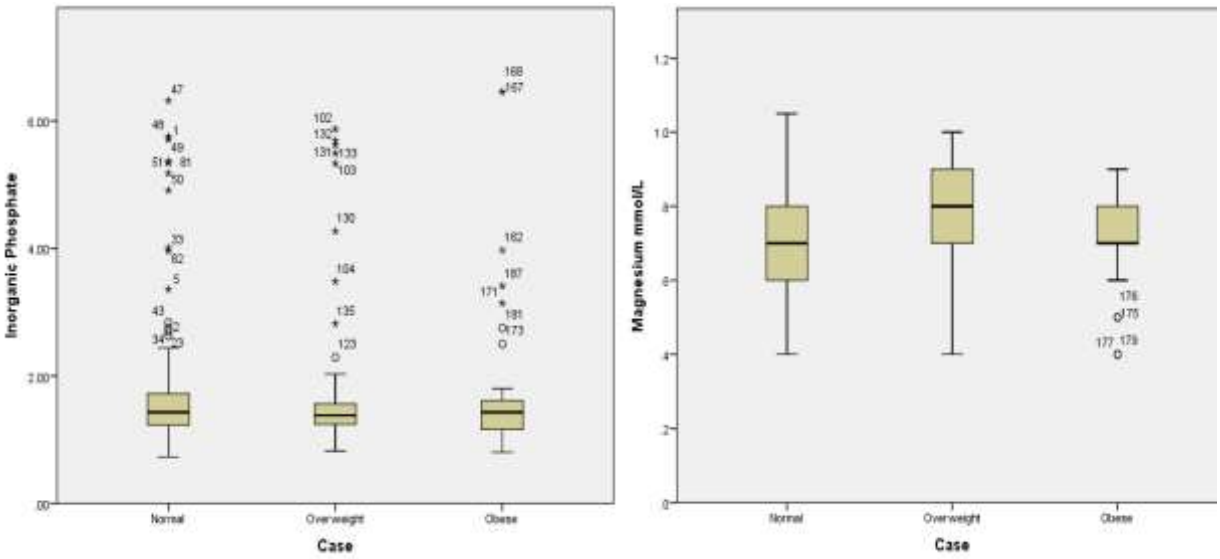

Figure 1. Boxplots for Age and Metabolic Variables

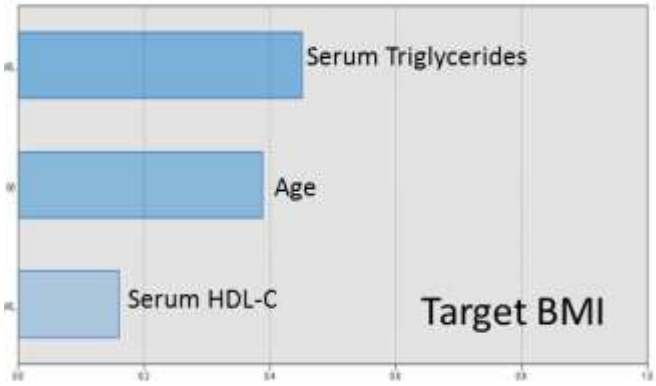

Figure 2. Predictor Importance of Variables Affecting BMI

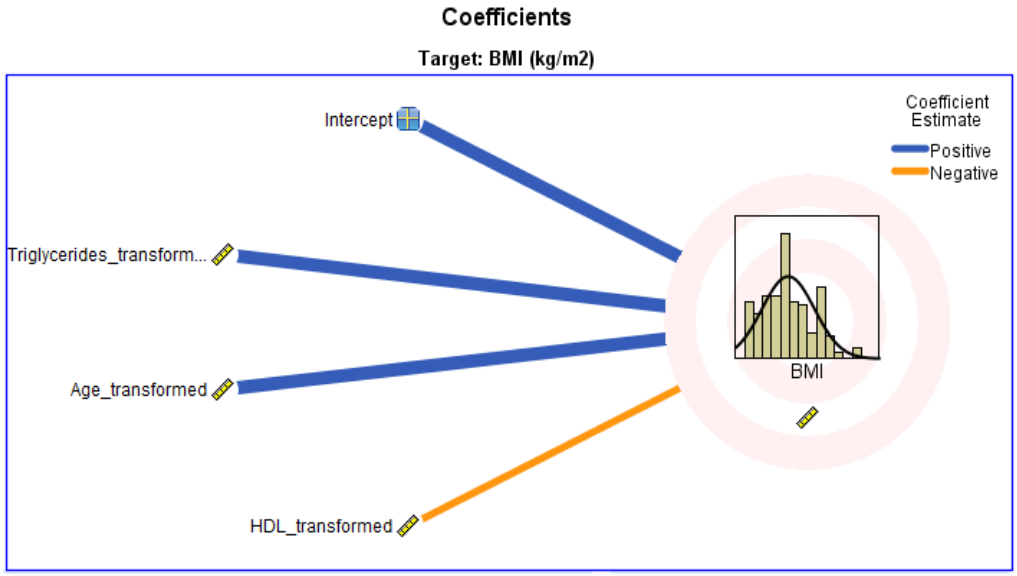

Figure 3. Coefficient Estimation of Predictors Affecting BMI

VII. DISCUSSION

The prevalence of overweight and obesity is increased in recent years [14] within large range of age since age may interact with ethnicity to predict regional body fat distribution [15]. Our result showed that age is a significant variable among BMI groups and it is an important positive predictors affecting BMI, although, our sample was composed of young women with the same range of age (20-40 years). This result is consistent with previous studies [16-17]. A study of Reas *et al*. [18] suggested that adults aged (20–40 years) gain of approximately 1.8 to 2.0 pounds/ year (0.8 to 1.0 kg) while women aged (20–29 years) gain weight of 1.5 pounds/year (0.7 kg). This study suggested that weight gains as a result of annual reducing in basic metabolic rate without decreasing the calorie intake or changing the life style.

On other hand, metabolic characteristics did not showed significant differences between control, overweight and obese groups except triglycerides which is also considered as a positive predictor of obesity; High triglycerides are usually caused by other conditions such as obesity [6,19] or may be associated with vitamin D deficiency [20-21] or insulin resistance [22] which are not measured in this study. Obese subjects especially with metabolic syndrome are suffering from insulin resistance, hypertriglyceridemia, reduced HDL-C level, elevated blood pressure and glucose intolerance [23].

HDL-C level is a common lipid disorder in obese subjects and is associated with degree and distribution of obesity [23]. This study revealed that HDL-C is a significant negative predictor of obesity. The negative relation between HDL-C and obesity is demonstrated in many studies [24-26].

The mechanisms that explained the low concentration of HDL-C in obese people: changes in HDL component and its role in rapid clearance, cholesterol efflux and influx of adipocytes, the alternations of adipokines in obesity as well as hepatic miR-33b and its host gene SREBP-1c coordinate HDL-C and TG variations [23].

Some of the limitations of this study included insulin and insulin resistance, very low density lipoprotein cholesterol (VLDL-C), adiponectin and leptin.

In conclusion, the present study has demonstrated that age, serum triglycerides are positive predictive factors of obesity while and serum HDL-C is a negative predictive factor affecting BMI.

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REFERENCES

[1] Harvey RA, Ferrier DR. Lippincott's Illustrated Reviews Biochemistry: Obesity. 5th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2011, PP. 349-355.

[2] Custódio Afonso Rocha V, Ramos de Arvelos L, Pereira Felix G, Nogueira Prado de Souza D, Bernardino Neto M, Santos Resende E, Penha-Silva N. Evolution of Nutritional, Hematologic and Biochemical Changes in Obese Women During 8 Weeks after Roux-En-Y Gastric Bypasss. Nutrición Hospitalaria 2012; 27(4):1134-1140.

[3] Lau DCW, Douketis JD, Morrison KM, Hramiak IM, Sharma AM, Ur E and for members of the Obesity Canada Clinical Practice Guidelines Expert Panel. 2006 Canadian Clinical Practice Guidelines on the Management and Prevention of Obesity in Adults and Children. Canadian Medical Association Journal 2007; 176(8): S1–S13.

[4] Hill JO, Wyatt HR, Peters JC. Energy Balance and Obesity. Circulation/ American Heart Association 2012; 126: 126-132.

[5] Champe PC, Harvey RA, Ferrier DR. Biochemistry: Obesity. 4th ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2008. PP. 350- 355.

[6] Foss YJ. Vitamin D Deficiency is the Cause of Common Obesity. Medical Hypotheses 2009; 72: 314–321.

[7] Shukla G, Sarika M, Saritha D, Sampath Kumar CJ. A Realistic Way to Win the War against Obesity Naturally. International Journal of Preclinical & Pharmaceutical Research 2014; 5(1): 51-56.

[8] Babalola JF. Health Consequences of Obesity and Overweight. Pakistan Journal of Social Sciences 2011; 8(3): 118-124.

[9] Redinger RN. The Pathophysiology of Obesity and its Clinical Manifestations. Gastroenterology and Hepatology 2007; 3(11): 856–863.

[10] Srivastava N, Lakhan R, Mittal B. Pathophysiology and Genetics of Obesity. Journal of Experimental Biology 2007; 45: 929-936.

[11] Gurevich-Panigrahi T, Panigrahi S, Emilia Wiechec E, Marek Los M. Obesity: Pathophysiology and Clinical Management. Current Medicinal Chemistry 2009; 16: 506-521.

[12] Wilborn C, Beckham J, Campbell B, Harvey T, Galbreath M, La Bounty P, Nassar E, Wismann J, Kreider R. Obesity: Prevalence, Theories, Medical Consequences, Management, and Research Directions. Journal of the International Society of Sports Nutrition 2005; 2(2): 4-31.

[13] Finer N. Medical Consequences of Obesity. Medicine 2011; 39(1): 18–23.

[14] WHO: World Health Organization. Obesity and Overweight. Available at: http://www.who.int/mediacentre/factsheets/fs311/en/. #Last Editorial Review (January, 2015).

[15] Stults-Kolehmainen MA, Stanforth PR, Bartholomew JB. Fat in Android, Trunk, and Peripheral Regions Varies by Ethnicity and Race in College Aged Women. Obesity 2012; 20: 660–665.

[16] Wang Y, Beydoun MA. The Obesity Epidemic in the United States- Gender, Age, Socioeconomic, Racial/Ethnic, and Geographic Characteristics: A Systematic Review and Meta-Regression Analysis. Epidemiologic Reviews 2007; 29:6-28.

[17] Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of Obesity in the United States, 2009–2010. National Center for Health Statistics Data Brief 2012; 82:1-7. Retrieved from http://www.cdc.gov/nchs/data/databriefs/db82.pdf.

[18] Reas DL, Jan F Nygard JF, Svensson E, Sørensen T, Sandanger I. Changes in Body Mass Index by Age, Gender, and Socio-Economic Status among A Cohort of Norwegian Men and Women (1990–2001). BMC Public Health 2007; 7:269-275.

[19] Lagunova Z, Porojnicu LC, Lindberg F, Hexeberg S, Moan J. The Dependency of Vitamin D Status on Body Mass Index, Gender, Age and Season. Anticancer Research 2009; 29(9): 3713–3720.

[20] Botella-Carretero JI, Alvarez-Blasco F, Villafruela JJ, Balsa JA, Va´zquez C, Escobar-Morreale HF. Vitamin D Deficiency is Associated with the Metabolic Syndrome in Morbid Obesity. Clinical Nutrition 2007; 26: 573–580.

[21] Wamberg L, Kampmann U, Stødkilde-Jørgensen H, Rejnmark L, Pedersen SB, Richelsen B (2013) Effects of Vitamin D Supplementation on Body Fat Accumulation, Inflammation, and Metabolic Risk Factors in Obese Adults with Low Vitamin D Levels — Results from A Randomized Trial. European Journal of Internal Medicine 2013; 24(7):644-649.

[22] Novotny R, Going S, Teegarden D, Van Loan M, McCabe G, McCabe L, Daida YG, Boushey CJ; ACT Research Team. Hispanic and Asian Pubertal Girls Have Higher Android/Gynoid Fat Ratio than Whites. Obesity 2007; 15(6): 1565-1570.

[23] Wang H, Peng D-Q. New Insights into the Mechanism of Low High-Density Lipoprotein Cholesterol in Obesity. Lipids in Health and Disease 2011; 10:176- 185.

[24] Mooradian AD, Haas MJ, Wehmeier KR, Wong NC. Obesity-Related Changes in High-Density Lipoprotein Metabolism. Obesity 2008; 16(6):1152-1160.

[25] McGillicuddy FC, Reilly MP, Rader DJ. Adipose Modulation of High-Density Lipoprotein Cholesterol: Implications for Obesity, High-Density Lipoprotein Metabolism, and Cardiovascular Disease. Obesity 2008; 16: 1152–1160.

[26] Head GA. Cardiovascular and Metabolic Consequences of Obesity. Frontiers in Physiology 2015; Retrieved from http://dx.doi.org/10.3389/fphys.2015.00032.