

**COMPRATIVE STUDY OF REMNANT CHOLESTEROL, SOME LIPID FRACTION AND FBS LEVEL IN OVERWEIGHT AND OBESE PARTICIPANTS IN SOUTH EAST NIGERIA.**

**ABSTRACT:**

**BACKGROUND:** Obesity is one of the features of metabolic disorders, and has been shown to be involved in cardiovascular diseases. Remnant cholesterol has been suggested to be a link from obesity to coronary heart diseases, hence a predisposing factor in the development of cardiovascular disease by obese individuals. We compared the level of calculated non-fasting remnant cholesterol, fasting and non-fasting lipid profile and fasting blood sugar in overweight, obese and normal weight participants in Nnewi, a town in South East Nigeria.

**METHODS:** this is a cross- sectional study. A total of 90 apparently healthy obese, overweight and normal weight participants who met the inclusion criteria were randomly enrolled into the study. They were grouped using their body mass index of 18.5 to 24.9 kg/m<sup>2</sup> (normal weight), 25 to 29.9 kg/m<sup>2</sup> (overweight) and ≥30 kg/m<sup>2</sup> (obese). The parameters were analyzed using standard methods.

**RESULTS:** Our results shows that there was progressive increase of remnant cholesterol among the group but there was no statistical difference ( $p>0.05$ ) in the mean calculated remnant cholesterol (mmol/l) between the obese ( $0.72\pm0.4$ ), overweight ( $0.68\pm0.46$ ), and normal weight ( $0.50\pm0.28$ ) participants. Mean fasting very low density lipoprotein (VLDL) and triglyceride (TG) (mmol/l). Were significantly higher ( $p<0.05$ ) in overweight compared to normal weight participants ( $0.76\pm0.31$  vs.  $0.47\pm0.22$ ) and ( $1.72\pm0.60$  vs.  $1.09\pm0.51$ ) and obese participants ( $0.82\pm0.23$  vs.  $0.47\pm0.22$ ) and ( $1.85\pm0.59$  vs.  $1.09\pm0.51$ ) when compared to normal weight participants respectively. While plasma glucose FBS levels (mmol/l) were significantly lower in normal weight participants when compared to overweight participants ( $3.45\pm0.79$  vs.  $4.46\pm1.82$ ) and obese participants ( $3.46\pm 0.79$  vs.  $4.94\pm1.26$ ). Remnant cholesterol was not statistically significant among the obese, overweight and normal weight participants.

**CONCLUSION:** The results obtained from this study demonstrated a progressive increase in the level of remnant cholesterol which was not statistically significant among apparently healthy individuals across the group. Furthermore we did not observed Remnant cholesterol as a sole predictor to obesity which will lead to cardiovascular diseases, with the rising prevalence of obesity in developing country as well as its associated complications and risks.

Keyword: Remnant cholesterol, obesity, BMI, Cardiovascular disease, lipid profile.

## INTRODUCTION

Obesity arise when there is an imbalance between food intake and energy metabolism (1), which gives rise to increased deposition of fat in adipose tissue, liver, muscle, pancreatic islets, and other organs involved in metabolic processes(1,2). Obesity elevates the risk of diabetes, fatty liver, coronary artery disease, gall stones, sleep apnea, arthritis and various types of cancer. (2). Metabolic consequences such as hyperlipidemia, diabetes, hypertension which are the important comorbidity of cardiovascular usually results from this metabolic imbalance. (3,8) These extremes of weight (obesity) tend to indicate the nutritional status of individuals and indirectly, the socioeconomic status. (4,5). studies has shown that Urban and rural residences are usually associated with higher and many a times, poorer socioeconomic status(5,6), leading to inappropriate unbalanced food intake, resulting to high prevalence of overweight/obesity may be seen in urban dwellers. (6,7,37). In accordance with the Global Health Observatory Data, unsuitable metabolic effects such as blood pressure, cholesterol, triglycerides and insulin resistance can result from overweight and obesity,(7,38) with increased risks of coronary heart disease, stroke and type-2 diabetes mellitus as body mass index increases. also raised body mass index increases the risk of cancer of the breast, colon, prostate, endometrium, kidney and gall bladder, and mortality rates increases as the degrees of overweight increases, (8,9,10).

Furthermore, despite the adverse risk associated with obesity, adults in developing countries believes that overweight and obesity is of no-concern and only affects the developed world, (9) and culturally in Nigeria there is this notion that any individual with huge body mass index is a sign that the individual is rich and is living well (9). This misunderstanding helped to elevates the dangerous silence as to the health effects/implications of obesity, which has been shown to be on the elevation trajectory in both urban and semi-urban areas. (8,9,10,4,11,38). Cardiovascular diseases (CVDs), which is the leading cause of morbidity and mortality in the western World, are now super-heading public health challenges in developing countries, (12,39,40,41).

Remnant cholesterol has been suggested as a link from obesity to ischemic disease, i.e., a part of the elevation risk of ischemic heart disease observed in obese individuals was explain by higher remnant cholesterol concentration (3,25,28,29,42). it is also known as remnant lipoprotein, it has been said to be very atherogenic lipoprotein and comprised primarily of very low density lipoprotein (VLDL), Intermediate density lipoprotein (IDL), and chylomicrons. Hence, can be stated that, remnant cholesterol is all plasma cholesterol that is not LDL or HDL cholesterol, because LDL and HDL are triglyceride rich lipoproteins. (13,49,50).

Lipids are found in the body tissue as they have an important role in virtually all aspects of life acting as energy stores (Triglycerides), important structural component of cells (Cholesterol)

and they could also have special functions (Hormones) (14,40). The serum concentrations of Very low density lipoproteins, cholesterol and triglycerides are positively correlated with obesity.(48) A study in twins has shown that for an average increase of 7.3% in body mass index (BMI) there were increases of 2.5% in total cholesterol concentration, 3% in low density lipoprotein (LDL) cholesterol concentration, and 18.2% in triglyceride concentration(45,43). The elevation in the concentration of cholesterol is has been linked to LDL cholesterol because the high density lipoprotein (HDL) cholesterol is typically reduced (15,48). Obesity is associated with hyperlipidemia (typically mixed) although studies has dyslipidemia is low among younger ages(1). The propensity of these conditions is greater among obese individuals. However, not only does the prevalence of these metabolic imbalances increases with age, but the rate of increase is more adverse among obese individuals (16,43). The regulation of body weight comprises a complex homeostatic system that results in a balance between energy intake and energy expenditure and the hypothalamus plays a huge role as the central coordinating base for this system (17,18,51).

## **MATERIALS AND METHODS**

This is a cross-sectional study, designed to compare the serum level of some lipid fractions and Remnant Cholesterol, in normal, overweight and obese participant. Based on the calculated sample size, 90 consenting participants that fulfill the inclusion criteria were randomly recruited for the study.

Informed consent of the participant was obtained before the enrollment into the study. Ethical approval was obtained from the Ethics Committee (NAUTH/CS/66/VOL.11/154/2018/088) of Nnamdi Azikiwe University Teaching Hospital Nnewi.

Questionnaires were administered and it served as a primary instrument for this study. The questionnaires were structured to reflect the health issues relevant to the objectives of the study. The study comprised of 90 obese and non-obese subjects. Among them, 30 participant were known obese, 60 participant were non-obese and between 20-50 years of age. The participants were labeled Normal weight (BMI 18.5-24.9  $\text{kg}/\text{m}^2$ ), over-weight (BMI 25.0-29.9 $\text{kg}/\text{m}^2$ ) and Obese ( $\geq 30\text{kg}/\text{m}^2$ ).

BMI was measured as weight in kilogram 0.01 divided by height squared ( $\text{m}^2$ ), WC was measured with a tape to the nearest 0.1cm at the end of a normal expiration at the midpoint between the subcostal plane and the iliac crest of an exposed abdomen. HC was also measured at the largest standing horizontal circumference of the buttock to determine the waist to hip ratio (WHR=WC/HC). Hypertension was defined as, systolic blood pressure $\geq 140\text{mmHg}$ , diastolic blood pressure $\geq 90\text{mmHg}$ .

A fasting blood sample was obtained in the morning between 0800-0900 hours about 7ml, through venipuncture from each participant. Aliquots were drawn into fluoride oxalate

container for plasma glucose estimation and later on, a non-fasting blood sample was obtained from the participant and aliquots drawn into plain tubes and allowed to clot at room temperature.

The serum for remnant cholesterol was separated after collection by centrifugation at 5000g for 5min and Serum samples aliquoted and frozen at -20°C for analysis later on. To ensure proper collection of fasting and non-fasting sample, the participant fast overnight and blood sample collected in the morning between 0800-0900 hours. after which the participant had his/her breakfast followed by the collection of non-fasting blood samples.

Fasting lipid profile was assessed using commercially available kits (Randox). serum total cholesterol and high density lipoprotein HDLc, was determined by cholesterol oxidase method of Allain *et al* (19), serum triglyceride by glycerol kinase method of Tindler (20) and LDLc was calculated using Friedwald formula. Friedwald, (21). and remnant cholesterol was calculated using the formula.  $Rc = Tc - HDLc - LDLc$ . (feldman, 2017). Fasting plasma glucose was determined using the enzymatic colorimetric method of Trinder (20).

Statistical analysis was performed to compare the two groups using ANOVA. The values were expressed as the Mean  $\pm$  S.D. Values were regarded as significant if  $p$  is  $\leq 0.05$ . Pearson correlation coefficient was used to correlate the parameters estimated.

## RESULTS;

**Table 1** shows the mean anthropometric markers of all participants. There was no significant difference in the diastolic and systolic blood pressures among the groups. There were significant increase in the body mass index among the groups ( $<0.001$ ). There was significance difference in the waist to hip ratio among the groups. **Table 2;** shows the mean of the various fasting biochemical parameters of Obese, Overweight and Normal weight group. Significant change was observed in the fasting plasma Triglyceride (TG) and very low density lipoprotein (VLDL) ( $p<0.001$ ), in the obese subjects when compared with the corresponding control values. No significant change was observed in other lipid parameters. **Table 3;** show the mean of the various Non-fasting biochemical parameters of obese, overweight and normal weight group. Significant change was observed in the Non-fasting plasma Triglyceride (TG) ( $p<0.005$ ) in obese subjects when compared with the corresponding control values. No significant change was observed in other lipid parameters. **Table 4;** Show the mean serum remnant cholesterol, fasting blood sugar, in obese, overweight and normal weight participant. There was no significant difference in the mean remnant cholesterol between obese and overweight subjects when compared to the normal weight control. There was statistical significance in the fasting blood sugar level between the normal weight participants ( $3.46\pm 0.79$ ) and overweight ( $4.46\pm 1.82$ ) at ( $P<0.021$ ), also between the normal weight and obese participants ( $3.46\pm 0.79$  vs  $4.94\pm 1.26$ ) at ( $P<0.001$ ). No significant difference was observed in the level of the calculated remnant

cholesterol among the groups. **Table 5;** shows the correlation of the measured parameters with BMI in Obese participant. There was a positive correlation of the lipid profile in overweight and obese participants but not statistically significant. There was no significant correlation of BMI with calculated remnant cholesterol.

## DISCUSSION

Obesity is a well-documented risk factor for various chronic medical conditions like diabetes, hypertension, dyslipidemia and cardiovascular diseases(41,42,46,48). In this study, a total of 90 participants were recruited, and classified according to groups using BMI. BMI was used as a standard for general obesity. WHR were used as predictors of cardiovascular risk factor and measure of central obesity. This study is in agreement with previous studies that reported unpleasant plasma lipid profile among subjects with higher BMI. (22,23,37,39), the levels of fasting serum VLDLc and TG were observed to be significantly higher in overweight ( $0.76\pm 0.31$ ,  $1.72\pm 0.60$   $P<0.001$ ) and obese ( $0.82\pm 0.23$ ,  $1.85\pm 0.59$   $p<0.001$ ) participants when compared to normal healthy controls ( $0.47\pm 0.22$ ,  $1.09\pm 0.51$ ) which was in accordance to the study findings of Gupta and Mukherjee, (24) and Haddad *et al.*, (25), that observed higher TG and VLDL-C in obese subjects when compared with the controls, and contradicted the study by Zavorini *et al* (26), who found no increase significant difference between the normal weight and overweight subjects. There was no observed significant change in other lipid fraction (TC, LDL-C and HDL-C) amongst all the different groups. Furthermore there was positive correlation of the lipid fractions (TG, VLDLc, LDLc, TC, and HDLc) with BMI in the obese group but not significant, this finding was in corroboration with the study of Ugwuja *et al* in which they observed a positive correlation of lipid in obese subjects without starting if its significant or not, but it reaffirms the role of lipids in the pathophysiology of overweight and obesity (24,46).

From our results, there is a progressive increased of remnant cholesterol level in the groups ( $0.50\pm 0.28$ ) in normal weight to ( $0.69\pm 0.46$ ) in overweight to ( $0.72\pm 0.40$ ) in obese participants but there is no significant difference as shown by the analysis of variance (ANOVA) at 2.853 and the p value at 0.064. it shows that the relationship of remnant cholesterol and cardiovascular disease is not caused by adiposity, but it can be explained by remnant cholesterol ability to

cause atherosclerosis in the arterial wall which is a process that has been assumed to be driven by remnant cholesterol concentration in the blood stream, independent of its reasons for elevations in overweight and obesity,(27,49) this finding is in correspondence with Varbo *et al* and chinonso *et al* (27,31). RC has considered to be the main factor mediating the residual risk of major cardiovascular events. (27,28,31,32,33,36) It has been stipulated that remnants cholesterol can be taken up directly by the macrophages, thereby converting such cells into foam cells, which is the hallmark of atherosclerosis lesions and the hydrolysis of triglyceride in remnant particles at the endothelial surface or within the arterial intima may generate local inflammation. (29,42). However our findings showed that the risk of cardiovascular accidents by high levels of remnants cholesterol may not be solely driven by BMI and level of fat tissues, this was observed in some overweight and normal weight individual with high level of remnant cholesterol, this was in accordance with the findings of Chinonso *et al.*, 2021. (31) this phenomenon can be explained by the attribute of metabolically obese normal weight,(30,31,47) but obese individual with higher remnant cholesterol levels are exposed to cardiovascular incidents due to remnant cholesterol mediated atherosclerotic process. The other lipid parameters, fasting TG and VLDL were significantly elevated in obese participants when compared to the corresponding groups (0.82±0.23) obese to (0.47±0.22) normal weight at p<0.001 for VLDL, and (1.85±0.59) obese to (1.09±0.51) normal weight at p<0.001 for TG. There was also a significance difference in the fasting blood glucose among the groups. There was no significance difference among the other lipid fractions measured in this study.

Furthermore, Retrospective Study (33), cohort studies (32, 34,35,36) and randomized clinical trials (27) investigating the relationship between RC, non-HDL-C, and the outcomes of CVDs have been carried out in recent years. Castañer *et al.* (34) enrolled 6,901 patients in the Prevención con Dieta Mediterránea (PREDIMED) research, while Langsted *et al.* enrolled patients from the Copenhagen General Population Study (CGPS), a cohort study involving a total of 109,574 individuals (35). and Xie *et al* in their study enrolled 15,464 individuals at Murakami Memorial Hospital, these studies showed that the high level of remnant-C was associated with the MACEs(32). In addition to the CGPS, Varbo *et al.* (27) included the patients from the CCHS Copenhagen City Heart Study (CCHS) and the Copenhagen Ischemic Heart Disease Study (CIHDS), a total of 73,513 subjects In which they demonstrate that a 1 mmol/L

(39 mg/dl) non-fasting remnant cholesterol increase may lead to a 2.8-fold causal risk for ischemic heart disease (27). but a cohort study on African/black population is lacking. In a study carried out in Nigeria by Chinonso *et al.*, 2021, in which they observed that the association of calculated remnant cholesterol with BMI is weak. Non-HDLc is associated with obesity. There was a negative correlation between calculated RC and non-HDLc. Implying that remnant cholesterol can be elevated in any individual without apparent increase in BMI. Suggesting, it to be predictive of cardiovascular incidents irrespective of BMI.(31).

Further studies can be carried out to determined Remnant cholesterol in metabolically obese normal weight and overweight individuals, this is because study design of this current research work limits our resources to determine this, and its attendant indices.

## **CONCLUSION**

This study demonstrated a progressive increase in the level of remnant cholesterol which was not statistically significant among apparently healthy individuals across the group, there was a significance difference in the level of FBS among the groups, also the total blood pressure (SBP and DBP) were elevated in the obese participants. Furthermore we did not observed Remnant cholesterol as a sole predictor to obesity which will lead to cardiovascular diseases. But with the increase in prevalence of obesity in developing country, with its associated complications and risks, large population cohort studies on remnant cholesterol in African/black population needs to be carried out to substantiate it as a marker towards the development of cardiovascular diseases in African population.

## **CONSENT**

Informed consent of the participants was sought and obtained before enrollment into the study.

## **ETHICAL APPROVAL**

Ethical approval was sought and obtained from the Ethics Committee (NAUTH/CS/66/VOL.11/154/2018/088) of Nnamdi Azikiwe University Teaching Hospital Nnewi

## REFERENCES

1. Finkelstein, E. A., Brown, D. S., Trogon, J. G., Segel, J. E., & Ben-Joseph, R. H. (2007). Age-specific impact of obesity on prevalence and costs of diabetes and dyslipidemia. *Value in Health*, 10(SUPPL. 1), S45-S51. <https://doi.org/10.1111/j.1524-4733.2006.00154.x>
2. Ogden, C.L., Yanovski, S.Z., Carroll, M.D., and Flegal, K.M. (2007). *The epidemiology of obesity. Gastroenterology*. 132:2087–2102.
3. Varbo A, Benn M, Davey Smith G, Timpson NJ, Tybjaerg-Hansen A, Nordestgaard BG.(2015). Remnant cholesterol, low density lipoprotein cholesterol, and blood pressure as mediators from obesity to ischemic heart disease. *Circulation Research*. 116:665–73.
4. Drewnowski A, Specter SE. (2004). Poverty and obesity: The role of energy density and energy costs. *American Journal of Clinical Nutrition*. 79:6–16.
5. Sola AO, Steven AO, Kayode JA, Olayinka AO. (2011). Underweight, overweight and obesity in adults Nigerians living in rural and urban communities of Benue State. *Annals of African Medicine*. 10:139–43.
6. Ayodele OE, Akinboro AO, Akinyemi SO, Adepeju AA, Akinremi OA, Alao CA, Popoola AA. Prevalence and clinical correlates of metabolic syndrome in Nigerians living with human immunodeficiency virus/acquired immunodeficiency syndrome. *Metab Syndr Relat Disord*. 2012 Oct;10(5):373-9. doi: 10.1089/met.2012.0050. Epub 2012 Jul 16. PMID: 22799758.
7. Ono T, Guthold R, Strong K. WHO Global Comparable Estimates: (2005). Global Infobase data for saving lives; <https://apps.who.int/infobase/Index.aspx>.
8. Nwachukwu Daniel C, Uchenna Francis Agu and Nkiru Zuada Nwachukwu. "Pattern of renal impairment among hypertensive subjects in Umuahia, South East, Nigeria." *International Journal of Medicine and Medical Sciences* 8.5 (2016): 45-50. <https://doi.org/10.5897/IJMMS2016.1229>
9. Akarolo-Anthony SN, Willett WC, Spiegelman D, Adebamowo CA. (2014). Obesity epidemic has emerged among Nigerians. *BioMedical Central Public Health*. 14:455.
10. Amole IO, OlaOlorun AD, Odeigah LO, Adesina SA. (2011). The prevalence of abdominal obesity and hypertension amongst adults in Ogbomosho, Nigeria. *African Journal of Primary Health Care. Family Medicine*. 3(1).
11. Ulasi II, Ijoma CK, Onodugo OD. (2010). A community-based study of hypertension and cardio-metabolic syndrome in semi-urban and rural communities in Nigeria. *Bio-Med Central of Health Service and Research*. 19(10):71.

12. World Health Organisation. (2002). The world health report: Reducing risks, promoting healthy lifestyles. Geneva.
13. Jorgensen AB, Frikke-schmidt R, West AS, Grand P, Nordestgaard BG, Tybjaerg-Hansen A (2013). "Genetically elevated non-fasting triglyceride and calculated cholesterol as a casual risk factor for myocardial infarction". *European Heart Journal*. 34(24):1826-1833.
14. Rifai N, Warnick GR. (2006). Lipids Lipoproteins, Apolipoproteins and other Cardiovascular Risk Factors. In: Burtis CA, Ashwood ER, Bruns DE. *Tietz Text Book of Clinical Chemistry*, 4<sup>th</sup> edition vol 3. Hsevier Saunders. 26:903-981.
15. Young DS, Bermes EW. (2006). Preanalytical variables and biological variation. In: Burtis CA, Ashwood ER, Bruns DE. *Tietz Text Book of Clinical Chemistry*, 4<sup>th</sup> edition vol 2. Hsevier Saunders. 17:449-473.
16. Finkelstein E, Fiebelkorn I, Wang G (2003). National medical spending attributable to overweight and obesity: how much and who is paying. *Health aff. (millwood)*. 3:219-26.
17. Flier JS, Flier ME, (2005). Obesity. In: kasper DL. *Harrison's principle of internal medicine*, 16th edition USA, McGraw-Hill Companies. 64:422-430.
18. Powell DW. (2000). In: Goldman L Bennett JL, *Cecil Textbook of Medicine*, 21<sup>st</sup>ed vol.1 W.D Sauders company. 228-1157.
19. Allen CC, Poon LS, Chan CS, Richmond W, Fu PC. (1974). Enzymatic determination of total Serum Cholesterol. *Clinical chemistry*. 20:470-475.
20. Trinder P (1969). Determination of Glucose in Blood Using Glucose Oxidase with an Alternative Oxygen Acceptor. *Annals of clinical biochemistry*. 6-24.
21. Friedwald WT, Levy RI, Fredrickson DS (1972). Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clinical Chemistry*. 18 (6):499-502.
22. Ugwuja E et al (2013). Overweight and obesity, lipid profile and athrogenic indices among civil servants in abakiliki, south east Nigeria. *Annals of medical and health sciences research*. 3(1):13-18.
23. Al-shareefi AN, et al (2015). Assessment of insulin resistant and related cardio-metabolic factors in overweight-obese women presented with missed abortion, Baghdad, Iraq. *Saudi journal of obesity*. 3:66-77.

24. Gupta M, Mukherjee R. (2018) study on the comparative study of serum leptin and lipid profile among non-diabetic obese subjects and type 2 diabetes patients due to obesity. *Biochemistry Indian Journal*. 12(3):132.
25. Haddad NIA, Nori E and Hamza SA (2017). Serum visfatin and chemerin levels in Iraqi diabetics and obese individuals. *RJPBCS*. 8(6):356-64.
26. Zavorini I, Dall'aglio E, Alpi O, et al (1985). Evidence for an independent relationship between plasma insulin and concentration of high density lipoprotein cholesterol and triglyceride. *Atherosclerosis*. 55:259-66.
27. Varbo A, Freiberg JJ, Nordestgaard BG (2018). "Remnant cholesterol and myocardial infarction in normal weight, overweight, and obese individuals from Copenhagen General Population Study". *Clinical Chemistry*. 64(1):219-230.
28. Varbo A, Benn M, Nordestgaard BG. Remnant cholesterol as a cause of ischemic heart disease: evidence, definition, measurement, atherogenicity, high risk patients, and present and future treatment. *Pharmacol Ther*. 2014 Mar;141(3):358-67. doi: 10.1016/j.pharmthera.2013.11.008. Epub 2013 Nov 26. PMID: 24287311.
29. Nakajima K, Nakano T, Tanaka A (2006). The Oxidative Modification hypothesis of Atherosclerosis: the comparison of atherogenic effects on oxidized LDL and remnant lipoprotein in the plasma. *Clinical Chemistry Acta*. 367:37-47.
30. Zheng et al. (2020). Prevalence and epidemiological determinants of metabolically obese but normal-weight in Chinese population. *BMC Public Health*. 20:487.
31. Nnamdi J. J. Chinonso<sup>1\*</sup>, U. K. Amah, J. A. Onuegbu, H. C. Okpara, J. E. Okwara, J. M. Olisekodiaka, R. A. Analike and E. C. Izuchukwu (2021). *Asian Journal of Cardiology Research*, 5(3): 42-49, 2021; Article no.AJCR.75546.
32. Xie et al (2021). Remnant Cholesterol is an Independent Predictor of New-Onset Diabetes: A Single-Center Cohort Study *Diabetes, Metabolic Syndrome and Obesity: Targets and Therapy* 2021:14 4735–4745.
33. Wang Kexin<sup>†</sup>, Ding Yaodong<sup>†</sup>, Gao Wen, Wang Rui, Yang Jiaxin<sup>1</sup>, Liu Xiaoli, Shen Hua and Ge Hailong<sup>\*</sup> (2021). Association of Increased Remnant Cholesterol and the Risk of Coronary Artery Disease: A Retrospective Study. *Front. Cardiovasc. Med*. 8:740596. doi: 10.3389/fcvm.2021.740596.
34. Castañer O, Pintó X, Subirana I, Amor AJ, Ros E, Hernáez Á, et al. Remnant Cholesterol, Not LDL cholesterol, is associated with incident cardiovascular disease. *J Am Coll Cardiol*. (2020) 76:2712–24. doi: 10.1016/j.jacc.2020.10.008.

35. Langsted A, Madsen CM, Nordestgaard BG. Contribution of remnant cholesterol to cardiovascular risk. *J Intern Med.* (2020) 288:116–27. doi: 10.1111/joim.13059.
36. Li W, Huang Z, Fang W, Wang X, Cai Z, Chen G, Wu W, Chen Z, Wu S, Chen Y. Remnant Cholesterol Variability and Incident Ischemic Stroke in the General Population. *Stroke.* 2022 Jun;53(6):1934-1941. doi: 10.1161/STROKEAHA.121.037756. Epub 2022 May 11. PMID: 35543132.
37. Amira CO, Sokunbi DOB, Dolapo D, Sokunbi A. (2011). Prevalence of obesity, overweight and proteinuria in an urban community in South West Nigeria. *Nigerian Medical Journal.* 52(2):110–113.
38. Amodu PH, Mba IO, Lawson L. (2005). Prevalence of obesity and dyslipidemia in hypertensive in Abuja Nigeria. *Scandinavia Journal of Clinical Laboratory Investigation.* 240:14-7.
39. Babu SV, Jagadeesan AR, Ramalingam J, (2017). A Comparative Study of Lipid Profile in Obese and Non-obese Men attending Master Health Checkup. *Indian Journal of Medical Biochemistry.* 21(2):73-75.
40. Copinschi G, Leproult R, Spiegel K (2014). "The important role of sleep in metabolism". *Front Hormonal Research.* 42: 59–72.
41. Criqui MH, Golom BA (2013). Epidemiologic aspects of lipid abnormalities. *American Journal of Medicine.* 105(Suppl 1A):48S–57S.
42. Dallinga-Thie GM, Kroon J, Boren J, Chapman MJ (2016). "Triglyceride-rich lipoproteins and Remnants: Targets for Therapy?". *Current Cardiology Reports.* 18(7):67.
43. Denke, M.A., C.T. Sempos, and S.M. Grundy, (1993). Excess body weight. An underrecognized contributor to high blood cholesterol levels in white American men. *Arch International Medicine.* 153(9): p. 1093-103.
44. Kinosian B, Glick H, Garland G. (2019) Cholesterol and coronary heart disease: predicting risks by levels and ratios. *Annals of Internal Medicine.* 121: 641–647.
45. Keaven M. Anderson, Patricia M. Odell, Peter W.F. Wilson, William B. Kannel (1991). Cardiovascular disease risk profiles.. *American Heart Journal*, Volume 121, Issue 1, Part 2, Pages 293-298, ISSN 0002-8703, [https://doi.org/10.1016/0002-8703\(91\)90861-B](https://doi.org/10.1016/0002-8703(91)90861-B).
46. Lopez JP, Juan B, Sandra M, Rueda Q, Cristina LL, Camilo F (2018). Obesity and preeclampsia; common pathophysiological mechanism. *Frontiers in physiology.* 9-1838

47. Mach F, Baigent C, Catapano A, et al. (2019). ESC/EAS Guidelines for the management of dyslipidaemias: lipid modification to reduce cardiovascular risk: The Task Force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS). *European Heart Journal*. 41:1,111-188

48. Manninen V, Tenkanen L, Koskinen P, et al. (2014). Joint effects of serum triglyceride and LDL cholesterol and HDL cholesterol concentrations on coronary heart disease risk in the Helsinki Heart Study. Implications for treatment. *Circulation*. 85:37–45.

49. Masuoka H, Kamei S, wagayama H, Ozaki M, Kawasaki A, Tanaka T, Kitamura M, Katoh S, Shintani U, Misaki M, Sugawa M, Ito M, Nakano T (2000). "Association of remnant-like particle cholesterol with coronary artery disease in patients with normal cholesterol total cholesterol levels". *American Heart Journal* 139(2 pt 1):305-310.

50. Mora S (2016). "Nonfasting for Routine Lipid Testing: From Evidence to Action". *Journal of American Medical Association Internal Medicine*. 176 (7): 1005–6.

51. Moraes J.C, Coope, A, Morari J, Cintra D.E, Roman E.A, Pauli J.R, Romanatto T, Carnevalheira J.B, Oliveira A.L, Saad M.J. et al. (2009). High-fat diet induces apoptosis of hypothalamic neurons. *PLoS ONE*, 4:e504.

**Table 1** Anthropometric maker and indices of obesity. it highlights the BMI, WHR, AGE, DBP and SBP (mean±sd)

GROUPS	SBP (mmHg)	DBP (mmHg)	BMI (Kg/m <sup>2</sup> )	WHR	AGE
NORMAL WEIGHT (A)	115.28±11.89	75.59±10.03	21.75±1.82	0.85±0.05	36.05±1.5
OVER WEIGHT (B)	118.92±25.02	78.40±9.55	27.11±1.02	0.86±0.07	36.54±0.7
OBESE (C)	127.00±10.86	85.64±12.25	33.85±4.51	0.96±0.10	37.85±2.1
f-test	3.310	6.246	123.387	18.038	150.044
p-value	0.042*	0.003*	<0.001*	<0.001*	0.556
A vs B	1.000	1.000	<0.001*	1.000	0.234
A vs C	0.040*	0.003*	<0.001*	<0.001*	0.083
B vs C	0.287	0.056	<0.001*	<0.001*	1.000

**Table 2** levels of fasting HDL-C, LDL-C, VLDL-C, TG, AND TC in normal weight, over weight and obese groups (mean±sd)

GROUPS	F-HDL-C (mmol/L)	F-LDL-C (mmol/L)	F-VLDL-C (mmol/L)	F-TG (mmol/L)	F-TC (mmol/L)
NORMAL WEIGHT (A)	1.00±0.21	1.77±0.67	0.47±0.22	1.09±0.51	3.39±0.66
OVER WEIGHT (B)	0.95±0.09	1.52±0.34	0.76±0.31	1.72±0.60	3.47±0.42
OBESE (C)	1.00±0.17	1.84±0.38	0.82±0.23	1.85±0.59	3.45±0.53
f-test	0.816	3.006	9.391	8.638	0.135
p-value	0.446	0.055	<0.001*	<0.001*	0.874
A vs B	0.732	0.201	<0.001*	<0.001*	1.000
A vs C	1.000	1.000	<0.001*	<0.001*	1.000
B vs C	0.906	0.067	0.019*	0.030*	1.000

**Table 3** levels of non-fasting HDL-C, LDL-C, VLDL-C, TG AND TC in normal weight, over weight and obese groups (mean±sd)

GROUPS	NF-HDL-C (mmol/L)	NF-LDL-C (mmol/L)	NF-VLDL-C (mmol/L)	NF-TG (mmol/L)	NF-TC (mmol/L)
NORMAL WEIGHT (A)	1.05±0.27	1.87±0.68	0.44±0.23	1.02±0.52	3.42±0.80
OVER WEIGHT (B)	1.08±0.30	1.56±0.47	0.66±0.35	1.67±0.93	3.32±0.47
OBESE (C)	1.07±0.23	1.95±0.58	0.62±0.22	1.37±0.53	3.66±0.75
f-test	0.088	3.167	5.031	6.023	1.625
p-value	0.916	0.048*	0.009*	0.004*	0.204
A vs B	1.000	0.176	0.014*	0.003*	1.000
A vs C	1.000	1.000	0.049*	0.182	0.253

B vs C	1.000	0.058	1.000	0.418	0.614
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**Table 4** levels of RC, AND FBS IN normal weight, over weight and obese groups (MEAN±SD)

GROUPS	RC(mmol/l)	FBS (mmol/L)
NORMAL WEIGHT (A)	0.50±0.28	3.46±0.79
OVER WEIGHT (B)	0.69±0.46	4.46±1.82
OBESE (C)	0.72±0.40	4.94±1.26
f-test	2.853	8.740
p-value	0.064	<0.001*
A vs B	0.199	0.021*
A vs C	0.095	<0.001*
B vs C	1.000	0.647

**Table 5** correlation of BMI with Lipid Profile, AND RC In Normal weight, Overweight and Obese individuals

PARAMETRS	NORNAL WEIGHT		OVERWEIGHT		OBESE	
	r	p-value	r	p-value	r	p-value
BMI vs F-HDL	-0.202	0.293	0.083	0.693	0.217	0.298
BMI vs F-LDL	-0.041	0.831	-0.108	0.607	0.089	0.672
BMI vs F-VLDL	-0.087	0.654	0.223	0.285	0.085	0.685
BMI vs F-TG	-0.134	0.490	0.024	0.909	0.031	0.882
BMI vs F-TC	-0.181	0.348	-0.141	0.502	0.141	0.503
BMI vs FBS	-0.103	0.593	0.010	0.962	0.021	0.919

BMI vs RC	-0.002	0.993	-0.312	0.129	-0.074	0.726
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\*\* Correlation is significant at 0.01 level (2tailed).

\* Correlation is significant at 0.05 level (2tailed)

UNDER PEER REVIEW