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Serum levels of adiponectin, lipid and plasma glucose in normal, overweight and obese individuals

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Abstract

Objectives: The levels of serum adiponectin, lipids and lipoproteins, and plasma glucose were compared in normal, overweight and obese adults residing in South-Eastern Nigeria.

Method: 168 subjects comprising of 56 each of normal weight, overweight and obese subjects participated in this study. Participant's weight and height were measured, recorded and Body Mass Index (BMI) was calculated. Fasting plasma glucose (FPG), serum total cholesterol (TC), triglycerides (TG) were assayed using colorimetric methods and adiponectin by ELISA technique. Low density lipoprotein cholesterol (LDL-C) and very low density lipoprotein cholesterol (VLDL-C) were calculated using the Friedewald'sformula.

*Results***:** The mean BMI observed in obese subjects (34.92±5.60) was significantly higher than the corresponding values in overweight (27.50±1.32) and control subjects (22.24±1.58) while the mean BMI in overweight subjects (27.50±1.32) was significantly higher than the mean value in controls (22.24±1.58). FPG, TC, TG, LDL-C and VLDL-C concentrations were significantly higher in obese group (5.33±0.77, 4.98±0.70, 1.26±0.30, 3.08±0.64 and 0.58±0.14) than in controls (4.45±0.57, 4.46±0.54, 1.03±0.26, 2.64±0.48, 0.47±0.12) respectively. A positive correlation was observed between the mean BMI of the subjects and their mean FPG, TC, TG, LDL-C and VLDL-C concentrations (r = 0.395, 0.257, 0.188, 0.231, 0.196; p = 0.0001, 0001, 0.015, 0.003, 0.011) respectively. There was no significant correlation between BMI and HDL-C levels (r = -0.073; p = 0.345). A negative correlation was however seen between adiponectin levels and BMI (r = -0.231, p = 0.003); and between adiponectinand fasting FPG, TC, TG and VLDL-C (r = -0.309, -0.153, -0.269, -0.274; p = 0.0001, 0.047, 0.0001, 0.0001) respectively.

Conclusion: The results obtained from this study suggest that with increase in body weight (estimated by body mass index), glucose levels and lipid parameters increased, while adiponectin levels decreased. It also illustrated that, with increase in adiponectin levels, certain lipids (TC, TG, VLDL-C) and glucose decreased. This infers that body mass index as well as adiponectin levels, are associated with glucose and lipid homeostasis.

However, further experimental studies are needed to further create enlightenment on the physiological and biochemical changes that take place in obesity and related health conditions.

Keywords: *Adiponectin, Adiposity, Body mass index, Glucose, Lipids, Obesity*

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Conclusion: The results obtained from this study suggest that with increase in body weight (estimated by body mass index), glucose levels and lipid parameters increased, while adiponectin levels decreased. It also illustrated that, with increase in adiponectin levels, certain lipids (TC, TG, VLDL-C) and glucose decreased. This infers that body mass index as well as adiponectin levels, are associated with glucose and lipid homeostasis.

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Introduction

Overweight and obesity are defined as abnormal or excessive fat accumulation (resulting from an imbalance between food intake and energy expenditure) that presents a risk to health [1]. A body mass index over 25 is considered overweight, and over 30 is obese. The issue has grown to epidemic proportions, with over 4million people dying each year as a result of being overweight or obese in 2017, according to the global burden of disease [1]. Body Mass Index can be an indicator of high body fatness, and may be associated with changes in lipid and lipoprotein and glucose levels. BMI is a person's weight in kilograms divided by the square of height in metres [2]. It is an inexpensive and easy-to-perform method of screening for weight categories [2]. Adipose tissue is now recognized not only as a main site of storage of excess energy derived from food intake but also as an endocrine oragan [3].

Adipose tissue produces a number of bioactive substances, known as adipocytokines or adipokines, which trigger chronic low-grade inflammation and interact with a range of metabolic processes in many different organs. Adipocytokines include inflammatory mediators (interleukin 6, interleukin 8), angiogenic proteins (vascular endothelial growth factor) and metabolic regulators (adiponectin and leptin) [3]. Adipose tissue, previously considered to be no more than an energy-storing depot, has also become a focus of intense scientific interest and is now thought to integrate a wide array of pathological processes, including altered nutrient homeostasis, immune response, blood pressure control, hemostasis, bone mass, and thyroid and reproductive function in both physological and pathological states. Some of these effects are brought about by adipogenic hormones including leptin, adiponectin, and other adipokines [3]. Amongst these adipokines produced by the adipose tissues, adiponectin is a well-known adipose-specific adipokines that is likely to produce insulin-sensitizing effects and may have beneficial effects on lipid metabolism [3]. But, a feedback inhibition in its production may occur during development of fat mass due to increase in the production of other adipocytokines [4].

Overweight and obesity are leading preventable causes of death worldwide, with increasing rates in adults and children [5]. According to WHO, in 2014, 600 million adults (13%) and 42million children under the age of five were obese [6]. In 2016, more than 1.9billion adults were overweight [3]. Overweight and obesity are more common in women than men [6]. Asides the known cardio-metabolic risks, maternal obesity has resulted in higher rates of miscarriages, still births and congenital disorders [7,8]. Obesity is seen as one of the most serious public health problems of the 21st century [9]. In 2013, the American Medical Association classified obesity as a disease [10]. Overweight and obesity are now so common within the world's population that they are beginning to replace under nutrition and infectious diseases as the most significant contributors to ill health [11]. In particular, obesity is associated with diabetes mellitus, coronary heart disease and certain forms of cancer and sleep-breathing disorders [11]. A study carried out in South Eastern Nigeria on overweight and obesity, gave the prevalence to be 7.8% [13]. Overweight and obesity should be regarded as an epidemic that threatens global well being. Previous studies have shown that several factors including age, gender, marital and socio-economic status, occupation, urban residence, dietary intake and physical

activity are associated with overweight and obesity [12,14,15].

The association of low adiponectin levels with obesity, insulin resistance, cardiovascular disease (CAD), and dyslipidemia indicates that this novel protein may be an important new marker of the metabolic syndrome [16]. Large epidemiological studies of adiponectin and obesity are few in populations of African ancestry [17-19]. This noted paucity of data is particularly relevant given that: there is increased prevalence of obesity in Africa, lower adiponectin levels have been reported in African populations compared with European and Asian populations. Remarkably, Africans have been reported to be more insulin resistant, hence making them a high risk group [17-19].

Therefore, the aim of this study was to assess serum adiponectin, lipids and lipoproteins, and plasma glucose levels in normal weight, overweight and obese adults.

Materials and methods

Study design/ Sampling technique

This study was a cross-sectional study in which subjects were recruited by simple random sampling. Each individual was chosen randomly and entirely by chance. Thereafter they were grouped according to their BMI. The weight categories include: underweight, described as BMI of less than 18.5, normal weight, BMI 18.5-24.9, overweight, BMI 25.0 - 29.9, and obese, BMI 30.0 and above [2]. Data were collected through use of questionnaires, anthropometric measurements and biochemical analysis. The informed consent of each participant was obtained before enrolment into the study. They were assured of confidentiality of the information provided.

Study subjects

Sample size was calculated using G*Power software version 3.0.10 (Universität Düsseldorf, Germany). A total of 168 apparently healthy adult subjects of both sexes, who were between the ages of 20-60 years consisting of 56 normal weight, 56 overweight and 56 obese subjects were enrolled in the study. The mean ages of the three weight categories (identified as A, B, C, respectively) were 32.84±10.04, 34.88±10.82 and 36.23±11.74 respectively. They were not on medication and none had a BMI of less than 18.5. Those diagnosed with hypertension, all forms of diabetes, HIV/AIDS, pancreatic disease, heart disease and kidney disorders, pregnant women, excessive alcohol consumers, smokers, professional athletes, those on special diet including persons who abstain from carbohydrate meals were excluded from the study.

Ethical consideration

Ethical approval for the study was obtained from Nnamdi Azikiwe University Teaching Hospital Ethics Committee (NAUTHEC) no. NAUTH/CS/66/ VOL.10/2017/005, Nnewi, Anambra State, Nigeria.

Samples methods of analyses

About 6ml of blood was collected from each subject by venopuncture from the cubital fossa and dispensed into labeled plain test tubes, and for the glucose samples, into fluoride oxalate containers. The samples in the plain containers were allowed to clot and centrifuged at 5000rpm for 5minutes. The sera were transferred into properly labeled plain containers and stored at 4ºC prior to analysis. Serum total cholesterol, and HDL-cholesterol obtained after precipitation of other lipoproteins[20] triglyceride, and plasma glucose were determined using the enzymatic colorimetric methods of Allain *et al.*,[21], Fossati and Prencipe [22], and Trinder[23] respectively. Both LDL-cholesterol and VLDL-cholesterol concentrations were calculated using the Friedewald's formular [24] while quantitative determination of serum adiponectin was carried out using the colorimetric and Human Adiponectin (ADP) ELISA method [25].

Statistical analysis

Statistical analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 25. Analysis of variance (ANOVA) was used for the analysis of means while pairwise T test was used for post-hoc. Pearson's correlation coefficient (r) was used to determine the relationship between variables. Values expressed as mean±sd were regarded as significant at $p<0.05$.

Results are expressed in the tables that follow:

Table 1: The mean height was significantly higher in normal on comparison with obese subjects ($p<0.05$), but did not differ significantly in comparison with overweight subjects ($p>0.05$). The mean height of overweight subjects did not differ significantly on comparison with obese subjects ($p>0.05$). On the other hand, as expected, the weight and BMI were significantly higher in obese subjects on comparison with normal weight and overweight ($p<0.0005$). No significant change was however seen when the mean age of subjects of the various groups were compared with each other ($p>0.05$)

Table 1: Height, weight, Body Mass Index and age in normal weight, over-weight and obese individuals Values are expressed as (mean±SD)

Parameters	Height	Weight	BMI	Age
Normal Weight (A)n = 56	1.70±0.10	64.63±8.67	22.24±1.58	32.84±10.04
Overweight (B)n = 56	1.68±0.10	78.21±10.35	27.50±1.32	34.88±10.82
Obese (C)n = 56	1.65±0.07	95.16±15.53	34.92±5.60	36.23±11.74
F-value	4.635	92.854	191.56	3.504
p-value	0.011	0.000	0.0001	-
A Vs B (p value)	-	p=0.0001	p=0.0001	-
A Vs C (p value)	p=0.010	p=0.0001	p=0.0001	p=0.059
B Vs C (p value)	-	p=0.0001	p=0.0001	-

Table 2: FPG, TCHOL, TG, VLDL-C, HDL-C, LDL-C and adiponectin in normal weight, over-weight and obese individuals (mean±SD)

Parameters	FPG	TC	TG	VLDL-C	HDL-C	LDL-C	ADIPO
Normal Weight (A) n = 56	4.45±0.57	4.46±0.54	1.03±0.26	0.47±0.12	1.34±0.21	2.64±0.48	86.24±6.67
Overweight (B) n = 56	5.00±0.54	4.78±0.68	1.22±0.32	0.56±0.15	1.30±0.21	2.92±0.63	75.98±6.67
Obese (C) n = 56	5.33±0.77	4.98±0.70	1.26±0.30	0.58±0.14	1.30±0.22	3.08±0.64	74.61±8.27
F-value	27.855	9.486	9.741	10.135	0.746	7.803	43.151
p-value	0.0001*	0.0001*	0.0001*	0.0001*	0.476	0.001*	0.0001*
A Vs B	P=0.0001*	P=0.023*	P=0.002*	P=0.002*	P=1.000	P=0.040*	P=0.0001*
A Vs C	P=0.0001*	P=0.0001*	P=0.0001*	P=0.0001*	P=0.775	P=0.0001*	P=0.0001*
B Vs C	P=0.018*	P=0.325	P=1.000	P=1.000	P=1.000	P=0.488	P=0.959

Key: TC – Total Cholesterol, HDL-C – High Density Lipoprotein-Cholesterol, LDL-C – Low Density Lipoprotein-Cholesterol,VLDL-C – Very Low Density Lipoprotein-Cholesterol, FPG- Fasting Plasma Glucose,TG – Triglyceride, ADIPO – Adiponectin

Table 3: Correlation of BMI with FBG, TC, TG, VLDL-C, HDL-C, LDL-C, and adiponectin and correlation of adiponectin with FBG, TC, TG, VLDL-C, HDL-C and LDL-C, in all subjects

		FPG	TC	TG	VLDL-C	HDL-C	LDL-C	ADIP
BMI	R	0.395**	0.257**	0.188*	0.196*	-0.073	0.231**	0.231**
	p-value	0.0001	0.001	0.015	0.011	0.345	0.003	0.003
Adiponectin	R	-0.309**	-0.153*	-0.269**	-0.274**	-0.010	-0.088	
(n=168)	p-value	0.0001	0.047	0.0001	0.0001	0.896	0.259	

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*(*pd"0.05 is considered significant; **pd"0.01)*

Table 2: Mean FPG, TC, TG, LDL-C and VLDL-C concentrations were significantly higher in overweight and obese individuals than in controls ($p<0.05$), but plasma glucose was significantly lower in overweight subjects compared to those that were obese. On the other hand, control subjects showed significantly higher mean adiponectin value on comparison to overweight and obese subjects respectively ($p<0.05$ in each case).

Table 3: Correlation analysis revealed that each of FPG ($r = 0.395$, $p = 0.0001$), TC ($r = 0.257$, $p=0.001$), TG ($r = 0.188$, $p=0.015$), VLDL-C ($r = 0.196$, $p=0.011$) and LDL-C ($r = 0.231$, $p=0.003$) were weakly but positively correlated with BMI,

while a weak negative correlation was observed between BMI and serum adiponectin (r = -0231, p=0.003). BMI and serum HDL-C were not significantly correlated (r =-0.073, p= 0.345). A weak negative correlation was found to exist between serum adiponectin and FBG (r = -0.309, p =0.0001), TC (r = -0.153, p =0.047), TG (r = -0.269, p =0.0001) and VLDL-C (r = -0.274, p=0.0001) but not with HDL-C (r=-0.010, p= 0.896) and LDL-C (r= -0.088, p=0.259)

Discussion

In this study, levels of serum adiponectin, lipid profile, and plasma glucose in obese, overweight and normal weight adults were assessed. There is paucity of data in this topic in Africa, given the high prevalence of diseases associated with glucose and lipid metabolism [17, 18,19]. . Abnormal weight changes can cause physical, psychosocial and physiological impairments leading to cardiovascular, renal, cerebral and thrombogenic anomalies [26]. These are important contributors to poor health outcomes, particularly for many cases of COVID-19 in African population groups [27]. Eventually, it is the cumulative effects of these anomalies that give rise to the increased morbidity and mortality associated with obesity, hyperglycemia and metabolic syndrome [28].

This study demonstrated significant changes in fasting plasma glucose amongst normal weight, overweight, and obese males and females, with FPG being highest in obese and lowest in normal weight individuals. A positive correlation was observed between the mean BMI levels of the subjects and their mean FPG levels. Insulin is responsible for glucose uptake into cells and tissues to be specific [31].Obesity can cause decreased ability of tissues to respond to insulin action. In obesity, free fatty acids can enter the liver directly through the portal circulation, and increased levels of hepatic free fatty acids induce increased lipid synthesis and gluconeogenesis as well as resistance in the liver [32].

Free fatty acids and various adipokines released from adipose tissue have been involved in abnormal insulin signaling. Schenk et al [33] postulated that fatty acids and their metabolites, such as acyl-coenzyme A, ceramides and diacyglycerol, can impair insulin signaling by promoting protein kinases such as protein kinase C, mitogen-activated protein kinases (MAPK), c-Jun *N*-terminal kinase (JNK), and the inhibitor of nuclear factor kappa-B kinase subunit beta (IKK-β). Saturated fatty acids induce the synthesis of ceramide, and inhibition of ceramide synthesis helps to slow saturated fatty acids-induced insulin resistance [34]. Tumor necrosis factor alpha (TNF-α) also promotes ceramide accrual by activating sphingomyelinase, an enzyme that catalyzes the hydrolysis of sphingomyelin to ceramide [35], and ceramide mediates TNF-α-induced insulin resistance in adipocytes [36].

Plasma ceramide levels are elevated in obese subjects [37] and it contributes to insulin resistance by activating inflammatory mediators, such as TNF-α. Thus, ceramide has been regarded as a mediator linking several metabolic stresses (*i.e.*, TNF-α and saturated fatty acids, but not unsaturated fatty acids) to the induction of insulin resistance [34, 38], although its role in insulin resistance had been described as partly controversial [39]. Obese subjects compared to lean persons seem to have greater whole body free fatty acids rates of appearance in plasma [40], and a sustained reduction in plasma free fatty acids levels after treatment of lipolysis inhibitor appear to be associated with an improvement of insulin sensitivity in diabetic obese subjects [41].

In a nutshell, increased BMI leads to increased circulating free fatty acids and their metabolites which can impair insulin signaling. Hence BMI being positively correlated with FPG. This is in line with what has been observed by other authors [29,30] whose studies also reported that blood glucose levels increased with increase in BMI. There was no significant correlation between BMI and HDL-C. This is in contrast to the work by Nevin and Nurcan [42] who reported that a negative correlation existed between BMI and HDL-C. The difference between these two studies may be due to the ages of the subjects. Whereas Nevin and his colleague worked on university students between the ages of 19 and 23years, our study involved subjects between the ages of 20 and 60years. Aging can possibly affect HDL metabolism.

Deeb *et al* [43] had proposed that HDL metabolism is strongly affected by obesity probably because of the increased number of remnants of chylomicrons and VLDL –C together with impaired lipolysis. The increased number of TG-rich lipoproteins results in increased CETP activity, which exchanges cholesterol esters from HDL for TG from VLDL and LDL [44]. Moreover, lipolysis of these TG-rich HDL-C occurs via hepatic lipase leading to occurrence of small HDL-C with a lowered affinity for apo A-I, which causes dissociation of apo A-I from HDL-C ultimately resulting to lower levels of HDL-C and a reduction in circulating HDL-C

particles with impairment of reversed cholesterol transport (RCT) [43].

This could explain the inverse relationship between HDL and increased BMI (characterized by increased lipolysis) seen in the work by Deeb et al [43]. Other serum lipids (TC, TG, LDL-C, VLDL-C), from this study, were positively correlated with BMI. This agrees with the findings of previous authors [30,10], which suggests that lipid metabolism is affected in obesity and overweight resulting in the increased circulating serum lipids stated. Excess fat accumulation promotes the release of free fatty acids into the circulation from adipocytes [45,32]. Increased delivery of fatty acids to the liver and synthesis of very-low-density lipoprotein cholesterol (VLDL-C) is often caused by uncontrolled fatty acid release from adipose tissue, especially visceral adipose tissue, through lipolysis, a very important contributing factor for obesity-related dyslipidemia. High levels of free fatty acids can decrease mRNA expression or activity of lipoprotein lipase (LPL) in adipose tissue and skeletal muscle, and increased synthesis of VLDL-C in the liver can inhibit lipolysis of chylomicrons, which promotes hypertriglyceridemia [46-48].

Hypertriglyceridemia further triggers a cholesterylester transfer protein-mediated exchange of triglycerides for cholesterol esters between triglyceride-rich lipoproteins (VLDL-C, immediate-density lipoprotein) and lipoproteins which are relatively richer in cholesterol esters [49]. This exchange ultimately leads to the formation of small dense LDL [50,51], eventually, chylomicrons and VLDL–C shrink in diameter during the process of lipolysis to form chylomicron remnants and dense LDL, respectively [52,44,53-55]. Therefore, increased fat deposits characterized by lipolysis could eventually lead to increased circulating lipids as seen in this study and works by previous researchers [30,13]

A negative correlation was demonstrated between adiponectin and BMI, and also between adiponectin and: FPG, TC, TG and VLDL in this study. It is possible that although adiponectin expression is activated during adipogenesis, a feedback inhibition in its production may occur during development of fat mass due to increase in the production of other adipocytokines. In addition, adipocytokines such as TNF-α may decrease adipocyte expression and secretion of adiponectin [4]. It is suggested that with increasing grades of obesity, there may be a decrease in the metabolic functioning of adipocytes, along with hypertrophy and/or aging of these cells [60]. There is another suggestion that adipose tissue within bone marrow is raised during caloric restriction, and therefore contributes to elevated circulating adiponectin in lean subjects [61].

Also studies have shown that adiponectin may stimulate fatty acid oxidation through activation of AMP-activated protein kinase (AMPK) in the liver and skeletal muscles which has been associated with many of the positive effects of adiponectin on lipid metabolism [62]. The negative correlation between adiponectin and FPG in this study might be because adiponectin modulates a number of metabolic processes, one of which is glucose regulation [63]. A study has shown that adiponectin in combination with leptin could completely reverse insulin resistance in mice [64]. It has been demonstrated that the high-molecular weight form of adiponectin could be the most biologically active form with to regards glucose homeostasis [65]. Adiponectin gene is localized at chromosome 3q27, a region highlighted as affecting genetic susceptibility to type 2 diabetes and obesity [66-71]. Supplementation by different forms of adiponectin has been shown to be capable of improving insulin control, blood glucose and triglyceride levels in mouse models [72].

Various researchers have observed that adiponectin may cause glucose flux, decreased gluconeogenesis, increase glucose uptake [73,66,19], lipid catabolism [69], β-oxidation [66], triglyceride clearance [66], protection from endothelial dysfunction (important facet of atherosclerotic formation), insulin sensitivity, weight loss, control of energy metabolism and upregulation of uncoupling proteins [74]. The works by other authors outside this environment [56,57,3,58,59] also suggests an inverse relationship between adiponectin and BMI; and between adiponectin and FPG,TC,TG and VLDL.

Conclusion

The results from this study show that, with increase in body weight (estimated by body mass index), glucose levels and lipid parameters increased, while adiponectin levels decreased. They also illustrated that, with increase in adiponectin levels, various lipids (TC, TG, VLDL-C) and glucose decreased. This suggests that body mass index as well as adiponectin levels, are associated with glucose and lipid homeostasis.

However, further experimental studies on the effects of body weight changes on adiponectin,

lipid and glucose parameters are needed to further create enlightenment on the physiological and biochemical changes that take place in obesity and related health conditions. This is pertinent because, with about 21 million overweight and 12 million obese persons in Nigeria in 2020, Nigeria possibly represents the most affected country in Africa. Low levels of physical activity, urban drifts, unhealthy diets, socio-economic changes, and psychosocial factors are largely responsible for this high burden [75]. Obesity and overweight are strongly linked with several cardio-metabolic disorders including high blood pressure, high blood glucose, insulin resistance, high blood cholesterol, coronary heart disease, stroke and cancers [76]. According to the results of this research, increase in body weight could elevate lipid and glucose parameters (TC, TG, LDL, VLDL and FPG), while seeming to suppress adiponectin levels. On the other hand, high adiponectin levels seemed to suppress lipid and glucose parameters (hence the negative correlation between them). Therefore, special attention should be given to preventive measures regarding overweight and obesity. Foods containing adiponectin (eg: avocados, nuts, olives, olive oil, safflower oil etc) should be consumed regularly. Healthy diets and lifestyles should be cultivated to maintain normal weights in individuals. Finally, it is recommended that adiponectin, lipid profile and glucose levels be measured routinely in medical laboratories and abnormal levels be considered as possible risk factors for obesity-related diseases.

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