

Burden of Dyslipidemia and Metabolic Syndrome among Indigenous Black African Secondary School Students in Lagos, Nigeria

Bamgboye M. Afolabi¹, Susan J. Holdbrooke¹

¹ Nigerian Institute of Medical Research

Funding: Nigerian Institute of Medical Research supported this article.

Potential competing interests: No potential competing interests to declare.

Abstract

Introduction: Metabolic syndrome (MetS) is a group of risk factors which together increase the chance of heart disease, stroke, and type 2 diabetes. In MetS, especially among overweight and obese people, blood pressure and blood glucose are elevated, excess body fat accumulates abdominally, and there is dyslipidemia – notably low level of high-density lipoprotein (HDL) cholesterol and high level of triglycerides. **Objectives:** This study has three objectives: (i) determine the prevalence of dysglycemia and of dyslipidaemia among Nigerian adolescents relative to gender (boys and girls), stage of adolescence (early and late) and BMI-for-age percentile (lean and overweight/obese) (ii) to quantify the burden of lipid abnormalities (excessive Total cholesterol (TC), Triglyceride (TG), High-density lipoprotein cholesterol (HDL) and Low-density lipoprotein (LDL) cholesterol) among Nigerian adolescents, which have appeared as substantial covariates of coronary heart disease (CHD) and (iii). To evaluate the association of overweight/obesity, dysglycemia and dyslipidaemia with MetS at ($\geq 90^{\text{th}}$ percentile). **Population and Methods:** An institution-based, cross-sectional, descriptive, epidemiological study was conducted from October 2019 to March 2020. A multi-stage sampling approach was used to recruit 650 students aged 10-19 years in secondary schools within Lagos State of Nigeria. Data were collected using a structured questionnaire and anthropometric measurements. Fasting venous blood samples were collected for plasma glucose and lipid profile analysis. Systolic and diastolic blood pressures were measured. A p-value < 0.05 was taken as statistically significant. Twenty-six questionnaires were discarded due to incomplete data. **Results:** A total of 650 adolescents aged between 10 and 19 years were included but 624 were analyzed with 26 discarded due to incomplete data. The overall means (\pm sd) of age was 14.7 ± 2.2 yrs. with no significant difference between boys (14.8 ± 2.2) yrs. and girls (14.7 ± 2.1) yrs.). Girls were significantly heavier (t-test = -2.24, P-value = 0.03) than boys (19.5 ± 4.1 vs 18.7 ± 4.5 kg/m²). The overall prevalence of hyperglycemia was 11.7%. The mean Systolic blood pressure (SBP) of overweight/obese subjects (113.6 ± 11.9 mm Hg) was significantly higher (t-test = -8.81, P-value < 0.00001) than that of lean subjects. Approximately 19% and 12% of the study subjects had impaired and diabetic fasting blood glucose (FBG), especially females, those in early-stage adolescence and the overweight subjects. The median lipid levels were 199.4 mg/dL for TC, 180.8 mg/dL for TG, 55.9 mg/dL for HDL and 289.6 mg/dL for LDL respectively. Only 53 (8.3%) subjects had abnormal levels of combined TC, TG, HDL, and LDL. Boys were 1.59 more likely to have dyslipidemia compared to girls ($\chi^2 = 2.66$, P-value = 0.10, OR = 1.59, 95% CI = 0.92, 2.76) and overweight/obese subjects were 1.53 more likely to develop dyslipidemia compared to lean subjects ($\chi^2 = 0.86$, P-value = 0.35, OR = 1.53, 95% CI = 0.62, 3.77). Overweight with dyslipidemia were significantly younger (t-test = 2.54, P-value = 0.04). The overall prevalence of metabolic syndrome (MetS) was 8.3% with roughly 15%, 44%, 32% and 9% of the study subjects having 0, 1, 2 or ≥ 3 risk factors for MetS. **Conclusion:** A higher proportion of male, mid-adolescent, and overweight students had a minimum of 3 risk factors for MetS. This is likely to impose a high burden on future health. Preventing the increasing burden of lipid abnormalities among Nigerian adolescents is essential. The extent to which the problem might affect other areas of Nigeria needs investigation. Multivariate regression analysis shows that the overall relationship between MetS $\geq 90^{\text{th}}$ percentile and five predictor variables – FBG, TC, TG, HDL, and BMI-for-age, was significant (P-value < 0.00001) and all of them contributed to the observed MetS $\geq 90^{\text{th}}$ percentile among the study subjects.

Bamgboye M. Afolabi^{1,2,3}, and Susan J. Holdbrooke¹

¹ Nigerian Institute of Medical Research, 6, Edmond Crescent, Yaba, Lagos, Nigeria.

² Health, Environment and Development Foundation, Lagos, Nigeria.

³ AfriHealth Optonet Association, Abuja

***Correspondence:**

Dr. Bamgboye M. Afolabi

Biochemistry and Nutrition Department

Nigerian Institute of Medical Research

6, Edmond Crescent,

Yaba, 101245, Lagos, Nigeria

Tel: +234 (0) 808 008 1946

Email: bmafolabi@gmail.com

www.heendef.org

Keywords: Adolescents, Dyslipidemia, Fasting Plasma glucose, Nigeria, Metabolic syndrome, Overweight/Obese, Sub-Saharan Africa.

Introduction

The alarming rise in the number of children and adolescents who are developing symptomatic diabetes and demonstrable precursors of cardiovascular disease calls for urgent action. Studies from the developed world have established that obesity among adolescents is associated with unfavorable lipid profiles [1]. The increasing frequency of obesity is becoming a vital public health issue. Globally in 2020, 38.9 million were overweight or obese [2]. Though the prevalence of child and adolescent obesity has flattened at elevated levels in most developed countries, it is rising in several low- and middle-income countries [3]. Health-related quality of life is reduced in those with obesity and comorbidities of obesity, such as type 2 diabetes mellitus, fatty liver disease and depression, are more probable in adolescents, especially among the obese [3]. The prevalence of obesity among adults in the 10 high-burden African countries ranges from 13.6% to 31%, while in children and adolescents it ranges from 5% to 16.5% [4]. There is a variation in obesity rates across Africa, though reports indicate that obesity on the continent is increasing [5]. In 2016, the average prevalence of obesity among boys and girls in Africa were 3.2% and 4.7% respectively [5]. Obesity in childhood and adolescence is a major risk factor for the development of obesity, premature death, and disability in adulthood. It is also a risk factor for breathing difficulties, hypertension, early markers of cardiovascular disease, insulin resistance and psychological consequences [6]. It has been earlier recognized that childhood and adolescent obesity is associated with unfavorable lipid profile, suggesting that obese children should be screened for hypercholesterolemia [7]. Lipids, circulating in the blood system as lipoproteins, are made up of unesterified cholesterol, triglycerides, phospholipids, and protein. Abnormalities in plasma lipids (the unfavorable lipid profile), such as (i) high total cholesterol (TC), (ii) high low-density lipoprotein cholesterol (LDL-C) (iii) high non-high-density lipoprotein cholesterol (non-HDL-C) (iv) high triglycerides and (v) low HDL-C, referred to as dyslipidemia, contributes to the occurrence of serious illnesses related to cardiovascular diseases (CVDs) [8]. Elevated serum lipid concentrations, including cholesterol and triglycerides, termed as hyperlipidemia, lead to a higher risk of developing atherosclerotic cardiovascular disease (ACVD). Potential reasons for the increase in CVD rates include lifestyle changes associated with urbanization and epidemiologic and nutritional transitions accompanying economic development now [9][10][11]. The etiology of dyslipidemia may be multifactorial, including sedentary life, dietary (excessive dietary intake of saturated and trans fats - an important contributor to elevated LDL and excessive intake of refined carbohydrates and simple sugars raises TG); secondary causes (exogenous e.g., alcohol, obesity, drug therapy; endocrine/metabolic e.g., hypothyroidism/hypopituitarism, diabetes mellitus types 1 and 2, Polycystic ovarian syndrome, lipodystrophy; renal e.g., nephrotic syndrome, chronic renal disease; Infections e.g., HIV, Hepatitis; hepatic e.g., obstructive liver disease, biliary cirrhosis; inflammatory disease e.g., Systemic lupus erythematosus; Storage disease e.g., Glycogen storage disease, and others such as idiopathic

hypercalcemia Klinefelter syndrome or Kawasaki disease), and genetic causes (monogenetic conditions such as familial hypercholesterolemia or polygenic hypercholesterolemia [12]. Thus, abnormal lipid profile in adolescents with dyslipidemia is like what is seen in adults with premature CVD. Elevated LDL cholesterol levels (hypercholesterolemia) are relatively common in populations consuming a western diet.[13]. Hypercholesterolemia is associated with the development of atherosclerosis, coronary heart disease (CHD), myocardial infarction (MI), and increased mortality [14] which are relatively common in the West[15] but not well studied in Africa. Closely associated with obesity and dyslipidemia (including hypercholesterolemia) is MetS, a subject that has received much attention in recent times, due to increasing awareness of its association with cardiovascular morbidity and mortality. However, it is a concept that dates back to over 5 decades now. Its existence was first observed as clustering of hypertension, hyperglycemia, and gout as described by Kylin in the 1920s. Later, Jean Vague in 1947 noted its association with android obesity [16][17][18]. Metabolic syndrome is an important public health problem that causes severe mortality and morbidity and is increasing all over the world with the realization that metabolic abnormalities such as insulin resistance (IR), obesity, dyslipidemia, and hypertension (HT) cluster in some patients [19] strongly associated with an increased risk of developing atherosclerotic cardiovascular disease (ACVD) [20]. Although MetS is often known as a health problem in adulthood, it has recently emerged as an essential problem in childhood and adolescence. It is known that an increasing number of children and adolescents are affected by MetS [21][22] though a definite consensus has yet to be reached in the definition and cutoff points for considered risk factors in childhood/adolescent MetS as in adults [23][24]. Studies have associated obesity [25][26] and dyslipidemia [27][28] as key features of MetS and its risk factors for CVD and type 2 diabetes (T2DM). There is paucity of data on lipid profiles and on MetS among adolescent Nigerian. Isolated studies have been conducted on some lipid profiles among Nigerian adolescents [29][30][31] emphasizing mainly those with diabetes or the relationship between body mass index and lipid profiles. However, most of these studies did not consider the Body Mass Index-for-age percentile of their study populations. Certain cardiovascular risk factor such as obesity, DM, and HT as well as some severe comorbidities and complications are associated with dyslipidaemia [32] requiring more attention to be paid to this increasing health burden among the African adolescents. Dyslipidaemia and associated disorders diseases are once said to be rare in children and adolescents in Africa [33] and up till now, there is a shortage of information on this topic. There is also scarcity of data on the triad of obesity, dyslipidaemia, and MetS among Nigeria adolescents and thus the burden of this syndrome is relatively unknown, leaving a vacuum in its clinical management. It is important to study MetS in adolescents to reduce adult morbidity and mortality due to its complications, reduce CVD, atherosclerosis and other co morbidities and to increase life-expectancy. The objectives of the present study were to determine the prevalence of (i) overweight/obesity and (ii) dyslipidaemia among Nigerian adolescents relative to gender and BMI-for-age percentile. In addition, this study determined, the association of overweight, obesity and dyslipidaemia with MetS at (≥ 90 th percentile). Early onset of dyslipidemia may affect the later life or overall life course of an individual, probably leading to other chronic conditions at such a young age which might hamper adulthood, highlighting how important it is to study this condition even among individuals as young as a 10-19-year-olds.

Populations and Methods

The present descriptive, epidemiological study was conducted by the Department of Biochemistry and Nutrition of the Nigerian Institute of Medical Research. Primary data were collected from recruited secondary school students, aged 10-19 years in Lagos State, Nigeria. Recruitment into the study, which included 624 participants (241 boys and 383 girls) was from October 2019 to March 2020. Written informed consents were obtained from parents and each participating student gave verbal assent. The study protocol was approved by the Institutional Review Board of the Nigerian Institute of Medical Research (NIMR IRB (IRB/18/062)) and the study was conducted in accordance with the Declaration of Helsinki (2000).

Study site

This descriptive, non-experimental, cross-sectional study was conducted in Lagos State, Southwest Nigeria. Lagos, the most economically important city in the country, is also the most populous, the second fastest growing city in Africa. The city had 616 registered public (372, 60.4%) and private (244, 39.6%) secondary schools in the selected Local Government Areas (LGAs) of study [34].

Study population

The target population was students in government-approved Secondary School Lagos State. The WHO defined the age range for adolescents to be between 10-19 years [35].

Sample size

The sample size was calculated for a single population with 95% confidence interval, 54% [36] proportion, a margin of error 5%, and allowing for 12% non-response. To ensure that results of the study are representative of all Nigerian ethnic groups resident in Lagos State, the sample size would then be 650 students to cater for attrition and missing data.

Sampling technique and procedure

Lagos State comprises of 3 Senatorial Districts (SDs) – Lagos East with 5 LGAs, Lagos Central with 5 LGAs and Lagos West with 10 LGA. Using simple random sampling technique (SRST), 1 LGA was selected from Lagos East SD (Ikorodu LGA with 222 secondary schools), 1 also from Lagos Central SD (Mainland LGA with 100 secondary schools) SD, and 2 LGAs (Ojo with 247 and Mushin with 47 secondary schools). Using probability proportional to size (PPS), different arms of classes – Year 1, 2 and 3 of Junior Secondary School (JSS) (mainly aged 10-15 years) and Year 1, 2 and 3 of Senior Secondary School (SSS) (mainly age 16-19 years), since there were many arms in either JSS or SSS. Finally, systematic sampling technique was used to select students in selected arms of each class (Figure 1). Thus, selection was stratified by junior and senior school years proportional to school size.

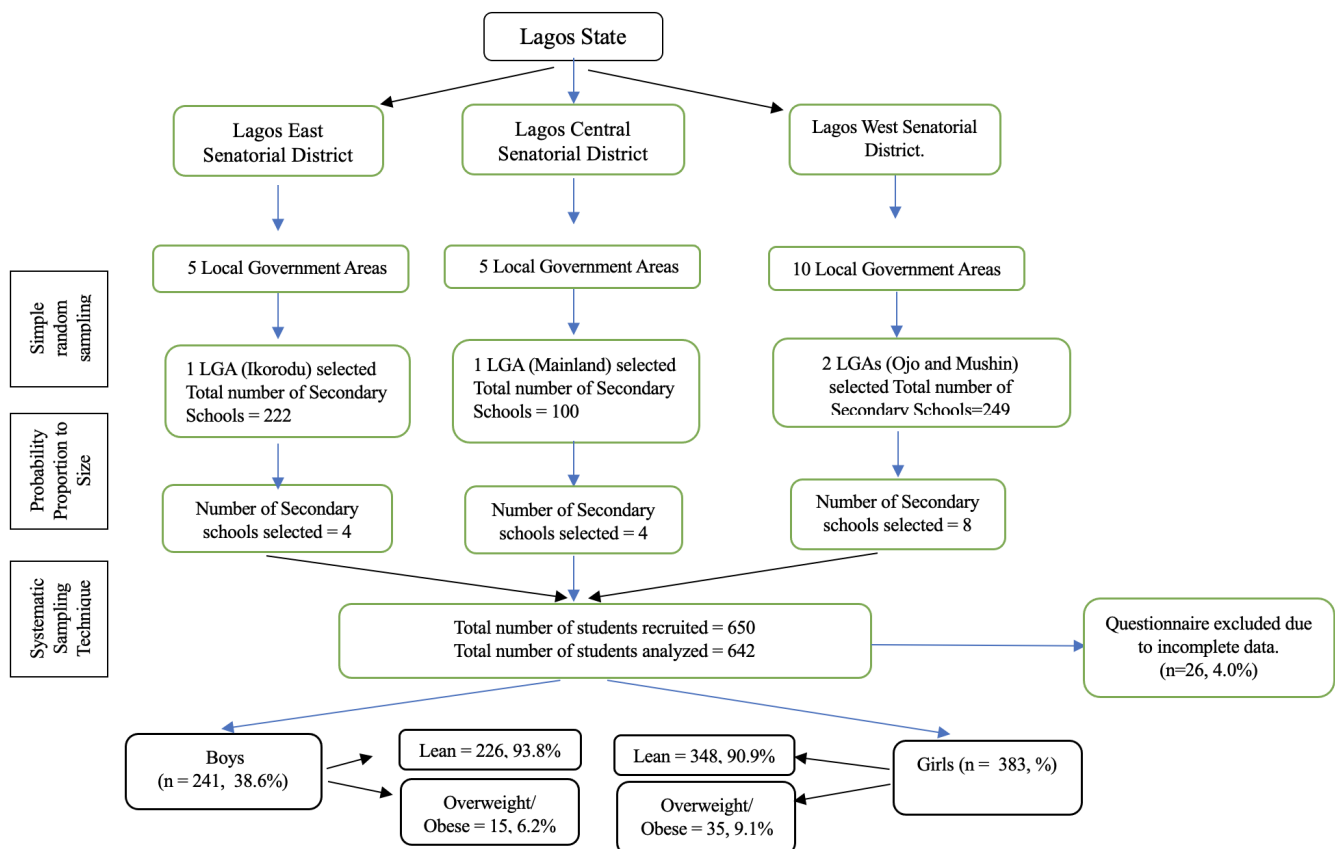


Figure 1. Flowchart of recruitment of study participants

Eligibility criteria

Inclusion criteria. Ten- to 19-year-old secondary school students Black, indigenous Nigerians were recruited into the study. The students must be resident in the community for a minimum of 2 years in the respective Local Government Areas of the study. They must also be regular students at the secondary schools identified for the study. Approval by the State Government Ministry of Education and by the parents were decisive inclusion criteria.

Exclusion criteria: Those on therapeutic diet or drugs, and those on admissions to a health facility in previous 6 month were excluded from the study. Thus, students with diabetes, those taking lipid-lowering medications, or with a history of vascular/liver/renal or other continuing illness were excluded. Pregnancy, suspected pregnancy, breastfeeding, or use of oral contraceptive were also exclusion criteria.

Measurements

Data on socio-demographic and economic characteristics were gathered from both parents and students. Body weight, height, waist, and hip circumferences were measured by trained field workers. Weight was measured with minimal clothing (no shoes) to the nearest 0.1 kg using an electronic scale (FBS machine Model HBF-514C and DP scale HN-283), and height was measured (without participants wearing shoes) to the nearest millimeter using a portable stature meter (SURGILAC). Waist and hip circumferences were also measured to the nearest millimeter over light clothing, waist midway between the lowest rib and the iliac crest, and hip at the widest part of the buttocks. World Health Organization (WHO) AnthroPlus V1.0.4 (Geneva, Switzerland) was used to calculate BMI-for-age and height-for-age percentiles for boys and girls separately, according to WHO [37]. Sex-specific categorization was used for BMI. Cut-offs were available for Nigerian or African for waist circumference were 0.94 m for boys and 0.80 m for girls, no Nigeria- or Africa- specific limits being available for this age group. Blood pressure (upper arm) and pulse rate were measured after 30 min sitting (aneroid sphygmomanometer for a very thin hand {SURGILAC CE 123-HS-20C. [Germany]}, automatic blood pressure monitor for moderate hand {Medical Instrument WUXI, Ltd, EN-BL-8030 [China]} or a mercury sphygmomanometer (long cuff Medical Instrument WUXI, Ltd, EN-BL-8030 [China]} machine for a very big hand). The average of the three measurements was used. Participants were asked to fast overnight, after which 5 ml of venous blood was taken. Fasting blood for glucose was collected into fluoride oxalate tubes while the fasting blood for lipids was collected in Lithium heparin tubes. Both were stored at -20°C before centrifuging for the production of plasma. Randox Glucose-PAP (Randox Laboratories, UK) reagent was used for analyzing FPG and lipid profile (total cholesterol, HDL, low-density lipoprotein (LDL) and triglycerides) were measured using a photo spectrometric analyzer (BioSystems EN ISO 13485 and EN ISO 9001 standards (Barcelona, Spain).

Statistical analysis

Data were presented in the three domains of gender (boys and girls), BMI-for-age percentile (Lean (<85th percentile), overweight/obese (≥85th percentile) and stage of adolescence (early=10-14.9 years of age; late=15-19.9 years of age). Descriptive statistics were performed for all anthropometric and biochemical data, and values were initially reported as mean (±) standard deviation (SD) and 95% CI for the continuous variables. Analyses were conducted using NCSS version 22 (Kaysville, Utah, USA) and Box plot was drawn using STATA version 13.1 (College Station, Texas, USA). Analysis of variance, Chi-square with odds ratios, bivariate, and multivariate logistic regression were performed. An unadjusted p-value <0.05 was considered statistically significant. Kolmogorov-Smirnov Normality test for normality of data distribution for continuous measures was conducted and when the test failed, Mann-Whitney U-test and Kruskal-Wallis one-way ANOVA were used to determine differences between 2 and 3 medians respectively. Independent Student's t-tests were used to identify differences in anthropometric measurements. MetS severity calculator [38], which considers the adolescent's height (cm), weight (kg), sex, serum triglyceride, HDL-C, fasting glucose, systolic blood pressure, and ethnicity (non-Hispanic, Black), was used to calculate continuous metabolic syndrome (cMetS) risk score for early occurrence of MetS.

Definitions

Dyslipidemia was defined as a combination of total cholesterol ≥200 mg/dL, low-density lipoprotein-cholesterol (LDL-C) ≥ 130 mg/dL,

triglycerides (TG) ≥ 130 mg/dL, or high-density lipoprotein cholesterol (HDL-C) < 40 mg/dL [39][40]. The NHLBI criteria specific for children and adolescents were used to identify MetS among participants aged 10 to 19 years [41]. This requires three or more of (i) BMI-for-age of $\geq 95^{\text{th}}$ percentile; fasting plasma levels of (ii) TG ≥ 130 mg/dL; (iii) HDL-cholesterol < 40 mg/dL; (iv) LDL-cholesterol ≥ 130 mg/dL; (v) total cholesterol ≥ 200 mg/dL; (vi) glucose ≥ 100 mg/dL; (vii) pre-hypertension as Systolic/Diastolic BP (SBP/SDP) 120-129/ < 80 mmHg, stage 1 hypertension, BP 130-139/80-89, and stage 2 $\geq 140/90$ mmHg [42]. However, for the purpose of this study, BMI-for-age percentile, fasting plasma levels of glucose, triglycerides, and total cholesterol were the variables taken for the assessment of MetS. Subjects with BMI for age $< 85^{\text{th}}$ and $\geq 85^{\text{th}}$ percentile were classified as lean, overweight/obese respectively using the BMI age chart [43]. Fasting blood glucose (FPG) of < 70 mg/dL, 70- < 100 mg/dL, 100-125.9 mg/dL and ≥ 126 mg/dL were taken as low, normal, impaired, pre-diabetic and diabetic [44].

Other Abbreviations

WHO – World Health Organization; NHLBI – National Heart, Lung, and Blood Institute; NCEP – National Cholesterol Education Program; IDF – International Diabetes Foundation

Results

Anthropometric and clinical characteristics of study subjects (Table 1, Figure 2).

A total of 650 students were recruited into the study but 624 (boys:241, 38.6%; girls: 383, 61.4%) were reported on after data from a negligible 26 (4.0%) students were discarded due to incomplete entry. Overall, 300 (48.1%) and 324 (51.9%) were in early or late adolescence, 574 (92.0%) were lean and 50 (8.0%) were overweight/obese (boys: n=9, 3.7%; girls=21, 5.5%) or obese (boys: n=6, 2.57%; girls=14, 3.7%). There was no significant difference in the means of age (years), weight (kg) and height (m) of boys and girls in the study subjects, though the mean BMI (kg/m^2) of girls (19.5 ± 4.1) was significantly higher (t-test=-2.24, **P-value=0.03**) than that of boys (18.7 ± 4.5). The means of SBP mm Hg (109.6 ± 12.2) of those in late adolescence was substantially higher BMI (t-test=-24.47, **P-value<0.00001**) than those in early adolescence. Students with overweight/obesity (n=50, 8.0%) were significantly younger (t-test = 4.022, **P-value=0.0002**), heavier (t-test=-11.1, **P-value<0.00001**), with significantly higher BMI (kg/m^2) (t-test=-24.47, **P-value<0.00001**), wider WC (cm) (t-test=-8.74, **P-value<0.00001**), higher SBP (t-test=-8.74, **P-value<0.00001**) and higher DBP (t-test=-3.50, **P-value<0.00001**). Figure 2. depicts box plot representing each lipid profile for boys and girls, for early- and late-stage adolescence and for lean and overweight/obese subjects. Girls had a significantly higher LDL (Mann-Whitney U test=-3.03, **P-value<0.002**) than boys, and TG was significantly higher (Mann-Whitney U test =-2.46, **P-value<0.01**) in early compared to late adolescence.

Table 1. Anthropometric and clinical characteristics of the study subjects, 2020.

Variable	Gender				Stage of adolescence			BMI-for-age		
	All (n=624)	Boys (n=241, 38.6%)	Girls (n=383, 61.4%)	t-test (P-value)	Early (n=300, 48.1%)	Late (324, 51.9%)	t-test (P-value)	Lean (n=574, 92.0%)	Overweight/ Obese (n=50, 8.0%)	t-test (P-value)
	Mean (±sd)				Mean (±sd)			Mean (±sd)		
Age (years)	14.7 (2.2)	14.8 (2.2)	14.7 (2.1)	0.56 (0.57)	12.9 (1.3)	16.4 (1.2)	-34.86 (<0.00001)	14.8 (2.1)	13.5 (2.2)	4.02 (0.0002)
Weight (Kg)	47.4 (11.6)	46.5 (12.5)	48.0 (11.0)	-1.53 (0.13)	45.2 (11.6)	49.4 (11.3)	-4.58 (<0.00001)	45.8 (10.1)	65.6 (12.2)	-11.1 (<0.00001)
Height (cm)	156.7 (12.3)	157.7 (13.0)	156.1 (11.9)	1.55 (0.12)	154.2 (13.4)	159.0 (10.8)	-4.90 (<0.00001)	156.9 (10.7)	154.6 (24.3)	0.66 (0.51)
BMI (kg/m ²)	19.2 (4.2)	18.7 (4.5)	19.5 (4.1)	-2.24 (0.03)	-0.21 (1.4)	-0.73 (1.2)	4.96 (<0.00001)	-0.69 (1.1)	1.98 (0.7)	-24.47 (<0.00001)
WC* (cm)	65.4 (6.7)	65.0 (6.6)	65.7 (6.7)	-1.28 (0.20)	64.9 (7.5)	66.2 (6.2)	-2.35 (0.02)	64.6 (5.5)	77.0 (9.9)	-8.74 (<0.00001)
SBP (mm Hg)	108.3 (12.4)	108.6 (13.9)	108.2 (11.4)	0.37 (0.71)	106.9 (12.5)	109.6 (12.2)	-2.72 (0.007)	107.9 (12.4)	113.6 (11.9)	-8.81 (<0.00001)
DBP (mm Hg)	66.2 (9.5)	65.1 (10.3)	67.0 (9.0)	-2.35 (0.02)	65.6 (9.4)	66.8 (9.7)	-1.57 (0.12)	65.8 (9.4)	70.8 (9.7)	-3.50 (<0.00001)
	Median			MW U-test (P-value)	Median		MW U-test (P-value)	Median		MW U-test (P-value)
FBG	87.5	89.7	86.3	0.66 (0.51)	65.0	87.3	0.45 (0.65)	87.6	87.4	0.26 (0.79)
T-Chol	199.4	204.7	198.0	0.69 (0.49)	207.3	197.3	1.81 (0.07)	198.9	221.2	1.06 (0.29)
Trig	180.8	187.1	179.5	0.84 (0.40)	156.6	190.1	-2.46 (0.01)	180.2	189.3	0.37 (0.71)
HDL	55.9	54.2	56.5	-0.58 (0.56)	56.8	55.5	0.95 (0.34)	55.8	57.4	0.04 (0.97)
LDL	289.6	271.5	295.4	-3.03 (0.002)	295.2	276.7	1.80 (0.07)	284.3	303.8	0.47 (0.64)

* Male Waist circumference ≥ 94 cm n=1 (0.4%); Mean (±sd) = 98.0 (0.0) and <94 cm n=240 (99.6%); Mean (±sd) = 64.9 (6.3); * Female Waist circumference ≥ 80 cm n=17 (4.4%); Mean (±sd) = 84.8 (4.2) and <80 cm n=366 (95.6%); Mean (±sd) = 64.8 (3.3). Boys were 1.52 times more likely to be lean than girls ($\chi^2=1.70$, P-value=0.19; OR=1.52, 95% CI=0.81, 2.84).

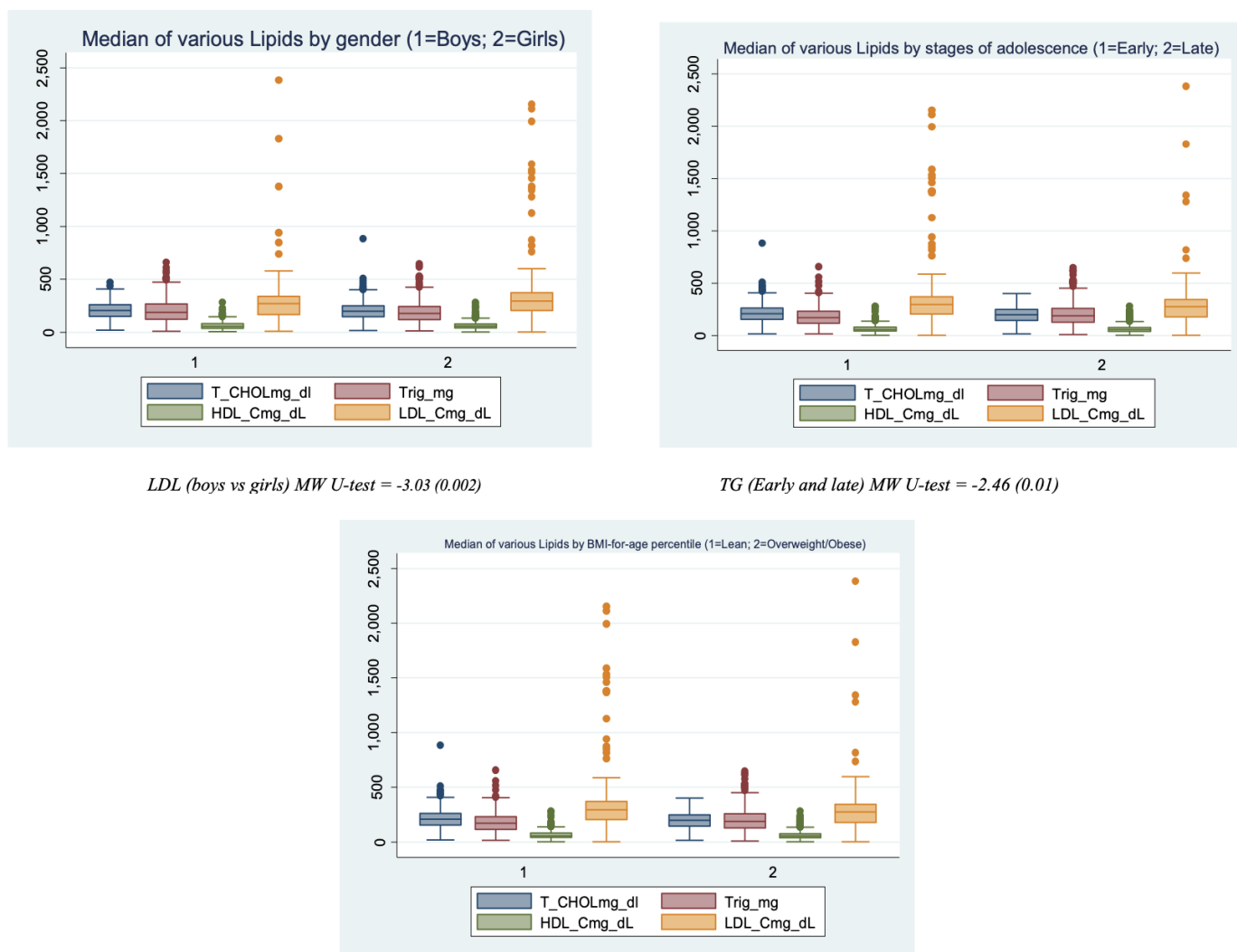


Figure 2. Box plot for lipid profiles by gender and stage of adolescence and BMI-for age percentile (Lean and Overweight/Obese) in Nigerian Secondary schools

Proportion of study subjects with borderline and high (low for HDL) Fasting Lipid or Blood Glucose (FBG) levels (Table 2).

Though there were no significant differences in their proportions, boys were however more likely to present with impaired FBG, borderline TC, TG and HDL respectively (statistical inferences for these are shown in Table 2). Those in early adolescence were also less likely to present with impaired FBG, but more likely to have borderline TC, TG, and HDL respectively while lean subjects were over two times more likely to have borderline TG compared to overweight/obese subjects. On the contrary, boys were unlikely to have diabetic FBG or high levels of lipids (TC, TG, HDL, LDL) compared to girls; those in early adolescence were over 1½ times more likely to present with diabetic FBG and TC, slightly more likely to have HDL compared to those in late adolescence; and lean subjects were less likely to have diabetic FBG but nine times more likely to present with high TC and about 1½ times more likely to have high HDL.

Table 2. Proportion of study subjects with borderline (impaired for FBG) and high (low for HDL) lipids and FBG

Variable	All (n=624)		Boys (n=241)		Girl (n=383)		χ^2 (P-value)	OR (95% CI)	Early adolescent (n=300)		Late adolescent (n=324)		χ^2 (P-value)	OR (95% CI)	Lean (n=574)		Overweight/Obese (n=50)		χ^2 (P-value)	OR (95% CI)
	Freq. (%)	Median	Freq. (%)	Median	Freq. (%)	Median			Freq. (%)	Median	Freq. (%)	Median			Freq. (%)	Median	Freq. (%)	Median		
Borderline (Impaired for FBG)																				
FBG	119 (19.1)	109.8	48 (19.9)	108.5	71 (18.5)	110.4	0.18 (0.67)	1.09 (0.73, 1.64)	53 (17.7)	110.3	66 (20.4)	109.8	0.74 (0.39)	0.84 (0.56, 1.26)	109 (19.0)	109.8	10 (20.0)	111.8	0.03 (0.86)	0.94 (0.45, 1.93)
T-Chol	72 (11.5)	172.4	35 (14.5)	168.0	37 (9.7)	173.5	3.42 (0.06)	1.59 (0.97, 2.60)	37 (12.3)	173.1	35 (10.8)	168.0	0.36 (0.55)	1.16 (0.71, 1.90)	69 (12.0)	172.5	3 (6.0)	167.2	1.63 (0.20)	2.14 (0.65, 7.06)
Trig	193 (30.9)	105.8	78 (32.4)	105.4	115 (30.0)	107.1	0.38 (0.54)	1.12 (0.79, 1.58)	96 (32.0)	102.0	97 (29.9)	103.4	0.31 (0.58)	1.10 (0.78, 1.55)	117 (59.0)	107.6	16 (32.0)	87.7	3.69 (0.05)	0.54 (0.29, 1.02)
HDL	2 (0.3)	53.3	1 (0.4)	53.5	1 (0.3)	53.2	0.00 (1.00)	1.59 (0.10, 25.57)	1 (0.3)	53.5	1 (0.3)	53.2	0.00 (1.00)	1.08 (0.67, 17.35)	2 (0.3)	53.3	0 (0.0)	0.0	0.00 (1.00)	-
LDL	1 (0.2)	87.8	1 (0.4)	87.8	0 (0.0)	0.0	0.05 (0.82)	-	0 (0.0)	0.0	1 (0.3)	87.8	0.00 (1.00)	-	1 (0.2)	87.8	0 (0.0)	0.0	0.00 (1.00)	-
High (Low for HDL, Diabetic for FBG)																				
FBG	73 (11.7)	149.2	24 (10.0)	158.4	49 (12.8)	140.1	1.15 (0.28)	0.75 (0.45, 1.26)	43 (14.3)	149.6	30 (9.3)	142.6	3.88 (0.049)	1.64 (1.00, 2.69)	66 (11.5)	149.4	7 (14.0)	145.2	0.28 (0.60)	0.80 (0.34, 1.85)
T-Chol	378 (60.6)	239.5	143 (59.3)	248.7*	235 (61.3)	231.2*	0.25 (0.61)	0.92 (0.66, 1.28)	43 (14.3)	207.4	30 (9.3)	192.7	3.88 (0.049)	1.64 (1.00, 2.69)	343 (59.8)	238.8	7 (14.0)	209.4	39.03 (<0.0001)	9.12 (4.03,20.63)
Trig	416 (66.7)	223.4	156 (64.7)	235.0!	260 (67.9)	218.5!	0.66 (0.42)	0.87 (0.62, 1.22)	196 (65.3)	214.3	220 (67.9)	228.1	0.46 (0.50)	0.89 (0.64, 1.24)	382 (66.6)	221.0	34 (68.0)	228.2	0.04 (0.83)	0.94 (0.50, 1.74)
HDL	268 (43.0)	36.6	103 (42.7)	33.1	165 (43.1)	38.9	0.01 (9.33)	0.99 (0.71, 1.37)	132 (44.0)	38.8	136 (42.0)	35.0	0.26 (0.61)	1.09 (0.79, 1.49)	249 (43.4)	37.0	19 (38.0)	29.5	0.54 (0.46)	1.25 (0.70, 2.27)
LDL	536 (85.9)	295.7	219 (90.9)	290.0**	358 (93.5)	302.1**	1.43 (0.23)	0.70 (0.38, 1.26)	277 (92.3)	306.0	300 (92.6)	290.9	0.01 (0.90)	0.96 (0.53, 1.75)	529 (92.2)	295.1	48 (96.0)	307.7	0.61 (0.43)	0.47 (0.11, 1.98)

Triglyceride: Kolmogorov-Smirnov Normality test value =0.093, Decision (Alpha =5%) Reject normality; Total cholesterol: Kolmogorov-Smirnov Normality test value =0.055, Decision (Alpha =5%) Reject normality; Low-density lipoprotein: Kolmogorov-Smirnov Normality test value =0.204, Decision (Alpha =5%) Reject normality; High-density lipoprotein: Kolmogorov-Smirnov Normality test value =0.153, Decision (Alpha =5%) Reject normality. * ($\chi^2=4.70$, P-value=0.03, OR=2.48, 95% CI=1.08, 5.69). *, ! In the "high" category, T-Chol and Trig. were significantly higher (Mann-Whitney U=1.97, 2.36; P-value=0.04, 0.02 respectively) in boys than girls but ** LDI was significantly higher (Mann-Whitney U=-2.80, P-value=0.005) among girls than boys

Comparative analysis of presence or absence of dyslipidemia relative to gender, BMI-for-age, and stage of adolescence of study participants (Table 3).

The overall prevalence of dyslipidemia among the study subjects was 8.5% (53/624) – higher among boys (10.8%) than girls (7.1%), higher in late-stage (8.6%) than in early-stage (8.3%) adolescence and higher among overweight/obese (12.0%) than among those that were lean (8.2%). When the median of various lipid among those with and without dyslipidemia were compared, significant variations were apparent in the levels of TC (243.5 vs 192.4, U-test=5.92, **P-value=<0.00001**), TG (207.4 vs 176.5, U-test=3.67, **P-value=0.0002**) and HDL (28.6 vs 58.2, U-test=-9.53, **P-value=<0.00001**). This was also the case when the lipid levels of boys and of girls with dyslipidemia were compared with those without dyslipidemia. Boys and girls with dyslipidemia presented with similar lipid and FBG profiles. Boys were 1.59 more likely to have dyslipidemia compared to girls ($\chi^2=2.66$, **P-value=0.10**, Or=1.59, 95% CI=0.92, 2.76). Overweight/Obese subjects were 1.53 more likely to develop dyslipidemia compared to lean students ($\chi^2=0.86$, **P-value=0.35**, OR=1.53, 95% CI=0.62, 3.77). Compared with those without dyslipidemia, the

TC, TG and HDL of early adolescents and late adolescents with dyslipidemia were also significantly higher as illustrated in Table 3. Overweight subjects with dyslipidemia were significantly younger (t-test=2.54, **P-value=0.04**), had higher BMI-for age (-10.48, **P-value<0.00001**) and higher WC (cm) (t-test=-4.72, **P-value= 0.004**) than lean subjects with dyslipidemia. Median levels of TC, TG and HDL were significantly higher among lean subjects with, compared to those without dyslipidemia while median LDL was marginally significant as shown in the Table. Only HDL was significantly higher among overweight obese subjects with dyslipidemia while TG levels between the two groups was marginally significant.

Table 3. Comparative analysis of presence or absence of dyslipidemia relative to gender, lean or overweight/obese and stage of adolescence of the study subjects.

	All	Boys	Girls	Boys vs. Girls
--	-----	------	-------	----------------

Variable	Sub-variable	Dyslipidemia			Dyslipidemia			Dyslipidemia			Dyslipidemia		
		Yes (n=53, 8.5%)	No (n=571, 91.5%)	t-test (P-value)	Yes * (n=26, 10.8%)	No (n=215, 89.2%)	t-test (P-value)	Yes * (n=27, 7.1%)	No (n=356, 92.9%)	t-test (P-value)	Yes	No	
		Mean (±sd)			Mean (±sd)			Mean (±sd)			t-test (P-value)		
All and Gender	Age	14.6 (2.1)	14.7 (2.1)	-0.33 (0.74)	14.9 (2.2)	14.8 (2.2)	0.22 (0.83)	14.3 (2.0)	14.7 (2.1)	-0.99 (0.33)	1.04 (0.30)	0.54 (0.59)	
	BMI (kg/m ²)	20.3 (7.9)	19.1 (3.7)	1.09 (0.28)	20.0 (10.8)	18.5 (2.9)	0.71 (0.49)	20.6 (3.9)	19.5 (4.1)	1.41 (0.17)	-0.26 (0.79)	-3.40 (0.0007)	
	WC (cm)	66.6 (8.3)	65.4 (6.7)	1.02 (0.31)	64.7 (8.5)	65.1 (6.7)	-0.23 (0.82)	68.5 (7.8)	65.7 (6.8)	1.81 (0.08)	-1.69 (0.10)	-1.03 (0.30)	
	Systolic BP	109.2 (12.3)	108.2 (12.4)	0.56 (0.57)	110.1 (14.8)	108.4 (13.8)	0.56 (0.58)	108.4 (9.6)	108.2 (11.6)	0.15 (0.88)	0.52 (0.60)	0.18 (0.86)	
	Diastolic BP	66.1 (9.5)	66.2 (9.6)	-0.07 (0.94)	66.0 (10.9)	65.0 (10.2)	0.45 (0.66)	66.1 (8.0)	67.0 (9.1)	-0.56 (0.57)	-0.04 (0.97)	-2.36 (0.02)	
		Median value		MW U (P-value)	Median value		MW U (P-value)	Median value		MW U (P-value)			
	FBG	81.3	88.1	-1.09 (0.27)	79.9	89.8	-1.12 (0.26)	82.9	86.7	-0.47 (0.64)	-0.22 (0.82)	0.80 (0.44)	
	TC	243.5	192.4	5.92 (<0.00001)	263.2	193.9	4.25 (0.00002)	235.6	192.4	4.07 (0.00005)	1.33 (0.18)	0.13 (0.90)	
	Triglyceride	207.4	176.5	3.67 (0.0002)	212.1	172.8	2.59 (0.01)	205.3	176.6	2.43 (0.01)	0.64 (0.52)	0.45 (0.66)	
	HDL	28.6	58.2	-9.53 (<0.00001)	27.1	70.4	-6.66 (<0.00001)	29.5	58.4	-6.81 (<0.00001)	-1.48 (0.14)	0.20 (0.84)	
LDL	281.0	290.0	1.04 (0.30)	276.2	269.4	1.65 (0.10)	292.4	295.5	0.22 (0.82)	-0.21 (0.83)	-3.16 (0.002)		
Stage of adolescence		Early adolescent stage (10-14 years)			Late adolescent stage (15-19 years)			Early vs. Late adolescent stages					
		Yes (n=25, 8.3%)	No (n=275, 91.7%)	t-test (P-value)	Yes (n=28, 8.6%)	No (n=296, 91.4%)	t-test (P-value)	Yes		No			
								t-test (P-value)					
	Age	12.8 (1.3)	12.9 (1.2)	-0.37 (0.71)	16.3 (1.1)	16.4 (1.2)	-0.46 (0.65)	-10.71 (<0.00001)		-34.85 (<0.00001)			
	BMI (kg/m ²)	20.1 (4.6)	18.6 (3.4)	1.59 (0.12)	20.5 (10.2)	19.5 (3.9)	0.52 (0.61)	-0.18 (0.86)		-2.94 (0.003)			
	WC (cm)	67.8 (10.7)	64.6 (7.1)	6.12 (<0.0001)	65.6 (5.2)	66.2 (6.3)	-0.57 (0.57)	0.93 (0.36)		-2.84 (0.005)			
	Systolic BP	106.2 (11.4)	107.0 (12.6)	-0.33 (0.74)	112.0 (12.7)	109.4 (12.2)	1.04 (0.31)	-1.75 (0.09)		-2.31 (0.02)			
	Diastolic BP	66.3 (8.5)	65.5 (9.5)	0.45 (0.66)	65.9 (10.4)	66.9 (9.6)	-0.49 (0.63)	0.15 (0.88)		-1.75 (0.08)			
		Median value		MW U (P-value)	Median value		MW U (P-value)						
	FBG	79.0	89.8	-1.27 (0.20)	83.8	87.7	-0.26 (0.79)	-0.70 (0.48)		0.67 (0.50)			
	TC	253.9	197.5	3.60 (0.0003)	241.1	183.9	4.75 (<0.00001)	0.24 (0.81)		1.91 (0.06)			
Triglyceride	204.2	171.0	2.62 (0.009)	209.7	185.4	2.55 (0.01)	-0.93 (0.35)		-2.34 (0.02)				
HDL	29.5	58.6	-6.71 (<0.00001)	28.1	57.2	-6.75 (<0.00001)	0.78 (0.44)		0.88 (0.38)				
LDL	295.7	295.1	0.34 (0.73)	280.0	275.7	1.12 (0.26)	0.20 (0.84)		1.80 (0.07)				
(contd.)													

		Lean			Overweight/Obese			Lean vs. Overweight/Obese		
		Yes ! (n=47, 8.2%)	No (n=527, 91.8%)	t-test (P-value)	Yes ! (n=6, 12.0%)	No (n=44,88.0%)	t-test (P-value)	Yes	No	
								t-test	(P-value)	
BMI-for-age	Age	14.9 (2.0)	14.8 (2.1)	0.33 (0.74)	12.4 (2.3)	13.7 (2.2)	-1.31 (0.24)	2.54 (0.04)	3.20 (0.002)	
	BMI (kg/m ²)	19.5 (8.0)	18.4 (2.4)	0.94 (0.35)	27.3 (2.5)	26.9 (6.4)	0.29 (0.78)	-5.03 (0.00005)	-8.756(<0.00001)	
	WC (cm)	64.5 (4.9)	64.6 (5.6)	-0.13 (0.90)	83.7 (9.8)	76.1 (9.7)	1.78 (0.12)	-4.72 (0.004)	-7.76 (<0.00001)	
	Systolic BP	108.5 (12.3)	107.8 (12.4)	0.37 (0.71)	115.0 (12.3)	113.5 (11.9)	0.28 (0.79)	-1.22 (0.27)	-3.04 (0.004)	
	Diastolic BP	65.4 (9.3)	65.9 (9.5)	-0.35 (0.73)	71.2 (9.6)	70.7 (9.9)	0.12 (0.91)	-1.40 (0.21)	3.10 (0.003)	
		Median value			MW U-test (P-value)	Median value		MW U-test (P-value)	MW U-test (P-value)	
	FBG	81.3	88.1	0.38 (0.71)	76.9	88.5	-0.39 (0.70)	0.08 (0.93)	0.28 (0.78)	
	TC	247.6	191.8	2.76 (0.006)	230.3	215.3	0.66 (0.51)	-0.94 (0.35)	1.14 (0.25)	
	Triglyceride	207.1	176.5	3.24 (0.001)	308.4	179.8	1.94 (0.05)	1.12 (0.26)	-0.05 (0.96)	
	HDL	28.9	57.8	-4.43 (0.00001)	25.2	58.4	-3.22 (0.001)	-1.01 (0.31)	0.50 (0.62)	
	LDL	281.0	287.7	-0.57 (0.057)	334.2	303.8	0.27 (0.79)	-0.03 (0.98)	0.42 (0.67)	

*Boys were 1.59 more likely to have dyslipidemia compared to girls ($\chi^2=2.66$, P -value=0.10, $OR=1.59$, 95% $CI=0.92, 2.76$). ! Overweight/Obese students were 1.53 more likely to develop dyslipidemia compared to lean students ($\chi^2=0.86$, P -value=0.35, $OR=1.53$, 95% $CI=0.62, 3.77$).

Specific risk factors for MetS (Table 4)

The overall prevalence of MetS in this study was 8.3% more prominent in boys (14.%) than girls (4.7%), in overweight/obese (26.0%) than among lean subjects (6.8%) and slightly more predominant in late (8.6%) than in early adolescence. BMI-for age (kg/m²) and WC (cm) were significantly higher (t-test=-3.05, **P-value=0.004** and t-test=-2.16, **P-value=0.04** respectively) among girls than boys. Overweight/obese subjects with MetS were significantly younger (t-test=3.46, P -value=0.003), with higher BMI-for-age (t-test=-9.90, **P-value<0.00001**) and WC (cm) (t-test=-4.08, **P-value=0.001**). Among all those with MetS, there was no significant difference in the FBG or lipid levels relative to gender or stage of adolescence. However, the median TG level of subject with overweight/obese status (296.4) was significantly higher (t-test=-2.35, **P-value=0.02**) than the median value (214.0) of lean subjects.

Table 4. Specific risk factors for MetS and its prevalence as demarcated by the occurrence of known potential risk factors relative to gender, stage of adolescence and BMI-for-age.

Variable	Mets $\geq 90^{\text{th}}$ percentile									
	All (n=52, 8.3%)	Gender			BMI-for-age percentile			Stage of adolescence		
		Boys (n=34, 14.1%)	Girls (n=18, 4.7%)	t-test (P-value)	Lean (n=39, 6.8%)	Overweight / Obese (n=13, 26.0%)	t-test (P-value)	Early (n=24, 8.0%)	Late (n=28, 8.6%)	t-test (P-value)
Mean (\pm sd)										
Age	14.4 (2.2)	14.4 (2.2)	14.5 (2.4)	-0.15 (0.88)	15.0 (2.0)	12.7 (2.1)	3.46 (0.003)	12.4 (1.3)	16.2 (1.0)	-11.7 (<0.00001)
BMI-for-age	0.28 (1.4)	-0.13 (1.4)	1.0 (1.2)	-3.05 (0.004)	-0.30 (1.1)	2.1 (0.6)	-9.90 (<0.00001)	0.78 (1.4)	-0.14 (1.3)	2.44 (0.02)
WC (cm)	69.5 (9.0)	67.5 (8.1)	73.2 (9.5)	-2.16 (0.04)	66.3 (5.9)	78.8 (10.5)	-4.08 (0.001)	69.1 (10.8)	69.7 (7.4)	-0.23 (0.82)
Systolic BP	113.6 (11.6)	112.4 (12.9)	115.8 (8.3)	-1.15 (0.26)	113.4 (11.7)	114.1 (11.5)	-0.19 (0.85)	113.0 (11.8)	114.1 (11.6)	-0.34 (0.74)
Diastolic BP	68.1 (10.1)	67.4 (10.1)	69.5 (10.2)	-0.71 (0.48)	66.9 (9.3)	71.9 (11.7)	-1.40 (0.18)	69.6 (10.9)	66.9 (9.3)	0.95 (0.35)
Median values (Mann-Whitney U test instead of t-test)										
FBG	115.0	111.9	120.7	0.87 (0.39)	113.6	116.4	-0.98 (0.33)	121.2	112.7	0.06 (0.96)
TC	164.1	165.7	161.4	0.14 (0.89)	163.2	176.1	0.61 (0.54)	179.5	151.1	1.49 (0.14)
Trig.	234.1	247.7	234.1	0.18 (0.86)	267.1	188.6	-1.04 (0.30)	214.0	296.4	-2.35 (0.02)
HDL	35.1	35.1	35.8	0.11 (0.92)	34.9	36.8	0.00 (1.00)	32.7	35.1	0.39 (0.69)
LDL	325.0	333.1	298.6	-1.45 (0.15)	332.8	315.2	-0.93 (0.35)	341.9	318.9	1.91 (0.06)

Frequency distribution of risk factors for MetS relative to gender, BMI-for-age percentile, and stage of adolescence (Table 5).

i. *Anthropometry*

Among all the study subjects, 8.0% had BMI-for-age $\geq 85^{\text{th}}$ percentile (6.2% in boys, 9.1% in girls; 0% in lean, 100% in overweight/obese; 11.3% in early and 4.9% in late adolescence). Girls with BMI $\geq 85^{\text{th}}$ percentile were about 1.2 times more at risk for MetS ($\chi^2=1.70$, **P-value =0.19**, RR=1.16, 95% CI=0.95, 1.40) than Boys. In all 0.2% (0.4% among boys alone) had WC (cm) ≥ 94 while 5.0% of all (5.0% among girls alone) had WC (cm) of ≥ 80 . The only boy with WC (cm) ≥ 94 was in early adolescence. Overall, 0.5% and 24.0% of lean and overweight/obese subjects were girls with WC (cm) ≥ 80 , while 3.3% and 1.5% respectively were in early or late-stage adolescence. Study subjects in early adolescence were approximately 2½ times more likely to be overweight/obese compared to those in late adolescence ($\chi^2=8.63$, P-value=0.003, OR=2.46, 95% CI=1.32, 4.56).

ii. *Pre-hypertension/Hypertension*

Of the 111 (7.8%) subjects with systolic pre-hypertension/hypertension, approximately 23% were boys while 14% were girls; 17% were in early- and 28% in late-stage adolescence and 14% were lean while 21% were overweight/obese. Boys with Pre-hypertension/ hypertension were 1.40 times more likely to be at risk for MetS ($\chi^2=7.96$, **P-value =0.005**, RR=1.40, 95% CI=1.13, 1.74) compared to girls.

iii. *FBG*

FBG of ≥ 100 mg/dL was more prevalent among girls (31.3%) than boys (29.9%), among overweight/obese (34.0%) compared to lean (30.5%) subjects and in early (32.0%) compared to late (29.6%) adolescence. Girls with FBG ≥ 100 mg/dL were slightly more likely to be at risk for MetS ($\chi^2=0.15$, **P-value =0.70**, RR=1.02, 95% CI=0.90, 1.17) compared to boys.

1. *HDL < 40 mg/dL*

A total of 43% (42.7%, and 43.1% among boys and girls; 43.4% and 38.0% among lean and overweight/obese; 44.0% and 42.0% in early and late adolescence) subjects had low HDL. Boys with HDL < 40 mg/dL, were about 1.2 times more likely to be at risk for MetS ($\chi^2=2.05$, **P-value =0.15**, RR=1.18, 95% CI=0.95, 1.46) than girls.

2. *TG ≥ 130 mg/dL*

TG level was the highest in the lipid profile, recorded by 70.5% of study subjects, including about 71% each of boys and girls, 67%, 68% of

lean and overweight/obese and 65%, 68% in early and in late adolescence. Boys with TG \geq 130 mg/dL were slightly more likely to be at risk for MetS ($\chi^2=0.14$, **P-value =0.71**, RR=1.04, 95% CI=0.84, 1.30) than Girls.

The proportion of boys and girls with 3 or more risk factors MetS was similar (9.1 vs 9.4) as was the proportion of those in early and late adolescence (10.3 vs 8.3) but very dissimilar (5.7% vs 50.0%) among lean and overweight/obese subjects respectively. Those in early adolescence were approximately 1½ times more likely to develop \geq 3 risk factors for MetS ($\chi^2=0.74$, P-value=0.39, OR=1.27, 95% CI=0.74, 2.18) compared to those in late adolescence. In all, 92 (14.7%) had no risk factor (about 13% boys, 16% girls; 16% lean, 2.0% overweight/obese; 14% early adolescents, 15.1 late adolescents).

Table 5. Frequency distribution of risk factors for MetS relative to gender, BMI-for-age percentile, and stage of adolescence.

Risk factors for MetS	All	Gender		BMI-for-age		Stage of adolescence		
		Boys	Girls	Lean	Overweight/Obese	Early	Late	
n (%)	624 (100.0)	241 (38.6)	383 (61.4)	574 (92.0)	50 (8.0)	300 (48.1)	324 (51.9)	
BMI-for-age \geq 85 th percentile †	50 (8.0)	15 (6.2)	35 (9.1)	0 (0.0)	50 (100.0)	34 (11.3)	16 (4.9)	
WC \geq 94 cm (boys)	1 (0.2)	1 (0.4)	-	0 (0.0)	1 (2.0)	1 (0.3)	0 (0.0)	
WC \geq 80 cm (girls)	19 (3.0)	-	19 (5.0)	3 (0.5)	12 (24.0)	10 (3.3)	5 (1.5)	
Pre-HT/HT *	111 (17.8)	56 (23.2)	55 (14.4)	97 (16.9)	14 (28.0)	43 (14.3)	68 (21.0)	
FBG glucose \geq 100 mg/dL ††	192 (30.8)	72 (29.9)	120 (31.3)	175 (30.5)	17 (34.0)	96 (32.0)	96 (29.6)	
HDL <40 mg/dL **	268 (43.0)	103 (42.7)	165 (43.1)	249 (43.4)	19 (38.0)	132 (44.0)	136 (42.0)	
TG \geq 130 mg/dL ***	440 (70.5)	172 (71.4)	268 (70.0)	382 (66.6)	34 (68.0)	196 (65.3)	220 (67.9)	
Number of risk factors for MetS	\geq 3	58 (9.3)	22 (9.1)	36 (9.4)	33 (5.7)	25 (50.0)	31 (10.3)	27 (8.3)
	2	202 (32.4)	77 (32.0)	125 (32.6)	187 (32.6)	15 (30.0)	93 (31.0)	109 (33.6)
	1	272 (43.6)	111 (46.1)	161 (42.0)	263 (45.8)	9 (18.0)	133 (44.3)	139 (42.9)
	0	92 (14.7)	31 (12.9)	61 (15.9)	91 (15.9)	1 (2.0)	43 (14.3)	49 (15.1)

WC=Waist circumference; HT=Hypertension, HDL=High-density lipoprotein; LDL= Low-density lipoprotein; TG= Triglyceride. *Boys with Pre-hypertension and stages 1 and 2 hypertension were 1.40 times more likely to be at risk for MetS ($\chi^2=7.96$, P-value =0.005, RR=1.40, 95% CI=1.13, 1.74) than Girls;**Boys with HDL<40 mg/dL, were about 1.2 times more likely to be at risk for MetS ($\chi^2=2.05$, P-value =0.15, RR=1.18, 95% CI=0.95, 1.46) than Girls; ***Boys with TG \geq 130 mg/dL were slightly more likely to be at risk for MetS ($\chi^2=0.14$, P-value =0.71, RR=1.04, 95% CI=0.84, 1.30) than Girls. †Girls with BMI \geq 85th percentile were about 1.2 times more at risk for MetS ($\chi^2=1.70$, P-value =0.19, RR=1.16, 95% CI=0.95, 1.40) than Boys. †† Girls with serum glucose \geq 100mg/dL were slightly more likely to be at risk for MetS ($\chi^2=0.15$, P-value =0.70, RR=1.02, 95% CI=0.90, 1.17) than Boys. Those in early adolescence were approximately 2½ times more likely to be overweight/obese ($\chi^2=8.63$, P-value=0.003, OR=2.46, 95% CI=1.32, 4.56) and were 1½ times more likely to develop \geq 3 risk factors for MetS ($\chi^2=0.74$, P-value=0.39, OR=1.27, 95% CI=0.74, 2.18).compared to those in late adolescence

Multivariate regression analysis (Table 6).

Among all the study subjects, a significant 25.5% of the variance in Mets \geq 90th percentile is explained by five predictor variables - FBG, TC, TG, HDL, and BMI-for-age percentile after a backward elimination was conducted. The overall relationship between MetS \geq 90th percentile and these

five predictor variables was significant and all of them contributed to the observed MetS $\geq 90^{\text{th}}$ percentile among study subjects. Similarly, among boys, a significant 38.9% of these same predictor variables explained the variations in MetS $\geq 90^{\text{th}}$ percentile whereas among girls, a significant 21.5% of the variance in MetS 90^{th} percentile is explained by four predictor variables – FBG, TG, BMI-for-age percentile, and WC.

All study subjects					
Dependent variable: MetS $\geq 90^{\text{th}}$ percentile	R ² =0.2554	F-test =42.40	P-value <0.00001	t-test	P-value
Independent variables	Regression coefficient	Standard error	95% CI		
Intercept	-0.128	0.042	-0.210, -0.045	-3.04	0.002
BMI-for-age percentile	0.002	0.0003	0.001, 0.002	5.00	<0.00001
Fasting Blood Glucose (mg/dL)	0.002	0.0001	0.002, 0.003	10.67	<0.00001
Total cholesterol (mg/dL)	-0.0004	0.0001	-0.0006, -0.0001	-3.21	0.001
Triglyceride (mg/dL)	0.0004	0.00009	0.0002, 0.0005	4.28	<0.00001
High-density lipoprotein (mg/dL)	-0.001	0.0002	-0.001, -0.0007	-4.39	<0.00001
Boys					
Dependent variable: MetS $\geq 90^{\text{th}}$ percentile	R ² =0.3890	F-test =29.92	P-value <0.00001	t-test	P-value
Independent variables	Regression coefficient	Standard error	95% CI		
Intercept	-0.137	0.078	-0.290, 0.017	-1.76	0.08
BMI-for-age percentile	0.003	0.0006	0.002, 0.004	4.51	<0.00001
Fasting Blood Glucose (mg/dL)	0.004	0.0005	0.003, 0.005	8.67	<0.00001
Total cholesterol (mg/dL)	-0.0006	0.0002	-0.001, -0.0002	-2.99	0.003
Triglyceride (mg/dL)	0.0005	0.0001	0.0002, 0.0007	3.12	0.002
High-density lipoprotein (mg/dL)	-0.002	0.0004	-0.003, -0.001	-4.82	<0.00001
Girls					
Dependent variable: MetS $\geq 95^{\text{th}}$ percentile	R ² =0.2151	F-test = 25.89	P-value <0.00001	t-test	P-value
Independent variables	Regression coefficient	Standard error	95% CI		
Intercept	-0.479	0.110	-0.695, -0.264	-4.37	<0.00001
BMI-for-age percentile	0.0009	0.0004	0.0002, 0.0017	2.36	0.019
Waist circumference	0.004	0.002	0.0009, 0.0079	2.47	0.014
Fasting Blood Glucose (mg/dL)	0.002	0.0002	0.001, 0.002	7.65	<0.00001
Triglyceride (mg/dL)	0.0003	0.0001	0.0001, 0.0004	2.82	0.005

Table 6. Multivariate regression analysis with MetS 90^{th} percentile as dependent variable and key predictor variables for all subjects, all boys, and all girls respectively in the study.

For all the study subjects, five predictor variables - Fasting blood glucose, Total cholesterol, Triglyceride, High-density lipoprotein, and BMI-for-age percentile - significantly (P -value<0.00001) explain 25.5% of the variance in the outcome variable - MetS $\geq 90^{\text{th}}$ percentile. One unit change in MetS 95^{th} percentile is associated with decrease in age and increase in other predictor variables.

Discussion

Published literature and data on severe dyslipidemia among secondary school students in Africa, including Nigeria is rare. This current study investigated the prevalence of overweight/obesity, dyslipidemia and of MetS among 10-19-years-old indigenous Black Nigerian adolescent secondary school students in Lagos, Southwest Nigeria. The application of various criteria in the numerous definitions of MetS has rendered the estimation of its prevalence problematic [45]. At present, there are no agreed guidelines or diagnostic criteria for MetS in the pediatric/adolescent population [46]. There are some key findings in this study that merit discussion. First, the overall prevalence of MetS was 8.3%, higher than the 5.2% reported from India [47] or 1.4% from Tanzania [48]. In consonance with a South African study [49], MetS was more prevalent among boys than girls, but much higher than the 6.1% among boys and 3.1% among girls reported in South Africa. Studies in different parts of Nigeria have reported different prevalence for MetS among adolescents under different criteria such as HIV [50], diabetes [29], and obesity [51] and others with a range from 1.9% to 23.8%. The overall 8.3% prevalence of MetS in this study falls within the range of MetS reported in Nigeria, though it is higher than the 2.2% among 10-17 years reported in Turkey [52]. Further, the 26% prevalence of MetS among overweight/obese subjects falls

within the 3-60% earlier reported [53]. National Health and Nutrition Examination Survey I (NHANES I) study (1988-1994), conducted in the USA, demonstrated that MetS features were found in 3% to 14% of all children and adolescents. These rates were reported as 13% to 37% in obese patients in the study [54]. The exact pathophysiology and mechanism of MetS seems complex and are yet to be clearly elucidated. However, Çelebi proposed that abdominal obesity and insulin resistance effectively develop MetS [55] while Rochlani et al suggested that macrophage infiltration occurs in adipose tissue with intra-abdominal obesity, that various cytokines such as TNF- α and IL6 released from macrophages cause inflammation and that this inflammation leads to lipolysis in adipose tissue, increasing circulating free fatty acids, decreased insulin signaling, and decreased glucose transporter (GLUT4) synthesis, resulting in insulin resistance [56]. The preponderance of impaired/diabetic FBG in this study's multivariate regression analysis may attest to the finding of a study which suggested that IR is the key mechanism thought to underlie metabolic syndrome [57][58]. An alternative view of pathway in the development of MetS is activating the renin-angiotensin system (RAS). Obesity and IR cause an elevated production of angiotensin-2 secreted by adipose tissue [56]. It is also suggested [59] that increased lipolysis in IR causes an elevation of free fatty acids in the circulation. The liver produces triglycerides using excess concentration of fatty acids in the blood with simultaneous increase in very low-density lipoprotein (VLDL) production, leading to dyslipidemia [59]. Possibly, adipokines released from visceral adipose tissue may be associated with the development of MetS and CVD [60] and stimulation of the renin-angiotensin system (RAS) may play a vital role in neurohumoral pathway that promotes the development of MetS [61]. The 11.7% prevalence of DM in this study is higher than the 0.4% prevalence reported in Cote d'Ivoire [62]. The prevalence of DM, a syndrome with various linked sub-phenotypes such as mitochondrial disorders, lipodystrophies, and inflammatory disorders involving cytokines [63] is already recognized to increase the probability of developing metabolic syndrome [64]. Foremost phenotypes correlated with DM are dyslipidemias, notably hypertriglyceridemia and low HDL cholesterol levels. Both diabetes and dyslipidemia are strongly associated with increased risk for atherosclerotic vascular disease [63]. Dyslipidemia, recognized as a global disease of epidemic proportion is also known as a major risk factor for cardiovascular disease with an enormous burden in terms of morbidity, mortality, and medical costs [32]. Atherosclerosis and cardiovascular disease (CVD) are the major health problems associated with dyslipidemia. These disorders are vascular problems associated with more than 17 million deaths worldwide in 2015, a rise of 12.5% from 2005 onwards [65]. The prevalences of TC \geq 200 mg/dL (60.6%), TG \geq 130 mg/dL (66.7%), HDL $<$ 40 mg/dL (43.0%) and LDL \geq 130 mg/dL (85.9%), in this study are higher than the 2.8%, 19.1%, 58.4% and 3.2% reported from Ethiopia [66] or the 12.1% 4.5%, 28.4% and 9.2% reported from a Ghana [46]. The most prominent dyslipidemia in this study was LDL, contrary to HDL reported in Ethiopia [66], Ghana [46] and in another study in Nigeria [67]. In variance with another study in India, LDL was significantly higher among boys than girls in this study [68]. High concentration of LDL cholesterol are presently believed to be the primary risk factor for the emergence of atherosclerosis coronary heart disease (ASCHD), myocardial infarction (MI), and increased mortality. Raised LDL cholesterol levels (hypercholesterolemia) are relatively common in populations consuming a western diet and is associated with the development of atherosclerotic plaque (AP) [14], leading to life-threatening myocardial infarction [69]. The precise pathophysiology of AP - local inflammation, LDL oxidation, macrophage activation, and necrotic core formation - has already been elucidated, though its molecular mechanisms are still unknown [68]. Hypertriglyceridemia is also an independent risk factor for CVD [70] which occurs through (i) abnormalities in hepatic VLDL production, and intestinal chylomicron synthesis (ii) Dysfunctional LPL-mediated lipolysis or (iii) Impaired remnant clearance. Liang et al [71] suggested that hypertriglyceridemia may intensify the risk of ischemic stroke by promoting not only atherosclerosis but also augmenting thrombosis and blood viscosity. On the contrary, hypertriglyceridemia may have been beneficial in patients who have already suffered a stroke [71]. Thus dyslipidemia, diabetic blood glucose and obesity are the triad for MetS and obesity on its own, is linked to non-alcoholic fatty liver disease (NAFLD), to abnormal lipid profiles and liver enzyme abnormalities [72]. The overall 43.0% prevalence of abnormally low HDL in this study is lower than the 61.2% reported from another Nigerian study [67] but higher than the 12.8% reported from Saudi Arabia [73], and the 40.6% reported from Brazil [74]. The principal function of HDL, composed of cholesterol, triglycerides, and various apolipoproteins, is to transport cholesterol from the peripheral tissues to the liver, thus performing biodistribution of lipids [75] and in the process, scavenge excess cholesterol through reverse cholesterol transport (RCT) or exchange it with apoB particles (e.g., LDL) [76] for disposal. Its anti-atherogenic and anti-inflammatory properties serve in lowering the size of the plaque and its associated inflammation [77]. In this study, there was no significant variation in SBP or DBP (mm Hg) among those with and without dyslipidemia. However, SBP (mm Hg) was significantly higher among those in late compared to those in early adolescence and among overweight/obese subjects than among the lean subjects. The relationship between hypertension and dyslipidemia, obesity, as well as insulin resistance (MetS), further increases the overall cardiovascular risk of an individual. Although SBP is recognized as a risk factor that increases

the chance of MetS, it was not associated with that condition in the adolescent population of this study.

Study limitations

There are some limitations in the study that need consideration. First, the study's main limitation is its relatively small sample size, which may restrict its ability to fully uncover the deteriorating metabolic conditions in this population. Blood pressures were measured with different measuring instruments in different adolescent stages, and this could have resulted in a bias. In the methodology, bias may have been introduced in the sample selection as the proportion of girls outweighed that of boys and some of the overweight or obese students may have been left out. Also, the study was conducted in the southwestern area bordering the Atlantic ocean and not in the northern arid area or the mountainous middle area of the country. For this reason, the results may not be generalized or interpreted as national, requesting for a national study on dyslipidemia and MetS among secondary schools' adolescents. NHLBI guide for references of MetS in adults was used as a reference. While this may be justifiable due to lack of established cut-off values for adolescents, especially indigenous Black Africans, the application may result in an underestimation of the prevalence of central obesity in this population of study. In general, there is no agreed definition of MetS among adolescents globally and hardly any clinical guidelines or cut-off points for any of the variables for MetS among Black Africans. For this reason, the definitions, cut-off points and variables used for the identification of MetS in American and European literatures were used in this study. Lastly, nutrition, dietary and physical exercise aspects of the participants have not been considered in this study though these play in MetS but will be considered in a future study. This study only focused on quantity of lipoproteins. As such, the quality and functionality of HDL or its dysfunctionality such as a lower apoA-I content, lower antioxidant ability, smaller size, and ambiguous shape were not considered. These will be addressed in a future study.

Conclusion and Recommendation

This study has documented the prevalence of dyslipidemia and that of MetS as 8.5% and 8.3% respectively among the adolescents in Lagos Nigeria. Findings from this study suggest that appropriate and satisfactory monitoring of lipid profile among adolescents in Nigeria is urgently needed. The elevated prevalence of lipid anomalies and MetS in the cohort of this study demands decisive lifestyle intervention approaches and policies for the prevention and control of significant cardiovascular and other risk factors. Health promotion, especially in regard to nutrition, diet and eating habits among adolescents should be an integral aspect of educational curriculum in Nigerian secondary schools. Governments at all levels and other service providers should not only frequently screen and instruct adolescents with diabetes and hypertension towards adopting healthy lifestyle but also make them recognize the eventual outcome of lipid abnormalities and their comorbidities in their adulthood. Decision-makers should lay stronger emphasis on establishing and reinforcing the early detection and prevention of lipid abnormalities among adolescents in Nigeria. Hence, periodic fasting lipid profile screening for adolescents, especially secondary school students is recommended to be mandatory biennially during the students' tenure. This will enhance early detection and treatment and reduce the high burden of this underdiagnosed and undertreated disease. Results from this study would be useful contribution towards enhancing knowledge towards dyslipidemia and MetS and perhaps can be utilized for effective policies and programs implications and in proposing guidelines for the diagnosis of MetS in Nigerian and African children and adolescents. Finally, health care providers caring for youth should adopt effective treatment for those at highest risk.

Statements and Declarations

Declaration of Competing Interest

The authors declare no competing interest.

Acknowledgments

Dr. Bamgboye M. Afolabi is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. This project was supported by the Nigerian Institute of Medical Research (NIMR). We would like to thank the Ministries of Education, Health and Public Service and their districts heads for making this study possible. We also thank the parents of the students as well as the students for making this study possible.

Data for reference

The data used to substantiate and validate the findings of this study are available from the corresponding author upon request.

References

1. [^] Mangili L. High prevalence of dyslipidemia in children and adolescents: opportunity for prevention. *Arq Bras Cardiol.* 2020;114(1):57–8.
2. [^] WHO, 2023 www.who.int/news-room/fact-sheets/detail/malnutrition. (Accessed on September 14, 2023).
3. ^{a, b} Lister NB, Baur LA, Felix JF, Hill AJ, Marcus C, Reinehr T, Summerbell C, Wabitsch M. Child, and adolescent obesity. *Nat Rev Dis Primers.* 2023;9(1):24
4. [^] www.afro.who.int/news/obesity-rising-africa-who-analysis-finds, (Accessed on September 14, 2023).
5. ^{a, b} globalnutritionreport.org/resources/nutrition-profiles/africa/ (Accessed on September 16, 2023).
6. [^] www.who.int/news-room/fact-sheets/detail/obesity-and-overweight. (Accessed on September 14, 2023).
7. [^] Friedland O, Nemet D, Gorodnitsky N, Wolach B, Eliakim A. Obesity and lipid profiles in children and adolescents. *J Pediatr Endocrinol Metab.* 2002;15(7):1011-6.
8. [^] Al Ashoor M, Al Hamza A, Zaboon I, Almomin A, Mansour A (2023) Prevalence and risk factors of diabetic retinopathy in Basrah, Iraq. *J Med Life* 16(2): 299.
9. [^] Ojiambo RM, Easton C, Casajús JA, Konstabel K, Reilly JJ, Pitsiladis Y. Effect of urbanization on objectively measured physical activity levels, sedentary time, and indices of adiposity in Kenyan adolescents. *J Phys Act Heal.* 2012;9(1):115–23.
10. [^] Gulati S, Misra A. Abdominal obesity, and type 2 diabetes in Asian Indians: Dietary strategies including edible oils, cooking practices and sugar intake. *Eur J Clin Nutr.* 2017;71(7):850–7.
11. [^] Popkin BM. The nutrition transition and its health implications in lower-income countries. *Public Heal Nutr.* 1998;1(1):5-21.
12. [^] de Ferranti SD, Newburger J. Dyslipidemia in children and adolescents: Definition, screening, and diagnosis. In *UpToDate* www.uptodate.com/contents/dyslipidemia-in-children-and-adolescents-definition-screening-and-diagnosis. Section Editor, David R. Fulton, Deputy Editor Carrie Armsby. (Accessed on September 20, 2023).
13. [^] Lecerf J, de Lorgeril M. Dietary cholesterol: from physiology to cardiovascular risk. *Br. J. Nutr.* 2011;106(1):6-14.
14. ^{a, b} Shao W, Espenshade PJ. Lipids | Cholesterol Synthesis and Regulation. Editor (s): Joseph Jez, *Encyclopedia of Biological Chemistry III (Third Edition)*, Elsevier, 2021:732-738, ISBN 9780128220405.
15. [^] Edwards PA. Cholesterol Synthesis. In: *Encyclopedia of Biological Chemistry*. Elsevier 2004; Editors WJ Lennarz WJ and Daniel Lane, 451-455.
16. [^] Albert RG, Zimmet P, Shaw J. Metabolic syndrome: A new world-wide definition: A Consensus Statement from the International Diabetes Federation. *Diabet Med* 2006;23:469-80.
17. [^] Kylin E. Studies of the hypertension-hyperglycemia-hyperuricemia syndrome (German). *Zentralbl Inn Med* 1923;44:105-27.
18. [^] Vague J. Sexual differentiation: A factor affecting the forms of obesity. *Presse Med* 1947;30:339-40
19. [^] Barutcu A, Ornek C, Kozanoglu E. A growing problem in childhood and adolescents: Metabolic syndrome and its relationship with physical activity and fitness. *Marmara Med J* 2023; 36(2):255-261
20. [^] Grundy SM, Hansen B, Smith SC, Jr, et al.; American Heart Association, National Heart, Lung, and Blood Institute, American Diabetes Association. *Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood*

- Institute/American Diabetes Association conference on scientific issues related to management. *Arterioscler Thromb Vasc Biol* 2004; 24(2): e19–e24.)
21. [^] Brambilla P, Pozzobon G, Pietrobelli A. Physical activity as the main therapeutic tool for metabolic syndrome in childhood. *Int J Obes (Lond)* 2011; 35: 16-28.
 22. [^] da Penha JT, Gazolla FM, de Miranda Carvalho CN, et al. Physical fitness and activity, metabolic profile, adipokines and endothelial function in children. *J Pediatr (Rio J)* 2019; 95: 531-37.
 23. [^] Andaki ACR, Tinôco ALA, Mendes EL, Júnior RA, Hills AP, Amorim PRS. Anthropometry and physical activity level in the prediction of metabolic syndrome in children. *Public Health Nutr* 2014; 17: 2287-94.
 24. [^] Martino F, Puddu PE, Pannarale G, et al. Metabolic syndrome among children and adolescents from Southern Italy: contribution from the Calabrian Sierras Community (CSCS). *Int J Cardiol.* 2014;177(2): 455–460.
 25. [^] Litwin M, Kułaga Z. Obesity, metabolic syndrome, and primary hypertension. *Pediatr Nephrol.* 2021 Apr;36(4):825-837.
 26. [^] Gobato AO, Vasques AC, Zambon MP, Barros Filho Ade A, Hessel G. Metabolic syndrome and insulin resistance in obese adolescents. *Rev Paul Pediatr.* 2014;32(1):55-62.
 27. [^] Lebovitz HE. Rationale for and role of thiazolidinediones in type 2 diabetes mellitus. *Am J Cardiol.* 2002 Sep 5;90(5A):34G-41G.
 28. [^] Pedrinelli R, Dell'Omo G, Di Bello V, Pontremoli R, Mariani M. Microalbuminuria, an integrated marker of cardiovascular risk in essential hypertension. *J Hum Hypertens,* 2002;16(2): 79-89.
 29. ^{a, b} Jaja TC, Yarhere IE. Dyslipidaemia in Nigerian children and adolescents with Diabetes Mellitus: Prevalence and Associated risk Factors. *Int J Diabetes Metab* 2019;25:45-51.
 30. [^] Odey FA, Ekanem EE, Udoh AE, Bassey IE. Lipid profile of apparently healthy adolescents in Calabar, Nigeria. *Centr Afr J Med,* 2010;53(1-4):11-18.
 31. [^] Eke CB, Ogbodo SO, Onyire NB, Muoneke UV, Ukoha MO, Amadi OF, Eze JN, Ibekwe RC. Association of Boddy Mass Index and Serum Lipid Profile among Adolescents in Enugu, Nigeria. *Ann Med Health Sci Res.* 2018;8:404-410.
 32. ^{a, b} Orimadegun BE. Dyslipidaemia in African Children and Adolescents. In *Management of Dyslipidaemia.* IntechOpen; 2021 Rijeka. Editor: Wilbert S. Aronow. <https://doi.org/10.5772/intechopen.96804> (Accessed on October 13, 2023)
 33. [^] Sliwa, K., *The heart of Africa: succeeding against the odds.* *Lancet,* 2016. 388(10063): p. e28-e36).
 34. [^] Lagos State Ministry of Education Directory, 2011.
 35. [^] Adolescent health [Internet]. Available from: <https://www.who.int/southeastasia/health-topics/adolescent-health>. (Accessed on October 13, 2023).
 36. [^] Adcock CJ. Sample size determination: A Review *Journal of the Royal Statistical Society, Series D. The Statistician* 1997, 46 (2):261-283.
 37. [^] World Health Organization. AnthroPlus V1.04. WHO 2014.
 38. [^] Gurka MJ, DeBoer MD, Filipp SL, Khan JZ, Rapczak TJ, Braun ND, Hanson K S, Barnes CP. MetS Calc: Metabolic Syndrome Severity Calculator. 2019., doi: 10.5281/zenodo.2542213
 39. [^] American Heart Association. Cholesterol Statistics. <http://www.americanheart.org/presenter.jhtml?identifier=536>. Accessed on 10th November 2022.
 40. [^] National Cholesterol Education Program (NCEP). Expert Panel on Blood Cholesterol Levels in Children and Adolescents: Highlight of the reports of the Expert Panel. *Pediatrics* 2012.
 41. [^] NHLBI. Obesity Education Initiative. *The practical guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults.* National Institute for Health, Bethesda MD, USA. (NIH Publication Number 004084), 2021. nhlbi.nih.gov
 42. [^] Lande MB, Batsky DL. New American Academy of Pediatrics Hypertension Guideline, *Hypertension* 2019;73(1):31-32.
 43. [^] CDC. Healthy weight, Nutrition, and Physical activity. cdc.gov/healthyweight/assessing/bmi/childrens_bmi/about_children_bmi.html. Accessed on 10th November 2022
 44. [^] WHO. Mean fasting blood glucose. www.who.int/data/gho/indicator-metadata-registry/imr-details/2380 (accessed on September 12, 2023).
 45. [^] Weiss R, Bremer AA, Lustig RH. What is metabolic syndrome, and why are children getting it? *Ann N Y Acad Sci* 2013;1281:123-40.
 46. ^{a, b, c} Al-Hamad D, Raman V. Metabolic syndrome in children and adolescents. *Transl Pediatr* 2017;6(4):397-407.

47. [^]Ramesh S, Abraham RA, Sarna A, Sachdev HS, Porwal A, Khan N, Acharya R, Agrawal PK, Ashraf S, Ramakrishnan L. Prevalence of metabolic syndrome among adolescents in India: a population-based study. *BMC Endocr Disord.* 2022;22(1):258.
48. [^]Lwabukunaw C, Mgonday. (). Early clinical markers of metabolic syndrome among secondary school adolescents in Dar es Salaam, Tanzania. *Tanza J Health Res,* 2021.
49. [^]Sekokotla MA, Goswami N, Sewani-Rusike CR, Iputo JE, Nkeh-Chungag BN. Prevalence of metabolic syndrome in adolescents living in Mthatha, South Africa. *Ther Clin Risk Manag* 2017;13 131–137.
50. [^]Salako AO, Gbaja-Biamila TA, Ezemelue PN, Musari-Martins TS. Metabolic syndrome among adolescents and young adults living with HIV in Lagos: A cross-sectional study. *Global Pediatrics,* 2021; 1, 100001.
51. [^]Onyenekwu CP, Dada AO, Babatunde OT. The prevalence of metabolic syndrome and its components among overweight and obese Nigerian adolescents and young adults. *Niger J Clin Pract.* 2017;20(6):670-676.
52. [^]Agirbasli M, Cakir S, Ozme S, Cilliv G. Metabolic syndrome in Turkish children and adolescents. *Metabolism* 2006; 55: 1002- 06.
53. [^]Steele RM, Brage S, Corder K, Wareham NJ, Ekelund U. Physical activity, cardiorespiratory fitness, and the metabolic syndrome in youth. *J Appl Physiol* 2008; 105: 342-51.
54. [^]Brambilla P, Pozzobon G, Pietrobelli A. Physical activity as the main therapeutic tool for metabolic syndrome in childhood. *Int J Obes (Lond)* 2011; 35: 16-28.
55. [^]Çelebi MM. Metabolic syndrome and physical activity. *Turkiye Klinikleri J Sports Med-Special Topics* 2015; 1: 13-23.
56. ^{a, b}Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. *Ther Adv Cardiovasc Dis* 2017; 11: 215- 25.
57. [^]Adediran OS, Jimoh AK, Ogbera AO. Metabolic syndrome: The pathogenesis of its predictors. *Postgrad Doctor Caribbean* 2006;22:35-45.
58. [^]Mufunda J, Sigola LB, Chifamba J, Vengesa PM: Hyperinsulinemia: Possible cause of high blood pressure in unemployed urban black women. *High Blood Pressure* 1995;4:137-40.
59. ^{a, b}Bharti A, Kushwaha A. Metabolic syndrome: pathophysiology and consequences. *Int J Curr Microbiol App Sci* 2020; 9: 3723- 28.
60. [^]Wallace AM, McMahon AD, Packard CJ, et al. on behalf of the WOSCOPS Executive Committee. Plasma leptin and the risk of cardiovascular disease in the west of Scotland coronary prevention study (WOSCOPS). *Circulation* 2001; 104(25): 3052–3056.
61. [^]Rochlani Y, Pothineni NV, Kovelamudi S, Mehta JL. Metabolic syndrome: pathophysiology, management, and modulation by natural compounds. *Ther Adv Cardiovasc Dis.* 2017;11(8):215-225.
62. [^]Agbre-Yace ML, Oyenusi EE, Oduwale AO, Ake MD, Abodo JR. Prevalence of diabetes mellitus among children and adolescents in the district of Abidjan in Cote d'Ivoire: a population-based study. *J Diabetes Metab Disord.* 2016;15:38.
63. ^{a, b}Kane JP, Pullinger CR, Goldfine ID, Malloy MJ. Dyslipidemia and diabetes mellitus: Role of lipoprotein species and interrelated pathways of lipid metabolism in diabetes mellitus. *Curr Opin Pharmacol.* 2021;61:21-27.
64. [^]Ogbera A. Prevalence and gender distribution of the metabolic syndrome. *Diabetol Metab Syndrome* 2010;2:1.
65. [^]GBD 2015 Mortality and Causes of Death Collaborators, Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet,* 2016, 388(10053): p. 1459-1544.
66. ^{a, b}Mohammed O, Kassaw M, G/Egzeabher L, Fekadu E, Bikila D, Getahun T, Challa F, Abdu A, Desta K, Wolde M, Tsegaye A. Prevalence of Dyslipidemia among School-Age Children and Adolescents in Addis Ababa, Ethiopia. *J Lab Physicians.* 2022;14(4):377-383.
67. ^{a, b}Onyiriuka AN, Iduoriyekemwen NJ, Sadoh WE. (2021 Prevalence and pattern of dyslipidaemia among Nigerian adolescents living in Benin City: A school-based cross-sectional study. *Sri Lanka J Child Health.* 2021;50(3):385-392.
68. ^{a, b}Puri S, Puri S, Rehan HS, Sabharwal A, Nanda R, Aggarwal SK, et al. Prevalence and pattern of Dyslipidemia in 2500 adolescents in suburban India. *J Am Coll Cardiol.* 2015;65(10):A1486.
69. [^]Kowara M, Cudnoch-Jedrzejewska A. Pathophysiology of Atherosclerotic Plaque Development-Contemporary Experience and New Directions in Research. *Int J Mol Sci.* 2021;29;22(7):3513.
70. [^]Hassing HC, Surendran RP, Mooij HL, Stroes ES, Nieuwdorp M, Dallinga-Thie GM. Pathophysiology of hypertriglyceridemia. *Biochim Biophys Acta.* 2012;1821(5):826-32.

71. ^{a, b}Liang HJ, Zhang QY, Hu YT, Liu GQ, Qi R. Hypertriglyceridemia: A Neglected Risk Factor for Ischemic Stroke? *J Stroke*. 2022;24(1):21-40.
72. [^]Altalebi RR, Al-hussaniy HA, Al-tameemi ZS, AL-Zobaigy MA4, Albu-Rghaif AH, et al. Non-alcoholic fatty liver disease: relation to juvenile obesity, lipid profile, and hepatic enzymes. *J Med Life*, 2023;16(1):42-47.
73. [^]AlMuhaidib S, AlBuhairan F, Tamimi W, AlDubayee M, AlAqeel A, Babiker A, AlFaraidi H, AlJuraibah F, Badri M, Al Alwan I. Prevalence, and factors associated with dyslipidemia among adolescents in Saudi Arabia. *Sci Rep*. 2022;12(1):16888.
74. [^]Bauman CD, Bauman JM, Mourão DM, Pinho L, Brito MFSF, Carneiro ALG, Silveira MF, Silva CSOE. Dyslipidemia prevalence in adolescents in public schools. *Rev Bras Enferm*. 2020;73(3):e20180523.
75. [^]Zhou L, Li C, Gao L, Wang A. High-density lipoprotein synthesis and metabolism (Review). *Mol Med Rep*. 2015;12(3):4015-4021.
76. [^]Li Y, Xu Y, Jadhav K, Zhu Y, Yin L, Zhang Y. Hepatic forkhead box protein A3 Regulates ApoA-I (Apolipoprotein A-I) expression, cholesterol efflux, and atherogenesis. *Arterioscler Thromb Vasc Biol*. 2019;39:1574–87.
77. [^]Connelly MA, Shalurova I, Otvos JD. High-density lipoprotein and inflammation in cardiovascular disease. *Transl Res*. 2016;173:7-18.