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Evaluation of Serum Lipid Profile at Different Trimesters among Pregnant Women in Ovia North-East Local Government Area, Edo State, Nigeria

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Abstract

The aim of this study was to evaluate the lipid profile of pregnant women at different trimester in Ovia North-East Local Government Area, Edo State, Nigeria. A total of 200 blood specimens (150 pregnant women and 50 non-pregnant women), aged between 18-49 years, were randomly collected from consenting women. Biochemical parameters including: Total Cholesterol (TCHL), Triglycerides (TG), High density lipoprotein (HDL) and Low density lipoprotein (LDL) were analyzed using standard spectrophotometric technique. A standard Height and Weight Measurement Unit (HWMU) was used to measure the height (m) and weight (Kg) of the participants and their BMI thereafter calculated using the formula: Weight/Square Height (Kg/m2). Data were entered into Microsoft Excel and statistical analysis was carried out using SPSS-18.0 (Statistical Package for Social Sciences - Version 18.0) Software Package. In comparison with the control, the serum levels of Triglycerides (TG), High density lipoprotein (HDL), as well as Very Low density lipoprotein (VLDL) changed significantly (P=0.00) in pregnant women with progression in pregnancy particularly in the third trimesters. While on the other hand, the serum levels of Total Cholesterol (TCHL) and the Low density lipoprotein (LDL) of the pregnant women were significantly (P=0.00) elevated in the second trimester compared to the control. TCHL (189±4.23 mg/dl), TG (166±5.12 mg/dl), HDL (78±2.59 mg/dl), VLDL (33.0±1.02 mg/dl), and BMI (24.2±0.4 Kg/m2) were significantly (P=0.00) elevated in the pregnant women than the control (142±5.34 mg/dl, 108±5.02 mg/dl, 39±3.20 mg/dl, 23.1±1.5 mg/dl, and 22.1±0.3 Kg/m2, respectively), except LDL (P=0.90). The outcome of this study shows that hyperlipidemia is highest in the third trimester of pregnancy and pregnant women

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suffering from atherosclerosis, are at risk of coronary heart disease this period; hence the need for close monitoring of the lipid profile during this phase of pregnancy.

Keywords: Lipid profile, BMI, Pregnancy, Trimester, Women, Edo State, Nigeria

1. Introduction

Significant variations in maternal lipid metabolism has been associated with pregnancy. In early pregnancy, there is increased body fat accumulation associated with both hyperphagia and increased lipogenesis; while in late pregnancy there is an accelerated breakdown of fat depots, which plays an important role in foetal development. Inconsistent observations on normal and abnormal pregnancies have been reported in previous studies (Fahy et al., 2009). Increase in maternal lipid profile during pregnancy differs with trimester. It has been observed that the concentration of serum total cholesterol, serum triglyceride, high density lipoprotein cholesterol and low density lipoprotein cholesterol in normal pregnant women increased with progression in pregnancy (Hunter, 2006). According to Guan and Wenk (2008) increase in the maternal lipid profile in the third trimester is in response to the maternal switch from carbohydrate to fat metabolism which is an alternative pathway for energy generation due to high energy demand (Russell, 2003).

During pregnancy, lipids in addition to carbohydrates are necessary classes of food that provide energy for the increased cellular proliferation of the fetus and the multisystem adaptation of the mother. However, to be able to perform their physiological functions, lipids are bound to specific proteins to form lipoproteins which provide solubility in the aqueous environment for metabolism (Ojule et al., 2005). Abnormal lipid metabolism with increased serum lipid profile creates a setting for atherosclerosis which is a risk factor for the development of coronary artery disease (CHD). Studies have shown that the circulating concentrations of triglycerides, low density lipoprotein, high density lipoprotein and total cholesterol rise during pregnancy. This is necessary because of the high energy required for the maternal cellular proliferation, uterine enlargement, blood volume expansion, as well as fetal implantation, formation of blood vessels in the utero placenta area, feto-placenta development and growth (Deepak and Digisha, 2011). Nevertheless, a high lipid profile is a risk factor for CHD and can adversely affect the health of the pregnant woman and her fetus (Glew et al., 2004). The increased lipid profile in pregnancy is traceable to pancreatic beta cell hyperplasia, hyperinsulinemia, hyperestrogenemia and hyperprogesteronemia. Hyperinsulinemia leads to an increase in peripheral glucose utilization, a decline in fasting plasma glucose levels, increased tissue storage of glycogen, increased storage of fats and decreased lipolysis Deepak and Digisha, 2011).

Furthermore, pregnancy has been described as a state of increased insulin resistance, insulin secretion and of reduced hepatic insulin extraction. The principal modulator of this hypertriglyceridemia is oestrogen as pregnancy is associated with hyperoestrogenaemia. The hyperinsulinism found in pregnancy modulates the estrogen induced hepatic biosynthesis of endogenous triglycerides, which is carried by VLDL (Adegoke et al., 2003). Changes in the plasma lipids during pregnancy have been recognized and thought to be mostly due to alterations

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in the hormonal milieu in the form of rise in insulin, progesterone, 17-B estradiol and human placental lactogen. Progesterone and estrogen are modulators of lipids metabolism and their concentration increase as pregnancy advances (Coleman and Lee, 2004).

Cardiovascular disease (CVD) on the other hand, is the leading global cause of death, accounting for 17.3 million deaths per year. Currently, 0.2–4% of all pregnancies in western industrialized countries are complicated by CVD, and the number of the patients who develop cardiac problems during pregnancy is increasing. Furthermore, pregnancy may cause specific cardiovascular disorders, which can impose a risk to the pregnant woman and her fetus. This risk is in the ascending order as the age of first pregnancy is increasing and as the number of cardiovascular risk factors is rising (e.g. smoking, hypercholesterolemia, diabetes, hypertension, obesity (Cribbs et al., 2017).

Previous studies have shown that low serum concentrations of high density lipoproteins (HDL) together with high serum concentrations of total cholesterol (TC), triglycerides (TG) and lipoproteins are associated with an increased risk of coronary heart disease (Cribbs et al., 2017). There is growing body of evidence showing that hyperglycaemia and hyperlipidemia are linked to increased cardiovascular risk. And that high levels of serum TC, triglycerides, LDL, VLDL, micro-albuminuria, low concentration of HDL and increased body mass index (BMI) are significantly associated with coronary heart disease. Hypertension, dyslipidemia and excess body weight are among the most potent accepted risk factors for CVD and atherogenesis. Pregnancy has been identified as a factor unique to women that may be associated with dyslipidaemia and obesity, and several studies have shown that it may affect them unfavourably. Lipids have been observed to be high or elevated during pregnancy and abnormal level of total cholesterol and triglyceride predisposes an individual to the development of atherosclerotic coronary artery disease (Applebaum et al., 1997).

Cardiovascular disease is common among pregnant women due to excess accumulation of lipids in blood vessels. Pregnancy has been described as a state of increased insulin resistance and insulin secretion and of reduced hepatic insulin extraction. The principal modulator of this in the plasma lipids during pregnancy have been recognized and thought to be mostly due to alterations in the hormonal milieu in the form of rise in insulin, progesterone, 17-B estradiol and human placental lactogen (Cribbs et al., 2017). Thus, it is important to find out the extent to which different trimesters affects the parameters of lipid profile. Lack of extensive studies on the effect of pregnancy on lipid profile in relation to body mass index, particularly among pregnant women dwelling in Ovia North-East Local Government Area, Edo State, Nigeria, necessitates this study. This study was therefore undertaken to ascertain the lipid profile pattern in the three trimester of pregnancy and to determine which of the trimester that is most predisposed to Coronary artery disease. It is hoped that, the outcome of this study will make a valuable contribution to previous studies in order to have a more efficient and appropriate respond to the health needs of the growing population. The aim of this study is to assess the effect of pregnancy on the lipid profile and body mass index (BMI) of pregnant women in Ovia North-East Local Government Area, Edo State, Nigeria.

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2. Method

2.1 Study Design

This is a cross-sectional descriptive-analytical study.

2.2 Study Area

The study was carried out at the Primary Health Care Centres in Ovia North East Local Government Area (LGA) of Edo-State which lies between latitude $5^{o}40^{1}$ North and Longitude $5^{0}00^{1}$ and East. The Local Government has an estimated population of 155,846 persons (NPC, 2006). Majority of the residents are farmers with few Civil Servants, Lecturers and Students making less than 5%. The Ethnic groups found in this area are the Bini tribe (who are the major tribe) and the minor tribes which include the Yorubas, Ijaw, Urhobo, Itsekiris, and Ogojas.

2.3 Duration of study

This study was carried out between the months of June and August, 2019.

2.4 Study population

This cross-sectional descriptive-analytical study was carried out among pregnant women receiving anti-natal care at various Primary Health Care Centres in Ovia North-East Local Government Area (LGA) of Edo-State.

2.5 Ethical consideration

Ethical clearance was sought from the Igbinedion University Health Research Ethics Committee (IUHREC2019034) before the commencement of the study. Also, administrative permission for the study was obtained from the Management of the Primary Health Care Centres in Ovia North-East Local Government Area (LGA) of Edo-State.

2.6 Eligibility of Subjects

2.6.1 Inclusion Criteria

Consenting pregnant women at various trimesters without history of steroids and anti-hyperlipidaemia drugs use in the preceding two weeks, as well as non-pregnant women which served as control were randomly recruited for the study.

2.6.1 Exclusion criteria

Women with history of steroids and anti-hyperlipidaemia drugs use in the preceding two weeks of the study were excluded from the study.

2.7 Informed Consent

Informed Consent was obtained from each patient and all participants were requested to voluntarily sign the consent forms in their own handwriting as proof of willingness to provide samples for the tests. The objective and the method of this study were explained to all participants to gain their consent.

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2.8 Sample Size

Minimum sample size was calculated using Fisher's formula for cross-sectional descriptive study; where minimum size is calculated using the formula:

$$N = \frac{Z^2pq}{d^2} = \frac{1.962 \text{ x } 0.20 \text{ x } (1\text{-}0.8)}{(0.05)^2} = 63.8$$

Where N= The desired sample size if population is more than 10,000

Z= The Standard Normal Deviate usually set at 1.9 corresponding to the 95% confidence level.

p= The population in the target population estimated to have a particular characteristic, p=1-p

d= degree of accuracy desired set at 0.05 (Peter et al., 2002).

A total of 200 blood specimens were randomly collected from consenting 200 women receiving health care in Primary Health Care Centres in Ovia North-East Local Government Area (LGA) of Edo-State. They consisted of 150 pregnant women (First trimester: n=50, Second trimester: n=50 and Third trimester: n=50) and 50 non-pregnant women which served as control.

2.9 Data Collection

Information was obtained from the participants through administration of a structured questionnaire. Interpreter was provided for translation in local dialect where necessary. The first part of the questionnaires contained the biodata of the patients e.g. Personal Identification Number (PIN), Age etc. Second part includes: trimester pregnancy, history of anti-hyperlipidaemia drugs use etc. For reasons of privacy, all data were kept confidential in accordance with World Medical Association declaration of Helsinki (WMA, 2008).

2.10 Measurement of anthropometric parameters

A standard Height and Weight Measurement Unit (HWMU) was used to measure the height (m) and weight (Kg) of the participants. The participants wearing light clothes were instructed to stand on the Unit without shoes, heels together and the head in the horizontal plane. Afterwards, measurements were taken and recorded appropriately.

2.11 Calculation of Body Mass Index (BMI)

The BMI of the individual participant was calculated using the formula:

Weight/Square Height (Kg/m^2).

2.12 Specimen Collection

The arm of each participant was tied with a tourniquet to make the veins prominent. The median antecubital vein was then selected and the area was disinfected with 70% alcohol. Five millilitres (5 ml) venous blood was then collected with the aid of syringe and needle. This was transferred

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into a plain bottle and allowed to clot in about 30min. It was centrifuged for 5 minutes at 3000 revolutions per minute. The serum was separated from the cells into another plain container with label corresponding to initial blood sample bottle. The serum samples were stored frozen at -20^{o}C until use for estimation of lipid profile (total cholesterol, high density lipoprotein cholesterol, low density lipoprotein cholesterol).

2.13 Laboratory Analyses

2.13.1 Determination of Serum Total Cholesterol
The serum total cholesterol (TC) was estimated by the enzymatic endpoint method using a spectrophotometer as described by Allain *et al.* (1974).

Principle:
Cholesterol is measured enzymatically in serum or plasma in a series of coupled reactions that hydrolyze cholesteryl esters and oxidize the 3-OH group of cholesterol. One of the reaction by-products, H_2O_2 is measured quantitatively in a peroxidase catalyzed reaction that produces a color. Absorbance is measured at 500 nm. The color intensity is proportional to cholesterol concentration. The reaction sequence is as follows:

$$\text{Cholesteryl ester} + H_2O \xrightarrow{\text{Cholesteryl ester hydrolase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + O_2 \xrightarrow{\text{Cholesterol oxidase}} \text{Cholest-4-en-3-one} + H_2O_2$$

$$2H_2O_2 + \text{4-aminophenazone} + \text{phenol} \xrightarrow{\text{Peroxidase}} \text{4-(p-benzoquinone-monoimino)-phenazone} + 4\ H_2O$$

2.13.2 Determination of Serum High Density Lipoprotein Cholesterol
The serum high density lipoprotein Cholesterol (HDL-C) was estimated by precipitation method using a spectrophotometer as described by Lopes-Virella (1977).

Principle:
Low density lipoprotein cholesterol (LDL-C), Very Low density lipoprotein cholesterol (VLDL-C) and Chylomicron fractions were precipitated quantitatively by the addition of phosphotungstic acid in the presence of magnesium ions. After centrifugation, the HDL-C fraction which remained in the supernatant was determined by cholesterol assay. The reagents were purchased from Roche/Boehringer-Mannheim Diagnostics. The method uses sulfated alpha-cyclodextrin in the presence of Mg+2, which forms complexes with apoB containing lipoproteins, and polyethylene glycol-coupled cholesteryl esterase and cholesterol oxidase for the HDL-cholesterol measurement. The reactions are as follows:
(1) ApoB containing lipoproteins + á-cyclodextrin + Mg^{+2} + dextran SO_4 ---> soluble non-reactive complexes with apoB-containing lipoproteins

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(2) HDL-cholesteryl esters PEG-cholesteryl esterase > HDL-unesterified cholesterol + fatty acid
(3) Unesterified chol + O_2 PEG-cholesterol oxidase > cholestenone + H_2O_2
(4) H_2O_2 + 5-aminophenazone + N-ethyl-N-(3-methylphenyl)-N'_succinyl ethylene diamine + H_2O + H+ peroxidase > qunoneimine dye + H_2O. Absorbance was measured at 600 nm.

2.13.3 Determination of Triglyceride

The serum triglyceride (TG) was estimated using glucerol-3-phosphate oxidase- peroxidase (GPO-PAP) method as described by Jacobs and Vandermark (1960) using a Spectrophotometer.

Principle:

Triglycerides were measured enzymatically in serum or plasma using a series of coupled reactions in which triglycerides are hydrolyzed with lipases to produce glycerol. Glycerol is oxidized using glycerol oxidase, and H_2O_2, one of the reaction products, is measured as described above for cholesterol. The indicator is a quinone imine formed from hydrogen peroxide, 4-aminophenazone and chlorophenol under the catalytic influence of peroxidase. Absorbance is measured at 500 nm. The reaction sequence is as follows:

$$\text{Triglycerides} + 3H_2O \xrightarrow{\text{Lipase}} \text{glycerol} + \text{fatty acids}$$

$$\text{Glycerol} + \text{ATP} \xrightarrow{\text{Glycerokinase}} \text{glycerol-3-phosphate} + \text{ADP}$$

$$\text{Glycerol-3-phosphate} + O_2 \xrightarrow{\text{Glycerophosphate oxidase}} \text{dihydroxyacetone phosphate} + H_2O_2$$

$$H_2O_2 + \text{4-aminophenazone} + \text{4-chlorophenol} \xrightarrow{\text{Peroxidase}} \text{4-(p-benzoquinone-monoimino)-phenazone} + 2H_2O + HCl$$

2.13.4 Determination of Low Density Lipoprotein Cholesterol

The serum low density lipoprotein (LDL-C) was calculated using Friedwald's formula (Friedewald et al., 1972).
LDL-C = Total cholesterol - (HDL + Triglyceride/2.2).

2.13.5 Determination of Very Low Density Lipoprotein Cholesterol

The serum very low density lipoprotein Cholesterol (VLDL-C) was calculated using the formular as described by Friedewald *et al.* (1972):

LDL-Cholesterol (mg/dl) = Total cholesterol – HDL cholesterol – TG/5

2.14 Statistical Analysis

Statistical data for the parameters were analyzed using mean, standard deviation, students t-test and p-value. The ordinal or quantitative data were separated according to the ordinal

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classification using SPSS-18.0 (Statistical Packages for Social Science Version-18.0). Events difference were considered with significant when probability of equality was lower than 0.05 ($p<0.05$), while events were considered related when the probability of absence of relationship was lower than 0.05 ($p<0.05$).

3. Results

This present study assessed the effect of pregnancy on the lipid profile and body mass index (BMI) of pregnant women in Ovia North-East Local Government Area, Edo State, Nigeria. A total of 200 women (150 pregnant and 50 non-pregnant) receiving health care in Primary Health Care Centers in Ovia North-East Local Government Area (LGA) of Edo-State were recruited for the study.

The socio-demographic characteristics of the study participants are presented in Table 1. Thirty-three, 33 (16.5%) of them were between 18-25 years, 58 (29.0%) between 26-33 years, 90 (45.0%) between 34-41 years, and 19 (9.5%) between 42-49 years. Majority of them (75.0%) were Christians by religion, followed by Muslims (17.5%), while the remaining 7.5% were Traditional Worshippers. On the basis of tribal distribution, most of them were Binis (58.3%), followed by the Yorubas (18.4%), Igbos (17.5%), and others who were neither of the above mentioned tribes (5.0%). Based on their educational status, most of them had secondary education (47.5%), followed by primary education (25.0%), no formal education (15.0%) and lastly, tertiary education (12.5%). Considering their marital status, most of them were married (92.5%), followed by those who indicated that they were singles (5.0%), the separated (1.5%) and then the divorced (1.0%).

The anthropometric parameters of the pregnant women, particularly their heights, height squares, weights and body mass index (BMI) at different trimester of pregnancy in comparison to the non-pregnant women are presented in Table 2. The weights and the BMIs of the pregnant women particularly in the second (63.25±4.20 Kg and 24.7±0.82 Kg/m^2, respectively) and third (67.25±4.20 Kg and 26.0±0.51 Kg/m^2, respectively) trimesters were significantly higher ($p<0.05$) than the control (62.24±5.43 Kg and 22.1±0.31 Kg/m^2).

Table 3 shows the lipid profile of the pregnant women at different trimester of pregnancy in comparison to the non-pregnant women. In comparison with the control, the serum levels of Triglycerides (TG), High density lipoprotein (HDL), as well as Very Low density lipoprotein (VLDL) increased significantly (P=0.00) in pregnant women with progression in pregnancy particularly in the third trimesters. While on the other hand, the serum levels of Total Cholesterol (TCHL) and the Low density lipoprotein (LDL) of the pregnant women were significantly (P=0.00) elevated in the second trimester compared to the control.

The lipid profile and BMI of the 150 pregnant women in comparison to the 50 non-pregnant women (control) assessed are presented in Table 4. All the lipid parameters under consideration: TCHL (189± 4.23 mg/dl), TG (166±5.12 mg/dl), HDL (78±2.59 mg/dl), VLDL (33.0±1.02

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mg/dl), and BMI (24.2±0.4 Kg/m^2) were significantly (P=0.00) elevated in the pregnant women than the control (142±5.34 mg/dl, 108±5.02 mg/dl, 39±3.20 mg/dl, 23.1±1.5 mg/dl, and 22.1±0.3 Kg/m^2, respectively), except LDL with no significant difference (P=0.90).

Table 5 shows the lipid profile and Body mass index of pregnant women in their first trimester as compared to those in their second trimester. While on one hand, all the lipid parameters assessed were significantly (P=0.00) elevated in the second trimester compared to the first trimester, on the other hand, the BMI did not (P=0.47).

Table 1: Socio-demographic characteristics of the study participants

Socio-demographic Characteristics	Category	Frequency (N)	Percentage (%)
Age Range	18-25yrs	33	16.5
	26-33yrs	58	29.0
	34-41yrs	90	45.0
	42-49yrs	19	9.5
	Above 50yrs	0	0
	Total	**200**	**100.0**
Religion	Christianity	150	75.0
	Islam	35	17.5
	Traditional	15	7.5
	Total	**200**	**100.0**
Tribe	Yoruba	37	18.4
	Igbo	35	17.5
	Hausa	2	0.8
	Bini	116	58.3
	Others	10	5.0
	Total	**200**	**100.0**
Educational Status	None	30	15.0
	Primary	50	25.0
	Secondary	95	47.5
	Tertiary	25	12.5
	Total	**200**	**100.0**
Marital status	Single	10	5.0
	Married	185	92.5
	Divorced	2	1
	Separated	3	1.5
	Widow	0	0
	Total	**200**	**100.0**

Table 2: Anthropometric parameters of pregnant women based on their trimester of pregnancy in comparison to the non-pregnant women.

Parameters	Test (Pregnant) Trimesters			Control (Non-pregnant)	F-value	p-value	Remark
	1st (n=50) Mean±SEM	**2nd (n=50) Mean±SEM**	**3rd (n=50) Mean±SEM**	**(n=50) Mean±SEM**			
Weight (Kg)	61.08±6.57	63.25±4.20*	67.25±4.20*	62.24±5.43	12.1	0.000	S
Height (m)	1.65±0.08	1.65±0.08	1.65±0.08	1.68±0.10	18.4	1.000	NS
Height^2 (m^2)	2.72±0 .05	2.72±0 .05	2.72±0 .05	2.82±0.01	18.4	1.000	NS
BMI (Kg/m^2)	21.9±0.51	24.7±0.82*	26.0±0.51*	22.1±0.31	12.5	0.000	S

Keys: *Body Mass Index = BMI, SEM = Standard Error of Mean, n = Number of subjects, S = Significant, NS = Non-significant. NB: *P<0.05 is considered statistically significant. The weights and the BMIs of the pregnant women in the second and third trimesters were significantly higher (p<0.05) than the control.*

TABLE 3: Lipid profile of pregnant women based on their trimester of pregnancy in comparison to the non-pregnant women

Parameters	**Test (Pregnant) Trimester**			**Control (Non-Pregnant)**	**F-value**	**p-value**	**Remark**
	1st (n=50) Mean±SEM	**2nd (n=50) Mean±SEM**	**3rd (n=50) Mean±SEM**	**(n=50) Mean±SEM**			
TCHL (mg/dl)	162±3.41	214±8.42*	191±7.42*	142±5.33	24.4	0.000*	S
TG (mg/dl)	116±3.41	160±6.81*	222±8.021*	109±5.02	73.3	0.000*	S
HDL (mg/dl)	52.2±2.71*	90.0±4.83*	93.0±4.83*	38.5±2.60	63.0	0.000*	S
LDL (mg/dl)	23.1±0.68*	92.1±8.12	57.2±8.11*	81.0±4.51	10.9	0.000*	S
VLDL (mg/dl)	23.1±0.68	32.0±1.40*	43.9±1.70*	23.1±1.50	52.4	0.000*	S

Keys: *TCHL = Total Cholesterol, TG = Triglycerides, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very Low density lipoprotein, SEM = Standard error of mean, n = Number of subjects, S = Significant. *P<0.05 is considered statistically significant. NB: TG, HDL, and VLDL increased significantly (P=0.00) in the third trimester. While TCHL and the LDL were significantly (P=0.00) elevated in the second semester compared to the control.*

TABLE 4: Lipid profile and Body Mass Index of pregnant women in comparison to the non-pregnant women

PARAMETER	PREGNANT (±SEM) n=150	NON-PREGNANT (±SEM) n=50	F-value	p-value	Remark
TCHL (mg/dl)	189± 4.23	142±5.34	3.05	0.00	S
TG (mg/dl)	166±5.12	108±5.02	17.6	0.00	S
HDL (mg/dl)	78±2.59	39±3.20	7.03	0.00	S
LDL (mg/dl)	83±4.2	82±4.5	6.5	0.902	NS
VLDL (mg/dl)	33.0±1.02	23.1±1.5	6.5	0.00	S
BMI (Kg/m^2)	24.2±0.4	22.1±0.3	23.4	0.00	S

Keys: *TCHL = Total Cholesterol, TG = Triglycerides, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very Low density lipoprotein, SEM = Standard error of mean, n = Number of subjects, S = Significant. NB: *P<0.05 is considered statistically significant. TCHL, TG, HDL, VLDL and BMI were significantly (P=0.00) elevated than the control, except LDL with no significant difference (P=0.90).*

TABLE 5: Lipid profile and Body mass index of pregnant women in their first trimester as compared with second trimester

Parameters	Trimester		p-value	Remark
	1st (n=50) Mean±SEM	2nd (n=50) Mean±SEM		
TCHL (mg/dl)	162±3.41	214±8.42*	0.00*	S
TG (mg/dl)	116±3.41	160±6.81*	0.00*	S
HDL (mg/dl)	52.2±2.71*	90.0±4.83*	0.00*	S
LDL (mg/dl)	23.1±0.68*	92.1±8.12*	0.00*	S
VLDL (mg/dl)	23.1±0.68	32.0±1.40*	0.00*	S
BMI (Kg/m^2)	21.9±0.51	24.7±0.82	0.47	NS

Keys: *TCHL = Total Cholesterol, TG = Triglycerides, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very Low density lipoprotein, SEM = Standard error of mean, n = Number of subjects, S = Significant. NB: *P<0.05 is considered statistically significant. All the parameters were significantly (P=0.00) elevated in the second trimester compared to the first trimester, except BMI (P=0.47).*

TABLE 6: Lipid Profile and Body Mass Index of pregnant women in their first trimester as compared with third trimester.

Parameters	**Trimester**		**p-value**	**Remark**
	1st (n=50) Mean±SEM	**3rd (n=50) Mean±SEM**		
TCHL (mg/dl)	162±3.41	191±7.42*	0.00*	S
TG (mg/dl)	116±3.41	222±8.021*	0.00*	S
HDL (mg/dl)	52.2±2.71	93.0±4.83*	0.00*	S
LDL (mg/dl)	23.1±0.68	57.2±8.11*	0.00*	S
VLDL (mg/dl)	23.1±0.68	43.9±1.70*	0.00*	S
BMI (Kg/m^2)	21.9±0.51	26.0±0.51	0.00*	S

Keys: *TCHL = Total Cholesterol, TG = Triglycerides, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very Low density lipoprotein, SEM = Standard error of mean, n = Number of subjects, S = Significant. NB: *P<0.05 is considered statistically significant. All the parameters were significantly (P=0.00) elevated in the third trimester compared to the first trimester.*

TABLE 7: Lipid Profile and Body Mass Index of pregnant women in their second trimester as compared with third trimester.

Parameters	**Trimester**		**p-value**	**Remark**
	2nd (n=50) Mean±SEM	**3rd (n=50) Mean±SEM**		
TCHL (mg/dl)	214±8.42	191±7.42*	0.00*	S
TG (mg/dl)	160±6.81	222±8.021*	0.00*	S
HDL (mg/dl)	90.0±48.3	93.0±4.83*	0.00*	S
LDL (mg/dl)	92.1±8.12	57.2±8.11*	0.00*	S
VLDL (mg/dl)	32.0±0.14	43.9±1.70*	0.00*	S
BMI (Kg/m^2)	24.7±0.82	26.0±0.51	0.47	NS

Keys: *TCHL = Total Cholesterol, TG = Triglycerides, HDL = High density lipoprotein, LDL = Low density lipoprotein, VLDL = Very Low density lipoprotein, SEM = Standard error of mean,*

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*n = Number of subjects, S = Significant. *P<0.05 is considered statistically significant. All the parameters were significantly (P=0.00) elevated in the third trimester compared to the second trimester, except BMI (P=0.47*).

Similarly, the lipid Profile and Body Mass Index of pregnant women in their first trimester as compared to those in their third trimester are presented in Table 4.6 All the lipid parameters, as well as the BMI were significantly (P=0.00) elevated in the third trimester compared to the first trimester.

Still, the lipid Profile and Body Mass Index of pregnant women in their second trimester as compared to those in their third trimester are presented in Table 7. While all the lipid parameters assessed were significantly (P=0.00) elevated in the third trimester compared to the second trimester, the BMI did not (P=0.47)

Figure 1 shows the correlation and regression analysis between BMI and TCHL. It shows that there is no relationship between total cholesterol of pregnant women and BMI (R^2=0.00; p>0.05).

Figure 2 shows the correlation and regression analysis between BMI and TG. It shows that there is a strong relationship between triglyceride of pregnant women and BMI (R^2=0.12; p=0.00).

Figure 3 shows the correlation and regression analysis between BMI and HDL-C. It shows that there is a no relationship between HDL-C of pregnant women and BMI (R^2=0.00; p=0.86).

Figure 4 shows the correlation and regression analysis between BMI and LDL-C. It shows that there is a no relationship between LDL-C of pregnant women and BMI (R^2=0.02; p=0.10).

Figure 5 shows the correlation and regression analysis between BMI and VLDL-C. It shows that there is a strong relationship between VLDL-C of pregnant women and BMI (R^2=0.12; p=0.00).

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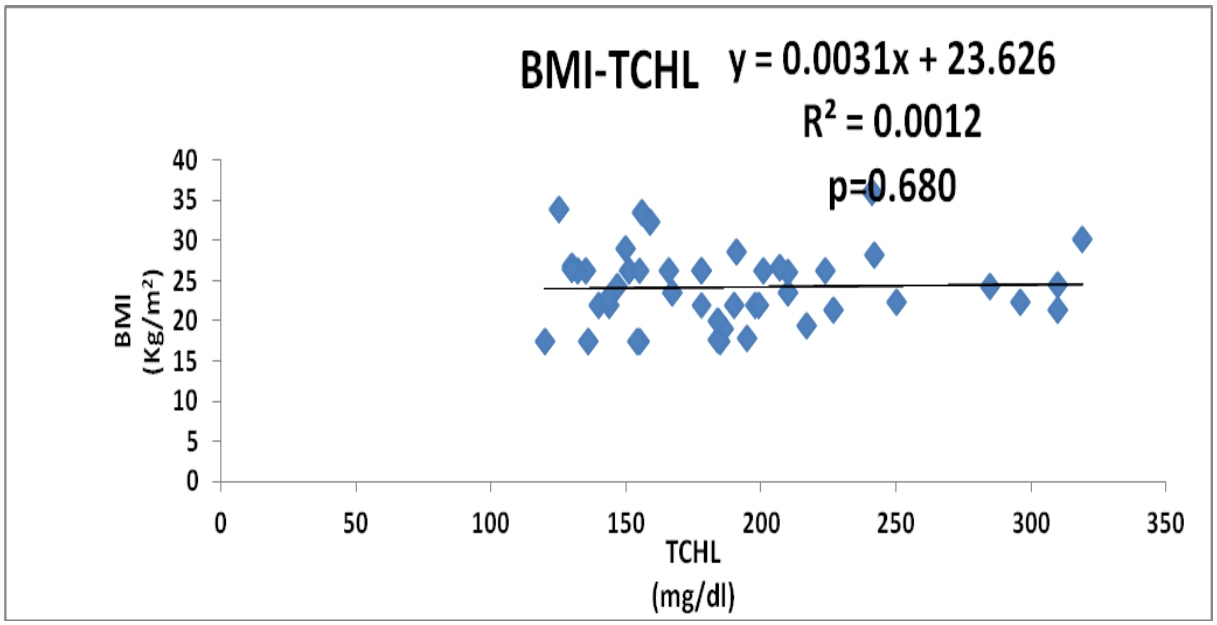

Figure 1: Correlation and regression analysis between BMI and TCHL of pregnant women.

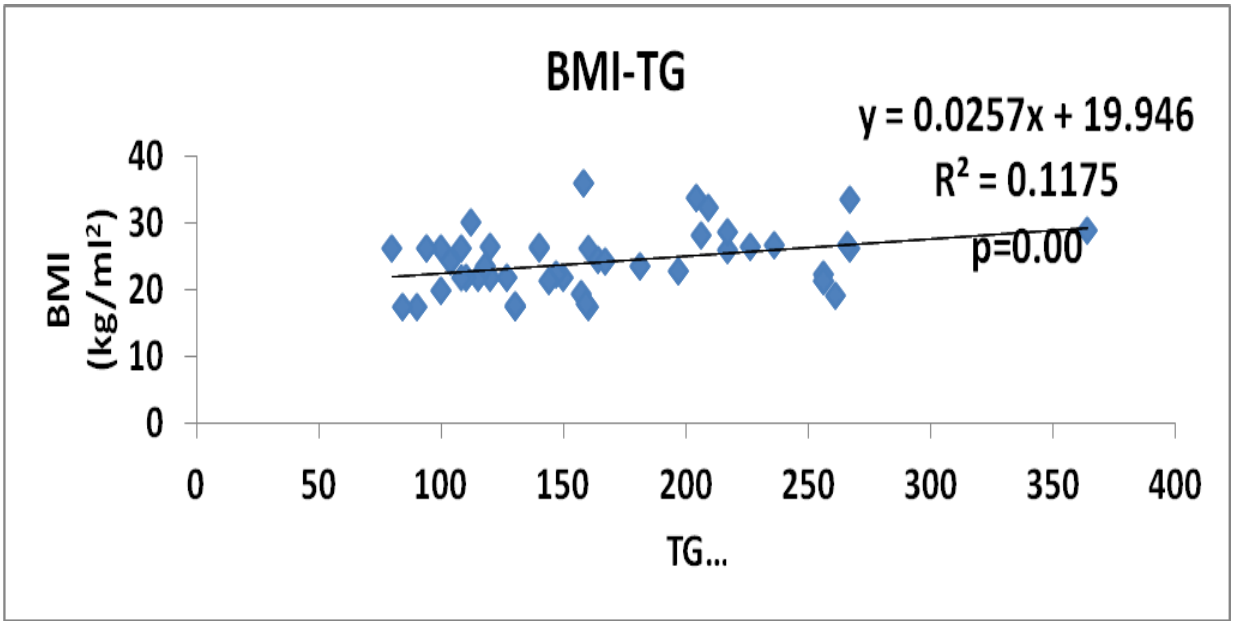

Figure 2: Correlation and regression analysis between BMI and TCHL of pregnant women.

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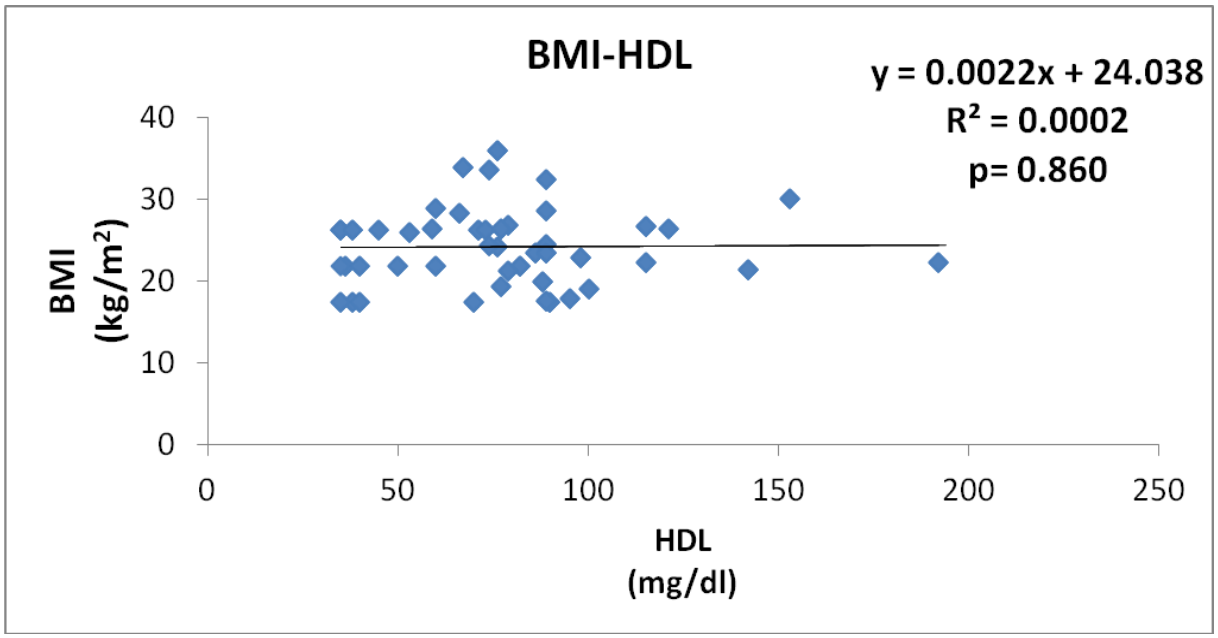

Figure 3: Correlation and regression analysis between BMI and HDL of pregnant women.

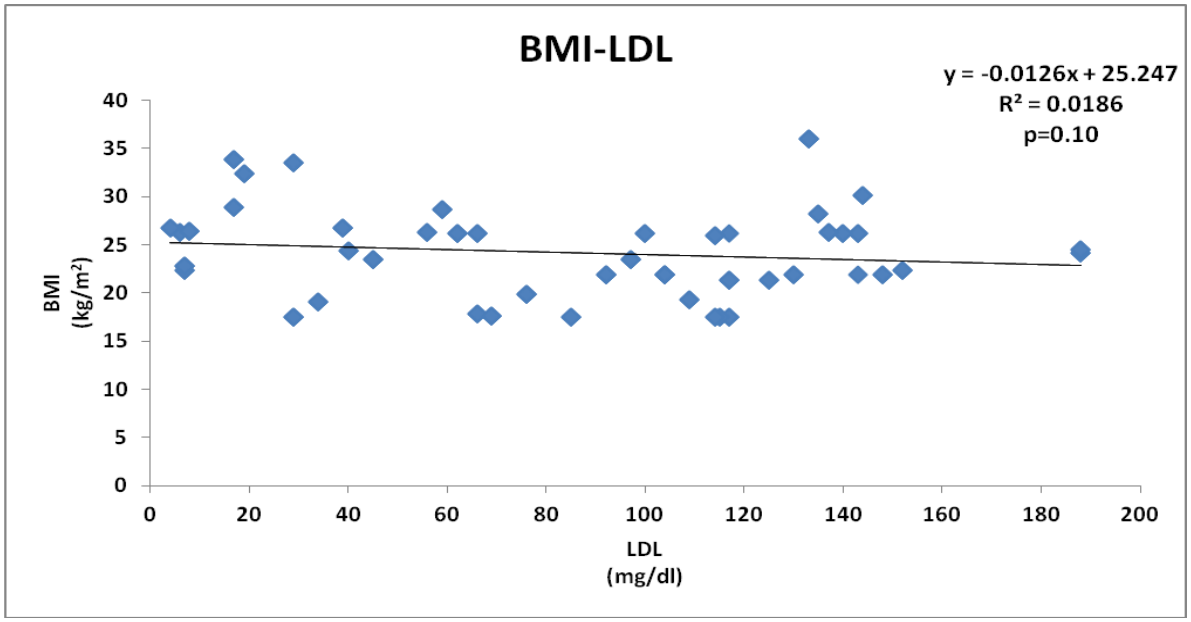

Figure 4: Correlation and regression analysis between BMI and LDL-C of pregnant women.

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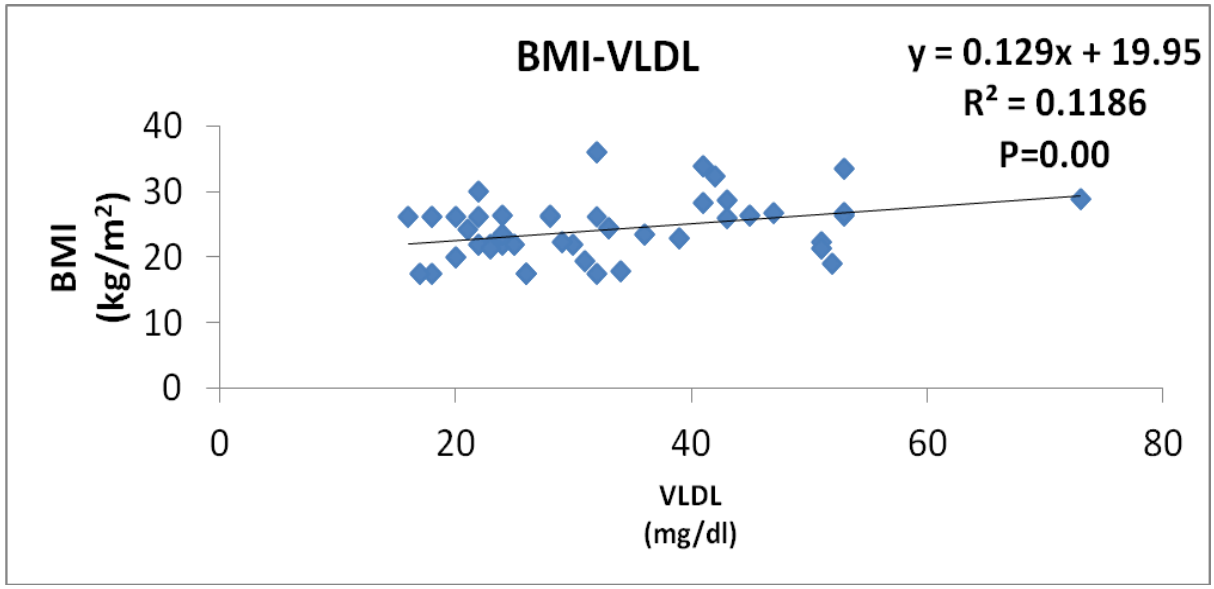

Figure 5: Correlation and regression analysis between BMI and VLDL-C of pregnant women.

4. Discussion

Some previous studies showed that many changes in lipid profile in normal pregnancy results in increased serum triglyceride level which may double in the third trimester (Pusukuru et al., 2016). In this study, our observation holds truth. It was observed that the concentration of serum Total cholesterol(TCHL), Triglyceride (TG), Very Low Density Lipoprotein Cholesterol (VLDL-C) High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol and BMI in pregnant women increased with increasing gestational age although LDL-C decrease in the third trimester of normal pregnant than in non-pregnant women. Similar observation was reported by Parchwani and Patel (2011) and Pusukuru et al. (2016). The increase in TG level is oestrogen modulated as pregnancy is associated with increase in serum oestrogen level. The endogenous biosynthesis of TG is induced by oestrogen which is carried by VLDL (Chatuphonprasert et al., 2018).

Furthermore, the study shows that TC, HDL-C, TG, BMI and VLDL of pregnant women were significantly higher than that of non-pregnant women. This is similar to the study by Salisu and Atiku (2009) which explains that an increased BMI, TC and HDL of pregnant women compared with non-pregnant women. Our study also showed that BMI, TC, HDL-C, LDL-C, VLDL-C and TG levels of pregnant women in second trimester were higher than the non-pregnant women. This is in consonance with the findings of Wald and Guckle (1988) which explains that increase in maternal lipid profile is in response to maternal switch from carbohydrate to fat metabolism which is an alternate pathway or energy due to high energy demand as pregnancy advances.
BMI, TC, TG, VLDL-C and HDL-C of the test subject in third trimester were higher than those of non-pregnant subjects. A study carried out by Pusukuru et al. (2016) reported similar findings.

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This study also showed a significant increase ($p< 0.05$) in TCHL, TG, VLDL-C, HDL-C, and LDL-C, levels during the second trimester of pregnancy when compared to the first trimester though the BMI of the second trimester is not significantly different. This study also showed significant increase ($p< 0.05$) in TCHL, TG, VLDL-C, HDL-C, and LDL-C levels during the third trimester when compared with the second trimester of pregnancy. This agrees with the study conducted by Salisu and Atiku (2009) in which a significant increase in these parameters where in the third trimester when compared with the second trimester.

In addition, maternal BMI was associated with blood lipid concentrations among the pregnant women as a higher BMI was significantly ($p<0.05$) associated with higher concentrations of triglyceride and VLDL-C among the pregnant participants. Our result is in accord with the study by Geraghty et al. (2016) in which significant ($p< 0.05$) between BMI and TG as pregnancy progresses. Pregnancy is accompanied by extra demand of energy with a well-integrated metabolic shift to ensure adequate supply of nutrients to a constantly feeding fetus from an intermittently fasting and feeding mother (Deepak and Digisha, 2011). Maternal hyperlipidemia and accumulation of fats in maternal tissues and are two consistent manifestations of altered metabolism of fat during uncomplicated pregnancy. The increase in adipose tissue store as anticipation for fetal growth spurt is indicated in this study by the concomitant increase in cholesterol concentration as pregnancy advanced. The high energy demand associated with advancing pregnancy necessitates an increase in maternal lipid profile and metabolism which is a collateral pathway for production of energy. This maternal switch from carbohydrate to fat metabolism is accompanied by an increase in hepatic lipase activity and a decrease in in lipoprotein lipase activity (Patrizia et al., 1999). Furthermore, the increase in cholesterol may be an adaptation by the body to serve its function as a precursor for the formation of the steroid hormones of pregnancy. Cholesterol is also the precursor of steroid hormones such as progesterone and of metabolic mediators such as oxysterol (Woolet, 2007). Hyperinsulinemia of pregnancy leads to an increase in peripheral glucose utilization, a decline in fasting plasma glucose levels, increased tissue storage of glycogen, increased storage of fats and decreased lipolysis. Maternal fuel adjustments during late pregnancy include a sparing of glucose (for the fetus) and an increased concentration of fatty acids in plasma. Estrogen and progesterone rise considerably during pregnancy to modify the maternal metabolic environment (Deepak and Digisha, 2011). With reference to the control, the pattern of triglyceride increase in this study was about three-fold increase in the first trimester and about four fold increases for the second and the third trimesters. Generally, normal pregnancy is also associated with high concentrations of estrogens which may contribute to the rise in total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglyceride especially in the late half of pregnancy. The concentrations of lipids, lipoproteins and apolipoproteins in the plasma increase appreciably during pregnancy because of the rise in insulin, progesterone, 17-β estradiol and Human Placental Lactogen (Wakatsuk et al., 1998; Adank et al., 2020).

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5. Conclusion

This study underscores the outcome of previous work that revealed general increase in serum lipid in pregnancy which is required to meet up with the energy needs of the pregnant state. The hyperlipidemia was highest in the third trimester of pregnancy with triglycerides having the highest percentage increase. Thus the third trimester of pregnancy may be the most vulnerable for coronary heart disease for women who are atherosclerotic. Lipid profile should therefore be included as part of the antenatal test in most of our healthcare units. To this end, the government should assist by providing the basis and essential equipment needed to facilitate this even at the primary healthcare level. Further studies should be done to compare the lipid profile between the nulligravida, gravida and post-menopausal women.

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Disclosure of Conflict of Interest

All authors declare no form of conflict of interest in this study.

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Author's Contributions

This work was carried out in collaboration among all authors. Authors CBE and HBO designed the study, wrote the protocol and the first draft of the manuscript. Authors KAD, EOO and JA managed the analyses of the study. Authors ENA, OAL, AA and CU managed the literature searches. Authors JA FAO and CVO performed the statistical analysis. All authors read and approved the final manuscript.

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