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# Lipid Profile and Some Parameters of Lipid Peroxidation in Pregnancy Trimesters

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## Abstract

Pregnancy is known to create profound metabolic, hormonal, and physiological changes in the body. Lipid profile and some parameters associated with lipid peroxidation play crucial roles in the sustenance of pregnancy and delivery. The biochemical changes resulting from pregnancy could be physiological or pathological depending on the parameter's concentration and other ancillary considerations. This study was therefore intended to evaluate lipid profiles and lipid peroxidation parameters in the three trimesters of pregnancy. The study population comprised one hundred women equally divided into pregnant and non-pregnant groups. One half made up of 50 pregnant women was monitored from the first to third trimester of pregnancy while the other half of 50 non-pregnant women were controls. Blood samples were collected into plain tubes after an overnight fast by venepuncture and thereafter standard biochemical procedures for lipid profile and lipid peroxidation parameters were done. The result revealed a significant decrease ($P < 0.05$) in serum cholesterol, triacylglycerol, low-density lipoprotein (LDL), superoxide dismutase, glutathione reductase, and catalase concentrations, whereas high-density lipoprotein, very LDL, and malondialdehyde exhibited a significant increase ($P < 0.05$) when compared to the controls and within trimesters multiple comparisons using one-way analysis of variance (*post hoc*-least significant difference). Conclusively, the alterations in serum lipid profiles and lipid peroxidation parameters are pointers to the predisposition of pregnancy to lipid dysfunction and oxidative stress phenomenon. Hence monitoring of these parameters during pregnancy is apt.

**Keywords:** Lipid peroxidation, Lipid profile, Pregnancy, Trimester of pregnancy

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## Introduction

Pregnancy is the term used to describe the period in which a fetus develops inside the uterus. It is a physiological process accompanied by alterations in the body's metabolic processes. These alterations are essential adaptations to accommodate the developing fetus, therefore as the size of the fetus increases with each trimester so also will there be variations in the metabolic processes.

Lipids are biomolecules that are soluble in non-polar solvents.[1] The functions of lipids include storing energy, signaling, insulation, and acting as structural components of cell membranes.[2-4] Lipid peroxidation is the chain of reactions of oxidative degradation of lipids containing polyunsaturated fatty acids. It is the process in which free radicals "steal" electrons from the lipids in cell membranes, resulting in cell damage.[5] This process is preceded by a free radical chain reaction mechanism. As with any radical reaction, the reaction consists of three major steps: Initiation, propagation, and termination. The chemical products of this oxidation are known as lipid peroxides or lipid oxidation products. This mechanism is employed by both physiological and pathological processes of the body utilizing molecules sourced from lipids.[6]

Advancement of pregnancy is complemented by extra demand of energy and stress on physiological processes.[7] As pregnancy progresses, a well-integrated metabolic shift ensues to guarantee an adequate supply of nutrients to a constantly feeding and growing fetus from an intermittently fasting mother.[7] Pregnancy not only initiates increased demand for adequate nutrients for fetal growth and development, but it also causes hormonal changes in the body which may lead to changes in lipid profile during different trimesters of the pregnancy.[8]

Basic research in the past two decades has led to an increased awareness of the role of lipid peroxidation in various physiologic and pathophysiologic processes Kong and Lin,[5]. Pregnancy, though not a disease state, is a stressful condition with considerable alterations in physiological and metabolic functions.[9] Every physiological and pathological process of the body has an interface with lipids and lipid peroxidation. This could be attributed to their integral roles in metabolism and health. A lot of processes linked to pregnancy such as vascular endothelial dysfunction may be caused by uncontrolled lipid peroxidation.[10] Free radical generation is a normal physiological process with a variety of effects. However, increased production of these free radicals will render lipids susceptible to lipid peroxidation in individuals with low intake of anti-oxidants.[11] *In vivo*, or *in vitro* generation of anti-oxidants is the major mechanism employed by the body to counteract free radicals generation and the deleterious effect of lipid peroxidation.[12]

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Lipids are crucial in pregnancy sustenance due to their energy-generating capacity, insulation capacity, and their use as a synthetic precursor of reproductive hormones. Lipid peroxidation has the preponderance of distorting the native configuration of lipids with negative consequences on its numerous functions. Similarly, pregnancy is a physiological process with attendant effects on almost all molecules that drive metabolic processes. There exists a gap in the effect of the various trimesters of pregnancy on lipid and extension indicators of lipid peroxidation. This study was therefore aimed at interrogating the effects of various trimesters of pregnancy on lipid profile and some biochemical indicators of lipid peroxidation. The result of this study could elucidate the biochemistry of lipids and lipid peroxidation as it concerns pregnancies.

## Methodology

### Study Area

The study was conducted at Federal Medical Centre, Yenagoa, Bayelsa State. Bayelsa state is located within latitude 4° 15′ North and Latitude 5°, 23′ South, 5° 22′ West and 6° 45′ East. It is bounded by Delta state on the North, Rivers state on the East, and the Atlantic Ocean on the western and southern part. According to the 2006 Census figures Bayelsa has a population of about 1.7 million people.[13]

### Experimental Design

This study patronized a cross-sectional study design coupled with a control. The controls served as a baseline, whereas the pregnant subject constituted the experimental group. The pregnant subjects were followed through the pregnancy period (9 months). Samples were collected on Thursdays of the 4$^{th}$ week of the first, second, and third trimesters consecutively.

### Study Population and Sampling

A total of 100 subjects were used as indicated using the Cochran formula for calculating the minimum sample size for cross-sectional studies.[14] The study population was equally divided into the control and experimental groups. The control group was constituted mainly of non-pregnant women as indicated by a negative HCG test. On the contrary, the experimental group was comprised of pregnant subjects recruited at first antenatal care with a scan indicating a 1-month pregnancy.

### Selection Criteria

Subjects with confirmed positive pregnancy tests and or ultra-sonological evidence of pregnancy were recruited as the test group, while healthy non-pregnant women were the control group. The confirmation was determined by an obstetrician.

Subjects with intrauterine fetal death, any systemic illness, preeclampsia, gestational diabetes, or under some drugs prescribed by a clinician or confirmed cases of diabetes or hypertension were excluded.

### Ethical Approval

Ethical clearance was duly approved by the Ethics Committee of the Federal Medical Centre Yenagoa. Similarly, individual informed consent was obtained from the subjects before sample collection and laboratory analysis. Ethical clearance protocol under the Helsinki Declaration of 1975, as revised in 1983 was adhered to World Medical Association.[15]

### Sample Collection

Blood collected between 8 am and 10 am after overnight fasting (of 10–12 h, the patients were instructed not to eat after 10 pm) by venepuncture was placed into a 5-ml vacuum plain tube and transported at ambient temperature to the laboratory. The samples were centrifuged at 3000 rpm and the serum was separated into plain containers and stored at −2°C in the refrigerator. A total of about 500 μL of the serum was used for all the laboratory analysis. The laboratory analysis was carried out within 12 h of the receipt of the samples.

### Laboratory Procedure

Serum total cholesterol and triacylglycerol concentrations were estimated quantitatively using the Agappe kit.[16] Serum high-density lipoprotein (HDL)-cholesterol concentration was estimated quantitatively using the Randox Kit as modified by Randox Laboratories.[16] Serum low-density lipoprotein (LDL) and very LDL (VLDL) concentrations were derived mathematically from the formula by Friedewald *et al.*[17] Serum catalase (CAT) and superoxide dismutase (SOD) were estimated according to the methods of Aebi[18] and Xin *et al.*,[19] respectively. Serum malondialdehyde (MDA) analysis was performed by determining the concentration of MDA formed using the method of Varshney and Kale.[20] The concentration of glutathione was determined according to the method of Habig *et al.*[21]

### Statistical Analysis

The Statistical Package for the Social Sciences (SPSS), version 25 (SPSS Inc., Chicago, IL, USA), and Microsoft Excel version 2010 were used for all the analyses. Results were expressed as mean ± standard deviation while comparisons were made between different groups using one-way analysis of variance (least significant difference-*post hoc*). The level of statistical significance was set at 95% confidence interval.

## Results

Table 1 shows the sample size distribution and age brackets of the subjects of the various groups. Table 2 shows a comparison of the lipid profiles of the studied groups. The concentration of cholesterol is lower in the first trimester when compared to the baseline (controls). In a similar vein, a comparison with the three trimesters showed that cholesterol was significantly higher in the third trimester when compared to the first and second. Serum triacylglycerol was lower in the first and second trimesters when compared to the third trimester and control. Serum HDL was lower in first trimester when compared to the control, second, and third trimesters. Serum LDL was lower across all trimesters when compared with the control and this fall was more pronounced in the second trimester. The serum VLDL concentrations were lower in the second and third trimesters when compared to the control and third trimesters. Serum SOD, glutathione reductase (GPX), and CAT were lower consistently in the trimesters of pregnancy when

compared to the control. Serum MDA concentration was higher across trimesters when compared to the control.

## Discussion

The findings on serum cholesterol concentration revealed a fall in the first trimester with a consequential significant increase in the second and third trimesters [Table 2]. The initial fall could be attributed to hormonal variations resulting from the pregnancy process. In a similar vein, the significant increase observed in the second and third trimesters could be a product of an increase in the production of cholesterol for the sustenance of pregnancy. All steroid hormones are derived from cholesterol and these are essential for maintenance of the of both the fetus and placenta. It is also needed for the production of estrogen, progesterone, and other essential hormones that play key roles in maintaining a healthy pregnancy and ensuring homeostasis. The first trimester of pregnancy is characterized by increased synthesis and pregnancy, hence the facilitated production. Extra cholesterol is needed to fuel the rapid growth circulation of estrogen, progesterone, and hyperinsulinemia.[9,22,23] These have a direct effect on the fall of serum cholesterol observed in the first trimester. This finding agrees with the reports by other authors.[24-27]

However, serum triacylglycerol exhibited a significant decrease in the first and second trimesters, with subsequent increases in the third trimester [Table 2]. The fall in the first and second trimester could be due to the geometric increase in energy need of the body. Energy expenditure has a direct effect on triacylglycerol which is used as a source of energy via gluconeogenesis. The findings of this study contrasted with a handful of studies.[25-28] However, the finding concurred with that of Raghuram *et al.*[24]

Serum HDL and VLDL exhibited an initial fall in the first trimester and subsequently increased significantly in the second and third trimesters. VLDL is composed mainly of triacylglycerol. The increase in concentration of VLDL is in line with the increase earlier observed in triacylglycerol. VLDL is a transporter of triacylglycerol. Similarly, the increase in HDL could be attributed to increase in the concentration of cholesterol. HDL is a major transporter of cholesterol. The increase in HDL concentration parallels lower risk of atheroma progression and this reflects that pregnancy is not a risk factor for cardiovascular compromise. This finding is in line with that of Okojie *et al*.[29] and Phuse,[30] but contradicts that of Raghuram *et al*.[24]

Serum LDL) exhibited a significant decrease comparable to the control and the first trimester concentrations are higher than what was obtained in the other trimesters [Table 2]. The finding is further proof of the non-vulnerability of pregnancy to cardiac diseases and dysfunction. The finding contradicted the reports of Okojie *et al*.[29] and Phuse.[30]

This study revealed lipid peroxidation in the trimesters of pregnancy [Table 3]. Serum SOD, GPX, and CAT decreased consistently in the trimesters of pregnancy when compared to the control. On the contrary, serum MDA increased across the trimesters when compared to the control.

A specific biomarker of lipid peroxidation is MDA. This study has shown that MDA level significantly increases progressively during first, second, and third trimesters of pregnancy. It is consistent with previous studies that reported increase in MDA concentration during late pregnancy.[31] On the contrary, studies on animal models during late pregnancy failed to show any significant changes in MDA levels.[32] However, this discrepancy could have been mainly due to individual variations in MDA concentration. The increase observed is a further validation that lipid peroxidation is part and parcel of pregnancy.

Moreover, this study shows that serum SOD, GPx, and CAT, activities tend to decrease progressively from the first, second, and third trimesters. The decrease in CAT and GPx activity during pregnancy is consistent with previous reports by Erisir *et al*.[33] and Myat.[34] Similarly, studies by Amer *et al*.[35] and Öztabak *et al*.[36] showed that plasma CAT activity was lower during late pregnancy.

SOD is well known to be a superoxide radicals' scavenger which is an essential factor in the protection against free radical damage and is considered the first line of defense against pro-oxidants.[37] In our study, the decrease SOD activity observed during pregnancy was consistent with previously reported observations by Celi *et al*.[38] and Amer *et al*.[35] This could also be attributed to erythrocyte SOD's role in pregnancy, red blood cells

**Table 1:** Demographic data of studied subjects

| *Demography data* | *C1 n=50* | *FT n=50* | *ST n=50* | *TT n=50* |
|---|---|---|---|---|
| Subject (n) | 50 (20%) | 50 (20%) | 50 (20%) | 50 (20%) |
| Age (year) | 25.4±2.6 | 27.7±4.7 | 27.7±4.7 | 27.7±4.7 |
| Age range (years) | 22–28 | 21–32 | 21–32 | 21–32 |

C1: Control group (non-pregnant/non lactating mothers), FT: First trimester pregnancy, ST: Second trimester pregnancy, TT: Third trimester pregnancy

**Table 2:** Comparisons of lipid profiles between controls and pregnancy trimesters (*N*=50)

| *Parameters* | *Control* | *1st Trimester* | *2nd Trimester* | *3rd Trimester* | *F-value* | *P-value* |
|---|---|---|---|---|---|---|
| Cholesterol (mmol/L) | 4.32±1.41 | 3.32±0.84[a] | 4.08±0.75[b] | 4.49±0.24[b] | 4.204 | 0.004 |
| Triacylglycerol (mmol/L) | 2.33±1.89 | 0.70±0.25[a] | 1.36±0.70[a] | 2.56±1.63[b,c] | 6.672 | 0.000 |
| HDL (mmol/L) | 0.99±0.65 | 0.34±0.15[a] | 1.92±0.72[a,b] | 1.56±0.59[a,b] | 23.098 | 0.000 |
| LDL (mmol/L) | 2.66±1.38 | 1.42±1.28[a] | 0.64±0.31[a,b] | 1.12±0.67[a] | 12.234 | 0.000 |
| VLDL (mmol/L) | 0.74±0.58 | 0.32±0.11[a] | 0.56±0.22 | 3.78±0.36[a,b,c] | 153.973 | 0.000 |

HDL: High-density lipoprotein, LDL: Low-density lipoprotein, VLDL: Very low-density lipoprotein

**Table 3:** Comparisons of some peroxidation parameters between controls and pregnancy trimesters (*N*=50)

| *Parameters* | *Control* | *1st Trimester* | *2nd Trimester* | *3rd Trimester* | *F-value* | *P-value* |
|---|---|---|---|---|---|---|
| MDA | 5.01±2.61 | 9.90±7.54[a] | 10.67±9.01[a] | 20.19±7.30[a,b] | 10.430 | 0.000 |
| GPX | 53.30±20.65 | 35.56±20.34[a] | 30.25±18.82[a] | 22.26±19.48[a,b] | 7.927 | 0.000 |
| SOD | 195.45±28.58 | 179.75±21.56[a] | 149.90±16.89[a,b] | 125.10±14.75[a,b,c] | 67.907 | 0.000 |
| CAT | 63.27±11.62 | 50.85±8.80[a] | 50.44±29.89[a] | 33.86±6.84[a,b,c] | 8.321 | 0.000 |

MDA: Malondialdehyde, GPX: Glutathione reductase, SOD: Superoxide dismutase, CAT serum: Catalase

(RBC) tends to deplete during pregnancy as a result of oxidative stress damage to RBC productions. A larger effect of oxidative stress occurs in the third trimester with consequent production of biogenic antioxidants defense systems and this results in the rapid decline in SOD values observed in the third trimester as compared to the second and first trimesters.

Based on the premise of the discourse, lipid peroxidation, and oxidative stress phenomenon are resultant fallout of pregnancy. However, the advantages and disadvantages of the phenomenon need further studies to buttress whether it is pathological or physiological.

## Conclusion

This study revealed significant alterations in lipids and lipid peroxidation biochemical parameters. Alterations in lipid and oxidative marker concentrations are manifestations of a lot of physiological and pathological processes and mechanisms. The alterations observed for oxidative markers are indicative of the preponderances of pregnancy in generating free radicals. Free radicals are culprits of a lot of disease conditions and in the same vein, are used in halting pathological processes. Based on this premise, lipids, and lipid peroxidation parameters could form a battery of medical laboratory investigations in antenatal care. Finally, the need for further investigations is apt to determine whether the alterations observed in this study are physiological or pathological.

### Ethical Approval

The study protocol was approved by the Department of Medical Laboratory Science of the Niger Delta University, Wilberforce Island, Bayelsa State. In a similar vein, individual consents were sought and granted before the research. The standard established by the Helsinki Declaration was strictly followed.

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