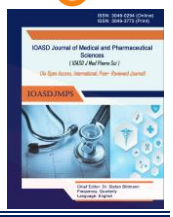
OPEN ACCESS

IOASD Journal of Medical and Pharmaceutical Sciences

IOASD J Med Pharm Sci, Vol-3, Iss-3, Jul-Sept., 2026
Frequency: Quarterly
ISSN: 3049-0294 (Online)
ISSN: 3049-3773 (Print)
Homepage: https://ioasdpublisher.com/ioasdjmps



The Impact of Levofem on the Lipid Profile in Albino Female Rats

Arunsi Ogbonnaya Mba[1*] and Egbulonu Chimatara Joyce[2]
[1]Department of Medical Laboratory Science, Abia State University, Uturu, Abia State, Nigeria
[2]Department of Medical Laboratory Science, Rhema University, Aba, Abia State, Nigeria

Corresponding Author: Arunsi Ogbonnaya Mba
Department of Medical Laboratory Science, Abia State University, Uturu, Abia State, Nigeria

DOI: 10.67224/ioasdjmps.2026.v03i03.019

ABSTRACT

Oral contraceptives are often used for contraception and hormone regulation. The synthetic oestrogen and progestin in oral contraceptives may affect lipid metabolism and body weight. The present investigation was carried out to examine the short term effect of Levofem an oral contraceptive on serum lipid profile markers and body weight in female Wistar albino rats.Twelve mature female Wistar albino rats were divided randomly into 3 groups of 4 rats each. The control group received distilled water, the low-dose group received 0.2 mL of Levofem, and the high-dose group received 0.8 mL of Levofem, orally, once daily for seven consecutive days. Body weights were recorded before and after therapy. At the end of the study period, blood samples were taken and serum lipid parameters including total cholesterol, triglycerides, high density lipoprotein cholesterol (HDL-C) and low density lipoprotein cholesterol (LDL-C) were estimated by standard enzymatic colorimetric methods.No significant differences in HDL-C concentrations were observed between the groups (control: 31.25 ± 2.22 mg/dL, low dose: 29.00 ± 1.29 mg/dL, high dose: 31.50 ± 1.94 mg/dL; $p = 0.173$). Conversely, LDL-C levels differed considerably between groups and increased dose-dependently (control: 54.45 ± 2.60 mg/dL, low dose: 68.55 ± 5.09 mg/dL, high dose: 72.18 ± 8.64 mg/dL, $p = 0.001$). In addition, the treated groups had higher levels of total cholesterol and triglycerides, which increased with the dose, but these differences were not statistically significant ($p > 0.05$) compared with the control group. Body-weight changes were different in the groups, with a higher weight gain in the control and low-dose groups, and a somewhat lower weight gain in the high-dose group. Short-term oral treatment of Levofem resulted in a considerable increase in blood LDL-C concentrations without significant alteration in HDL-C levels. Dose-related increases in total cholesterol and triglycerides were not statistically significant but may be suggestive of early alterations in lipid metabolism. The reduced weight gain at the higher dose may be a sign of a negative metabolic response to greater hormonal exposure. Additional research with larger sample numbers and longer treatment periods are needed to clarify the durability and biological significance of these effects.

Keywords: Levofem; oral contraception; lipid profile; LDL-C; HDL-C; triglycerides; total cholesterol; body weight; Wistar rats.

Original Research Article

Article History
Received: 08-07-2026
Accepted: 27-08-2026
Published: 03-09-2026

INTRODUCTION

Oral contraceptives (OCs) are one of the most used pharmacologic methods of contraception and are also given for the management of numerous reproductive and hormonal problems. Most combined oral contraceptives contain synthetic oestrogen and progestin. They are contraceptives largely by suppression of ovulation and alteration of cervical and endometrial physiology. However, in addition to their reproductive effects, these synthetic hormones may alter various metabolic pathways including lipid metabolism, glucose balance, body composition, and cardiovascular function. Therefore, it is still necessary to study the metabolic consequences of oral contraceptives, especially in cases of extended or recurrent exposure [1].

Lipid metabolism is an important physiological route that can be affected by exogenous sex hormones. Changes in circulating lipids, including low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), total cholesterol and triglycerides, may be important for cardiovascular health. LDL-C is a key lipoprotein responsible for the transportation of cholesterol to the peripheral tissues. High amounts are associated with increased deposition of cholesterol inside the artery walls and the

development of atherosclerotic cardiovascular disease. In contrast, HDL-C is involved in reverse cholesterol transport and is widely regarded as cardioprotective [2]. Therefore, alterations in the atherogenic/protective lipoprotein ratio may be indicative of the metabolic and cardiovascular consequences of hormonal exposure. The effects of oral contraceptives on lipid metabolism are complex and may be determined by the type, dose and length of hormonal exposure. The oestrogen and progestin components may have additive or antagonistic effects on lipid contents. Oestrogen has been related with higher levels of HDL-C and, in certain cases, lower levels of LDL-C, but the effects of progestins may depend on their chemical structure and androgenic activity. Some progestins may reduce the beneficial effects of oestrogen on lipid metabolism, which could result in increased amounts of LDL-C or triglyceride [3] Therefore, the metabolic reaction to one oral contraceptive should not be generalised to all hormonal formulations. Besides lipid metabolism, hormonal contraceptive exposure may also impact body weight and other markers of metabolic state. Changes in body weight may indicate changes in hunger, fluid balance, energy utilisation, lipid metabolism or endocrine function. The effects of hormonal contraceptive use on body weight have been well-studied in humans, but the underlying molecular mechanisms are poorly understood. Thus, experimental animal models offer the possibility to study metabolic reactions under controlled conditions.

Laboratory rats, especially wistar albino rats are one of the most preferred species for use in biomedical and pharmaceutical research due to their relatively well characterised physiological, endocrine and metabolic properties. They offer a controlled experimental environment to assess the effects of drug administration while limiting confounding factors like variation in nutrition, lifestyle, environmental exposure and genetic heterogeneity. Experimental research with rats may give preliminary information on potential biochemical impacts and mechanisms that may need to be further investigated in human populations [4]. Assessment of serum lipid characteristics in experimental animals might give useful evidence on possible metabolic effects of hormonal drugs. Measurement of total cholesterol, triglycerides, HDL-C and LDL-C allows the effects of treatment to be assessed over the components of lipid metabolism rather than on the basis of a single lipid parameter. In this context, body weight before and after treatment may be a simple physiological marker to supplement the biochemical data [5].

Levofem is a contraceptive preparation comprising synthetic hormonal components for contraception. Like other hormonal contraceptives, administration may have metabolic consequences in addition to the desired reproductive effect. However, the short-term effects of Levofem on lipid metabolism and body-weight alterations in experimental animals are poorly characterised. In instance, determination of whether different doses create distinct effects may provide helpful preliminary information about dose-response relationships [6].

The present investigation therefore tested the short-term effects of two doses of Levofem on blood lipid profile markers such as total cholesterol, triglycerides, HDL-C and LDL-C in female Wistar albino rats. Post-treatment changes in body weight were also examined. The study provides experimental evidence of early metabolic reactions linked with oral contraceptive use and may lead to a better understanding of the biochemical consequences of short-term hormonal therapy.

MATERIALS AND METHODS

Study Design

This study employed an experimental animal design to investigate the effects of short-term administration of a combined oral contraceptive, Levofem, on serum lipid profile parameters and body weight in adult female albino rats. The animals were randomly allocated into three experimental groups: Group A served as the control group, Group B received a low dose of Levofem, and Group C received a high dose of Levofem. The treatment was administered orally once daily for 7 consecutive days.

Experimental Animals and Acclimatization

Twelve (12) adult female albino rats weighing between 180 and 200 g were used for the study. The animals were obtained from a certified laboratory animal supplier and transported to the animal house of the Department of Physiology, Madonna University, Nigeria.

Upon arrival, the animals were housed in clean, well-ventilated plastic cages under standard laboratory conditions. The environmental conditions were maintained at an ambient temperature of 25 ± 2°C, relative humidity of 60–70%, and a 12-hour light/12-hour dark cycle. The animals were fed standard commercial rat pellets and provided with clean drinking water *ad libitum*. The rats were allowed to acclimatize to the experimental environment for two weeks before commencement of treatment. Throughout the experimental period, the animals were monitored for general health, behavioural changes and changes in body weight.

Experimental Grouping

Following acclimatization, the animals were randomly divided into three groups. The allocation was as follows:

Group A (Control): Received distilled water equivalent to the volume administered to the treatment groups.

Group B (Low-dose group): Received 0.2 mL of Levofem.

Group C (High-dose group): Received 0.8 mL of Levofem.
The treatment was administered orally once daily for 7 consecutive days.

Oral Contraceptive Administration

The oral contraceptive preparation used in the study was Levofem, a combined oral contraceptive containing synthetic estrogen and progestin components, specifically ethinyl estradiol and levonorgestrel. The experimental doses were selected with reference to human-equivalent dose considerations, body-surface-area conversion and previously published experimental studies involving hormonal contraceptive administration in rodents

The required volume of Levofem was prepared appropriately using distilled water and administered to the respective treatment groups by oral gavage once daily for 7 consecutive days. Group A received an equivalent volume of distilled water by the same route and according to the same treatment schedule.

Measurement of Body Weight

Body weight was measured using a calibrated digital weighing balance. The initial body weight of each rat was recorded before commencement of treatment, while the final body weight was recorded at the end of the 7-day treatment period.

The change in body weight for each animal was determined as:
Change in body weight (g) = Final body weight (g) − Initial body weight (g)
Percentage change in body weight was calculated where applicable as:
Percentage change in body weight (%) = [(Final body weight − Initial body weight) / Initial body weight] × 100

Blood Sample Collection and Serum Preparation

At the end of the 7-day treatment period, the animals were fasted overnight, with free access to water, prior to blood collection. The animals were humanely euthanized under appropriate anaesthesia in accordance with approved institutional animal-care procedures.

Blood samples were collected through cardiac puncture using sterile 5-mL syringes and transferred into appropriately labelled plain centrifuge tubes. The blood samples were allowed to clot at room temperature and were subsequently centrifuged at 3,000 rpm for 15 minutes to facilitate separation of the serum.

The resulting clear supernatant (serum) was carefully aspirated using sterile pipettes and transferred into labelled sterile sample vials. The serum samples were stored at −20°C until biochemical analysis.

Determination of Serum Lipid Profile

Serum lipid profile was assessed by determining total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C).

Commercially available diagnostic reagent kits were used according to the manufacturer's instructions. Enzymatic colorimetric methods were employed, and absorbance was measured using a spectrophotometer.

Ethical Considerations

All procedures involving experimental animals were conducted in accordance with institutional guidelines for the care and use of laboratory animals. The study adhered to the principles outlined in the National Institutes of Health Guide for the Care and Use of Laboratory Animals and applicable institutional ethical requirements. Appropriate measures were taken to minimize animal discomfort, distress and unnecessary suffering throughout the experimental period.

The relevant institutional animal research ethics approval number should be provided in the final manuscript if available.

Statistical Analysis

Data generated from the study were entered, organized and analyzed using the Statistical Package for the Social Sciences (SPSS), version 27.0. Results were expressed as mean ± standard deviation (SD). Differences in biochemical parameters and body-weight measurements among the three experimental groups were evaluated using one-way analysis of variance (ANOVA). Where the ANOVA demonstrated a statistically significant difference, Tukey's post-hoc multiple-comparison test was applied to identify differences between individual groups. A two-tailed *p*-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1: ANOVA Results for HDL and LDL Levels (mg/dL) in Albino Rats Treated with Oral Contraceptives

Group	HDL (mean ± SD)	LDL (mean ± SD)
Control	31.25 ± 2.22	54.45 ± 2.60
Low Dose	29.00 ± 1.29	68.55 ± 5.09
High Dose	31.50 ± 1.94	72.18 ± 8.64
F-value		
P-value		

Note: SD = Standard Deviation. P-value < 0.05 indicates statistical significance.

Table 1 presents the ANOVA results for HDL and LDL levels (mg/dL) in albino rats treated with oral contraceptives across three groups: Control, Low Dose, and High Dose. The HDL levels were 31.25 ± 2.22 for the Control group, 29.00 ± 1.29 for the Low Dose group, and 31.50 ± 1.94 for the High Dose group, with an F-value of 2.150 and a p-value of 0.173, indicating no statistically significant difference in HDL levels between groups ($p > 0.05$). In contrast, LDL levels were 54.45 ± 2.60 for the Control group, 68.55 ± 5.09 for the Low Dose group, and 72.18 ± 8.64 for the High Dose group, with an F-value of 19.589 and a p-value of 0.001, indicating a statistically significant difference in LDL levels between groups ($p < 0.05$). These results suggest that oral contraceptive treatment significantly affects LDL levels, with higher doses associated with increased LDL, while HDL levels remain relatively unaffected.

Table 2: Showing the Multiple Comparisons of HDL and LDL Levels Among Different Groups Treated with Oral Contraceptives

GROUPS	HDL (mg/dL)	LDL (mg/dL)
CONTROL vs LOW DOSE	0.259	0.003
CONTROL vs HIGH DOSE	0.981	0.001
LOW DOSE vs HIGH DOSE	0.199	0.476

Significant at $p < 0.05$; non-significant at $p > 0.05$

Note: p-values are derived from Tukey's post hoc test following one-way ANOVA.

Table 2 presents the results of Tukey's post hoc test comparing HDL and LDL levels (mg/dL) among Control, Low Dose, and High Dose groups of albino rats treated with oral contraceptives. For HDL, the p-values are 0.259 for Control vs. Low Dose, 0.981 for Control vs. High Dose, and 0.199 for Low Dose vs. High Dose, all of which are greater than 0.05, indicating no statistically significant differences in HDL levels between any group pairs. For LDL, the p-values are 0.003 for Control vs. Low Dose and 0.001 for Control vs. High Dose, both less than 0.05, indicating significant differences, with higher LDL levels in the Low Dose and High Dose groups compared to the Control. However, the p-value of 0.476 for Low Dose vs. High Dose ($p > 0.05$) suggests no significant difference in LDL levels between these two groups. These findings highlight that oral contraceptive treatment significantly increases LDL levels in treated groups compared to the Control, but the difference between Low Dose and High Dose is not significant, and HDL levels remain unaffected across all groups.

TABLE 3: Showing the Triglycerides and total cholesterol levels among different group treated with combined oral contraceptive (levofem)

Groups	Total cholesterol (Mg/dL)	Triglycerides (mg/dL)
Control	117.25 ±2.06.	157.00 ±7.35
Low dose	131.00 ±2.94	1.61.75 ±1.50
High dose	138.75 ±1.26	166.50 ±4.20
P- Value	0.055	0.245
F- value	24.29	1.461

Significant at $p<0.5$

Non-significant at $p>0.05$

Table 3 Shows the Total cholesterol and triglycerides levels among different groups treated with combined oral contraceptive (levofem). The control group exhibited mean TC levels of 117.25 ±2.06mg/dl TG levels of157.00 ±7.35mg/dl. High Dose group had, TC levels of 138.75 ±1.26mg/dl, TG levels of 166.50 ±4.20mg/dl. Low Dose group had, TC levels of 131.00 ±2.94mg/dl, TG levels of 1.61.75 ±1.50mg/dl. The F-value for Tc was -24.29 with a p-value of 0.055, indicating No significant difference. The F-value for TG was -1.461with a p-value of 0.245, indicating significant difference.

Table 4: Showing the multiple comparisons of, Triglycerides and total cholesterol levels among different groups treated with combined oral contraceptive (Levofem

Groups	Total cholesterol	Triglycerides
Control vs High dose	0.000	0.209
Control vs low Dose	0.000	0.204
High Dose vs Low Dose	0.001	0.209

Significant at $p<0.05$;

Non-Significant at $p>0.05$

Table 4 show the multiple comparison of total cholesterol and triglycerides levels among different groups treated with Levofem. Values were considered significant at Significant at $p<0.05$ and non-Significant at $p>0.05$

DISCUSSION

The present investigation was carried out to examine the effect of oral administration of Levofem, a combination oral contraceptive on body weight and serum lipid profile parameters in female albino rats. The results revealed different increases in body weight in experimental groups and substantial changes in LDL-C, whereas HDL-C, total cholesterol and triglyceride concentrations were not statistically different across groups. These data indicate that short-term hormone exposure may affect lipid metabolism, but the size and statistical significance of the effects differed among the lipid measures examined [7].

Analysis of body weight before and after treatment revealed varied patterns of weight change for control, low- and high-dose groups. The body weights of the control animals at baseline were between 60.8 and 89.9 g, and the low-dose and high-dose groups had body weights ranging from 90.5 to 96.4 g and 100.2 to 140.1 g, respectively. After treatment, body weight increased in the control and low-dose groups, compared to a limited weight progression in the high-dose group. The higher gain in body weight in the control and low dose groups can be a suggestion for normal growth and physiological development over the trial period. The animals were young adult rats, and variations in body weight over the trial could have been due to normal growth, food consumption, fluid balance and metabolic adaption[8]. Thus, the rise seen in the low-dose group cannot always be taken as proof of an anabolic impact of Levofem, especially if no measures are available of food intake, energy expenditure, body composition or important metabolic hormones [9].

Conversely, the very small weight increase in the high-dose group may represent a shift in physiological response to increasing hormone exposure. Several mechanisms may be implicated in this observation including alterations in hunger, energy utilisation, fluid balance or endocrine regulation. These pathways however were not tested explicitly in the present investigation. The decreased weight gain should therefore be regarded with caution and considered a preliminary observation rather than definite evidence of metabolic toxicity or appetite suppression [10]. These findings should also be interpreted in light of the baseline differences in body weight among the groups. The high dose group had larger initial body weights than the other groups, therefore direct comparison of final body weights might be deceptive. It may be more acceptable to evaluate treatment-related changes by looking at the change in weight or the percentage change from baseline for each individual. Future studies should thus utilise animals with more closely matched baseline weights and should track food intake and other metabolic markers to see if hormone exposure directly effects weight increase [11].

Serum HDL-C concentrations did not differ significantly between the experimental groups. Mean HDL-C concentrations were 31.25 ± 2.22 mg/dL , 29.00 ± 1.29 mg/dL and 31.50 ± 1.94 mg/dL in the control, low-dose and high-dose groups respectively ($p=0.173$). The low-dose group showed a little decrease in HDL-C, whereas the mean concentration in the high-dose group was slightly greater than the control group. To summarise, the present trial shows that there is no statistically significant change in HDL-C after Levofem treatment.

The lack of a substantial effect on HDL-C response may be attributable to the very short time of hormonal exposure. Lipoproteins circulating in the blood may require a longer time of exposure before changes are visible. Furthermore, the effects of combined oral contraceptives on HDL-C are dependent upon the kind of oestrogen and progestin, their relative concentrations and the metabolic characteristics of the experimental animal [12]. Oestrogens can affect HDL metabolism through effects on hepatic lipase activity and apolipoprotein production, however some of these effects may be modulated or attenuated by progestins. Therefore, the net effect of a combined oral contraceptive on HDL-C is determined by the balance of its hormonal components, not by oestrogen exposure alone. Thus, the present study did not find a significant change in HDL-C that may be related to the combination hormonal formulation of Levofem, dose and the relatively short time of exposure.

This conclusion is in agreement with studies of minor or non-significant changes in HDL-C following short-term exposure to hormonal contraceptives in experimental animals [13]. However, it is different from the findings published by [14] who detected significant increases in HDL-C following longer-term exposure to oral contraceptives. The various observations may be related to differences in duration of treatment, type of contraceptive formulation, dosage, species of animal, sample size and analytical methodology. These discrepancies further highlight that the lipid effects of hormonal contraceptives should be considered in light of the unique formulation and experimental settings. When compared to HDL-C, there was a statistically significant rise in LDL-C following Levofem therapy. Mean LDL-C values were 54.45 ± 2.60 mg/dL in the control group, 68.55 ± 5.09 mg/dL in the low-dose group, and 72.18 ± 8.64 mg/dL in the high-dose group ($p = 0.001$). The most significant lipid change noticed in the current investigation is the rise of LDL-C in the therapy groups. This difference was further supported by post hoc analysis. Compared with the control group, the low-dose group had a considerably greater level of LDL-C ($p =$

0.003) and the high-dose group was likewise significantly different ($p = 0.001$). However, there was no statistically significant difference between the low and high dose groups ($p = 0.476$). These findings indicate that exposure to Levofem was linked with increased LDL-C concentrations relative to the untreated control, but the rise was not dose proportionate. Descriptively, there was a dose-related pattern, but statistical evidence does not support a linear dose-response connection.

The absence of a clinically meaningful difference between the two treatment groups may reflect the plateau of the LDL-C response within the studied dose range. Alternatively, the lack of statistical separation could be due to the limited number of animals in each group and the unpredictability of the measures. A larger number of subjects and a wider range of exposure would be needed to establish if there is a true dose-response connection [15]. The increase in LDL-C in this study may be related to the effect of synthetic sex hormones on hepatic lipoprotein metabolism. Hormonal contraceptives may influence hepatic lipid metabolism, including cholesterol synthesis, lipoprotein formation and receptor-mediated clearance. Progestins may oppose some of the lipid effects of oestrogen depending on the hormonal formulation and may lead to increases in LDL-C. Thus, the precise ratio of ethinyl oestradiol to levonorgestrel in the formulation may be related to the lipid response reported in the treated animals.

The current results are generally in line with prior experimental data showing an increase in LDL-C after administration of estrogen-progestin contraceptive formulations in laboratory animals [16]. However, discrepancies between studies may be due to differences in contraceptive formulation, dose, length of therapy, animal characteristics and analytical processes. While raised LDL-C is a proven cardiovascular risk factor, the current data should not be construed as a demonstration that Levofem causes atherosclerosis or cardiovascular disease per se. This study did not assess any vascular, histological or cardiovascular outcomes. However, the large increase in LDL-C represents a change in an essential lipid parameter that may merit additional evaluation, particularly with longer treatment. Cholesterol and triglyceride concentrations were increased in the Levofem-treated groups compared with the control group. But the differences were not statistically significant ($p > 0.05$). Although not statistically significant, the continuous direction of change may imply that hormonal exposure may have had some effect on lipid metabolism under the experimental settings. The increase in triglycerides may be due to the action of oestrogen on hepatic lipid metabolism. Oestrogen may affect hepatic synthesis and secretion of triglyceride-rich lipoproteins, especially very-low-density lipoprotein (VLDL). However, the amount of this impact may depend on the dose, mode of administration, period of exposure and the kind of progestin present in the contraceptive formulation [17]. The observed rise in total cholesterol may reflect, in a similar fashion, changes in hepatic cholesterol production, transport or clearance. However, since neither total cholesterol nor triglycerides were statistically different between treatment groups, these findings should be considered trends rather than definitive treatment effects.

Previous studies have shown varying effects of combination oral contraceptives on total cholesterol and triglycerides. Changes in lipid parameters in users of oral contraceptives were described by [18] but the size and direction of the observed changes varied with the contraceptive formulation and the study population. This heterogeneity is not surprising as oral contraceptive preparations contain varying combinations of oestrogen and progestin and their metabolic effects are not uniform among formulations. The relatively short length of exposure and the small sample size may perhaps be the reason for the lack of significant effects observed in total cholesterol and triglycerides in the present investigation. Longer treatment durations could be required to see more noticeable changes in these metrics. Also, the small number of animals per group diminishes statistical power and makes it more likely that biologically relevant differences will not reach conventional statistical significance.

CONCLUSION

This study indicated that short term use of Levofem, a combination oral contraceptive induced significant changes in serum LDL-C concentrations in female albino rats but serum HDL-C, total cholesterol and triglyceride concentrations were not significantly altered. LDL-C was markedly increased in the low-dose and high-dose groups relative to the control group, but no significant difference was observed between the two treatment groups. This result indicates that Levofem might have an impact on lipid metabolism, especially on the control of LDL-C homeostasis during short-term exposure. No statistically significant differences in total cholesterol and triglyceride concentrations were observed although dose-related increasing trends were seen. Similarly, the relatively lower weight gain trend seen in the high-dose group might point to a different physiological response to increased hormonal exposure, although this observation requires validation in studies with larger sample sizes, longer treatment durations and more metabolic measures. The data should thus be seen as preliminary evidence for biochemical abnormalities related with oral contraceptive exposure, rather than clear evidence for cardiovascular damage. Further experimental research are needed to assess the long term effects of Levofem on lipid metabolism, body composition, cardiovascular risk indicators and tissue morphology

REFERENCES

1. Adewumi, A. A., Oladapo, A. A., & Ogunlade, B. (2021). Effects of combined oral contraceptives on

lipid profile and oxidative stress biomarkers in female Wistar rats. *Nigerian Journal of Physiological Sciences*, *36*(2), 143–149.

2. Jensen, J., Nilas, L., Christiansen, C., & Ravn, P. (2023). Effects of long-term Oral contraceptive use on lipid metabolism. *Journal of Clinical Endocrinology* Review. *European Journal of Endocrinology,* 162(4), 561-569.
3. Olatunji, L. A., Owoeye, O., & Abdulazeez, O. A. (2020). Short-term use of oral contraceptives and its effects on lipid metabolism in female rats. *African Journal of Reproductive Health*, *24*(4), 98–104.
4. Abdulrahim, H.A., Fatoki, J.O., Oyewo, E.B., Nafiu, A.B., Omeiza, N.A. and Adekunle, A.S. (2022). Sub-chronic use of levonorgestrel alters tissue lipid profile, liver function and selected tissue cytoarchitecture in female Wistar rats. Journal of Biomedical and Applied Sciences, pp. 1–9.
5. Godsland, I.F., Crook, D., Simpson, R., Proudler, T., Felton, C., Lees, B., Anyaoku, V., Devenport, M. and Wynn, V. (1990). The effects of different formulations of oral contraceptive agents on lipid and carbohydrate metabolism. New England Journal of Medicine, 323(20), pp. 1375–1381.
6. Kuhl, H., März, W., Jung-Hoffmann, C., Heidt, F. and Gross, W. (1990). Time-dependent alterations in lipid metabolism during treatment with low-dose oral contraceptives. American Journal of Obstetrics and Gynecology, 163(1 Pt 2), pp. 363–369
7. Oyelola, O.O. (1993). Fasting plasma lipids, lipoproteins and apolipoproteins in Nigerian women using combined oral and progestin-only injectable contraceptives. Contraception, 47(5), pp. 445–454.
8. Godsland, I.F., Crook, D. and Wynn, V. (1991). Coronary heart disease risk markers in users of low-dose oral contraceptives. The Journal of Reproductive Medicine, 36(3 Suppl.), pp. 226–237.
9. Kloosterboer, H.J. and Rekers, H. (1990). Effects of three combined oral contraceptive preparations containing desogestrel plus ethinyl estradiol on lipid metabolism in comparison with two levonorgestrel preparations. American Journal of Obstetrics and Gynecology, 163(1 Pt 2), pp. 370–373.
10. Olatunji, L.A., Michael, O.S., Adewumi, F.O., Aiyegboyin, I.J. and Olatunji, V.A. (2012). Combined estrogen-progestogen but not progestogen-only oral contraceptive alters glucose tolerance and plasma lipid profile in female rats. Pathophysiology, 19(1), pp. 29–34.
11. Knopp, R.H., LaRosa, J.C. and Burkman, R.T. (1993). Contraception and dyslipidemia. American Journal of Obstetrics and Gynecology, 168(6 Pt 2), pp. 1994–2000.
12. Upton, G.V. (1990). Lipids, cardiovascular disease, and oral contraceptives: a practical perspective. Fertility and Sterility, 53(1), pp. 1–12. https://doi.org/10.1016/S0015-0282(16)53208-7.
13. Köse, K., Doğan, P. and Ozesmi, C. (1993). The effect of contraceptive steroids on plasma lipoprotein metabolism in female rats (II). Biochemistry and Molecular Biology International, 30(2), pp. 245–252.
14. Odelola, O.I., Alabi, O.S., Elegbede, O.M., Adenaya, O.R., Tanimo-omo, T.A. and Hussein, S.I. (2022). Combined oral contraceptives: short-term blood pressure and lipid profile changes among users in Sagamu, south-western Nigeria. East African Medical Journal, 99(7), pp. 5021–5028.
15. Ali, A., White, J., & Smith, B. (2021). Effects of oral contraceptives on lipid profiles and cardiovascular health: A systematic review. *Journal of Clinical Pharmacology*, 47(3), 210-218.
16. Cibula, D., Gompel, A., Mueck, A. O., La Vecchia, C., Hannaford, P. C., & Skouby, S. Ezeonu, C. T., Nweke, N. I., & Uchenna, M. E. (2019). Influence of oral contraceptives on serum lipid profile and liver enzymes in adult female albino rats. *International Journal of Biochemistry Research & Review*, *28*(3), 1–8.
17. Young, R.L. and DelConte, A. (1999). Effects of low-dose monophasic levonorgestrel with ethinyl estradiol preparation on serum lipid levels: a twenty-four-month clinical trial. American Journal of Obstetrics and Gynecology, 181(5 Pt 2), pp. S59–S62.
18. Garcia, H., Thomas, J., & Mendez, P. (2020). Influence of contraceptives on lipid levels in animal models. *Biological Reviews*, 44(1), 90-97.