# Impact of Hormonal Contraceptive on Body Mass Index, Lipid Profile and Blood Pressure of Women at Bearing Age in Rural Area, Northern Sudan

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# Introduction

The oral contraceptive hormones, introduced in 1960, have under gone much modification. The term "contraception" is defined as the intentional prevention of pregnancy. Contraceptives are therefore pharmaceuticals or devices that prevent pregnancy. The goal of contraception is to prevent unintended pregnancy without causing adverse effects and to preserve fertility, when desired. Worldwide, the contraceptive pills are used by 100 millions of women in child bearing age [1]. Two types of oral contraceptive pills are available in market, the combined oral contraceptives containing both estrogen and progesterone and progestin containing only progesterone. The combined oral contraceptive pills are prepared in three different formulations, i.e. monophasic, biphasic, triphasic depending on the concentration of estrogen and progestin in menstrual cycle [2].

Estrogens facilitate the growth of the ovarian follicles and increase the motility of the uterine tubes. The estradiol secretion rate is  $36\mu g/dl$ . Almost estrogen comes from ovary, and there are 2 peaks of secretion; one just before ovulation ( $380\mu g/dl$ ) and one during the mid luteal phase ( $250\mu g/dl$ ). The principle target organs of progesterone are the uterus, the breasts, and the brain. In men the plasma progesterone level is

approximately 0.3ng/mL and in women is 0.9ng/mL [3]. The action of contraceptive pills depending on that estrogen inhibits the release of follicle-stimulating hormone (FSH) and thus suppresses the development of the ovarian follicle. The progesterone inhibits the release of luteinizing hormone (LH) and thus prevents ovulation, and it also makes the cervical mucus less suitable for the passage of sperm. Together they alter the endometrium in such a way as to discourage implantation [4].

All other types of hormonal contraception in current use in the UK are progestogen-only and share many similar features in the terms of mode of action and side effects. Because they don't contain estrogen, they are extremely safe and can be used if a woman has cardiovascular risk factors [5]. The current mode of progestogen-only contraception are progestogen-only pills, subdermal implant, injectable, hormonesreleasing intrauterine system. Females rats implanted with progesterone gained weight more rapidly than control animals and had an increased proportion of total body fat. Restriction of food intake to control levels demonstrated that the weight changes were not dependent on increased energy intake [6]. In vitro studies have demonstrated that estrogen exerts favorable actions on the endothelium, for instance, stimulating the production of nitric oxide [7] and reducing the expression of adhesion molecules [8]. Moreover, estrogen is capable of promoting in vivo protective actions, such as the decrease of soluble adhesion molecules [9] and reduction of hepatic triglyceride lipase, which degrades HDL (High Density Lipoprotein), stimulating, thus, the production of HDL cholesterol and reducing the production of LDL (Low Density Lipoprotein) cholesterol [10]. Estrogen also affects the cardio vascular (CV) system through its impact on CV risk factors such as the lipid profile. Oral contraceptives (OCs) alter the lipid profile through the genomic pathway, in which estrogen receptor alterations affect hepatic apolipoprotein up regulation [11,12]. Large clinical assays show that the extended use of estrogen associated with progesterone cannot be beneficial and may, also, compromise the efficiency of the autonomic modulation of the heart rate [13].

Some studies show no evidence of unfavorable effects due to estrogenic therapy on autonomic heart rate control with women who practice regular physical training, in the rest condition [14]. Studies report that there are alterations of the metabolism of plasmatic lipids of young, females using oral contraceptives, and that the estrogen and progesterone dosages are related [15]. However, other authors report that low-dose, combined contraceptives reduce the adverse effects of the increases of the total triglyceride and cholesterol serum levels [16].

Santos, *et al*, 2008 [17], conducted study to detect the influence of oral contraceptive use on lipid levels and cardio-respiratory responses among healthy sedentary women, their study revealed that low doses of estrogen/ progesterone did not affect the aerobic capacity and autonomic modulation in this selected age range. However, it does contribute to modifications of the lipoprotein metabolism related to increases of the total cholesterol and triglycerides levels. Mohammad, *et al*, 2013 [18], studied the effect of combined oral contraceptive pills on lipid profile, blood pressure and body mass index in women of child bearing age, in family planning department of tertiary referral health care hospitals of Peshawar. They found that contraceptive had significantly elevated lipid profile, blood pressure and body mass index (BMI). Scocco, *et al.*, 2013 [19], studied the effect of different contraceptive drugs on the lipid profile of Brazilian women, authors found that the anti-androgenic progestogen drospirenone was not effective to counterbalance the beneficial effect of the estrogen on the levels of the high density lipoprotein (HDL). Asare *et al*, 2014 [20], studied the effect of hormonal contraceptives on lipid profile and the risk indices for cardiovascular disease

in a ghanaian community, they found that hormonal contraceptive use is associated with significant increases in body mass index, diastolic blood pressure, total cholestrol, low density lipoprotein. Mohamed, *et al.*, 2016 [21], conducted study on impact of using hormonal progestogen only as contraceptive treatment on plasma lipids, their study showed that the levels of plasma total cholesterol, triglycerides and low density lipoprotein increased by using hormonal contraceptives and this increase were directly proportional to the duration of using hormonal contraceptives. Mohammed, *et al.*, 2017 [22], conducted a study to assess the association between body mass index, lipid profiles and total cholesterol among married versus spinster and they found that upper body mass index was inversely associated with high density lipoprotein (HDL) and total glycerides (TG) in the benefit of a spinster. Although the association between BMI and both HDL and TG may be explained by oral contraceptives causing a decrease in apoprotein, cholesterol, high density lipoprotein, triglycerides the case of married women.

## Methods

The volunteer women were subjected to two types of measurements:

#### Anthropometric Measurements

women in study sample before weighing was wearing light clothing and without shoes. Anthropometric measurements; weight (nearest 0.1kg), height (nearest 0.5cm) were measured by using standard medical balance and measuring tape respectively. Body mass index (BMI) was calculated as sample weight (kg) divided by height (m) squared (kg/m<sup>2</sup>).

#### **Clinical Analysis**

The women were subjected to fasting process about 12 hours before biochemical testing. Total cholesterol and total triglyceride concentrations were investigated by using the automatic colorimetric enzymatic method. Also blood pressure was assessed.

#### **Statistical Analysis**

SPSS software was used for data analysis and the results were calculated as mean ± standard deviation. Differences between groups were compared using student's t-test, considered significant at the 5% level of probability.

### Results

Anthropometric	Weight (kg)		Height (m)		Body mass index (kg/m <sup>2</sup> )	
measurements	Test	Control	Test	Control	Test	Control
Mean	66.18	62.90	1.60	1.53	25.52	27.70
S.D.	1.27	1.18	0.10	0.16	1.83	7.52
Sig.	0	.138	0	0.074	0	.321

Table 1: Anthropometric measurements of test and control group

Lipid	Total ch	olesterol	Total triglyceride		
profile	Test	Control	Test	Control	
Mean	132.55	151.17	74.90	94.50	
S.D.	2.17	2.56	2.87	2.87	
Sig.	0.038		0.050		

Table 2: Lipid profile of study groups

I ::: d Drofts	Systolic B	B.P(mg/dL)	Diastolic B.P (mg/dL)		
Lipid Prome	Test	Control	Test	Control	
Mean	125.67	122.07	84.04	82.90	
S.D.	9.48	5.22	5.16	5.18	
Sig.	0.255		0.315		

Table 4: Comparison of anthropometric and clinical parameters for 20 - 30 years age group

Group	Test	Control	Sig
Weight (kg)	65.90±1.30	$62.28 \pm 1.52$	0.094
Height (m)	$1.61 \pm 1.33$	$1.51\pm0.33$	0.087
Body mass index (kg/m <sup>2</sup> )	25.45±4.68	$26.85\pm9.85$	0.231
Total cholesterol (mg/dL)	125.90±3.06	$143.12\pm1.50$	0.049
Total glycerides (mg/dL)	72.20±3.99	$92.71\pm2.28$	0.037
Systolic B.P (mmHg)	123.15±8.16	$122.48\pm5.77$	0.276
Diastolic B.P (mmHg)	82.67±5.16	$82.53\pm0.00$	0.782

Table 5: Comparison of anthropometric and clinical parameters for 30 - 40 years age group

Group	Test	Control	Sig
Weight (kg)	$67.16 \pm 1.33$	$63.46 \pm 1.07$	0.165
Height (m)	$1.59\pm00.16$	$1.52. \pm 0.49$	0.098
Body mass index (kg/m <sup>2</sup> )	$25.57\pm4.42$	$28.54\pm6.09$	0.217
Total cholesterol (mg/dL)	$139.20\pm1.92$	$159.21\pm2.61$	0.042
Total glycerides (mg/dL)	$77.50\pm2.20$	$96.31\pm3.89$	0.016
Systolic B.P (mmHg	$127.18\pm6.33$	$123.62\pm1.00$	0.856
Diastolic B.P (mmHg)	$85.33 \pm 5.16$	$83.26 \pm 5.77$	0.512

Group	3 <sup>rd</sup> decade	4 <sup>rd</sup> decade	Sig
Weight (kg)	65.90±1.30	$67.16 \pm 1.33$	No
Height (m)	$1.61 \pm 1.33$	$1.59\pm00.16$	No
Body mass index (kg/m <sup>2</sup> )	25.45±4.68	$25.57\pm4.42$	No
Total cholesterol (mg/dL)	125.90±3.06	$139.20\pm1.92$	No
Total glycerides (mg/dL)	72.20±3.99	$77.50\pm2.20$	No
Systolic B.P (mmHg)	123.15±8.16	$127.18\pm6.33$	No
Diastolic B.P (mmHg)	82.67±5.16	$85.33\pm5.16$	No

Table 6: Comparison between two groups of test sample according to age

Table 7: Comparison between two groups of control sample according to age

Group	3 <sup>rd</sup> decay	4 <sup>rd</sup> decay	Sig
Weight (kg)	$62.28 \pm 1.52$	$63.46 \pm 1.07$	No
Height (m)	$1.51\pm0.33$	$1.52 \pm 0.49$	No
Body mass index (kg/m <sup>2</sup> )	$26.85\pm9.85$	$28.54\pm6.09$	No
Total cholesterol (mg/dL)	$144.12\pm1.50$	$158.21\pm2.61$	No
Total glycerides (mg/dL)	$92.71\pm2.28$	$96.31\pm3.89$	No
Systolic B.P (mmHg)	$122.48\pm5.77$	$123.62\pm1.00$	No
Diastolic B.P (mmHg)	$82.53\pm0.00$	$83.26 \pm 5.77$	No

# Discussion

The study did not show a significant difference for body mass index (BMI) (Table 1) although there was increase in BMI average for control group compared with sample. The study revealed that the averages for cholesterol and triglycerides for test group were less than that of control group and they showed significant differences (P value = 0.038 and 0.050) respectively, while average of systolic, diastolic blood pressure were close in both groups and weren't show significant difference (Tables 2&3 respectively). The same results of previous measurements were represented when the two groups compared according to age (Tables 4&5). All values of anthropometric and clinical measurements showed an increase with age progress, but did not show a significant difference, Tables (6&7). Our findings disagree with that studies conducted by [17, 18, 20, 21] where their results were revealed that hormonal contraceptive had significant effect on the BMI and some clinical investigation. On other hand our results were matched with those conducted by [22] which showed that hormonal contraceptive drugs had no effect on the lipid profile and anthropometric measurements, and partially it agree with study conducted by [19] which revealed that the progestogen was not effective to counterbalance the beneficial effect of the estrogen on the levels of the high density lipoprotein (HDL) and the lipid profile.

Estrogen gains weight through their action on retention fluid and lipogenesis increase. The high oral dose of estrogen can cause fluid retention and symptoms of weight gain or obesity (estrogen obesity). The study was showed that there is no significant difference in anthropometric measurements, although the study questionnaire revealed that some women were gained weight but others loss weight after contraceptive intake. This may be attributed to low concentration of estrogen in the dose (<50mg). Light elevation in blood pressure of test group can be attributed to increase of renine enzyme secretion in the renal under effect of contraceptive. On the other hand, the total cholesterol and triglycerides parameters appeared that hormonal contraceptive had no clear effect on the target measurements. In general, the elevation in measurements value in the benefit of test group could be attributed to absence of estrogen obesity, nutritional status variance and lipoprotein lipase activity.

# Conclusion

Many studies have shown that there is a close relationship between hormonal contraceptives and an increase in body mass index, lipid profile and blood pressure, but this study comes in line with a few of them indicating that hormonal contraceptives have no significant effect on anthropometric and clinical measurements.

## Bibliography

1. Margolis, K. L., Adami, H. O., Luo, J., Ye, W. & Weiderpass, E. (2007). A prospective Study of oral contraceptive use and risk of myocardial infarction among Swedish women. *J Fertil Steril.*, 88, 310-316.

2. Petitti, D. B. (2003). Clinical practice combination estrogen-progestin oral contraceptives. *NEngl J Med.*, *349*(15), 1443-1450.

3. Ganong, W. F. (1995). *Review of medical physiology*, alange medical book. 17<sup>th</sup> edition, Appleton & Lange, Norwalk, CT. (pp. 406-407).

4. Whalen, K. (2015). *Pharmacology, Lippincott's illustrated reviews*. 6th ed., Wollers Kluwer, London, (pp. 351-359).

5. Edmonds, K., Gebbie, A. E., Hay, P., Ingamells, S., Monga, A., et al. (2006). Gynecology by Ten Teachers.

6. Shirling, D., Ashby, J. P. & Baird, J. D. (1981). Effect of progesterone on lipid metabolism in the intact rat. *J Endocrinol.*, 90(2), 285-294.

7. Chow, R. W., Handelsman, D. J. & Ng, M. K. (2010). Minireview: rapid actions of sex steroids in the endothelium. *Endocrinology*, 151(6), 2411-2422.

8. Andrade, C. M., Sa, M. F. & Toloi, M. R. (2012). Effects of phytoestrogens derived from soy bean on expression of adhesion molecules on HUVEC. *Climateric*, 15(2), 186-194.

9. Stocco, B., Fumagalli, H. F., Franceschini, S. A., Martinez, E. Z., Marzocchi-Machado, C. M., *et al.* (2012). Drospirenone and levonorgestrel in combination with either 30 or 20mcg ethinylestradiol reduce soluble adhesion molecules in Brazilian women; cross-sectional study. *Contraception*, *86*(5), 506-510.

10. Knopp, R. H., Zhu, X. & Bonet, B. (1994). Effects of estrogens on lipoprotein metabolism and cardiovascular disease in women. *Atherosclerosis*, *110*, S8391.

11. Tsismenakis, A. J., Christophi, C. A., Burress, J. W., Kinney, A. M., Kim, M. & Kales, S. N. (2009). The obesity epidemic and future emergency responders. *Obesity*, *17*(8), 1648-1650.

12. Jones, D. R., Schmidt, R. J., Pickard, R. T., Foxworthy, P. S. & Eacho, P. I. (2002). Estrogen receptormediated repression of human hepatic lipase gene transcription. *J Lipid Res.*, 43(3), 383-391.

13. Schueller, P. O., Feuring, M., Sharkova, Y., Grimm, W. & Christ, M. (2006). Effects of synthetic progestagens on autonomic tone, neurohormones and C-reactive protein levels in young healthy females in reproductive age. *Int J Cardiol.*, *111*(1), 42-48.

14. Perini, R., Fisher, N., Veicsteinas, A. & Pendergast, D. R. (2002). Aerobic training and cardiovascular responses at rest and during exercise in older men and women. *Med Sci Sports Exerc.*, 34(4), 700-708.

15. Davy, K. P., DeSouza, C. A., Jones, P. P. & Seals, D. R. (1998). Elevated heart rate variability in physically active young and older adult women. *Clin Sci.*, *94*(6), 579-584.

16. Bergink, E. W., Kloosterboer, H. J., Lund, L. & Nummi, S. (1984). Effects of levonorgestrel 12. and desogestrel in low-dose oral contraceptive combination on serum lipids, apolipoproteins A-I and B and glycosylated proteins. *Contraception*, *30*(1), 61-72.

17. Santos, M. C., Rebelo, A. C., Zuttin, R. S., César, M. C., Catai, A. M. & Silva, E. (2008). Influence of oral contraceptive use on lipid levels and cardio-respiratory responses among healthy sedentary women. *Rev Bras Fisioter*, *12*(3), 188-194.

18. Mohammad, N. S., Nazli, R., Khan, M. A., Akhtar, T., Ahmad, J. & Zafar, Z. (2013). Effect of combined oral contraceptive pills on lipid profile, blood pressure and body mass index in women of child bearing age. *KMUJ.*, *5*(1), 22-26.

19. Stocco, B., Fumagalli, H. F., Franceschini, S. A., Machado, C. M. & Toloi, M. R. (2013). The Effect of Different Contraceptive Drugs on the Lipid Profile of Brazilian Women. Pharmaceutica Analytica Act, 4(1), 1-4.

20. Asare, G., Sheila, Santa, S., Ngala, R., Asiedu, B., Afriyie, D. & Amoah, A. B. (2014). Effect of hormonal contraceptives on lipid profile and the risk indices for cardiovascular disease in a ghanaian community. *International Journal of Women's Health*, 6, 597-603.

21. Mohamed, N. A. A., Amanullah, M., Ibrahim, M. A. & Mohamed, E. A. (2016). Impact of Using Hormonal Progestogen-only as Contraceptive Treatment on Plasma Lipids. *J Women's Health Care.*, *5*, 313.

22. Mohammed, Z., Idriss, M. M., Nora, B. A. & Mabrouki Fatiha, M. (2017). The association between body mass index, lipid profiles and total cholesterol among married versus spinster. *J Gastroenterol Dig Dis.*, 2(1), 18-21.