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Original Article

The effect of lipid disturbance and vitamin D on the fertility in male albino rats

Shimaa F. Hikal[1], Mona M. El-Bayoumi [1], Samah E. Ibrahim[1], Mohammad M. EL-Shawwa[1]

[1]Physiology Department, Faculty of Medicine for Girls, Cairo, Al-Azhar University, Egypt.

ABSTRACT

Background: Vitamin D has multiple biological effects on male reproductive system. Vitamin D deficiency (VD−) and disturbance of lipid metabolism can induce changes in testicular hormone production and seminal parameters that relate to male infertility.

Objective: To investigate the role of vitamin D and lipid metabolism on fertility in male albino rats.

Methods: The study was performed on 60 male rats, divided into 6 groups; GI: control group, GII: orlistat group, GIII: orlistat and vitamin D group, GIV: hyperlipidemic group, GV: hyperlipidemic orlistat group and GVI: hyperlipidemic orlistat and vitamin D group. Serum total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-c) and high density lipoprotein cholesterol (HDL-c), 25-hydroxyvitamin D (25(OHD), testosterone (T), inhibin B, follicle-stimulating hormone (FSH) and estradiol (E) levels were determined. Sperm count and viability were also analyzed.

Results: Administration of orlistat in GII caused significant alterations of the serum lipid profile, compared to control group. There was interaction between dietary fat and VD− on serum 25 (OHD). A significant VD− was occurred in both GII and GIV, compared to GI. VD− in these groups consequently caused significant decrease in serum T, inhibin B, sperm count and viability and significant increase in FSH and E levels. Administration of orlistat in GV caused significant changes in serum lipid profile, a decrease in E, increase in 25(OHD) and inhibin B, compared to IV. On the other hand, administration of vitamin D with orlistat in GIII and GVI caused significant increase in HDL-c, 25(OHD), T, sperm count and viability and significant decrease in TC, TG, LDL-c, FSH and E.

Conclusion: Both hyperlipidemia and hypolipidemia were associated with vitamin D deficiency. Moreover, vitamin D has a positive potential effect on male fertility in either hyperlipidemic or hypolipidemic rats.

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Keywords: Lipids, male fertility, vitamin D.

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Corresponding Author: Shimaa FM. Hikal; physiology department, faculty of medicine for girls, Cairo, Al-Azhar University, Egypt. **Tel.** 01002993474. **E-mail:** drshimaafouad88@gmail.com -samahelmetwally.medg@azhar.edu.eg

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INTRODUCTION

For many years, fats were considered as dangerous substances to our health and consequently should be minimized or avoided in our diet [1]. Currently, all the guidelines since 2000 changed from maintaining low fat to moderate fat. This includes guidelines issued by the American Heart Association and the 2000 Dietary Guidelines for Americans. Obese individuals practice an excess of dietary calorie intake but mostly suffer from micronutrient deficiencies, including vitamin D, vitamin C, biotin, chromium and thiamine [2]. Reproductive functions are mediated by the hypothalamic-pituitary-gonadal (HPG) axis in both males and females [3]. Both vitamin D receptor (VDR) and vitamin D metabolizing enzymes have been localized throughout the hypothalamus and the pituitary gland [3]. Thus vitamin D might improve gonadal function by improving pituitary function. There is a clear relationship between dietary lipid supplementation and sperm fatty acids (FAs) composition [4]. Dietary FAs play an important role in the modulation of sperm metabolism and affect all sperm parameters [5]. In the last years, growing evidence has linked dyslipidemia to male infertility [5]. So, our study was conducted to investigate the effect of fat in the form of hyperlipidemia or hypolipidemia and vitamin D on fertility markers in male rats.

MATERIALS AND METHODS

Sixty male albino rats weighing 130g to 140g were used. They were housed in plastic cages (10 rats/each) and were kept at standard room temperature (22-25 Cº) and 12 hours light/dark cycle. Rats were left 10 days for adaptation before the start of the experiment and had free access to food and water.

I. Experimental diet: Balanced rat chow diet and high fat diet (HFD) were used (table 1). Hyperlipidemia was induced in thirty rats by administration of HFD **(** 30%**)** for 8 weeks, as evidenced by elevation of lipid profile analysis [8]. Fat was in the form of beef tallow. Beef tallow contains 46g saturated FAs (palmitic, stearic and myristic acids), 50g monounsaturated FAs (oleic and palmitoleic acids) and 4g polyunsaturated FAs (linoleic and linolenic acids.

Table (1): Diet composition

Components %	Balanced rat chow diet [6]	High fat diet [7]
Fat	5.4	33.78
Carbohydrate	53.8	37.66
Protein	21.9	15.33
Fiber	2.9	2.03
Minerals, added vitamins A, D and E	15.8	11.06
Cholesterol	0.2	0.14

II. Experimental design: The rats (normal and hyperlipidemic) were classified into 6 equal groups and subjected to the following regimens for 8 weeks:

1. Group I (Control group): Rats were kept on balanced rat chow diet. They received daily 1 ml 0.9% saline solution orally and injected intraperitoneally (i.p.) with 0.1 ml 0.9% saline every other day.
2. Group II (Orlistat group): Rats were kept on balanced rat chow diet. Each rat was supplemented orally with 1 ml of orlistat solution (6 times/week) for 8 weeks, at a concentration of 12 mg/kg B.W/ml according to Amin et al.[8] and injected i.p. every other day with 0.9% saline solution.
3. Group III (Orlistat and Vitamin D group): Rats were kept on balanced rat chow diet and received both orlistat solution as previously described and injected i.p. every other day with 0.1 ml 1,25-dihydroxyvitamin D_3 (1,25$(OH)_2D_3$) solution for 8 weeks, at a dose of 5 µg/kg B.W/ml according to Yin et al.[9].
4. Group IV (Hyperlipidemic group): Rats were kept on HFD and received 0.9% saline solution as described above for GI.
5. Group V (Hyperlipidemic and Orlistat group): Rats were kept on HFD and received orlistat solution as GII.
6. Group VI (Hyperlipidemic, Orlistat and Vitamin D group): Rats were kept on HFD and received both orlistat and 1, 25 $(OH)_2 D_3$ solutions as GIII.

At the end of the experimental period, blood samples of overnight fasted rats were collected from retro-orbital sinuses by heparinized capillary tubes under light ether anesthesia [10]**.** Blood samples were centrifuged at 3000 rpm for 15 minutes for serum collection. Sera were separated and stored at -80 Cº till the time of analysis.

III. Biochemical analysis:

- Serum level of 25 (OHD) was assayed by rat 25 hydroxy vitamin D ELISA kit of DIA-Source Immuno-Assays S.A., Belgium[11].
- Serum testosterone (T) was assayed by rat testosterone ELISA kit of DRG International, Inc., USA [12].
- Serum estradiol (E) was determined by estradiol ELISA kit, Bio-Line, USA [13].
- Serum inhibin B was assayed by rat inhibin B ELISA kit of Bio-Line, USA [14].
- Serum follicle stimulating hormone (FSH) was assayed by rat FSH ELISA kit of Bio-Line, USA [15].
- Lipid profile: Serum total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-c) and high density lipoprotein cholesterol (HDL-c) were measured by quantitative enzymatic colorimetric method using kits of Spin-React, S.A. ctra, Spain [16-19].

IV. Collection of epididymal sperms: Sperms were collected by cutting the caudal region of the epididymis into small pieces and flushing the sperms in 15 ml Ringer's solution at 32 Cº. The collected sperms were used for estimation of sperm count and viability [20].

- **Estimation of sperm count:** By haemocytometer [21].
- **Estimation of sperm viability:** By sperm viability test Principle: Sperm vitality was determined by eosin-nigrosin stains. Eosin stained the head of dead sperms with red color, but it did not stain living sperms which remained white [22].

Statistical analysis

Statistical analysis was done by using Statistic Package for Social Science (SPSS) software version 18 [23]. Quantitative data were expressed as mean ± standard deviation (SD). Differences among experimental groups were analyzed using one-way variance analysis (ANOVA) followed by post hock test for multiple comparison of groups [24].

RESULTS

Changes of serum 25 (OHD) and lipid profile

Our results showed that administration of orlistat to normal rats (GII) caused significant decrease in serum 25 (OHD), TC, TG and LDL-c and insignificant change in HDL-c, compared to the control group (GI). Co-administration of orlistat and vitamin D to normal rats (GIII) caused a significant increase in serum 25 (OHD), compared to GII and significant decrease in serum TC, TG and LDL-c, compared to GI, but insignificant changes in HDL-c**.** Rats fed on HFD (GIV) had significant decrease in serum 25 (OHD) and HDL-c and significant increase in TC, TG and LDL-c, compared to GI. Administration of orlistat alone (GV) or combined with vitamin D (GVI) to the hyperlipidemic rats caused significant increase in serum 25 (OHD) and HDL-c and significant decrease in TC, TG and LDL-c, compared to GIV. In addition, the co-administration of orlistat and vitamin D in GVI caused the same significant changes compared to GV as well **(Table 2).**

Changes of serum testosterone, estradiol, inhibin B and follicle stimulating hormone levels

Administration of orlistat to normal rats (GII) caused significant decrease in serum T and inhibin B and insignificant changes in E and FSH, compared to control group (GI). Co-administration of orlistat and vitamin D to normal rats (GIII) caused significant increase in serum T and inhibin B compared to GII and insignificant changes in E and FSH. Feeding HFD to GIV caused significant decrease in serum T and inhibin B and significant increase in E and FSH, compared to control group (GI). Administration of orlistat to hyperlipidemic rats (GV) caused a significant increase in serum inhibin B and a significant decrease in E but insignificant changes in T and FSH levels, compared to hyperlipidemic rats (GIV). While co-administration of orlistat and vitamin D to hyperlipidemic rats (GVI) caused significant increase in serum T and inhibin B and significant decrease in E and FSH, compared to both GIV and GV **(Table 3)**.

Changes of sperm count and viability %

Administration of orlistat to normal rats (GII) had significant decrease in sperm count and sperm viability percentage, compared to control group (GI). Co-administration of orlistat and vitamin D to normal rats (GIII) caused significant increase in sperm count and sperm viability, compared to GII. While sperm count and sperm viability in hyperlipidemic group (GIV) had significantly decreased, in comparison to GI. Administration of orlistat to the hyperlipidemic rats (V) caused insignificant increase in both sperm count and sperm viability, compared to hyperlipidemic group (IV). While, co-administration of orlistat and vitamin D to the hyperlipidemic rats (VI) had significantly increased both sperm count and sperm viability as compared to either hyperlipidemic group (IV) or hyperlipidemic orlistat fed group (V) **(Table 4).**

Table (2): Changes of serum 25 (OHD) and lipid profile in different experimental groups

Groups / Parameters	Group I Mean ±SD	Group II Mean ±SD	Group III Mean ±SD	Group IV Mean ±SD	Group V Mean ±SD	Group VI Mean ±SD
25 (OHD) (ng/ml)	47.51± 2.76	27.01 ± 2.9 [a]	50.08± 3.04[b]	21.24±2.71 [a,b,c]	36.94 ± 2.25 [a,b,c,d]	51.31 ±3.24 [b,d,e]
TC (mg/dL)	138.87± 8.02	116.1±4.24[a]	118.51±6.82 [a]	303.78±8.33 [a,b,c]	195.57 ±5.64 [a,b,d]	164.8 ±13.95 [a,b,c,d,e]
TG (mg/dL)	62.24±9.94	34.42± 2.5[a]	32.21±1.66[a]	123.42 ±7.3 [a,b,c]	74.48 ±7.15 [a,b,c,d]	57.52±3.01 [b,c,d,e]
HDL-c (mg/dL)	60.92±4.12	60.21±6.7	69.62±11.6	21.98±1.8 [a,b,c]	53.82 ±11.1 [c,d]	60.61 ±7.17 [b,c,d,e]
LDL-c (mg/dL)	65.69±10.5	48±5.98[a]	51.24±10.08[a]	259.11 ±8.98 [a,b,c]	127.24 ±11.92 [a,b,c,d]	92.68±18.6 [a,b,c,d,e]

a: Significant values vs. group I, b: Significant values vs. group II, c: Significant values vs. group III, d: Significant values vs. group IV, e: Significant values vs. group V.

Table (3): Changes of serum testosterone, estradiol, inhibin B and follicle stimulating hormone levels in different experimental groups

Groups / Parameters	Group I Mean ±SD	Group II Mean ±SD	Group III Mean ±SD	Group IV Mean ±SD	Group V Mean± SD	Group VI Mean ±SD
Testosterone (ng/ml)	1.94±0.41	1.15±0.15[a]	2.3±0.22[b]	0.56±0.14 [a,b,c]	0.98± 0.14[a]	1.55± 0.32 [c,d,e]
Estradiol (pg/ml)	51.6± 6.63	45.9± 4.43	40.25±3.06	101.77±9.53 [a,b,c]	71.32± 2.99 [a,b,c,d]	57.47± 4.3 [b,d,e]
Inhibin B (pg/ml)	38.74±3.43	27.65±6.21 [a]	36.54 ±2.7 [b]	18.28± 2.1 [a,b,c]	35.44 ±3.28 [d]	43.14± 3.61 [b,d,e]
FSH (uIU/ml)	0.82± 0.2	0.46± 0.14	0.53± 0.09	4.83± 1.12 [a,b,c]	1.73±0.29[a,b,c,d]	0.97± 0.04 [d, e]

a: Significant values vs. group I, b: Significant values vs. group II, c: Significant values vs. group III, d: Significant values vs. group IV, e: Significant values vs. group V.

Table (4): Changes of sperm count and viability % in different experimental groups

Groups / Parameters	Group I Mean ±SD	Group II Mean ±SD	Group III Mean ±SD	Group IV Mean ±SD	Group V Mean ±SD	Group VI Mean ±SD
Sperm count (million/ml)	5215714± 589543.8	2698571± 337314.3 [a]	5884286± 579507.1[b]	3161429± 602755.5[a,c]	3374286± 385437.2 [a]	5724286± 401366.7[b,d,e]
Viability (%)	0.74±0.01	0.54±0.02 [a]	0.77±0.01 [b]	0.49±0.01[a,c]	0.59±0.01 [a]	0.78±0.01[b,d,e]

a: Significant values vs. group I, b: Significant values vs. group II, c: Significant values vs. group III, d: Significant values vs. group IV, e: Significant values vs. group V.

DISCUSSION

The HFD is an important factor in the functional disturbance of male reproductive system [25], since it leads to the development of hyperlipidemia, and abnormal lipid metabolism. In the present study, feeding of HFD to male rats increased plasma TC, TG and LDL-c, while it results in a decrease of HDL-c, compared to control rats. There is consistency between our findings and that reported by Yu et al. [26] and Barakat and Mahmoud [27]. The observed dyslipidemia was explained by the disorders of lipid metabolism by HFD [26]. High levels of FAs can result in increased intracellular TG synthesis and formation of lipid droplets in serum and liver [28]. Barakat and Mahmoud [27] attributed the increased TC level in liver and plasma to increased cholesterol deposition and decreased its catabolism as evidenced by a reduction in bile acid production and turnover of bile acids. Dietschy et al. [29] reported that excessive dietary intake of fat led to down regulation of the LDL receptor synthesis and subsequently an increase of blood cholesterol level. In the current study, 25(OHD) was significantly decreased in rats fed HFD, compared to the control group and the normal orlistat group. This revealed that the hyperlipidemic effect of HFD on decreasing serum 25(OHD) level was more than the hypolipidemic effect of orlistat. These results are consistent with the studies of Karanova et al. [30] and Fu et al. [31]. The decrease in serum 25(OHD) level might be attributed to the increase of 25 (OHD) sequestrated in adipose tissue of HFD fed rats [32]. Roizenet al. [33] demonstrated that the levels of CYP2R1 mRNA were significantly reduced in mice fed HFD. CYP2R is a gene that encodes the principal hepatic vitamin D 25-hydroxylase, which converts calciferol to 25(OHD) [34]. Testosterone and inhibin B were significantly decreased in the present study with administration of HFD, while FSH and E were significantly increased, compared to control. These findings are consistent with the studies of Jia et al. [35] and Abdel-Fadeil et al. [36]. Hormonal disturbances in the present study can be explained based on vitamin D deficiency (VD−) that occurred in this hyperlipidemic group. VDR stimulates testosterone production directly in human Leydig cells and improve other gonadal functions [37]. The link between lower levels of 25(OHD) vitamin D and these hormonal disturbances was reported in several studies [38,39]. Another possible mechanism of the decreased T and increased E levels is the high expression of aromatase by excessive fat tissue which converts excess T to E [40].

Sperm count and viability were significantly decreased with administration of HFD in our study, compared to control. These results agree with that of Elmas et al. [41] and Demirci and Sahin [42]. Our findings could be referred to the decrease in T and the increase in E levels which could disrupt the negative feedback loop of the HPG axis and disturb Sertoli cell function [43]. Merino et al. [44] showed that VD− influenced DNA status, inducing fragmentation of DNA in rat epididymal sperm.

Oral administration of orlistat to rats fed ordinary diet caused significant decrease in serum TC, TG and LDL-c and insignificant decrease in HDL-c. While administration of orlistat to hyperlipidemic rats fed HFD caused significant decrease in TC, TG and LDL-c and a significant increase in HDL-c, when compared to hyperlipidemic rats fed HFD only, but could not reach the normal level. This proved that when orlistat given with HFD could abolish its hyperlipidemic effects. These results are consistent with the studies of Wafa [45] and Othman et al. [46]. The effect of orlistat could be related to its potent and long-lasting gastrointestinal lipase inhibiting effect, thus blocks intestinal absorption of dietary fat [47].

25 (OHD) was significantly decreased in the current study with administration of orlistat to rats fed ordinary diet, compared to the control. These results are in line with the study of McDuffie et al. [48], they found that serum 25 (OHD) levels were significantly reduced in subjects received orlistat despite a daily oral multivitamin supplement containing vitamin D. Orlistat interferes with the absorption of vitamin D along with the impairment of fat absorption [49]. While administration of orlistat to hyperlipidemic rats fed HFD caused significant increase in serum 25 (OHD) level compared to hyperlipidemic rats fed HFD only. Since vitamin D is fat soluble, it is sequestered and stored in fat tissues [32]. Accordingly, greater loss of body adipose tissue would result in a greater release of vitamin D in the circulation [50]. Adipose tissue increases vitamin D metabolism due to activity of 24-hydroxylase in adipocytes [51].

Serum T and inhibin B levels were significantly decreased in the present study, with administration of orlistat to rats fed ordinary diet, while E and FSH levels were insignificantly changed, compared to control group. These results are in line with that of Rehm et al. [52]. We attributed these hormonal disturbances to VD− that occurred in this hypolipidemic group, as explained before in hyperlipidemic group. This was evidenced by their return to the normal levels with vitamin D supplementation.

In the current study, administration of orlistat to hyperlipidemic rats fed HFD showed insignificant increase in serum T level compared to rats fed HFD only. Although serum 25 (OHD) was increased in this group, its level remains insufficient to increase serum T. In contrary to our results, the study of Corona et al. [53] on obese men showed significant increase in their serum T after taking low calorie diet. This discrepancy might be related to different study methods with respect to treatments, baseline levels of 25 (OHD) and T, sample size, experimental model, as well as co-morbidities. Inhibin B was significantly increased while FSH and E were significantly decreased compared to the hyperlipidemic rats fed HFD only. These results are consistent with Molina-Vega et al. [54]. The effects of orlistat on E and FSH might be mediated via its weight reducing effect. As previously mentioned, excess adiposity is associated with increased aromatase and consequently high E level. Thus, with weight loss by orlistat the reverse occurs. In addition, Inhibin B exerts a negative feedback response on the pituitary, decreasing FSH production and secretion [54].

In addition, we found that sperm count and viability were significantly decreased with administration of orlistat to rats fed ordinary diet, compared to control level. These results are consistent with that of Jensen et al. [55]. They reported that overweight as well as underweight men had lower total sperm counts and motile spermatozoa compared to men with ideal weights. The low sperm count and viability in these rats was supported by lower T and inhibin B levels with VD−. Moreover, hypolipidemia could affect sperm parameters since dietary FAs play an important role in the modulation of sperm metabolism [5].

Moreover, we found that administration of orlistat and, 25 dihydroxyvitamin (1, 25-$(OH)_2$ D_3) in rats fed ordinary diet caused significant decrease in the serum levels of TC, TG and LDL-c, while insignificant change in HDL-c, compared to the control group. On the other hand, their administration to hyperlipidemic rats fed HFD caused significant decrease in TC, TG and LDL-c and significant increase in HDL-c, when compared to both hyperlipidemic rats fed HFD only and hyperlipidemic rats administered orlistat. This revealed the effect of, 25 $(OH)_2$ D_3 in treating dyslipidemia that occurred by HFD. The results of the current work are in accordance with Alkhatatbeh et al. [56] and Mostafai et al. [57]. The hypolipidemic effect of, 25 $(OH)_2$ D_3 is through suppression of the gene expression involved in lipogenesis and hepatic steatosis [9]. Active vitamin D may influence HDL-c via its effect on apolipoprotein A-1 production or via its effect on cholesterol turnover and transport [58].

The 25 (OHD) level was significantly increased in the present study in either rats fed ordinary diet or those fed HFD after administration of orlistat and 1,25-$(OH)_2D_3$. These results are consistent with the studies of Ghaly et al. [59] and Jahn et al. [60].

Serum T and inhibin B levels were significantly increased in the current study after administration of orlistat and 1,25$(OH)_2D_3$ to rats fed ordinary diet, compared to the normal orlistat administered group, and could reach to the normal level. Their administration to hyperlipidemic rats caused significant increase in T and inhibin B levels and significant decrease in FSH and E, compared to both hyperlipidemic rats fed HFD only and hyperlipidemic rats administered orlistat and could nearly reach to the normal level. These results are in line with that of Canguven et al. [61] and Liu et al. [62]. VDR and the vitamin D metabolizing enzymes are expressed in sertoli cells, germ cells, leydig cells, spermatozoa and in the epithelial cells lining the male reproductive tract [37]. Thus, vitamin D has suggested stimulating testosterone production directly in human Leydig cells and improving other gonadal functions [37]. Moreover, vitamin D treatment upregulate certain genes in mice testis including ATP-binding cassette transporter 1[63]. De Angelis et al. [64] reported that vitamin D increases T secretion by inducing changes in intracellular calcium homeostasis in leydig cells via calbindin-D 28k, a cytosolic calcium-binding protein [64].

Sperm count and viability were significantly increased after administration of orlistat plus $1,25(OH)_2D_3$ to either rats fed ordinary diet or those fed HFD, compared to rats fed HFD and orlistat treated groups and could reach to the normal level. These results are in line with that of Jensen et al. [65] and Liu et al. [62]. Vitamin D regulates human sperm cholesterol outflows, affects sperm protein serine and threonine phosphorylation, and thus increases the survival ability of spermatozoa [66]. In contrary to the present results, the study of Jensen et al. [67] on infertile men showed that supplementation with vitamin D and calcium had no effect on semen quality or live birth rate in men with vitamin D insufficiency. This apparent conflict may be due to the differences in the study model, vitamin D levels, and supplementation protocols. It has been reported that the presence of VDR and vitamin D-metabolizing enzymes may reflect the quality of spermatozoa [68]. Therefore, the poor response of spermatozoa to vitamin D in a previous study may be due to the loss of function of the sperm VDR.

CONCLUSIONS

Lipid disturbance in the form of hyperlipidemia or hypolipidemia was associated with VD− and consequently altered male fertility. There is a link between lipid disturbance and vitamin D on male fertility. Future studies are needed to better clarify the molecular mechanism of vitamin D on hormonal and seminal panel in male fertility in cases of lipid disturbance.

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الملخص العربي

تأثير اضطراب الدهون وفيتامين د على الخصوبة في ذكور الجرذان البيضاء

شيماء فؤاد محمد هيكل[1]، منى محمود البيومى[1]، سماح المتولى ابراهيم[1]، محمد محمد الشو[1]
[1]قسم الفسيولوجيا، كلية طب البنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية.

الملخص

الخلفية: فيتامين د له تأثيرات بيولوجية متعددة على الجهاز التناسلي الذكري، يؤدي نقص فيتامين د واضطراب التمثيل الغذائي للدهون إلى إحداث تغييرات في إنتاج هرمون الخصية والخصائص المنوية التي تتعلق بالعقم عند الذكور.

الهدف: تهدف هذه الدراسة الى معرفة تأثير اضطراب الدهون وفيتامين د على مقاييس الخصوبة فى ذكور الجرذان.

الطرق: أجرى هذا البحث على ٦٠ من ذكور الجرذان، تم تقسيمهم الى ٦ مجموعات، المجموعة الأولى (المجموعة الضابطة)، المجموعة الثانية (المجموعة المعالجة بعقار الاورليستات)، المجموعة الثالثة (المجموعة المعالجة بعقار الاورليستات وفيتامين د)، المجموعة الرابعة (المجموعة عالية الدهون)، المجموعة الخامسة (المجموعة عالية الدهون المعالجة بعقار الاورليستات)، المجموعة السادسة (المجموعة عالية الدهون المعالجة بعقار الاورليستات وفيتامين د). وقد تم قياس نسب الكوليسترول الكلى والكوليسترول فى البروتينات الدهنية منخفضة الكثافة والكوليسترول في البروتينات الدهنية عالية الكثافة والدهون الثلاثية ومستويات فيتامين د والتستوستيرون وإنهيبين ب والهرمون المنشط للحوصلة والاستراديول. وتم ايضا حساب عدد ونسبة حيوية الحيوانات المنوية.

النتائج: أحدث إعطاء عقار الاورليستات للمجموعة الثانية تغيرات لها دلالة إحصائية فى مستويات الدهون في الدم مقارنة بالمجموعة الضابطة. واظهر البحث وجود علاقة بين اضطراب التمثيل الغذائي للدهون ونقص فيتامين د فى الدم والتي بدورها ادت الى نقص فيتامين د في المجموعتين الثانية والخامسة مقارنة بالمجموعة الضابطة. وقد احدث نقص فيتامين د في هاتين المجموعتين انخفاضاً ذو دلالة إحصائية فى مستوى التستوستيرون وإنهيبين ب وعدد ونسبة حيوية الحيوانات المنوية و زياده ذات دلالة إحصائية فى معدل الهرمون المنشط للحوصلة والاستراديول مقارنة بالمجموعة الضابطة. أحدث إعطاء عقار الاورليستات للمجموعة الخامسة تغيرات لها دلالة إحصائية فى مستويات الدهون فى الدم وانخفاضاً فى مستوى الاستراديول وزيادة فى مستوى فيتامين د وإنهيبين ب مقارنة بالمجموعة عالية الدهون. من ناحية اخرى فقد احدث إعطاء فيتامين د مع الاورليستات للمجموعة الثالثة والسادسة زيادة ذات دلالة إحصائية فى مستوى الكوليسترول فى البروتين الدهني عالي الكثافة وفيتامين د والتستوستيرون وإنهيبين ب وعدد ونسبة حيوية الحيوانات المنوية و انخفاضاً ذو دلالة إحصائية فى مستويات الكوليسترول الكلى والدهون الثلاثية والكوليسترول فى البروتين الدهنى منخفض الكثافة و الهرمون المنشط للحوصلة والاستراديول.

الاستنتاجات: اضطراب الدهون فى شكل زيادة الدهون او نقصها يؤدى الى نقص فيتامين د. علاوة على ذلك فيتامين د له تأثير إيجابي على الخصوبة لدى كلا من ذكور الجرذان عالية الدهن ومنخفضة الدهن.

الكلمات المفتاحية: الدهون، الخصوبة لدى الذكور، فيتامين د.

الباحث الرئيسي
الأسم: شيماء فؤاد محمد هيكل، قسم الفسيولوجيا، كلية الطب، بنات، القاهرة، جامعة الأزهر، جمهورية مصر العربية
الهاتف: 01002993474
البريد الإلكتروني: samahelmetwally.medg@azhar.edu.eg-drshimaafouad88@gmail.com