JMGCB

ISSN:3032-1085

https://doi.org/10.61796/jmgcb.v1i4.409

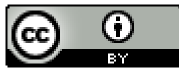
ASSOCIATION BETWEEN FEMALE HORMONES (FSH, LH, AND ESTROGEN) AS WELL AS HYPERLIPIDEMIA AND OBESITY IN AL- NAJAF HOLY CITY

Worood Muslem Jubair
Department of Medical Physics, Al-Mustaqbal University College

Received: Feb 22, 2024; Accepted: March 29, 2024; Published: Apr 30, 2024;

Abstract: An excessively high level of lipids, or fat, in the blood is called hyperlipidemia. The levels of hormones in females were examined, along with their correlation with elevated body fat and impact on cardiovascular conditions. The information was gathered in Najaf City between November 1, 2023, and December 20, 2023.

The results of this study showed that the concentrations for a hormone that promotes follicle development within samples had increased by 58%. LH increased in 50% of the samples that were analyzed. Better pubertal development has been associated with elevated FSH levels in the blood. Improved metabolism has been associated with an increase in blood FSH. Moreover, nonlinear correlations were found between adiposity measures and serum FSH levels.



This is an open-acces article under the CC-BY 4.0 license

Introduction

When blood lipid levels, such as cholesterol and triglycerides, are raised for various underlying reasons, it is referred to as hyperlipidemia (Brown & Valiere, 2004). Steroid hormone production disorder, atherosclerosis, and pancreatitis are the two major life-threatening disorders that are the clinically noticeable effects of hyperlipidemia. In order to be carried in plasma to different tissues, where circulating lipids are either used for cell function, energy usage, steroid hormone generation, and bile acid formation or stored as a form of energy storage because triglycerides and cholesterol are insoluble in plasma (Stewart et al., 2020). FSH is an alpha and beta subunit glycoprotein dimer. Whereas the alpha subunit is the same in TSH, hCG, and LH, the beta subunit is specific to FSH, FSH release is promoted by GnRH, GnRH is produced by the hypothalamus and delivered through the hypophyseal portal circulation to operate on gonadotropic cells in the anterior pituitary's G protein-coupled receptors (Haldar et al., 2022). FSH and luteinizing hormone. LH which are produced by those gonadotropic cells and released further peripheral circulation. Pulsatile GnRH release occurs, having low pulse frequencies activating more FSH and higher pulse frequency stimulating more LH. Chronic GnRH use limits the anterior pituitary's secretion of FSH and LH, which prevents women from ovulating and producing estrogen (Orlowski & Sarao, 2019). Hyperlipidemia refers to lipids (including cholesterol and triglycerides) in the blood due to various underlying causes. The clinically significant consequences of hyperlipidemia are two main life-threatening diseases: atherosclerosis and pancreatitis. Cholesterol and triglycerides are insoluble in the plasma, and circulatory lipids are linked to Apo lipoproteins to transport them to different tissues in which lipids are used for cell

function, energy use, steroid hormone production, and biliary acid formation, or as a form of energy storage (Stewart et al., 2020). A variety of lipoproteins of different chemical composition can be identified in plasma. Based on their density, lipoproteins are commonly described as very-low density lipoproteins (VLDLs), intermediate-density lipoproteins (IDLs), low- density lipoproteins (LDLs), high-density lipoproteins (HDLs), and chylomicrons classification of hyperlipidemias based on these discrete forms of lipoproteins. Chylomicrons are very large particles that carry dietary lipid. They are associated with a variety of Apo lipoproteins, including A-I, A-II, A-IV, B-48, C-I, C-II, C-III, and E (Bhavani, 2015).Very low-density lipoprotein carries endogenous triglycerides and to a lesser degree cholesterol. The major Apo lipoproteins associated with VLDL are B-100, C-I, C-II, C-III, and E. Intermediate-Density Lipoprotein Intermediate-density lipoprotein carries cholesterol esters and triglycerides. It is associated with Apo lipoproteins B-100, C-III, and E (Bhavani, 2015). Low-density lipoprotein carries cholesterol esters and is associated with Apo lipoprotein B-100(Chung et al., 2022). High-density lipoprotein also carries cholesterol esters. Apolipoproteins A-I, A-II, C-I, C-II, C-III, D, and E, are connected to it. Depending on the lipid content of the individual lipoprotein anomaly, abnormalities in plasma levels of VLDL, IDL, LDL, HDL, or chylomicrons might cause hypercholesterolemia, hypertriglyceridemia, or mixed hyperlipidemia. From a clinical perspective, assessing hyperlipidemias and hyper-lipoproteinemia based on their probable side effects, such as atherosclerosis and pancreatitis, is helpful. The risk of FSH, LH, and atherosclerotic diseases is correlated with abnormalities in the metabolism of VLDL, IDL, and LDL, which are apoprotein B-100 (apoB-100)-containing lipoproteins, and HDL, which is apoprotein A-containing lipoproteins. Pancreatitis risk is correlated with abnormalities in chylomicron (lipoproteins including apolipoprotein B-48) metabolism (Petersmann et al., 2019). Obesity, type II diabetes, thyroid dysfunction, alcoholism, renal disorders, jaundice, hormone therapy, a mutation in lipoprotein lipase, and drugs like diuretics, B-receptor blockers, & cyclosporine are indeed the main causes of hyperlipidemia. (Naser et al., 2021). Also it have a secondary causes tha The patient learns they have hyperlipidemia through routine blood testing because the disorder's signs and symptoms might not be obvious. Patients commonly have heart diseases, athermal plaques in blood vessels, hepatic enlargement, abdominal pain, spleen hypertrophy, and pain in the chest and. Several studies and researchers have attempted to describe the precise biology of hyperlipidemia in more depth, due to the disease's significant consequences and elevated rates of death and morbidity. To detect the condition early and begin effective therapy, this is necessary to reduce consequences and enhance the patient's condition (Brown & Valiere, 2004). According to recent studies and reviews, endothelial damage to blood vessels causes a loss of nitric oxide, which increases inflammation around the affected area and causes an increase in lipids within the deepest layer of the endothelial wall, where they are then engulfed by macrophage cells to form what is known as (the foam cell) with the cholesterol content. Foamy cell development will result in necrosis, apoptosis, and mitochondrial dysfunction. It may also result in hormonal disruption (FSH, LH) (Naser et al., 2021; Stewart et al., 2020). The liver and gastrointestinal tract both produce this discoidal lipoprotein. Unless it is supplemented with cholesterol esters, it is quickly hydrolyzed in the bloodstream. Via a sequence of processes that include fast esters of free cholesterol outside the cell and cholesterol outflow from cytoplasm through the plasma membrane, cholesterol ester absorption from the macrophage/foam cell can happen. The cholesterol efflux regulatory protein mediates the movement of cholesterol from across the plasma membrane (CERP) (Kaiser et al., 2017; Naser et al., 2021). The enzyme lecithin cholesterol acetyltransferase (LCAT) facilitates the transfer of fatty acids from the developing HDL's phospholipid core to the free cholesterol leaving the macrophage. As a result, developing HDL

particles gradually become more enriched with cholesterol and mature. Hepatocyte, adrenal, and gonad scavenger receptor class B type 1 (SR-B1) receptors identify mature HDL. As a result, HDL particles facilitate the transportation of reverse cholesterol from peripheral cells to the liver and steroidogenic tissues (Huang et al., 2015; Naser et al., 2021).The reported preventive effects of HDL on the development of atherosclerotic plaque appear to be predominantly mediated by this function. The antioxidant properties of HDL to inhibit LDL modification and detection of hepatic chylomicron remnant receptors by macrophages' scavenger receptors are two additional theories that have been put forth. Chylomicrons shouldn't be seen in post-absorptive circumstances (Naser et al., 2021).Elevated hepatocyte synthesis of VLDL and/or reduced clearance are two possible causes. Genetic diseases including familial mixed hyperlipidemia are known to cause an increase in VLDL production. The more frequent secondary causes of elevated VLDL levels are excessive consumption of fats, calories, and alcohol, in addition to diabetes, insulin resistance, nephrotic syndrome, hypothyroidism, and obesity. Genetic disorders like LPL deficiency and apo C-II deficiency are known to cause decreased clearance of VLDL. Insulin resistance, type 2 diabetes, and hypothyroidism are secondary causes of reduced VLDL clearance. These circumstances either directly reduce LPL activity or boost apoC- III production while decreasing apoC-II production. IDL frequently rises when isoform apoE-2 homozygosity is present (Goldberg & Chait, 2020). The isoform of apoE lowers the absorption of VLDL remnants and IDL because it has the lowest interaction for the LDL receptor. Unless a simultaneous secondary cause, such as hypothyroidism, diabetes, or postmenopausal status, is present, this hyper-lipoproteinemia typically does not show up (Packard et al., 2020). syndrome. When the liver's LDL receptor is not cleared properly, LDL levels rise. Both familial and polygenic high cholesterol exhibit defective receptor- mediated LDL elimination. Obesity and excessive consumption of saturated fats in the diet are secondary causes of impaired receptor-mediated clearance of LDL. The most significant risk factor for coronary artery disease is increased LDL. It may also show tendon symptoms(Armstrong & Krueger, 2016). Folliculogenesis in the ovary it is a phenomenon of incredible complexity, and our understanding of it is evolving constantly. A pathophysiological schema that focuses on the key players is necessary, even if it isn't full, to guide research and help with patient care. In order for human ovarian folliculogenesis to occur, androgens, FSH, anti-Müllerian hormones (AMH), and estradiol (E2) are all necessary. It is unclear how these four players interact with one another, though (Dewailly et al., 2016). The gonadotropin - releasing hormone cells in the adenohypophysis co- secrete luteinizing hormone (LH), a glycoprotein hormone, alongside follicle- stimulating hormone (anterior pituitary). The hypothalamus, the pituitary, and the gonads are all components of the neurological pathway that luteinizing hormone is a part of gonadotropin-releasing hormone (GnRH) stimulates LH release through this route, which is blocked by estrogen in females and androgen in males(Xu et al., 2022). LH performs a variety of tasks that are different for men and women. LH assists in the maturation of primordial germ cells in both genders. LH stimulates the production of testosterone in the testes' Leydig cells in men. LH causes the ovaries to produce steroid hormones in females. LH also assists in controlling the duration and timing of the Menopause is linked to negative changes in lipid levels and other risk factors for cardiovascular disease (CVD). Though reductions in estrogen levels have been linked to many of the physiological changes associated with menopause, there is mounting evidence that follicle-stimulating hormone (FSH) may have a separate impact on the risk of CVD (Lee et al., 2022). Throughout the perimenopausal years, FSH levels start to rise, and they fluctuate somewhat into postmenopause. There is debate regarding how the increase in FSH affects the risk of CVD. Some research has revealed a link between high FSH levels and a higher risk of CVD, whilst other studies

have found a link between high FSH levels and a decreased risk. There is growing evidence that FSH may be related to CVD risk through its connection, regardless of the direction of the link (Serviente et al., 2019).

. This study Aims to Observe the relation between hyperlipidemia and (FSH, LH) levels and serum lipid profiles in adult female. Observe the relation between hyperlipidemia and estrogen level in serum lipid profiles in adult female

Methods

3.2. Sample Collection

Thirty-four serum samples were collected from adult female from January to 1 February 2023. Analyzed by using kit of Elisa for detected FSH, LH and estrogen.

All serum sample were cheeked by the Elisa kit for detect the levels of FSH, LH and estrogen in female.

Figure (3.2) Elisa kit. Analysis positive, negative control and FSH, LH and estrogen

3.3. Assay Technique

Female E2 (Estradiol) ELISA KIT, FSH, LH, and estrogen values can be determined by:

1. Add the Standard working solution to the first eight columns: Each concentration of the solution is added, to well each, (from 200 uL to zero uL for each stander well). Add the samples to the other wells (50 uL for each well). Immediately add 50 µL of Biotinylated Detection Ab working solution to each well. Cover the plate with the sealer provided in the kit. Incubate for 45 min at 37°C. Note: solutions should be added to the bottom of the micro ELISA plate well, avoid touching the inside wall and causing foaming as much as possible.
2. Aspirate or decant the solution from each well add 350 uL of wash buffer to each well. Soak for 1~2 min and aspirate or decant the solution from each well and pat it dry against clean absorbent paper. Repeat this wash step 3 times. Note: a microplate washer can be used in this step and other wash steps.

3. Add 100 µL of HRP Conjugate working solution to each well. Cover with the Plate sealer. Incubate for 30 min at 37°C.
4. Aspirate or decant the solution from each well, repeat the wash process for five times as conducted in step 2.
5. Add 90 µL of Substrate Reagent to each well. Cover with a new plate sealer. Incubate for about 15 min at 37°C. Protect the plate from light. Note: the reaction time can be shortened or extended according to the actual color change, but not more than 30min.
6. Add 50 µL of Stop Solution to each well. Note: Adding the stop solution should be done in the same order as the substrate solution.
7. Determine the optical density (OD value) of each well at once with a micro-plate reader set to 450 nm.

Results and Discussion

Results

4.1. Sample of FSH

Our research facility examined 36 samples (FSH kit) Six (16%) of the samples were normal, while twenty-four (58%) a specimen indicated a rise in FSH, while fifteen (41%) indicated a drop that is shown in table (1).

Table (4.2) FSH sample. (a: refer to stander samples) (b: refer to samples with an increased level of FSH) (c: refer to sample with decreased FSH level)

Stander	No. Samples of FSH					
0.087	0.089[c]	0.091[c]	0.093[c]	0.103[c]	0.131[b]	0.086[a]
0.088	0.165[b]	0.085[c]	0.091[c]	0.097[c]	0.124[b]	0.105[a]
0.092	0.094[c]	0.098[c]	0.1[b]	0.099[c]	0.117[b]	0.132[a]
0.085	0.095[c]	0.105[b]	0.1[b]	0.017[c]	0.194[b]	0.173[a]
0.093	0.103[c]	0.105[b]	0.094[c]	0.124[b]	0.177[b]	0.127[a]
0.091	0.098[c]	0.106[b]	0.1[b]	0.126[b]	0.166[b]	0.116[a]
0.099	0.099[c]	0.11[c]	0.118[b]	0.096[c]	0.208[b]	0.145[b]
0.112	0.114[b]	0.132[b]	0.109[b]	0.125[b]	0.19[b]	0.162[b]

4.1.2. Sample of Estrogen

The experiment examined 36 sample data (estrogen kit) Six (16%) of the samples are normal, 18 (50%) of the cases exhibited an increase in estrogen, and 18 (50%) of the specimens indicated a drop in hormone levels.

Table (4.3) Estrogen sample. (a: refer to stander samples) (b: refer to samples with an increased level of Estrogen) (c: refer to sample with decreased Estrogen level)

Stander	No. Samples of Estrogen					
106	0.1^{b}	0.113^{c}	0.148^{b}	0.141^{b}	0.153^{b}	0.117^{a}
106	0.097^{c}	0.099^{c}	0.102^{c}	0.109^{c}	0.121^{b}	0.112^{a}
145	0.105^{c}	0.109^{c}	0.109^{c}	0.144^{b}	0.112^{c}	0.109^{a}
121	0.102^{c}	0.119^{c}	0.116^{c}	0.115^{c}	0.149^{b}	0.054^{a}
137	0.123^{b}	0.123^{b}	0.117^{c}	0.108^{c}	0.148^{b}	0.081^{a}
116	0.114^{c}	0.117^{c}	0.12^{c}	0.121^{b}	0.135^{b}	0.065^{a}
157	0.106^{c}	0.115^{c}	0.115^{c}	0.105^{c}	0.124^{b}	0.069^{c}
162	0.144^{b}	0.159^{b}	0.138^{b}	0.122^{b}	0.152^{b}	0.062^{c}

4.1.3. Sample of LH

The research examined 36 specimens (LH kit) Six (16%) of the measurements were normal, twelve (33%) demonstrated an upsurge throughout LSH, and 18 (50%) indicated a decline in LH levels.

Table (4.1) LH sample. (a: refer to stander samples) (b: refer to samples with an increased level of LH) (c: refer to sample with decreased LH level)

Stander	No. Samples of LH					
0.091	0.112^{b}	0.069^{c}	0.088^{c}	0.103^{c}	0.093^{c}	0.138^{a}
0.117	0.104^{c}	0.103^{b}	0.099^{c}	0.095^{c}	0.093^{c}	0.103^{a}
0.124	0.122^{b}	0.091^{c}	0.109^{b}	0.109^{b}	0.112^{b}	0.121^{a}
0.096	0.102^{c}	0.087^{c}	0.084^{c}	0.083^{c}	0.087^{a}	0.051^{a}
0.089	0.097^{c}	0.073^{c}	0.067^{c}	0.079^{c}	0.071^{c}	0.047^{a}
0.133	0.118^{b}	0.124^{b}	0.106^{b}	0.101^{c}	0.093^{c}	0.05^{a}
0.113	0.116^{b}	0.095^{c}	0.132^{b}	0.104^{b}	0.087^{c}	0.043^{c}

0.086	0.085[c]	0.074[c]	0.083[c]	0.086[c]	0.091[c]	0.053 c

4.2. Statistical analysis

Several specimens of the eighteen samples of the female rat's FSH level in the T-analysis had a huge value of (0.05%), while other samples had no substantial value of (0.05%). While other samples do not exhibit a significant value at 0.05%, an LH statistical test reveals a significant result for such twelve human samples, while other samples show no significant value at 0.05%. Six samples will have a significant value for the estrogen level at (0.05%), whereas the remaining samples will not have a substantial value at (0.05%). that was illustrated in table (4.4).

Table (4.4) Statistical analysis of (FSH, LH and Estrogen).

		FSH	FSH	FSH	LH	LH	Estrogen
N		6	6	6	6	6	6
		.278	.278	.348	.300	.348	.266
	Positive	.278	.278	.218	.300	.348	.266
	Negative	-.143	-.143	-.348	-.240	-.197	-.125
	Positive	.278	.278	.348	.300	.348	.266
Significant at p value at (0.05%)		.007[a]	.007[a]	.005[a]	.033[c]	.005[c]	.099[c]

Conclusion

1. An elevated blood FSH levels of rats was linked to improved pubertal development.
2. An elevated serum FSH is linked to an increase in metabolism.

Furthermore, we discovered nonlinear relationships among varying sera FSH levels and adiposity indicators.

3. FSH may play a physiological and/or pathological role in the regulation of glucose levels, hence regulating lipid levels in the body.
4. The association of LH levels and increased HDL cholesterol levels suggests that.

References

[1]. Aritonang, T. R., Rahayu, S., Sirait, L. I., Karo, M. B., Simanjuntak, T. P., Natzir, R., Sinrang, A. W., Massi, M. N., Hatta, M., & Kamelia, E. (2017). The Role of FSH, LH, Estradiol and Progesterone Hormone on Estrus Cycle of Female Rats. International Journal of Sciences: Basic and Applied Research (IJSBAR), 35(1), 92–100.

[2]. Armstrong, E. J., & Krueger, J. G. (2016). Lipoprotein Metabolism and Inflammation in Patients with Psoriasis. American Journal of Cardiology, 118(4), 603–609.

[3]. Aydin, B. K., Stenlid, R., Ciba, I., Cerenius, S. Y., Dahlbom, M., Bergsten, P., Nergårdh, R., & Forslund, A. (2022). High levels of FSH before puberty are associated with increased risk of metabolic syndrome during pubertal transition. Pediatric Obesity, 17(8), 1–12.
[4]. Bhavani, N. (2015). Hyperlipidemia. In PG Textbook of Pediatrics: SystemicDisorders and Social Pediatrics (Volume 3) (Vol. 28, Issue 3, pp. 2596–2596). Jaypee Brothers Medical Publishers (P) Ltd.

[5]. Brown, A. C., & Valiere, A. (2004). Probiotics and medical nutrition therapy. Nutrition in Clinical Care : An Official Publication of Tufts University, 7(2),56–68.
[6]. Brown, A. C., & Valiere, A. (2004). Probiotics and medical nutrition therapy. Nutrition in Clinical Care : An Official Publication of Tufts University, 7(2),56–68.
[7]. Chung, S. J., Lim, H. S., Lee, M. Y., Lee, Y. T., Yoon, K. J., & Park, C. H. (2022). Sex-Specific Associations between Serum Ferritin and Osteosarcopenic Obesity in Adults Aged over 50 Years. Nutrients, 14(19).
[8]. Coutinho, & Kauffman. (2019).The Role of the Brain in the Pathogenesis andPhysiology of Polycystic Ovary Syndrome (PCOS). Medical Sciences, 7(8), 84.
[9]. Dewailly, D., Robin, G., Peigne, M., Decanter, C., Pigny, P., & Catteau- Jonard, S. (2016). Interactions between androgens, FSH, anti-Mullerian

[10]. Estradiol level of male rat is correlated with depression and anxiety after traumatic stress. Journal of Medical Sciences, 38(4), 176.
[11]. Fourman, L. T., & Fazeli, P. K. (2015). Neuroendocrine causes of amenorrhea
[12]. Fraietta, R., Zylberstejn, D. S., & Esteves, S. C. (2013). Hypogonadotropic hypogonadism revisited. Clinics (Sao Paulo, Brazil), 68 Suppl 1(Suppl 1), 81–88.
[13]. Goldberg, R. B., & Chait, A. (2020). A Comprehensive Update on theChylomicronemia Syndrome. Frontiers in Endocrinology, 11(October), 1–13.
[14]. Haldar, S., Agrawal, H., Saha, S., Straughn, A. R., Roy, P., & Kakar, S. S. (2022). Overview of follicle stimulating hormone and its receptors in reproduction and in stem cells and cancer stem cells. International Journal ofBiological Sciences, 18(2), 675–692.
[15]. hormone and estradiol during folliculogenesis in the human normal and polycystic ovary. Human Reproduction Update, 22(6), 709–724.
[16]. Huang, L. H., Elvington, A., & Randolph, G. J. (2015). The role of the lymphatic system in cholesterol transport. Frontiers in Pharmacology, 6(SEP),1–11.
[17]. Kaiser, T., Kinny-Köster, B., Bartels, M., Berg, T., Scholz, M., Engelmann, C., Seehofer, D., Becker, S., Ceglarek, U., & Thiery, J. (2017). Cholesterol esterification in plasma as a biomarker for liver function and prediction of mortality. BMC Gastroenterology, 17(1), 1–7.
[18]. Kumar, P., & Sait, S. (2011). Luteinizing hormone and its dilemma inovulation induction. Journal of Human Reproductive Sciences, 4(1), 2–7.
[19]. Lan, H.-C., Lin, J.-H., Chen, C.-H., Chu, P.-W., & Cheng, C.-P. (2018).
[20]. Laqif, A., DJ Anwar, D. K., & Melinawati, E. (2018). Effect of ovarian auto transplantation on FSH levels in Wistar rats late menopause model. Majalah Obstetri & Ginekologi, 26(1), 42.
[21]. Lee, S. W., Hwang, I. S., Jung, G., Kang, H. J., & Chung, Y. H. (2022). Relationship between metabolic syndrome and follicle-stimulating hormone in postmenopausal women. Medicine (United States), 101(18), E29216.
[22]. Lee, S. W., Hwang, I. S., Jung, G., Kang, H. J., & Chung, Y. H. (2022). Relationship between metabolic syndrome and follicle-stimulating hormone in postmenopausal women. Medicine (United States), 101(18), E29216.
[23]. Martin, L. J., & Touaibia, M. (2020). Improvement of testicular steroidogenesis using flavonoids and isoflavonoids for prevention of late- onset male hypogonadism. Antioxidants, 9(3).
[24]. Naser, I. H., Alkareem, Z. A., & Mosa, A. U. (2021). Hyperlipidemia : pathophysiology, causes,

complications, and treatment. A review.

[25]. Orlowski, M., & Sarao, M. S. (2019). Physiology, Follicle Stimulating Hormone. StatPearls, December.

[26]. Packard, C. J., Boren, J., & Taskinen, M. R. (2020). Causes and Consequences of Hypertriglyceridemia. Frontiers in Endocrinology, 11(May), 1–15.

[27]. Petersmann, A., Müller-Wieland, D., Müller, U. A., Landgraf, R., Nauck, M.,Freckmann, G., Heinemann, L., & Schleicher, E. (2019). Definition, Classification and Diagnosis of Diabetes Mellitus. Experimental and Clinical Endocrinology & Diabetes : Official Journal, German Society of Endocrinology [and] German Diabetes Association, 127(S 01), S1–S7.

[28]. Santi, D., Crépieux, P., Reiter, E., Spaggiari, G., Brigante, G., Casarini, L., Rochira, V., & Simoni, M. (2020). Follicle-stimulating hormone (FSH) actionon spermatogenesis: A focus on physiological and therapeutic roles. Journal of Clinical Medicine, 9(4).

[29]. Sekar Mahadani, S., Herawati, L., & Ratna Dwiningsih, S. (2021). The Combination of High Calorie Diet and Moderate Interval Training DecreasesFollicle Stimulating Hormone (FSH) Level In Rats. International Journal of Research Publications, 91(1), 288–295.

[30]. Serviente, C., Tuomainen, T.-P., Virtanen, J., Witkowski, S., Niskanen, L., &Bertone-Johnson, E. (2019). Follicle-stimulating hormone is associated with lipids in postmenopausal women. Menopause, 26(5), 540–545.

[31]. Stewart, J., McCallin, T., Martinez, J., Chacko, S., & Yusuf, S. (2020). Hyperlipidemia. Pediatrics in Review, 41(8), 393–402.

[32]. Stewart, J., McCallin, T., Martinez, J., Chacko, S., & Yusuf, S. (2020). Hyperlipidemia. Pediatrics in Review, 41(8), 393–402.

[33]. Stewart, J., McCallin, T., Martinez, J., Chacko, S., & Yusuf, S. (2020). Hyperlipidemia. Pediatrics in Review, 41(8), 393–402.

[34]. Wopara, I., Modo, E. U., Mobisson, S. K., Olusegun, G. A., Umoren, E. B., Orji, B. O., Mounmbegna, P. E., & Ujunwa, S. O. (2021). Synthetic food dyescause testicular damage via up-regulation of pro-inflammatory cytokines and down-regulation of fsh-r and tesk-1 gene expression. Jornal Brasileiro deReproducao Assistida, 25(3), 341–348

[35]. Xu, Z., Gu, S., Wu, X., Zhou, Y., Li, H., & Tang, X. (2022). Association of follicle stimulating hormone and serum lipid profiles in postmenopausal women. Medicine (United States), 101(39), E30920.