ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

Male Obesity and Reproductive Health

Duo Li[1,a], Xianhui Dong[2,b], Li Li[1,c,*]

[1]*School of Nursing, Hangzhou Normal University, Hangzhou, Zhejiang, China*
[2]*Obesity Clinic, Affiliated Hospital of Hangzhou Normal University, Hangzhou, Zhejiang, China*
[a]*18079974441@163.com,* [b]*lislucy@163.com,* [c]*tcmdongxianhui@126.com*
[*]*Corresponding author*

Abstract: *Obesity is the most common chronic disease worldwide, with numerous adverse health effects. In addition to contributing to type 2 diabetes and cardiovascular disease, obesity increases the risk of conditions such as obstructive sleep apnea and osteoarthritis. Furthermore, it is closely associated with impaired male reproductive health. In recent years, as global obesity rates have risen dramatically, male reproductive health disorders have also increased significantly. This paper reviews the relationship between obesity and male reproductive dysfunction, explores the underlying mechanisms of obesity-related male infertility, and proposes potential interventions to improve reproductive health outcomes.*

Keywords: *Male Obesity; Reproductive Health; Male Infertility*

1. Introduction

A body mass index (BMI) of≧30 kg/m^2 indicates obesity, which is defined by the World Health Organization (WHO) as a metabolic disorder caused by excessive accumulation of body fat[1] .The human body has a diverse distribution of adipose tissue and one of the main characteristics of our population is the predominance of visceral fat in the abdominal cavity. Therefore, the most common form of obesity in our country is centripetal obesity, sometimes referred to as abdominal obesity[2] . Studies have shown a strong correlation between male reproductive health and the accumulation of abdominal fat[3] , which may lead to hormonal abnormalities, poor sperm quality and reduced fertility. China now has the largest obese population in the world[5] , as obesity rates have skyrocketed over the past few decades[4] . However, the impact of obesity goes far beyond this; it is strongly associated with the risk of a number of chronic diseases, including diabetes, heart disease, hypertension, and dyslipidemia[6] , which poses a serious problem for both individual health and the social healthcare system. Obesity is a major problem that needs to be addressed urgently, especially because of its distributional characteristics and impact on male reproductive health. According to recent data[7] , more than 20% of men of reproductive age are obese. In recent years, low fertility and obesity rates in men have steadily increased. In addition to the general decline in semen quality and the varying degrees of impact on sperm count, motility, and morphology, many studies have shown that the fertility of obese men is much lower than that of normal-weight men[8-10] . In this paper, by summarizing the relationship between male obesity and reproductive health, briefly exploring the intrinsic mechanism of male obesity leading to infertility, and combining with previous studies, organizing and sorting out the relevant preventive strategies that can improve male obesity, thus improving male fertility.

2. Effects of male obesity on reproductive health

2.1 Decline in sperm quality

Semen quality is an important reflection of testicular function and a key factor in assessing male fertility. Male infertility caused by obesity has complex mechanisms, including many possible influences. Obesity has a complex effect on semen quality, resulting in decreased sperm count, decreased motility, and increased malformations.

2.1.1 Decreased sperm count

(1) Oxidative stress

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

One of the key causes of male infertility is sperm destruction caused by reactive oxygen species (ROS). The imbalance between the body's antioxidant capacity and the production of reactive oxygen species is known as oxidative stress[11]. Studies have shown a significant positive correlation between the body mass index (BMI) of male spermatozoa and their level of oxidative stress[12]. Obesity exacerbates oxidative stress in spermatozoa, which can damage sperm DNA and drastically reduce their ability to fertilize[13]. This inhibits spermatogenesis and reduces sperm count.

(2) Hormonal Imbalance

Testosterone is essential to maintain healthy male reproductive function. Once testosterone binds and activates its receptors, it can initiate the process of spermatogenesis by binding to certain nucleotides in chromosomal DNA. Lack of testosterone affects the ability of spermatogenic cells to proliferate and undergo apoptosis, which prevents proper initiation of spermatogenesis and reduces sperm count[14]. In addition, testosterone deficiency leads to decreased sperm viability and quality. Functional gonadal hypoplasia or low testosterone is common in obese men and significantly impairs spermatogenesis and male fertility[15]. Spermatogenesis is inhibited by hypothalamic-pituitary-gonadal (HPG) axis dysfunction and elevated aromatase activity in adipose tissue, which reduces testosterone levels[16].

2.1.2 Decreased sperm motility

(1) Metabolic abnormalities

Insulin resistance disrupts the hormonal balance of the HPG axis, leading to hypogonadism and decreased testosterone synthesis, which affects spermatogenesis. Insulin resistance and hyperglycemia are common metabolic disorders associated with obesity, and they interfere with the energy metabolism of spermatozoa, thereby decreasing sperm motility[17].

(2) Inflammatory response

Obesity can reduce sperm quality by inducing an inflammatory response. Obese men have significantly higher levels of inflammatory factors in serum, testicular tissue and seminal plasma, including tumor necrosis factor α (TNF-α), interleukin-1 (IL-1), interleukin-6 (IL-6) and interleukin-18 (IL-18)[18]. By altering the blood-testis barrier, decreasing spermatogonia differentiation, inhibiting meiotic DNA synthesis, decreasing sperm viability, and interfering with gap junction communication in supporting cells, these inflammatory substances may have a direct effect on spermatogenesis[19].

2.1.3 Increased malformation rate

(1) DNA damage

Obesity is strongly associated with higher rates of sperm malformations and DNA damage, and there is growing evidence that fragmentation of sperm DNA in men is strongly associated with the accumulation of abdominal fat[20,21]. The main reason for this is increased oxidative stress and high levels of reactive oxygen species (ROS) leading to lipid peroxidation, DNA damage, enzyme inactivation, and oxidation of sperm proteins, which leads to sperm malformations[22].

(2) Epigenetic alterations

Through multiple pathways, including oxidative stress, chronic inflammation, hormonal imbalance, abnormal lipid metabolism, and insulin resistance, male obesity causes epigenetic changes, including abnormal sperm DNA methylation, altered histone modifications, and dysregulated noncoding RNA expression[18, 20]. These alterations affect sperm gene expression and function, decrease sperm quality, increase the likelihood of sperm abnormalities, and may negatively affect the health of the offspring.

2.2 Endocrine disruption

Obesity interferes with the endocrine system and affects the secretion of reproductive hormones, leading to significant changes in hormone levels in men, mainly in the form of decreased levels of androgens (e.g., testosterone) and increased levels of estrogens. The specific mechanisms are as follows:

2.2.1 Decrease of androgen

One of the main features of physiological changes in adipose tissue in the obese male group is the increase in aromatase activity. Adipose tissue is an active organ that has an endocrine function in addition to storing energy. Adipose tissue in the male body increases significantly in obesity, as does

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

the activity and amount of aromatase. Through a series of biochemical processes, this key catalytic enzyme is able to permanently convert testosterone, an important androgen in the male body, into estrogen. The effectiveness of the conversion of testosterone to estrogen increases with the increase in aromatase activity, which ultimately leads to a significant decrease in testosterone levels in the body. This can upset the natural hormonal balance and may lead to a number of health problems, such as loss of muscle mass, metabolic abnormalities and decreased sexual function[23] .

2.2.2 Hypothalamic-pituitary-gonadal axis dysfunction

Hypothalamic-pituitary-gonadal (HPG) axis dysfunction is the main way in which obesity affects the reproductive endocrine system. Obese individuals have insulin resistance and persistent low-grade inflammation due to excess adipose tissue, which prevents the hypothalamus from producing gonadotropin-releasing hormone (GnRH)[24] . Pituitary function is directly affected by the decline in GnRH, which leads to a decrease in follicle-stimulating hormone and luteinizing hormone (LH) release.This affects testicular function, creating a vicious cycle that further impairs male fertility.

2.2.3 Leptin resistance

Adipocytes secrete the important hormone leptin, which is essential for controlling the body's endocrine and metabolic processes. Leptin can reduce testosterone production through direct action on testicular interstitial cells, which interferes with intracellular signaling pathways and inhibits the expression and activity of important enzymes involved in testosterone synthesis. The accumulation of adipose tissue in obese individuals leads to a significant increase in leptin release[25] . However, prolonged high levels of leptin may lead to leptin resistance and reduce the body's sensitivity to leptin. The direct inhibitory effect of leptin on testicular interstitial cells may still exist and may even be enhanced due to elevated leptin levels in the body. Obesity negatively affects people's physical and mental health because it not only decreases testosterone production but may also lead to gonadal dysfunction, which can cause problems including decreased libido and decreased sperm quality.

2.2.4 Elevated estrogen

Estrogen levels in the body rise due to increased deposition of adipose tissue, which promotes the conversion of testosterone to estradiol[26] . The disruption of the negative feedback regulatory mechanism of the hypothalamic-pituitary-gonadal axis as a result of rising estrogen levels and decreasing testosterone levels triggers a vicious cycle of infertility and declining sperm counts.

These hormonal changes not only affect spermatogenesis but may also lead to decreased libido, decreased muscle mass, and other manifestations of diminished masculinity, further exacerbating reproductive health problems in obese men.

2.3 Sexual dysfunction

Obesity is an important risk factor for sexual dysfunction in men, which is mainly characterized by erectile dysfunction (ED) and decreased libido. The following are the specific mechanisms and manifestations of sexual dysfunction due to obesity:

2.3.1 Erectile dysfunction (ED)

(1) Endothelial dysfunction

Insulin resistance and metabolic syndrome, often associated with obesity, may impair endothelial function. Obesity-induced chronic inflammation and oxidative stress impair the normal function of endothelial cells, which in turn affects blood flow to the cavernous body of the penis. Endothelial cells are essential for controlling vasodilation and constriction[27] . Erectile dysfunction is directly caused by inadequate blood supply.

(2) Decreased nitric oxide (NO)

Nitric oxide (NO) is an important mediator of penile erection, as it relaxes vascular smooth muscle and promotes engorgement of the cavernous body of the penis. Increased inflammatory factors (including TNF-α and IL-6) in obese individuals can limit the function of nitric oxide synthase (NOS), thereby reducing NO production[28] . In addition, oxidative stress associated with obesity may accelerate the breakdown of nitric oxide, reducing its physiological effects and increasing the risk of erectile dysfunction.

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

2.3.2 Decreased libido

Hypoactive libido is another common manifestation of sexual dysfunction in obese men, which is mainly related to hormonal imbalance.

(1) Low testosterone level

The key hormone that maintains male desire and sexual function is testosterone. Functional hypogonadism, which is characterized by significantly lower testosterone levels, is common in obese men. Low testosterone levels affect the initiation and maintenance of sexual behavior[15], as well as directly reducing libido. Insufficient testosterone also leads to a further decline in sexual function, increased fatigue, and reduced muscle mass.

(2) High estrogen levels

Due to increased aromatase activity in adipose tissue, obese men convert more testosterone to estrogen, which raises estrogen levels in the body. Through a negative feedback mechanism, high estrogen levels inhibit the hypothalamic-pituitary-gonadal (HPG) axis, which further decreases testosterone production and begins a vicious cycle[26]. In addition to decreasing libido, high estrogen levels can exacerbate sexual dysfunction by affecting emotional and psychological well-being.

3. Intervention strategies

3.1 Lifestyle intervention

Lifestyle intervention is the basis for improving the reproductive health of obese men, mainly including diet control and exercise intervention, which can effectively regulate chronic inflammation and oxidative stress, thus improving sperm quality and sexual function.

3.1.1 Diet control and weight loss

(1) High-protein diet

Male reproductive health is significantly affected by dietary components, of which protein and antioxidant intake are particularly important. The major endocrine system controlling male reproductive function is the hypothalamic-pituitary-testicular axis. Hormone synthesis and secretion in this axis may be affected by inadequate protein intake, which is the basic building block of life. This may result in decreased secretion of luteinizing hormone and gonadotropin-releasing hormone, which reduces testosterone synthesis, impairs spermatogenesis, and decreases male fertility potential[29]. Obese men often suffer from chronic inflammation and oxidative stress due to fat accumulation, which leads to sperm DNA damage and decreased viability. Fertility in obese men can be improved by consuming a diet rich in antioxidants such as vitamins C and E, zinc, and selenium, which are found in fresh fruits and vegetables, nuts, and deep-sea fish. These antioxidants neutralize free radicals, reduce inflammatory responses, repair damaged germ cells, and significantly improve sperm quality and function[30].

(2) Mediterranean Diet

The Mediterranean diet has been recognized by UNESCO as an “intangible cultural heritage” and is known for its rich antioxidant and anti-inflammatory content[31]. Vegetables, fruits, legumes, whole grains, olive oil, and fish make up the majority of the diet, while red meat, dairy products, sweets, and highly processed foods are consumed to a lesser extent[32]. Increased testosterone levels and improved sperm quality are two of the main advantages of MD antioxidant ingredients, which include vitamin A, B vitamins, omega-3, zinc, selenium, N acetylcysteine and alpha-lipoic acid[33,34].

3.1.2 Exercise interventions

Regular exercise helps to improve sexual function and hormone levels. According to research[35], men who exercise regularly have higher sperm motility than sedentary individuals. Sperm quality can be effectively promoted by moderate-intensity aerobic exercise[36]. By enhancing the antioxidant defense of the testes, decreasing pro-inflammatory cytokines, and promoting steroidogenesis, exercise has significant potential for the treatment of lifestyle-related male infertility[37].

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

3.2 Pharmacologic therapy

Pharmacotherapy is an important intervention for obesity-related metabolic abnormalities and endocrine disruption.

3.2.1 Improvement of insulin sensitivity

(1) Metformin

Lowers blood glucose and insulin levels by reducing hepatic glucose production and improving insulin sensitivity in peripheral tissues[38].

(2) Thiazolidinediones (e.g., rosiglitazone, pioglitazone)

Activate PPAR-γ receptors, enhance insulin sensitivity and improve glycemic control[39].

3.2.2 Anti-inflammatory effects

SGLT2 inhibitors (e.g., dagliflozin, empagliflozin) reduce blood glucose by inhibiting renal glucose reabsorption while reducing inflammatory responses[40].

3.3 Testosterone replacement therapy (TRT)

Low testosterone levels are common in obese men, and testosterone deficiency can be treated with testosterone replacement therapy (TRT)[15]. In addition to improving metabolic syndrome, insulin sensitivity, and body fat distribution, testosterone replacement therapy significantly reduces erectile dysfunction, low libido, fatigue, depression, and other quality of life issues[41].

3.4 Surgical interventions

Bariatric surgery, such as gastric bypass, is a successful solution for extremely obese individuals. Significant weight loss, improved endocrine function, and increased libido and sexual satisfaction are all possible outcomes of this surgery[42]. According to a study, testosterone levels in men increased significantly after the procedure and semen quality improved[43]. This effect is directly related to the reduction of visceral fat, which decreases the activity of aromatase, thus reducing the conversion of testosterone to estradiol[44].

3.5 Assisted Reproductive Technology (ART)

IVF and ICSI are examples of assisted reproductive technologies that offer significant fertility prospects for obese male infertile patients. However, since obesity affects sperm quality and embryo development, it may reduce the success rate of assisted reproductive technology (ART)[45]. Therefore, improving the success rate of assisted reproductive technology (ART) requires multidisciplinary collaboration and comprehensive interventions (e.g., medication and lifestyle changes).

4. Conclusion

At this stage, obesity in adults is mainly caused by unhealthy eating habits and lifestyle decisions, and obesity is a major risk factor for male reproductive health. The reproduction of the species is crucial because reproductive function is the basis of its survival. Enhancing the health management of obese men is clinically important because obesity affects male fertility through complex interactions between multiple factors. The primary goal of treating overweight or obese infertile men is to modify their lifestyle (e.g., diet and exercise) to bring their weight down to the normal range. However, for men who are extremely obese, do not respond well to lifestyle changes, or have other difficulties, medical weight loss techniques such as medications or surgery may be considered. Keeping the weight of obese individuals in the normal range is critical to reducing the incidence of obesity-related problems.

The active prevention and treatment of obesity and the maintenance of reproductive health have become key priorities in contemporary society, and the number of obese men is steadily increasing. Studies have shown that chronic inflammation and oxidative stress in the gonadal tissues are important factors in the obese infertile population. In order to prevent and treat male infertility caused by obesity, future research should focus on methods to maintain redox balance and alleviate chronic inflammatory

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

diseases.

Acknowledgement

Ms. Li would like to thank Mentor Li Li and Teacher Dong Xianhui for their careful guidance and the Zhejiang Provincial Department of Science and Technology for funding the project.

This research was funded by Zhejiang Provincial Department of Science and Technology, “Leading Soldier, Leading Goose” Major Program (2022C03138).

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Kivimäki M, Strandberg T, Pentti J, et al. Body-mass index and risk of obesity-related complex multimorbidity: an observational multicohort study[J]. Lancet Diabetes Endocrinol. 2022 Apr; 10(4): 253-263. doi: 10.1016/S2213-8587(22)00033-X.

[2] Yang Na,Chen Wei. Interpretation of the Obesity Diagnosis and Treatment Guidelines (2024 Edition)[J]. Beijing Medicine,2025,47(03):248-252.DOI:10.15932/j.0253-9713.2025.03.001.

[3] Leisegang K, Henkel R, Agarwal A. Obesity and metabolic syndrome associated with systemic inflammation and the impact on the male reproductive system[J]. Am J Reprod Immunol. 2019 Nov; 82(5): e13178. doi: 10.1111/aji.13178.

[4] Xue Yaoyin, Fen Pingzheng, Zhao Jiaqiao, et al. Central obesity and metabolic risk factors in middle-aged Chinese[J]. Biomed Environ Sci. 2014;27(5):343-352. doi:10.3967/bes2014.059.

[5] Pan Xiongfei, Wang Limin, Pan An. Epidemiology and determinants of obesity in China[J]. Lancet Diabetes Endocrinol. 2021 Jun; 9(6): 373-392. doi: 10.1016/S2213-8587(21)00045-0.

[6] Zhang Lina,Pan Qi. Research progress of obesity and related diseases[J]. Chinese Journal of Frontiers of Medicine(Electronic Edition),2025,17(01):86-87.

[7] Meng Yuhao,XIAO Min,Jiang Xiaocui,et al. Potential mechanism of monogenic obesity on semen quality[J/OL]. Liaoning Journal of Chinese Medicine, 1-9[2025-05-25] .http: //kns. cnki. net/kcms/detail/21. 1128.R.20250214.1315.090.html.

[8] Zhang Qian, Du Chen, Sun Qi, et al. Analysis of correlation between semen quality and sex hormone level in 1310 men[J]. Journal of Inner Mongolia Medical University, 2024, 46(02): 180-183+187. DOI: 10.16343/j.cnki.issn.2095-512x.2024.02.023.

[9] Wang Shengyu, Xie Yichang, Ma Jing, et al. Correlation analysis of semen quality and reproductive hormones with adipokines in infertile men[J]. Chinese Journal of Family Planning, 2024, 32(07): 1613-1617.

[10] Chen Liang, Li Guodong, Li Zhijiang, et al. Comparison of testicular texture, sperm quality and reproductive hormone levels in men with different body mass indexes preparing for second or third pregnancy[J]. Journal of Reproductive Medicine, 2024, 33(09):1242-1245.

[11] Zhang X, Wu S, Qi X, et al. Effect of Paternal Body Mass Index on In Vitro Fertilization and Neonatal Outcomes among Oligozoospermia and Asthenospermia Patients[J]. World J Mens Health. 2024;42(1):216-228. doi:10.5534/wjmh.220286.

[12] Sahu A, Pajai S. The Impact of Obesity on Reproductive Health and Pregnancy Outcomes[J]. Cureus. 2023;15(11):e48882. doi:10.7759/cureus.48882.

[13] Pearce KL, Hill A, Tremellen KP. Obesity related metabolic endotoxemia is associated with oxidative stress and impaired sperm DNA integrity[J]. Basic Clin Androl. 2019; 29:6. doi:10. 1186/s12610-019-0087-5.

[14] Sun Wenlu, Xu Qingyu, Yu Long, et al. Research progress on the relationship between obesity and testosterone in men[J]. Heilongjiang Medicine, 2023, 47(03):371-373+379.

[15] Kelly DM, Jones TH. Testosterone and obesity[J]. Obes Rev. 2015;16(7):581-606. doi:10.1111/obr. 12282.

[16] Xu Xiaoling, Sun Minqi, Ye Jifeng, et al. The Effect of Aromatase on the Reproductive Function of Obese Males[J]. Horm Metab Res. 2017; 49(8): 572-579. doi: 10.1055/s-0043-107835

[17] Morrison CD, Brannigan RE. Metabolic syndrome and infertility in men[J]. Best Pract Res Clin Obstet Gynaecol. 2015;29(4):507-515. doi:10.1016/j.bpobgyn.2014.10.006.

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

[18] Pini T, Parks J, Russ J, et al. Obesity significantly alters the human sperm proteome, with potential implications for fertility[J]. J Assist Reprod Genet. 2020;37(4):777-787. doi:10. 1007/s10815-020-01707-8.

[19] Püschel GP, Klauder J, Henkel J. Macrophages, Low-Grade Inflammation, Insulin Resistance and Hyperinsulinemia: A Mutual Ambiguous Relationship in the Development of Metabolic Diseases[J]. J Clin Med. 2022;11(15):4358. doi:10.3390/jcm11154358.

[20] Service CA, Puri D, Al Azzawi S, et al.The impact of obesity and metabolic health on male fertility: a systematic review[J]. Fertil Steril. 2023;120(6):1098-1111. doi:10.1016/j. fertnstert. 2023. 10.017.

[21] Belan M, Carranza-Mamane B, Pesant MH, et al. Male partners of subfertile couples in which the spouse is obese display adverse weight and lifestyle associated with reduced sperm quality[J]. Obes Res Clin Pract. 2019;13(3):226-232. doi:10.1016/j.orcp.2019.03.001.

[22] Barati E, Nikzad H, Karimian M. Oxidative stress and male infertility: current knowledge of pathophysiology and role of antioxidant therapy in disease management[J]. Cell Mol Life Sci. 2020; 77(1):93-113. doi:10.1007/s00018-019-03253-8.

[23] Chesnokova V, Melmed S. Peptide Hormone Regulation of DNA Damage Responses[J]. Endocr Rev. 2020;41(4):bnaa009. doi:10.1210/endrev/bnaa009.

[24] Carrageta DF, Oliveira PF, Alves MG, et al. Obesity and male hypogonadism: Tales of a vicious cycle[J]. Obes Rev. 2019;20(8):1148-1158. doi:10.1111/obr.12863.

[25] Childs GV, Odle AK, MacNicol MC, et al. The Importance of Leptin to Reproduction[J]. Endocrinology. 2021;162(2):bqaa204. doi:10.1210/endocr/bqaa204.

[26] Katib A. Mechanisms linking obesity to male infertility[J]. Cent European J Urol. 2015;68(1):79-85. doi:10.5173/ceju.2015.01.435.

[27] Aversa A, Bruzziches R, Francomano D, et al.Endothelial dysfunction and erectile dysfunction in the aging man[J]. Int J Urol. 2010;17(1):38-47. doi:10.1111/j. 1442-2042. 2009. 02426. x.

[28] Rosselli M, Keller PJ, Dubey RK. Role of nitric oxide in the biology, physiology and pathophysiology of reproduction[J]. Hum Reprod Update. 1998;4(1):3-24. doi:10.1093/humupd/4.1.3.

[29] Ajuogu PK, Al-Aqbi MA, Hart RA, et al. The effect of dietary protein intake on factors associated with male infertility: A systematic literature review and meta-analysis of animal clinical trials in rats[J]. Nutr Health. 2020; 26(1): 53-64. doi: 10. 1177/0260106019900731.

[30] Wang XM, Ma ZY, Song N. Inflammatory cytokines IL-6, IL-10, IL-13, TNF-α and peritoneal fluid flora were associated with infertility in patients with endometriosis[J]. 2018;22(9):2513-2518. doi:10. 26355/eurrev_201805_14899.

[31] Gaskins AJ, Nassan FL, Chiu YH, et al. Dietary patterns and outcomes of assisted reproduction[J]. Am J Obstet Gynecol. 2019;220(6):567.e1-567.e18. doi:10.1016/j.ajog.2019.02.004.

[32] Lăcătușu CM, Grigorescu ED, Floria M, et al. The Mediterranean Diet: From an Environment-Driven Food Culture to an Emerging Medical Prescription[J]. Int J Environ Res Public Health. 2019;16(6):942. doi:10.3390/ijerph16060942.

[33] Mentella MC, Scaldaferri F, Ricci C, et al. Cancer and Mediterranean Diet: A Review[J]. Nutrients. 2019;11(9):2059. Published 2019 Sep 2. doi:10.3390/nu11092059.

[34] Ditano-Vázquez P, Torres-Peña JD, Galeano-Valle F, et al. The Fluid Aspect of the Mediterranean Diet in the Prevention and Management of Cardiovascular Disease and Diabetes: The Role of Polyphenol Content in Moderate Consumption of Wine and Olive Oil[J]. Nutrients. 2019;11(11):2833. doi:10.3390/nu11112833.

[35] Agarwal A, Mulgund A, Alshahrani S, et al. Reactive oxygen species and sperm DNA damage in infertile men presenting with low level leukocytospermia[J]. Reprod Biol Endocrinol. 2014;12:126. doi: 10. 1186/1477-7827-12-126.

[36] Gong Lamei, Sheng Jiazhi. Research progress on the effects of exercise intervention and dietary supplementation on sperm quality in men[J]. Chinese Journal of Male Science, 2021, 35(02): 83-88.

[37] Abedpoor N, Taghian F, Hajibabaie F. Exploring the dynamics of exercise intensity on male fertility and reproductive health: advancements and implications for fertility research[J]. Front Reprod Health. 2024;6:1423916. doi:10.3389/frph.2024.1423916.

[38] Zheng Yu, Deng Zidong, Yu Lijin. Comparison of the efficacy of high-dose and conventional-dose metformin in the adjuvant treatment of obese polycystic ovary syndrome and its effect on patients' endocrine and metabolism[J]. Evaluation and analysis of medication in Chinese hospitals, 2023, 23(04): 427-430.DOI: 10.14009/j.issn.1672-2124.2023.04.009.

[39] Zhang Miao, Guo Yifei, Shi Lixin. Effects of metformin and pioglitazone on lipids and uric acid in patients with primary diagnosis of abnormal glucose metabolism with abdominal obesity[J]. Shandong Medicine, 2012, 52(29):47-48.

[40] Wu XiaoShan, Li Ying. Research progress on the protective effect of sodium-glucose co-transporter protein 2 inhibitor on diabetic nephropathy[J]. Clinical meta-analysis, 2021, 36(11):

ISSN 2616-5791 Vol. 6, Issue 6: 24-31, DOI: 10.25236/AJMHS.2025.060604

1034-1040.
[41] Hackett G, Kirby M, Rees RW, et al. The British Society for Sexual Medicine Guidelines on Male Adult Testosterone Deficiency, with Statements for Practice[J]. World J Mens Health. 2023; 41(3):508-537.doi:10.5534/wjmh.221027.
[42] Wang Hao, Aqbayr A. Li, Maimaiti A. Maimaitimin, et al. Effect of laparoscopic sleeve gastrectomy on sex hormones in male obese patients[J]. China Clinical Research, 2024, 37(07): 1097-1100. DOI: 10.13429/j.cnki.cjcr.2024.07.022.
[43] Ren ZhenJu, Wang Chendi, Li Tiamin, et al. Meta-analysis of the effect of bariatric surgery on erectile function in men[J]. Chinese Journal of Evidence-Based Medicine, 2016, 16 (6) : 676-681.
[44] Lee Y, Dang JT, Switzer N, et al. Impact of Bariatric Surgery on Male Sex Hormones and Sperm Quality: a Systematic Review and Meta-Analysis[J]. Obes Surg. 2019; 29(1): 334-346. doi: 10. 1007/s11695-018-3557-5.
[45] Alibhai K, Churchill I, Vause T, et al. The impact of bariatric surgery on assisted reproductive technology outcomes: a systematic review protocol[J]. Syst Rev. 2022;11(1):1. Published 2022 Jan 3. doi:10.1186/s13643-021-01870-8.