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Impact of Moderate-Intensity Exercise on Reproductive Hormones and Zinc Levels in Obese Men

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ABSTRACT

Background: Due to its significant impact on endocrine function and micronutrient equilibrium, obesity is becoming recognized as a primary contributor to male infertility, which is emerging as a substantial global health issue.

Objective: This study aimed to evaluate the effect of moderate-intensity exercise on serum levels of testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and zinc in obese men, independent of semen analysis parameters.

Methods: A case-control study enrolled 90 obese men (BMI 30–35 kg/m², aged 20–40 years), divided into an exercise group (n=60) and a sedentary control group (n=30). Participants in the exercise group engaged in structured moderate-intensity exercise for three months. Serum levels of testosterone, FSH, LH, and zinc were measured and compared between groups using independent t-tests.

Results: Men in the exercise group had significantly higher serum testosterone (5.19 ± 1.31 ng/mL), FSH (4.77 ± 1.09 U/L), LH (4.12 ± 0.98 U/L), and zinc (86.77 ± 10.17 µg/mL) compared to controls ($p<0.05$ for all parameters). These findings suggest that moderate exercise can effectively improve the hormonal and micronutrient environment essential for male fertility in obese individuals.

Conclusion: Structured moderate-intensity exercise significantly enhances reproductive hormone and zinc levels in obese men, supporting the adoption of lifestyle interventions as a frontline strategy for improving male fertility associated with obesity.

INTRODUCTION

Male fertility is a crucial component of reproductive health, significantly influencing a couple's ability to conceive. Research indicates that male infertility may be associated with an increased risk of eventual health issues, including cardiovascular, metabolic, and oncological illnesses. This suggests that reproductive health may reflect overall male health, influenced by environmental exposures, lifestyle factors, and psychological well-being. Resolving these difficulties is crucial for improving fertility and general health[1].

Male fertility is significantly affected by lifestyle choices; numerous factors contribute to reproductive outcomes. This encompasses coverage, dietary habits, body weight, level of physical activity, and psychological stressors. Moreover, environmental and occupational exposure to pollutants, together with personal behaviors such as tobacco use, illegal drug usage, excessive alcohol intake, and high caffeine consumption, adversely impact hormonal balance, spermatogenesis, and overall male reproductive capacity[2].

Male infertility is a growing global concern, with obesity identified as a major contributor. It disrupts reproductive hormone levels and reduces essential micronutrients like zinc and selenium. These changes impair sperm production and function. Exploring how lifestyle changes, especially exercise, can restore hormonal and micronutrient balance is increasingly important [3].

Elevated oxidative stress can significantly disrupt the intricate equilibrium between reactive oxygen species and the body's antioxidant defense, thereby affecting male fertility. This metabolic imbalance diminishes the normal concentration, progressive motility, viability, and morphology of sperm cells. The capacity for fertilization is thereby diminished, resulting in subfertility or infertility in those affected[4]

The proper function and equilibrium of gonadal hormones in males is crucial for fertility regulation. These hormones are essential, especially concerning spermatogenesis—the complex process of sperm cell development—which commences in the testes. The hypothalamic-pituitary-gonadal axis orchestrates this regulation through a specific hormonal cascade. Gonadotropin-releasing hormone (GnRH), secreted by the hypothalamus, stimulates the anterior pituitary to create luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Luteinizing hormone (LH) stimulates Leydig cells to produce testosterone, whereas follicle-stimulating hormone (FSH) operates on Sertoli cells to facilitate sperm maturation. These hormones are necessary for optimal testicular function and male reproductive health[5]

The preservation of male fertility mostly relies on hormonal equilibrium, as adequate levels of reproductive hormones are crucial for spermatogenesis and testicular function. This fragile hormonal equilibrium can be disrupted by various factors, including unhealthy lifestyle choices, chronic diseases, and environmental exposures. Early identification of such imbalances and the initiation of therapeutic measures rely on frequent medical examinations. Restoring hormonal equilibrium has resulted from lifestyle modifications, including a well-structured, nutrient-dense diet, consistent physical exercise, and either the

elimination of alcohol and tobacco use or a reduction in their usage. Especially in those with obesity or endocrine dysfunction, these non-pharmacological approaches can greatly increase male fertility potential[6]

An imbalance between reactive species and their neutralizing capacity considerably affects male infertility, referred to as oxidative stress. Excessive generation of reactive oxygen species (ROS) adversely affects sperm DNA, lipid membranes, and proteins, resulting in infertility due to compromised sperm activities [7].

Regular participation in moderate-intensity physical activities—such as brisk walking, jogging, swimming, or cycling—has demonstrated improvements in male reproductive parameters. A study featured in a systematic review and network meta-analysis demonstrated that moderate intensity continuous training (MICT) enhances semen quality and elevates live birth rates[8]

MATERIALS AND METHODS

Study Design:

A controlled case-control research, meticulously conducted from December 2024 to March 2025, examined specific health markers in a limited cohort of obese male participants.

Participants:

Ninety obese men aged 20 to 40 years, having a body mass index (BMI) between 30 and 35 kg/m², were recruited for the study. Thirty volunteers served as inactive, non-exercising control subjects, whereas sixty people engaged in a structured, supervised moderate-intensity exercise regimen.

Inclusion Criteria:

- Male, 20–40 years
- BMI 30–35 kg/m²
- No regular physical activity in the last three months (controls) or consistent exercise (exercise group)
- No history of endocrine disorders or medication affecting fertility

Measurements:

Key reproductive hormones—testosterone, follicle-stimulating hormone (FSH), and luteinizing hormone (LH)—were measured using sophisticated immunoassay analyzer (Cobas e411, Roche Diagnostics, Germany), providing accurate and sensitive assessments of endocrine function. To evaluate micronutrient status, zinc concentrations in serum samples were measured via a conventional spectrophotometric method. In consideration of obesity-related dysfunction, these integrated investigations aimed to comprehensively assess hormone and trace element profiles essential for male reproductive health, spermatogenesis, and overall fertility potential.

Statistical Analysis

Independent sample t-tests were employed in the statistical analysis to assess differences between groups. Results were presented as mean ± standard deviation (SD), and a p-value of less than 0.05 was deemed statistically significant in all comparisons made during the study.

RESULTS

Three consecutive months of an organized moderate-intensity exercise regimen led to significantly elevated serum levels of zinc and crucial reproductive hormones in

obese males compared to their sedentary peers. The exercise group had significantly increased mean testosterone levels (5.19 ± 1.31 ng/mL) compared to the control group (4.10 ± 1.05 ng/mL), with a p-value less than 0.001. With p =< 0.001, follicle-stimulating hormone (FSH) levels significantly increased in the exercise group (4.77 ± 1.09 U/L) compared to controls (3.86 ± 0.97 U/L), and luteinizing hormone (LH) levels also significantly increased (4.12 ± 0.98 U/L) with p < 0.001. Zinc levels were significantly elevated in the exercise group (86.77 ± 10.17 µg/mL) compared to the control group (81.60 ± 8.09 µg/mL), with a p-value of 0.017. The findings suggest that, particularly in obese individuals predisposed to endocrine and trace element irregularities, moderate-intensity exercise positively influences the hormonal and micronutrient profiles essential for optimum spermatogenesis and male fertility.

DISCUSSION

This work provides compelling evidence that moderate-intensity exercise markedly enhances the hormonal and micronutrient profiles of obese men, hence presenting an effective non-pharmacological strategy to augment male fertility. Although semen analysis was excluded from this investigation, the observed elevations in serum testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and zinc levels suggest a beneficial alteration in the reproductive endocrine milieu. These alterations may indicate enhanced functionality of the hypothalamic-pituitary-gonadal axis, which is pivotal in regulating male reproductive quality. The findings align with recent research indicating that regular physical activity helps mitigate the adverse hormonal consequences of obesity, such as hypogonadism and deficiencies in trace elements.

Exercise may aid in restoring hormonal equilibrium and facilitating the biochemical conditions necessary for effective spermatogenesis by mitigating these adverse effects. These findings underscore the potential efficacy of structured exercise regimens in addressing obesity-related reproductive issues in men[9,10].

Considering that hypogonadism is prevalent in obese men, increased testosterone levels are particularly important. This disorder is mostly due to increased aromatization of testosterone to estradiol in excess adipose tissue, resulting in high estrogen levels that adversely affect the hypothalamic–pituitary–testicular axis. Decreased gonadotropin secretion, diminished endogenous testosterone production, and impaired spermatogenesis due to this hormonal disruption ultimately contribute to reduced reproductive potential in obese males[11,12]. Consistent exercise reinstates the hormonal milieu essential for optimal testicular function by reducing adipose tissue and enhancing insulin sensitivity. Collectively, these factors contribute to the normalization of testosterone levels and the enhancement of gonadotropin regulation, hence promoting optimal spermatogenesis and overall reproductive function in obese men suffering from endocrine and metabolic disorders[13] .

Elevated concentrations of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) observed in the exercise cohort contribute to the reestablishment of normal pituitary-gonadal axis signaling. LH enhances testosterone production by activating Leydig cells, while FSH directly

stimulates Sertoli cells to facilitate sperm formation; therefore, this hormonal recovery is crucial. Collectively, these systems enhance spermatogenesis and indicate improved endocrine function, which is often deficient in obese men with disrupted hormonal equilibrium and reduced fertility [14–16]. Notably, these alterations in reproductive hormone levels remained within recognized physiological limits, indicating a favorable adaptation rather than an abnormal or harmful hormonal response. This indicates that, in obese males, moderate-intensity exercise improves reproductive health without inducing endocrine disruption or supraphysiological alterations in hormone production, hence fostering natural hormonal equilibrium.

In addition to hormonal alterations, the elevation of serum zinc levels observed in the exercise group is particularly noteworthy. Zinc is an essential trace element that acts as a cofactor in various biological processes, including testosterone synthesis, spermatogenesis, and antioxidant protection in testicular tissue.

Optimal zinc levels are crucial for preserving sperm quality, safeguarding against oxidative stress, and facilitating enzyme activity related to reproductive function, all of which are frequently impaired in obese males. Prior research has unequivocally demonstrated that zinc deficiency, commonly found in obese men, adversely affects male reproductive health by compromising hormone synthesis and sperm quality [17,18].

Zinc is crucial for the proper functioning of the hypothalamic-pituitary-gonadal axis; its absence may result in less testosterone production, impaired spermatogenesis, and heightened oxidative stress. Research indicates that zinc supplementation or increased dietary consumption, along with physical exercise, might effectively mitigate these adverse effects and significantly enhance male reproductive potential and fertility[19,20].

The findings of this study align with existing evidence indicating that physical activity enhances zinc absorption, distribution, and cellular utilization. This improved zinc metabolism facilitates spermatogenesis, hormone production, and superior antioxidant defense, hence increasing reproductive health indicators. Exercise functions as a standard regulator of micronutrient bioavailability and reproductive health [21,22]. This study emphasizes the importance of upstream physiological parameters in male fertility by excluding direct semen analysis data. The observed elevations in testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and zinc levels indicate fundamental enhancements in the reproductive endocrine and micronutrient milieu due to exercise.

The support of spermatogenesis, testicular function, and overall reproductive capacity is contingent upon these components. The findings validate the notion that, even without immediate evaluations of semen parameters or interventions, rectifying hormonal and micronutrient deficiencies through lifestyle modifications—particularly organized physical activity—can serve as an effective, non-pharmacological primary approach for addressing obesity-related subfertility in men [23,24] .

These findings align with other scientific research indicating that comprehensive lifestyle changes—particularly a balanced diet and regular physical activity—can effectively restore metabolic and

reproductive health in males. Timely intervention via non-pharmacological methods has demonstrated efficacy in enhancing testicular function, rectifying hormonal imbalances, and modifying micronutrient profiles. Importantly, these benefits can be realized prior to the onset of permanent harm to spermatogenesis or endocrine function, underscoring the necessity for proactive health care in obese males with fertility issues [24,25].

The cumulative findings of this study indicate that moderate-intensity exercise is a safe, readily accessible, and effective strategy for improving critical hormonal and nutritional factors associated with male fertility.

The significant elevations in serum testosterone, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and zinc levels indicate a coordinated enhancement of the hypothalamic-pituitary-gonadal axis and trace element status. These alterations enhance spermatogenesis and optimize testicular function. For obese men seeking to enhance their reproductive health and fertility, this research provides compelling physiological rationale for recommending structured, moderate-intensity exercise as a primary lifestyle intervention [9,10,19].

Future research may elucidate the long-term effects of exercise and establish its comparative significance among other lifestyle factors such as nutrition, sleep, and stress management. Nevertheless, particularly concerning obesity, the current findings provide compelling and pertinent evidence endorsing exercise as a fundamental element in the treatment of male infertility. Enhancing hormonal equilibrium and micronutrient levels identifies moderate-intensity exercise as an effective, low-risk, and readily accessible intervention suitable for males with obesity-related reproductive challenges within fertility treatment protocols.

CONCLUSION

Structured moderate-intensity exercise has been shown to raise levels of important reproductive hormones like testosterone, follicle-stimulating hormone, and luteinizing hormone, as well as zinc levels in obese men. The enhancements in hormone and micronutrient levels are crucial as they directly contribute to improved sperm production and overall fertility. The findings robustly endorse lifestyle modifications, particularly consistent moderate exercise, as a primary non-pharmacological approach to enhance male fertility compromised by obesity. These modifications additionally confer several health and metabolic advantages.

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Authors' Declaration

Conflict of Interest: The authors declare that there is no conflict of interest regarding the publication of this manuscript.

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Ethical Clearance: The project was approved by the local ethical committee at University of Tikrit - College of Medicine, Iraq.

Clinical trial number: not applicable

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TABLES

Table 1: Summary statistics for T, FSH, LH, and Zinc and were calculated for each group.

variable	Groups	N	Mean	SD	p value
Testosterone ng/ml	control	30	4.10	±1.05	<0.05
	exercised	60	5.19	±1.31	
FSH u/l	control	30	3.86	± 0.97	<0.05
	exercised	60	4.77	± 1.09	
LH U/L	control	30	3.16	± 0.80	<0.05
	exercised	60	4.12	± 0.80	
Zinc µg/dL	control	30	81.60	± 8.09	<0.05
	exercised	60	86.77	± 10.17	

FIGURES

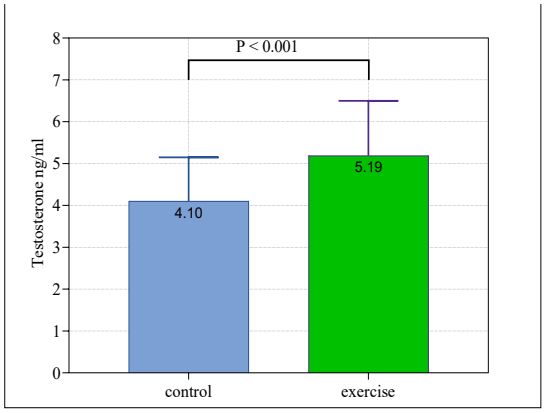

Figure 1: Comparison of Mean Testosterone by Group.