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The Relationship Between Body Mass Index (BMI) and Reproductive Hormone Levels and Sperm Parameters in Relatively Healthy Individuals

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<https://dx.doi.org/10.4314/sokjmls.v10i1.29>**Abstract**

Infertility affects approximately 13% to 15% of couples worldwide, with 1 in 5 couples unable to achieve pregnancy within the first year of trying. Obesity is recognized as a significant modifier of reproductive hormones, with evidence suggesting its role in declining sperm parameters, given the rising prevalence of obesity globally. This study aimed to evaluate the relationship between body mass index (BMI), sperm parameters, and reproductive hormone levels in relatively healthy individuals. Participants who met the inclusion criteria were categorized into four groups based on fertility status (normozoospermic fertile men and infertile patients) and BMI classification: normal weight (BMI: 18.5–24.9 kg/m²), overweight (BMI: 25–29.9 kg/m²), and obese (BMI 30 kg/m²). Semen parameters, reproductive hormone levels, and thyroid-stimulating hormone (TSH) levels were retrospectively compared across groups. There were no significant differences in age or height between the groups. However, sperm count, semen volume, and BMI varied significantly across the groups. The BMI values for non-obese infertile, obese infertile, non-obese fertile, and obese infertile men were 42.3 ± 5.7 , 89.4 ± 12.7 , 51.0 ± 9.8 , and 86.9 ± 10.9 , respectively ($p < 0.001$). Significant differences were also observed between groups in serum levels of follicle-stimulating hormone, luteinizing hormone, TSH, prolactin, semen volume, sperm concentration, and total sperm count ($p < 0.001$). These findings suggest that male obesity may influence semen characteristics and alter reproductive hormone levels.

Keywords: Body mass index, Reproductive hormones, Male infertility, Thyroid-stimulating hormone (TSH)

Introduction

Infertility affects approximately 13% to 15% of all couples globally, while 1 in 5 couples is unable to achieve pregnancy in the first year of trying (Esteves *et al.*, 2021). In 20% to 30% of infertility cases, males are solely responsible, with an overall contribution to infertility in couples of about 30% to 40% (Leslie *et al.*, 2025). Infertility is generally defined as the inability of a couple to conceive even after one year of unprotected, frequent sexual intercourse (Schlegel *et al.*, 2021). Male infertility, specifically, is defined by the World Health Organization (WHO) as the inability of a male to make a fertile female pregnant after at least one year of regular unprotected intercourse. The causes of male infertility can be broadly classified based on their general underlying etiology. These include; Endocrine disorders, usually due to hypogonadism, accounting for 2% to 5% of cases. Sperm transport disorders (e.g., vasectomy), responsible for 5%. Primary testicular defects, which include abnormal sperm parameters without any identifiable cause, comprising 65% to 80%. Idiopathic infertility, where an infertile male has normal sperm and semen parameters, representing 10% to 20% of cases (Kaltsas *et al.*, 2023).

A body mass index (BMI) over 25 is considered overweight, while a BMI over 30 is classified as obese. Obesity, defined as abnormal or excessive

fat accumulation that poses a risk to health, has become a significant global health issue. In 2019, an estimated 5 million noncommunicable disease (NCD) deaths were attributed to higher-than-optimal BMI (Okunogbe *et al.*, 2022). Obesity is an important modifier of reproductive hormones. Evidence for its role in the decline of sperm parameters is underscored by the exponential rise in obesity prevalence in the general population (Darand *et al.*, 2023).

Thyroid dysfunction is another factor that may impact male fertility. Thyroid hormones exert their effects through nuclear receptors expressed within the testis (Mazzilli *et al.*, 2023). They influence Sertoli cells, Leydig cells, and spermatogenesis by regulating gene transcription, protein synthesis, and cellular proliferation and differentiation (Cannerella *et al.*, 2024).

The relationship between Body Mass Index (BMI) and semen quality, along with reproductive hormone concentrations, remains inadequately elucidated. The objective of this investigation was to assess the relationship between BMI and sperm parameters, as well as reproductive hormone levels, in individuals devoid of identifiable risk factors for infertility.

Material and Methods

Study Design

This retrospective observational study assessed the relationship between body mass index (BMI), reproductive hormone levels, and sperm parameters in relatively healthy individuals. Patients who met the study's inclusion criteria were divided into four groups based on their fertility status (normozoospermic fertile men and infertile patients) and BMI values: normal weight (BMI: 18.5–24.9 kg/m²), overweight (BMI 25–29.9 kg/m²), and obese (BMI ≥ 30 kg/m²). Semen parameters and TSH levels were evaluated, and BMI was calculated as weight in kilograms divided by height in meters squared (kg/m²).

Two semen samples were collected per participant, with a one-week interval, following a minimum abstinence period of three days. Blood samples (5 mL) were also drawn between 8:00–10:00 a.m. for hormone analysis.

Study Area

The study was conducted at Rivers State University Teaching Hospital (RSUTH), located in Port Harcourt, Rivers State, Nigeria, where relevant patient records and samples were retrospectively analyzed.

Inclusion Criteria

Inclusion criteria encompassed men without identifiable infertility risk factors, aged 18 years and above, who provided informed consent.

Exclusion Criteria

Individuals undergoing assisted reproductive techniques, those with genital tract infections, severe chronic illnesses (e.g., cerebrovascular, cardiovascular diseases, diabetes), HIV, or those using specific medications (steroids, antihyperlipidaemics) were excluded.

Ethical Consideration

The study adhered to ethical guidelines, with approval from RSUTH's ethics committee. Data confidentiality was maintained, and informed consent was obtained where necessary for the retrospective use of data.

Sample Analysis

Two separate semen samples were collected and analyzed, separated by one week, after at least three days of abstinence, following Schlegel *et al.* (2021) guidelines. Blood samples (5 mL) were obtained between 8:00–10:00 am and the centrifuged sera were preserved at -4°C. Serum FSH, LH, PRL, testosterone (T), and estradiol (E2) levels were estimated using the ELISA method. Vitamin D3 concentration levels were estimated using the radioimmunoassay (RIA) method.

Statistical Analysis

Data were analyzed using SPSS version 22.0. Descriptive statistics (mean ± standard deviation) were calculated, and one-way analysis of variance (ANOVA) with Tukey's Post Hoc test was applied. A probability (p) value of less than 0.05 was considered statistically significant.

Analysis of Data

The SPSS version 22.0 statistical package was used to analyze the generated data. The mean ± standard deviation was determined. One-way

ANOVA with Tukey's Post Hoc test was performed for statistical decision-making and inferential evaluation. A p-value of less than 0.05 was considered statistically significant.

Results

The data for the groups studied are presented in Table 1. The mean sperm count, pH of the semen specimen, and volume of the specimen showed significant differences across the groups. In

infertile men, the sperm count was significantly lower than in fertile men. Table 2 highlights significant differences in TSH, PRL, LH, and serum T levels across the groups, while there was no significant difference in vitamin D3 levels. Table 3 demonstrates that BMI had a significant positive correlation with PRL and vitamin D3, and a significant negative correlation with FSH, LH, T, and vitamin D3.

Table 1: Seminal Parameters of Study participants

Variables	Group 1 (n = 30) Non-obese infertile	Group 2 (n = 30) Obese Infertile	Group 3 (n = 30) Non-obese fertile	Group 4 (n = 30) Obese fertile	P-value
Abstinence (Day)	5 ± 1	4 ± 2	6 ± 1	5 ± 2	0.240
Ph	7.9 ± 0.2	7.8 ± 0.3	8.4 ± 0.4	8.2 ± 0.3	0.702
Semen Volume (ml)	1.8 ± 0.5	1.2 ± 0.2	2.9 ± 0.5	2.5 ± 0.3	0.045
Sperm Count (Million/ml)	10.0 ± 2.5	8.6 ± 1.5	79.3 ± 10.4	55 ± 11.5	0.001

Table 2: Hormonal Parameters of Study Participants

Variables	Group 1 (n = 30) Non-obese infertile	Group 2 (n = 30) Obese Infertile	Group 3 (n = 30) Non-obese fertile	Group 4 (n = 30) Obese fertile	P-value
TSH (miu/ml)	1.9 ± 0.8	3.2 ± 0.8	3.1 ± 0.8	2.9 ± 0.7	0.043
FSH (miu/ml)	9.9 ± 3.3	5.3 ± 3.2	12.2 ± 0.7	32.2 ± 3.2	0.033
LH (miu/ml)	6.4 ± 1.7	4.0 ± 0.5	9.2 ± 0.3	5.1 ± 2.2	0.017
PRL (miu/ml)	10.7 ± 1.0	18.8 ± 1.3	11.9 ± 1.4	16.7 ± 1.2	0.009
Testosterone (miu/ml)	756.2 ± 43.9	433.1 ± 41.4	618.2 ± 44.2	519.6 ± 23.9	0.013
Vitamin D ₃ (miu/ml)	29.9 ± 8.9	12.2 ± 5.2	28.5 ± 3.1	19.1 ± 2.0	0.427

Table 3: Correlation Between BMI and Hormones of Participants.

Variables	R	P-value
TSH	0.059	0.002
FSH	-0.070	0.034
LH	-0.203	0.068
PRL	0.120	0.038
Testosterone	-0.336	0.002
Vitamin D ₃	-0.168	0.013

Key: TSH- Thyroid Stimulating Hormone, FSH- Follicle Stimulating Hormone, LH- Luteinizing Hormone, PRL- Prolactin.

Discussion

The present study showed that the mean BMI was inversely correlated with sperm count, sperm volume, and sperm normal morphology. Cannarella *et al.* (2024) reported that excess weight has been related directly or indirectly to biologic changes that could reduce male fertility. Chaudhuri *et al.* (2022) showed that overweight and obese men had reduced sperm motility and increased sperm DNA fragmentation. Chaudhuri *et al.* (2022) also reported a negative correlation between BMI and total number of normal spermatozoa. Loukil *et al.* (2014), demonstrated decreased sperm count in obese normozoospermic men compared with non-obese fertile subjects. Chaudhuri *et al.* (2022) reported higher prevalence of oligozoospermia in overweight and obese men than in normal weight men with a significant association between sperm count and BMI.

In addition, obesity was demonstrated to be associated with a significant disturbance in the hormonal milieu, which can affect the reproductive system. In the present study, the BMI was positively correlated with serum PRL and TSH levels and negatively correlation with T, FSH, LH and Vitamin D₃. Several reports showed that the accumulation of fatty tissue in men was associated with decreased total, free T, and increased E₂ serum levels. On the other hand, Chaudhuri *et al.* (2022) reported that BMI had no effect on serum LH or FSH.

Thyroid dysfunction is frequently associated with female infertility. Although the present study shows no significant difference with TSH and the male reproductive hormones. A study conducted

by La Vignera and Vita (2018), suggested that a significantly high level of TSH is also associated with the reduction of sperm concentration, motility and impairment of sperm morphology, as well as a reduction of semen volume. Mazzilli *et al.* (2022) reported that high levels of thyroid hormones can cause sperm DNA damage and infertility. Indeed, high levels of thyroid hormones promote an increase in reactive oxygen species (ROS) and consequently oxidative stress. Hypothyroidism is related to obesity as an increase in body weight gain is seen in hypothyroid patients. Moreover, an inverse correlation between free thyroxine values and body mass index has been reported (Meligi *et al.*, 2024).

Conclusion

It is determined in this study that male obesity may have an influence on semen characteristics and may also alter male reproductive hormone levels. Additional investigations aimed at exploring the effects of obesity on male infertility are warranted to clarify the intricate relationship between these variables.

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