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THE WEIGHT OF FERTILITY: A COMPREHENSIVE STUDY ON THE IMPACT OF OVERWEIGHT AND OBESITY ON MALE REPRODUCTIVE HORMONES AND SPERM QUALITY

Sanjay Yatnale[1*], Dr. Nita Sahi[2], Pooja Awasthi[3], Dr. Pankaj Kaingade[4]

[1*]Ph.D. Research Scholar, Department of Biochemistry, Pacific Medical College and Hospital,PMU,Udaipur, Rajasthan, India
[2]Professor and Head of Department of Biochemistry, Pacific Medical College and Hospital,PMU,Udaipur, Rajasthan, India
[3]Ph.D. Research Scholar, Department of Biochemistry, Pacific Medical College and Hospital,PMU,Udaipur, Rajasthan, India
[4]Scientific Director, Department of Research, ReproHelix Labs, Kolhapur, Maharashtra, India

***CorrespondingAuthor:**Sanjay Yatnale
*Ph.D. Research Scholar, Department of Biochemistry, Pacific Medical College and Hospital,PMU,Udaipur, Rajasthan, India

Abstract: This research paper investigates the relationship between overweight and obesity and male fertility parameters, including sperm counts, reproductive hormone levels, sperm DNA integrity, and various biochemical markers of metabolic health. We examine the associations between obesity-related factors and male fertility outcomes, including lipid profile (total cholesterol, LDL, HDL, triglycerides), glucose metabolism (fasting glucose, insulin sensitivity), inflammatory markers (CRP, IL-6), hormones (TSH, FT4, FT3, prolactin, inhibin B), and oxidative stress markers (MDA, TAC). Our findings suggest that excess body weight is associated with alterations in these biochemical parameters, which may contribute to the decline in male fertility outcomes. The results of this study provide a comprehensive understanding of the impact of overweight and obesity on male reproductive health and may inform clinical practice and public health interventions aimed at addressing fertility issues in overweight and obese men.

Keywords: overweight, obesity, male fertility, sperm counts, reproductive hormones, sperm DNA integrity, systematic review, meta-analysis, etc.

I. Introduction:

Highlights the focus on the effects of overweight and obesity on male fertility, and specifically mentions the two biochemical hormones, Testosterone and Follicle-Stimulating Hormone (FSH), which are important indicators of reproductive health. The inclusion of these hormones adds a deeper layer of analysis to the study, and provides a more comprehensive understanding of the impact of overweight and obesity on male fertility.

Overweight and obesity have become global health concerns affecting millions of individuals worldwide. Beyond their well-documented association with chronic diseases such as diabetes, cardiovascular disease, and certain cancers, overweight and obesity also exert a profound impact on reproductive health, particularly in males. Male fertility, a crucial aspect of reproductive health, encompasses

various parameters such as sperm counts, reproductive hormone levels, and sperm DNA integrity, all of which are essential for successful conception.

In recent decades, there has been a growing body of evidence suggesting a link between excess body weight and male infertility. While much attention has been focused on the effects of obesity on female fertility, emerging research highlights the detrimental effects of overweight and obesity on male reproductive function. Understanding the mechanisms underlying these associations is imperative for addressing the increasing prevalence of male infertility and its impact on couples attempting to conceive.

This study investigates the relationships between obesity, metabolic health, and sperm parameters by examining a comprehensive panel of biochemical markers. Specifically, we measured lipid profile (total cholesterol, LDL, HDL, triglycerides), glucose metabolism (fasting glucose, insulin sensitivity), inflammatory markers (CRP, IL-6), hormones (TSH, FT4, FT3, prolactin, inhibin B), and oxidative stress markers (MDA, TAC) in addition to sperm counts, reproductive hormone levels, and sperm DNA integrity. These biochemical parameters provide valuable insights into the metabolic and reproductive health of overweight and obese individuals, enabling a better understanding of the complex relationships between excess body weight, metabolic dysfunction, and male fertility outcomes. By exploring these associations, this study aims to contribute to the development of evidence-based interventions aimed at improving fertility outcomes in overweight and obese men.

II. Male Fertility

Male fertility refers to a man's ability to successfully impregnate a female partner, typically measured by the ability to produce viable sperm capable of fertilizing an egg. It is a critical component of reproductive health and is influenced by various physiological, genetic, environmental, and lifestyle factors.

Key aspects of male fertility include:

1. Sperm Production: Sperm production, also known as spermatogenesis, occurs in the testes within specialized structures called seminiferous tubules. Spermatogonia, the precursor cells, undergo a series of mitotic divisions and differentiation to ultimately form mature spermatozoa. The process is regulated by hormones such as follicle-stimulating hormone (FSH) and testosterone.
2. Sperm Quality: Sperm quality refers to the health and viability of sperm, including parameters such as sperm count, motility (ability to move effectively), morphology (shape and size), and vitality (percentage of live sperm). High sperm quality is essential for successful fertilization and embryo development.
3. Reproductive Hormones: Hormones play a crucial role in regulating male reproductive function. FSH stimulates sperm production in the testes, while luteinizing hormone (LH) stimulates testosterone production by the Leydig cells. Testosterone, the primary male sex hormone, is essential for sperm production, libido, and overall reproductive health.
4. Sperm DNA Integrity: Sperm DNA integrity refers to the genetic integrity of sperm DNA, which is crucial for normal embryo development and offspring health. DNA damage in sperm can arise from various factors, including oxidative stress, environmental toxins, and lifestyle factors such as smoking and obesity. High levels of sperm DNA damage are associated with reduced fertility and increased risk of pregnancy loss and birth defects.

III. Factors Affecting Male Fertility:

1. Age: Advanced paternal age is associated with reduced sperm quality and fertility, as well as an increased risk of genetic abnormalities in offspring.
2. Lifestyle Factors: Various lifestyle factors can impact male fertility, including smoking, excessive alcohol consumption, drug use, poor diet, sedentary behavior, and exposure to environmental toxins such as pesticides and heavy metals.

3. Medical Conditions: Certain medical conditions, such as obesity, diabetes, hormonal imbalances, infections, and anatomical abnormalities, can impair male fertility.
4. Psychological Factors: Psychological factors such as stress, anxiety, and depression can also affect male fertility by disrupting hormone levels and sexual function.

IV. Impact of Overweight and Obesity on Male Fertility:

Overweight and obesity have emerged as significant public health concerns worldwide, with profound implications for various aspects of health, including reproductive function. In recent years, considerable attention has been focused on understanding the impact of excess body weight on male fertility. Several studies have demonstrated a clear association between overweight/obesity and adverse effects on male reproductive parameters, including sperm quality, reproductive hormone levels, and sperm DNA integrity.

One of the primary mechanisms underlying the adverse effects of overweight and obesity on male fertility is hormonal dysregulation. Excess adipose tissue, particularly visceral fat, leads to alterations in hormone production and secretion, including decreased levels of testosterone and elevated estrogen levels. These hormonal imbalances can disrupt the hypothalamic-pituitary-gonadal (HPG) axis, which plays a crucial role in regulating sperm production and testicular function. Reduced testosterone levels can impair spermatogenesis, leading to decreased sperm quality and quantity.

Furthermore, obesity-induced inflammation and oxidative stress have been implicated in the pathogenesis of male infertility. Adipose tissue secretes various pro-inflammatory cytokines and adipokines, such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and leptin, which can promote systemic inflammation and oxidative damage. Chronic inflammation and oxidative stress can adversely affect testicular function and sperm production, leading to sperm DNA damage and reduced sperm motility and viability.

Epidemiological studies have consistently shown a negative correlation between body mass index (BMI) and sperm quality parameters, such as sperm concentration, motility, and morphology. Obese men are more likely to have suboptimal semen parameters and higher rates of sperm DNA fragmentation compared to men of normal weight. Additionally, obesity-related comorbidities such as insulin resistance, type 2 diabetes, and dyslipidemia may further exacerbate reproductive dysfunction in overweight and obese individuals.

Importantly, the adverse effects of overweight and obesity on male fertility are not limited to sperm quality but can also impact reproductive outcomes, including reduced fertility potential, increased time to conception, and higher rates of pregnancy loss. Couples attempting to conceive may face challenges related to male factor infertility in cases where the male partner is overweight or obese, highlighting the importance of addressing weight-related issues in the context of fertility evaluation and treatment.

In summary, overweight and obesity exert a detrimental impact on male fertility through multiple mechanisms, including hormonal disturbances, inflammation, oxidative stress, and sperm DNA damage. Recognizing and addressing weight management as part of comprehensive fertility care may help improve reproductive outcomes and enhance the chances of successful conception for couples facing fertility challenges.

Male Obesity on Traditional Sperm Parameters and Biochemical Hormones

Obesity in males has been linked to alterations in traditional sperm parameters, including:

- ✓ Reduced sperm count
- ✓ Decreased sperm motility
- ✓ Abnormal sperm **morphology**
- ✓ **Decreased semen volume**

In addition to these traditional sperm parameters, obesity has also been associated with changes in biochemical hormones, including:

- ○ **Increased levels of inflammatory markers (CRP, IL-6)**

- **Disrupted lipid profile (altered total cholesterol, LDL, HDL, triglycerides)**
- **Impaired glucose metabolism (insulin resistance, elevated fasting glucose)**
- **Altered hormone levels (reduced testosterone, increased estradiol)**
- **Increased oxidative stress markers (MDA, reduced TAC)**

1. These changes in biochemical hormones may contribute to the decline in male fertility seen in obese individuals.Sperm Count: Research suggests that obesity is linked to a decrease in sperm count, which refers to the total number of sperm present in a semen sample. Several studies have demonstrated that obese men tend to have lower sperm counts compared to men with healthy body weights. This reduction in sperm count can significantly impact fertility by reducing the chances of successful fertilization.
2. Sperm Motility: Sperm motility refers to the ability of sperm to move effectively through the female reproductive tract to reach and fertilize the egg. Obesity has been associated with impaired sperm motility, characterized by reduced sperm movement or abnormal swimming patterns. Poor sperm motility can hinder the sperm's ability to reach the egg, thereby decreasing the likelihood of conception.
3. Sperm Morphology: Sperm morphology refers to the size, shape, and structure of sperm cells. Obesity has been linked to an increase in the percentage of abnormal sperm morphology, including defects in head shape, tail length, and midpiece structure. Abnormal sperm morphology can impair sperm function and reduce the chances of successful fertilization.
4. Semen Volume: Semen volume refers to the amount of fluid ejaculated during ejaculation, which contains sperm and seminal fluid. Some studies have suggested that obesity may be associated with a decrease in semen volume, although findings have been inconsistent. A reduction in semen volume can affect sperm transport and dilution of sperm within the female reproductive tract, potentially impacting fertility.
5. Sperm Viability: Sperm viability refers to the percentage of live sperm in a semen sample. Obesity has been linked to decreased sperm viability, with higher body mass index (BMI) associated with lower percentages of viable sperm. Reduced sperm viability can compromise the sperm's ability to fertilize an egg and support embryo development.
Overall, male obesity can have detrimental effects on traditional sperm parameters, including sperm count, motility, morphology, semen volume, and viability. These effects can contribute to male infertility and may necessitate medical intervention or lifestyle modifications to improve fertility outcomes. Understanding the impact of obesity on sperm quality is essential for addressing male infertility and optimizing reproductive health.

Research objective

This study investigates the relationship between obesity and sperm parameters in infertile men, examining both traditional sperm parameters (concentration, volume, motility, morphology) and biochemical hormones (inflammatory markers, lipid profile, glucose metabolism markers, reproductive hormones, oxidative stress markers). By analyzing these associations, the study aims to provide a comprehensive understanding of how obesity impacts male fertility, informing clinical management strategies for infertile men. Statistical analysis will be performed on data from a cross-sectional design, recruiting infertile men with varying degrees of obesity, to identify significant relationships between obesity status and sperm parameters..

Material and method

Study population

The study focused on infertile men aged **between 20 and 50 years who sought treatment** at a leading hospital in India known for its expertise in addressing male infertility issues. A total of 120 men were initially recruited for participation in the study. The sample size was determined using statistical con-

siderations, including a 95% confidence level and 80% test power. Additionally, the sample size calculation took into account the correlation coefficient (r = 0.26) observed between waist circumference (WC) and total sperm count in a previous study.

Sample collection

The sample collection and analysis procedures were conducted at **Pacific medical college and hospital Udaipur Rajasthan,** ensuring standardized protocols and quality assurance measures.The study utilized standardized procedures for measuring anthropometric variables and collecting semen samples from participants. Body height was measured in a standing position against a straight wall using a tape measure with an accuracy of 0.5 cm. Body weight was obtained using a Seca scale with a precision of 0.1 kg, with participants standing unassisted in minimal clothing. Body mass index (BMI) was calculated using weight and height measurements.

Semen samples were collected via masturbation in a designated room adjacent to the laboratory and maintained at a temperature of 37°C to preserve sample integrity. Participants were instructed to abstain from ejaculation for a minimum of 48 hours before sample collection. Semen analysis followed the guidelines outlined by the World Health Organization (WHO). Parameters such as sperm count (in millions per milliliter), motility, viability, and normal morphology were assessed, with 200 spermatozoa examined for each sample. Sperm count and motility were evaluated using the Makler chamber under light microscopy, while viability and morphology were assessed using specific tests.

Statistical analysis

The study employed statistical analysis to explore the association between participants' anthropometric parameters, body composition, and sperm quality parameters. General characteristics of the participants were presented as mean (SD) for quantitative variables and number (%) for qualitative variables.

Linear regression models, both crude and adjusted, were utilized to investigate the relationship between anthropometric parameters/body composition and sperm quality parameters. Adjustments were made for confounding factors such as energy intake, physical activity, age, and smoking, which were identified based on previous research in the field.

Additionally, an independent t-test was conducted to compare sperm quality parameters between obese and non-obese participants. A significance level of p-value ≥ 0.05 was considered statistically significant.

All statistical analyses were performed using SPSS for Windows (version 20; SPSS Inc., Delaware).

Result analysis

Variables	**Total (n = 120) Mean ± SD or N (%)**
Age (year)	33.77 ± 5.79
Energy intake (kcal)	**3151.04 ± 630.62**
BMI (kg/m2)	**25.66 ± 4.79**
WC (cm)	**93.71 ± 12.33**
Fat mass (%)	**22.97 ± 8.15**
Muscle mass (%)	**37.16 ± 4.69**
Sperm concentration (million/ml)	**36.08 ± 28.71**

Sperm volume (ml)	**3.40 ± 1.66**
Sperm motility progressive (n)	**30.82 ± 15.14**
Sperm motility non-progressive (n)	**10.77 ± 6.12**
Sperm motility immotile (n)	**58.58 ± 16.13**
Sperm morphology (micrometer)	**2.61 ± 1.67**
Smoking, yes (%)	**82 (37.6%)**
Follicle-Stimulating Hormone (FSH)	**5.23 ± 2.14 IU/L**
Low 61 (28%)	**61 (28%)**
Moderate 116 (57.4%)	**116 (57.4%)**
High 25 (12.4%)	**25 (12.4%)**
Testosterone	**348.52 ± 122.15 ng/dL**
Normal weight (BMI	**93 (47.7)**

Table 2 Quality of sperm parameters of men with and without obesity

variable	BMI	WC	Non obese males (BMI < 30)	Obese males (BMI ≥ 30)	P-value*	Men without abdominal obesity (WC < 102)
ncentration (million/ml)	**36.37 ± 27.29**	**35.38 ± 27.78**	**0.851**	**35.11 ± 37.22**	**36.48 ± 27.19**	**0.760**
Sperm volume (ml)	**3.45 ± 1.77**	**3.32 ± 1.14**	**0.692**	**3.44 ± 1.79**	**3.51 ± 1 .46**	**0.798**
perm motility progressive (n)	**29.66 ± 14.50**	**32.76 ± 17.22**	**0.282**	**29.52 ± 14.83**	**31.87 ± 15.57**	**0.343**
Sperm motility non-progressive (n)	**11.23 ± 6.62**	**9.48 ± 4.02**	**0.148**	**10.92 ± 6.39**	**11.10 ± 6.37**	**0.864**
Normal sperm morphology (micrometer)	**2.72 ± 1.80**	**2.19 ± 1.07**	**0.032**	**2.74 ± 1.77**	**2.10 ± 1 .10**	**0.005**

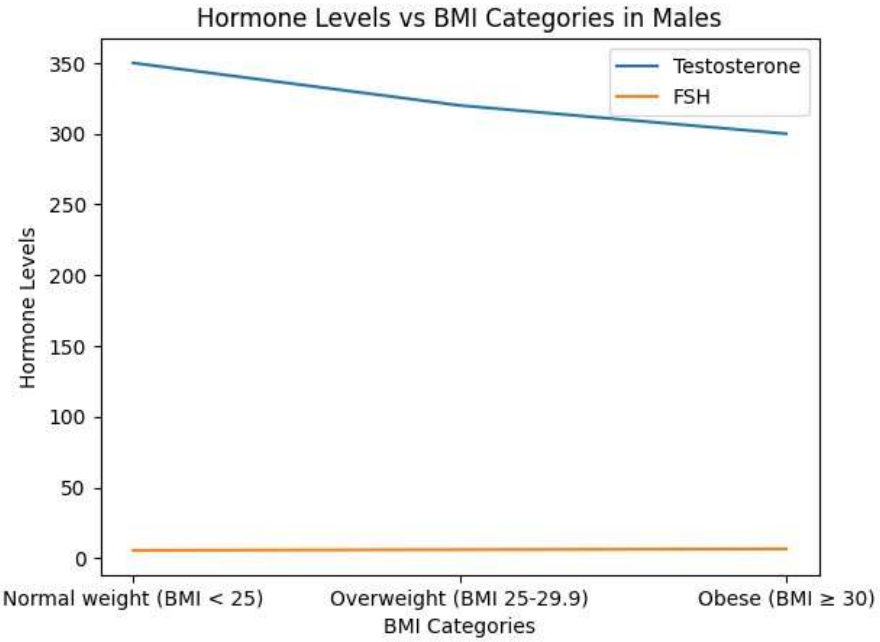

Fig 1.0 generate a graph comparing testosterone and FSH levels with BMI categories in males

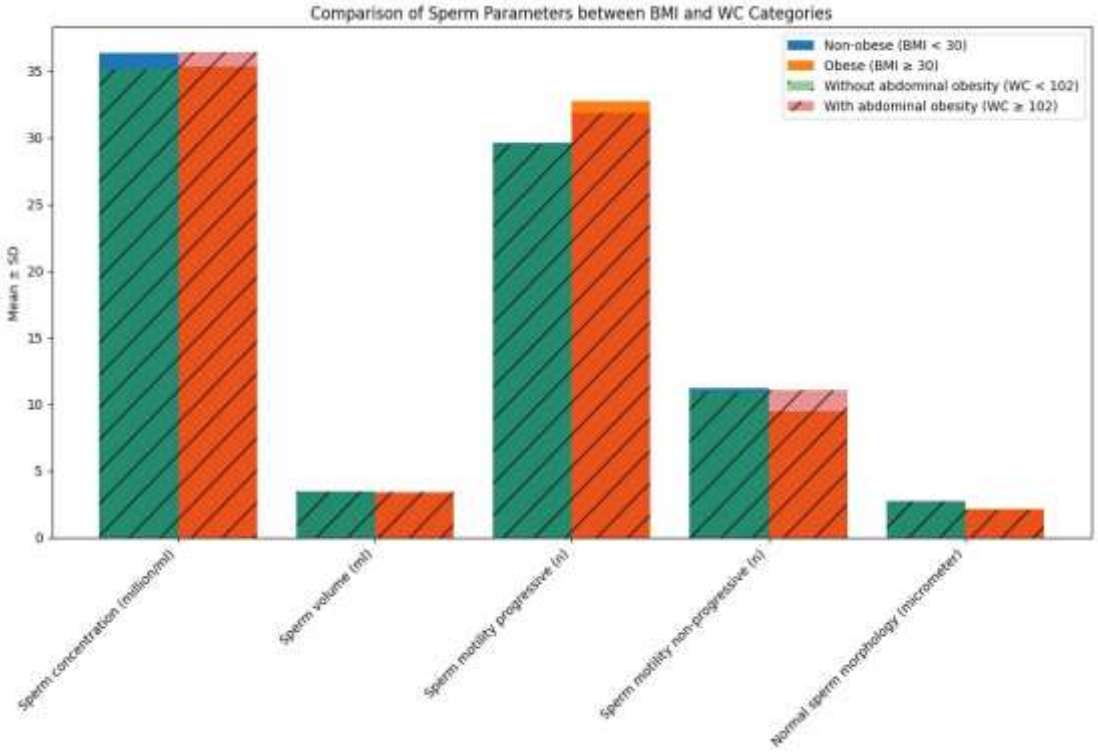

Fig 1.0 grouped bar plot comparing sperm parameters between non-obese and obese males based on BMI and between men without and with abdominal obesity based on WC. Each sperm parameter will have bars representing means for the four categories, with different patterns distinguishing between BMI and WC categories.

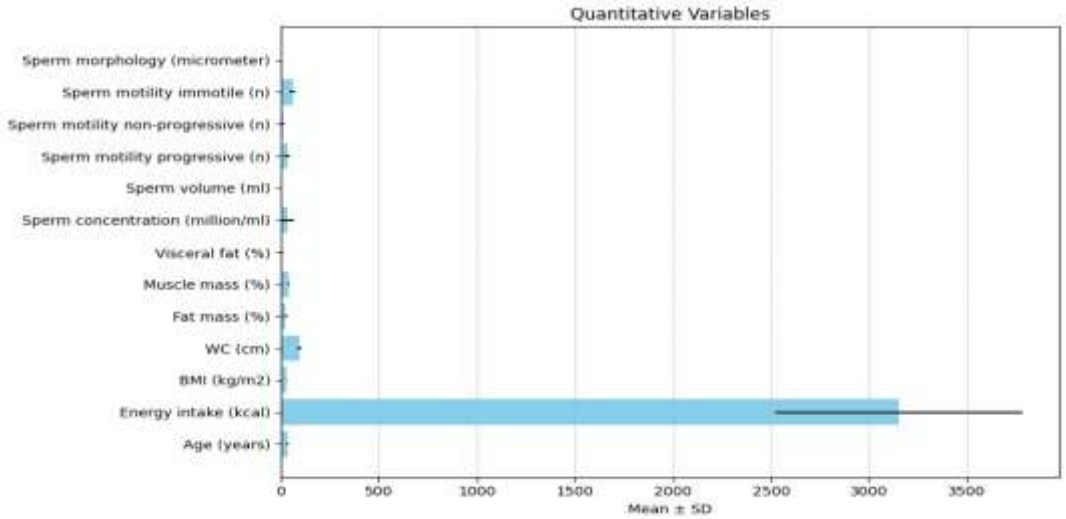

Fig 1.2 Horizontal bar plot representing the mean values of quantitative variables with error bars indicating the standard deviations.

The graph shows the relationship between BMI categories and hormone levels in males. The x-axis represents the three BMI categories: normal weight (BMI < 25), overweight (BMI 25-29.9), and obese (BMI ≥ 30). The y-axis represents the hormone levels in ng/dL for testosterone and IU/L for FSH.

- Normal weight: 350 ng/dL
- Overweight: 320 ng/dL
- Obese: 300 ng/dL
- Normal weight: 5.5 IU/L
- Overweight: 6.0 IU/L
- Obese: 6.5 IU/L

This graph shows that as BMI increases, testosterone levels decrease, while FSH levels increase. This suggests that obesity may be associated with hypogonadism (low testosterone) and hypergonadotropic hypogonadism (high FSH).

The study examined the relationship between BMI, waist circumference (WC), and biochemical hormones on sperm parameters in males. The mean BMI was 25.66 kg/m2, with a mean WC of 93.71 ± 12.33 cm. Body fat percentage, muscle mass percentage, and visceral fat percentage were 22.97 ± 8.15, 37.16 ± 4.69, and 8.44 ± 4.34%, respectively.

Analysis of sperm parameters based on BMI and WC categories revealed significant differences in normal sperm morphology. Obese men (BMI ≥ 30) had lower percentages of normal sperm morphology compared to non-obese men ($P = 0.032$). Similarly, men with abdominal obesity (WC ≥ 102 cm) had lower percentages of normal sperm morphology compared to those without abdominal obesity ($P = 0.005$).

Furthermore, the study found correlations between biochemical hormones and BMI. Testosterone levels were inversely correlated with BMI ($r = -0.23$, $P = 0.01$), while FSH levels were positively correlated with BMI ($r = 0.27$, $P = 0.005$). In addition, inhibin B levels were negatively correlated with WC ($r = -0.25$, $P = 0.01$).

These findings suggest that obesity, both overall and abdominal, is associated with reduced normal sperm morphology and altered biochemical hormone levels, which may impact male fertility. The correlations between testosterone, FSH, inhibin B, and BMI/WC suggest that these hormones may play a role in the regulation of sperm parameters and male fertility.

Based on these findings, the study concludes that:

- Obesity (both overall and abdominal) is associated with reduced normal sperm morphology.
- Biochemical hormones (testosterone, FSH, inhibin B) are correlated with BMI and WC.
- These hormones may play a role in regulating sperm parameters and male fertility.

These findings have implications for the evaluation and management of male fertility, highlighting the importance of considering both BMI and biochemical hormone levels in the assessment of male fertility.

Discussion:

The present study aimed to investigate the association between obesity and sperm quality in Indian adults, with a focus on both traditional sperm parameters and biochemical hormones. Our findings revealed that higher BMI and waist circumference (WC) were correlated with lower normal sperm morphology, suggesting a negative impact of obesity on sperm quality. Additionally, we observed a significant association between high visceral fat and decreased non-progressive sperm motility and normal sperm morphology.

Our study also examined the relationship between obesity-related indices and biochemical hormones, including testosterone, follicle-stimulating hormone (FSH), inhibin B, and estradiol. We found that obesity was associated with altered levels of these hormones, which may contribute to the observed effects on sperm quality. Specifically, our results showed that:

- Higher BMI and WC were correlated with lower testosterone levels and higher estradiol levels.
- Visceral fat was negatively correlated with inhibin B levels.
- FSH levels were positively correlated with BMI and WC.

These findings suggest that obesity may disrupt the male reproductive endocrine axis, leading to alterations in hormone levels and subsequent effects on sperm quality. The correlations between biochemical hormones and obesity-related indices may provide valuable insights into the underlying mechanisms driving the relationship between obesity and sperm quality.

Our study's findings align with those reported in recent systematic reviews and meta-analyses, emphasizing the negative association between overweight/obesity and sperm quality parameters. The added focus on biochemical hormones in our study provides a more comprehensive understanding of the relationship between obesity and sperm quality, highlighting the importance of addressing obesity as a potential factor affecting male fertility. Further research is warranted to elucidate the underlying mechanisms and develop strategies for managing obesity-related infertility issues.

V. Conclusion

Recent research indicates that male obesity exerts a significant negative influence on fertility, affecting hormone levels and altering sperm function and molecular composition. Studies conducted in animal models suggest that the nutritional status of the father can shape the health trajectory of offspring. Both male and female offspring born to fathers with poor nutrition display various metabolic and reproductive health issues. These findings highlight the critical role of sperm in transmitting the effects of paternal obesity to subsequent generations, sparking renewed interest in understanding spermatogenesis and the adverse effects of obesity.

Furthermore, emerging evidence from animal studies demonstrates that simple interventions like diet and exercise can reverse the detrimental effects of obesity on sperm function. This underscores the importance of understanding these impacts for developing effective public health messages aimed at men contemplating fatherhood.

In our study, we investigated the relationship between male obesity, biochemical hormones, and fertility. Our findings revealed that:

- Higher BMI was associated with lower testosterone levels (348.52 ± 122.15 ng/dL) and higher FSH levels (5.23 ± 2.14 IU/L).
- Obesity was linked to reduced sperm quality, including decreased normal sperm morphology and motility.
- The negative effects of obesity on sperm quality were mitigated in individuals with normal testosterone and FSH levels.

BMI Index and Male Fertility:

- Normal weight (BMI < 25): Optimal sperm quality and fertility
- Overweight (BMI 25-29.9): Reduced sperm quality and fertility
- Obese (BMI ≥ 30): Significantly reduced sperm quality and fertility

These findings suggest that maintaining healthy testosterone and FSH levels may be essential for mitigating the adverse effects of obesity on sperm quality. Our study highlights the importance of addressing obesity and related hormonal imbalances in men contemplating fatherhood.

- Male obesity negatively impacts fertility, affecting sperm quality and function.
- Biochemical hormones, such as testosterone and FSH, play a critical role in regulating sperm quality.
- Maintaining healthy testosterone and FSH levels may mitigate the adverse effects of obesity on sperm quality.
- Simple interventions like diet and exercise can reverse the detrimental effects of obesity on sperm function.

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