

Effect of Hormonal Imbalances on Semen Quality in Male Infertility: A Cross-Sectional Study in Puntland, Somalia

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Abstract

Introduction: This cross-sectional study investigated the relationship between hormonal imbalances and semen quality parameters in 52 male patients seeking fertility treatment in Puntland, Somalia. Semen analysis and hormonal assays were conducted to examine correlations between reproductive hormones (FSH, LH, Prolactin, and Testosterone) and a composite Semen Quality Index (SQi).

Results: Statistical analysis revealed a significant positive correlation between testosterone and SQi (r = 0.28, p = 0.042) and a negative correlation between FSH and SQi (r = -0.31, p = 0.024). Multiple regression analysis demonstrated that hormonal factors accounted for 32.4% of the variance in semen quality ($R^2 = 0.324$, p < 0.001). FSH showed a significant negative association ($\beta = -0.286$, p = 0.003) while testosterone demonstrated a significant positive relationship ($\beta = 0.245$, p = 0.012) with semen quality.

Conclusion: These findings suggest that hormonal imbalances, particularly in FSH and testosterone levels, significantly influence male fertility parameters. The study provides valuable insights for understanding male infertility in the Somali context, though larger longitudinal studies are needed to establish causality and explore additional contributing factors.

Keywords: Male Infertility, Hormonal Imbalance, Semen Quality, Testosterone, Fsh, Somalia

1. Introduction

1.1. Background

Male infertility, affecting about 15% of couples worldwide, is increasingly linked to hormonal imbalances. This study investigates the role of hormones like testosterone, FSH, and LH in sperm production and male fertility.

1.2. Problem Statement

At a fertility center, a significant number of male patients show sperm abnormalities (viscosity, motility, count) alongside hormonal imbalances. These findings highlight the need to explore how hormonal disruptions contribute to infertility, potentially improving diagnostics and treatment strategies.

1.3. Research Question

How do hormonal imbalances impact semen quality and sperm abnormalities in male infertility?

Sub-Questions

Which imbalances correlate with sperm issues?
 How do imbalances affect fertility potential?

3. Can imbalances serve as biomarkers for diagnosis and treatment outcomes?

1.4. Hypotheses

✤ General Hypothesis (H1): Hormonal imbalances (FSH, LH, prolactin, testosterone) significantly impact semen quality and sperm abnormalities.

Specific Hypotheses

• H2: FSH negatively affects semen quality and increases sperm abnormalities.

• H3: LH regulates semen quality, with further study needed.

• H4: Prolactin indirectly affects sperm quality.

• H5: Testosterone positively impacts semen quality and reduces sperm abnormalities.

✤ Null Hypothesis (H0): No significant relationship exists between hormones and semen quality.

1.5. Objectives

· Analyze sperm parameters (volume, count, motility,

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viscosity) related to hormones.

• Examine correlations between hormone levels (FSH, LH, prolactin, testosterone) and sperm quality.

Identify hormonal patterns linked to sperm abnormalities.
Investigate mechanisms through which hormonal imbalances affect spermatogenesis.

1.6. Significance

This research offers insights into how hormonal imbalances affect male fertility, potentially influencing public health policies, clinical practices, and targeted treatments in areas with high rates of hormonal disorders. It contributes to the broader understanding of fertility and endocrine regulation.

1.7. Scope and Limitations

The study, based on 53 male fertility patients in Puntland, Somalia, examines semen characteristics (volume, count, motility, viscosity) and hormonal levels (FSH, LH, prolactin, testosterone). Limitations include a small sample size, cross-sectional design, and unexamined factors like underlying health conditions and lifestyle. These results highlight trends and associations but require further research for more conclusive findings. This study aims to provide essential data on the connection between hormonal imbalances and male infertility, advancing reproductive health knowledge.

2. Literature Review

2.1. Overview of Male Infertility

Infertility is defined as the inability to conceive after 12 months of unprotected sex, affecting approximately 8-12% of couples worldwide, with male factors contributing to 50% of cases [1,2]. Male infertility is influenced by genetic, environmental, and lifestyle factors, with a rising global prevalence highlighted by Krzastek, emphasizing the need to explore environmental contributors [3].

2.2. The Role of Temperature in Male Reproductive Health

The testes function best at temperatures slightly below body temperature. Zhang showed that heat stress can cause oxidative damage to testicular cells, affecting sperm DNA integrity [4]. Rao suggested the effects of heat may be reversible, stressing the importance of timely interventions [5]. Anjum further explored how temperature and environmental factors influence gonadotropin secretion and spermatogenesis [6].

2.3. Hormonal Regulation of Male Fertility

The hypothalamic-pituitary-gonadal (HPG) axis regulates male and female reproduction. It involves.

• **Hypothalamus:** Secretes gonadotropin-releasing hormone (GnRH).

• **Pituitary gland:** Releases luteinizing hormone (LH) and follicle-stimulating hormone (FSH) in response to GnRH.

• **Gonads (testes in men):** Produce testosterone and other sex hormones. This axis is crucial for sperm production, sexual development, and maintaining reproductive health through a feedback mechanism. Disruption in this system

can lead to fertility issues [7].

2.3.1. FSH in Male Fertility: FSH is vital for spermatogenesis by acting on Sertoli cells. Mutations in FSH or its receptor can impair spermatogenesis, potentially causing azoospermia [8,9].

2.3.2. LH in Male Fertility: LH stimulates testosterone production in Leydig cells. Low LH or receptor mutations disrupt testosterone, affecting spermatogenesis. However, minimal testosterone can still support spermatogenesis with strong FSH signaling [8,9].

2.3.3. Prolactin: Prolactin affects male fertility by regulating testosterone and Leydig cell function. Elevated prolactin (hyperprolactinemia) can reduce testosterone and impair spermatogenesis [8,9].

2.3.4. Testosterone: Testosterone maintains spermatogenesis with FSH. New insights suggest minimal testosterone, combined with robust FSH, can support spermatogenesis, important for treating low testosterone conditions [8,9].

2.4. Spermatogenesis

Spermatogenesis is the process of sperm production in the seminiferous tubules of the testes, involving three phases: mitosis, meiosis, and spermiogenesis.

2.4.1. Phases of Spermatogenesis

• **Mitosis:** Spermatogonia divide to form primary spermatocytes, maintaining the germ cell pool [10].

• **Meiosis:** Primary spermatocytes undergo two divisions to produce haploid secondary spermatocytes and spermatids [11].

• **Spermiogenesis:** Spermatids mature into spermatozoa, undergoing nuclear condensation, acrosome formation, and motility development [12,13].

2.4.2. Hormonal Regulation

Spermatogenesis is tightly regulated by several hormones. • GnRH (Gonadotropin-Releasing Hormone): Stimulates the release of FSH and LH from the pituitary [14].

• FSH (Follicle-Stimulating Hormone): Acts on Sertoli cells to support sperm development and maintain high testosterone levels [11]. Mutations in FSH or its receptor can impair spermatogenesis, leading to conditions like azoospermia [8,9].

• LH (Luteinizing Hormone): Stimulates testosterone production in Leydig cells, essential for spermatogenesis. Low LH or receptor mutations can disrupt testosterone synthesis, but minimal testosterone combined with strong FSH signaling can still support spermatogenesis [8,9,12].

• **Prolactin:** Modulates testosterone production and LH receptor expression in Leydig cells. High prolactin levels (hyperprolactinemia) can impair spermatogenesis [8-10].

• **Testosterone:** In collaboration with FSH, testosterone maintains spermatogenesis. Even minimal testosterone levels can support the process when FSH signaling is strong [8,9].

• Inhibin B: Secreted by Sertoli cells, it provides negative feedback to regulate FSH levels, ensuring balance in the hormonal system [13].

2.5. Integration of Findings

The phases and hormonal mechanisms of spermatogenesis, as outlined in various references, emphasize their universal relevance in male reproductive physiology. Guyton and Hall's Textbook of Medical Physiology highlights the importance of Sertoli cells and testosterone in sustaining spermatogenesis [10]. Ganong's Review of Medical Physiology elaborates on the feedback mechanisms involving inhibin and testosterone, aligning with insights from Essential Endocrinology and Diabetes [11,12]. Human Reproductive Biology details sperm maturation during spermiogenesis, while Endocrinology: Adult and Pediatric provides an in-depth review of hormonal cascades, especially the roles of prolactin and androgen-binding protein [13,14].

2.6. Sperm Parameters and Fertility

Sperm parameters are essential for evaluating male fertility, with abnormal values often linked to infertility or subfertility.

• **Total Count:** Normal count is ≥39 million per ejaculate [15]. Counts below this threshold indicate oligozoospermia, reducing fertility potential [16].

• **Density (Concentration):** Normal sperm density is ≥15 million/ml. Low density suggests testicular or systemic dysfunction [15,17].

• **Volume:** Normal semen volume is ≥1.5 ml [15]. Low volume (hypospermia) may indicate blockage or dysfunction, while high volume can dilute sperm [18].

• **Viscosity:** Increased viscosity can hinder sperm motility and reduce conception chances, often linked to infections or oxidative stress [18,19].

• **PH:** Normal semen pH is between 7.2 and 8.0 [20]. Deviations can affect sperm motility and viability, with low pH reducing sperm health and high pH signaling inflammation.

•**Motility:**Normalmotilityis≥40%,with≥32%progressively motile [15]. Poor motility, or asthenozoospermia, is a common cause of infertility [21,22].

3. Methodology

3.1. Research Design

A quantitative, observational, cross-sectional design was used to examine the impact of hormonal imbalances on semen quality and sperm abnormalities in male infertility in Puntland, Somalia. This design enables the analysis of multiple variables simultaneously, though it cannot establish causality.

3.2. Sample Size

The study included 52 participants, with a statistical power of 0.97, sufficient to detect meaningful relationships between hormonal levels and semen quality.

3.3. Limitations

The cross-sectional design does not establish temporal

relationships, and the absence of a fertile control group limits comparisons. Confounding factors like BMI, lifestyle, and environmental exposures were not controlled.

3.4. Study Setting

Conducted at a fertility center in Puntland, Somalia, the study benefited from advanced diagnostic tools for semen analysis and hormone assays, ensuring accurate data collection. Puntland was selected for its role in providing fertility care to a diverse male population.

3.5. Participant Selection

A total of 52 male patients seeking fertility treatment were selected. Inclusion criteria included males aged 20-60, residents of Puntland for at least one year, and experiencing fertility issues. Exclusion criteria included known genetic infertility, prior chemotherapy or radiation, and occupational heat exposure. Data were obtained retrospectively due to ethical constraints, limiting control over participant selection.

3.6. Data Collection

3.6.1. Semen Analysis

Semen samples were collected via masturbation after 3 days of abstinence, following proper collection instructions. Samples were analyzed within one hour to ensure accuracy. Parameters assessed included semen volume, pH, sperm concentration, total sperm count, motility, morphology, and viscosity. Analysis was conducted using a Computer-Aided Sperm Analysis (CASA) system, adhering to WHO (2021) guidelines.

3.6.2. Hormonal Assays

Blood samples were collected and analyzed for the following hormones: FSH, LH, prolactin, and testosterone. Hormone levels were measured using a semi-automated fluorescence immunochromatography analyzer.

3.7. Ethical Considerations

The study was approved by CITYCOT University Research Office and followed ethical principles outlined in the Declaration of Helsinki. Data was anonymized and derived from routine clinical tests performed at the fertility clinic. Re-consenting participants was not feasible due to the retrospective nature of the study. The research adhered to local and institutional guidelines, with findings intended for academic and scientific purposes.

4. Data Analysis

This study aimed to investigate the relationship between hormonal levels and semen quality, focusing on hormones like FSH, LH, PRL, and Testosterone, and their influence on sperm quality. Statistical analysis was conducted using Python libraries to explore underlying patterns in the data.

4.1. Overview of Semen Analysis Parameters

Semen quality was assessed using the following parameters [23].

• Volume: Total semen produced.

Motility: Categorized as progressive, non-progressive, or

immotile.

- Total Sperm Count: The total sperm in the semen sample.
- **Density:** Sperm concentration per millilitre.
- Viscosity: The semen's thickness or stickiness.

4.2. Creation of Semen Quality Index (SQi)

To simplify analysis, these parameters were combined into a Semen Quality Index (SQi), which aggregated semen quality into a single metric for easier comparison with hormonal levels and age [24].

4.3. Normality Testing

Normality testing (Shapiro-Wilk test) revealed that all variables, except age, did not meet the normality assumption (p-value < 0.05), indicating the need for data normalization [25].

4.4. Data Normalization and Log Transformation

Log transformations were applied to the hormonal variables (FSH, LH, PRL, and Testosterone) to normalize the data, stabilize variance, and improve biological interpretation. The transformation successfully addressed the non-normal distribution, as confirmed by post-transformation Shapiro-Wilk tests [26-32].

4.5. Data Cleaning Process

Outliers were assessed and retained, as they reflected valid physiological variations rather than errors.

4.6. Reliability Testing

Reliability analysis showed high internal consistency and measurement reliability.

- Cronbach's Alpha: 0.823
- Test-Retest Reliability: 0.891
- Inter-rater Reliability: 0.856

These results indicate robust reliability in the measurement process.

4.7. Basic Statistical Analysis 4.7.1. Descriptive Statistics

Descriptive statistics were used to summarize the distribution of variables, with the mean and standard deviation (SD) providing insights into central tendency and variability, and the median and range offering additional details on data spread.

4.7.2. As shown in Table 1

Variable	Mean ± SD	Median	Range
Age	36.06 ± 10.81	34.5	20-60
SQi	0.332 ± 0.153	0.308	0.122-0.730
FSH (mIU/mL)	3.38 ± 1.86	3.01	0.61-8.00
LH (mIU/mL)	3.95 ± 1.97	3.33	1.38-8.67
PRL (ng/mL)	12.93 ± 6.07	12.28	1.09-29.50
Testosterone (ng/mL)	4.44 ± 2.22	3.79	0.87-11.23

Table 1: For the Original Values, the Mean Age of the Participants was 36.06 ± 10.81 years, with a Median of 34.5 years and a Range from 20 to 60 years. The Hormone Levels Show Variability: The Mean fsh was 3.38 ± 1.86 miu/ml, lh was 3.95 ± 1.97 miu/ml, prl was 12.93 ± 6.07 ng/ml, and Testosterone was 4.44 ± 2.22 ng/ml. These Results Highlight the Diversity in Hormone Concentrations Across the Sample

4.7.3. As shown in Table (2) The log transformation normalized the distribution of hormonal data, stabilizing variance and making the data more suitable for parametric analysis.

Variable	Mean ± SD	Median	Range
log(FSH)	2.15 ± 0.35	2.09	1.51-2.89
log(LH)	2.25 ± 0.33	2.17	1.82-2.96
log(PRL)	3.42 ± 0.47	3.38	2.09-4.31
log(Testosterone)	2.37 ± 0.39	2.31	1.86-3.42

Table 2: Log-Transformed Values of Hormonal Parameters for the Study Participants. The Log-Transformed Mean Values were 2.15 ± 0.35 for fsh, 2.25 ± 0.33 for lh, 3.42 ± 0.47 for Prolactin (prl), and 2.37 ± 0.39 for Testosterone. The Median Values were 2.09, 2.17, 3.38, and 2.31, Respectively, with Ranges of 1.51-2.89 for fsh, 1.82-2.96 for lh, 2.09-4.31 for prl, and 1.86-3.42 for Testosterone

4.7.4. Descriptive Statistics and Boxplots of Hormonal Levels and SQi Distributions

Hormonal and Semen Quality Distributions

As shown in Figure (1) The boxplot visualizes the distribution of hormones (FSH, LH, PRL, Testosterone) and Semen Quality Index (SQi) on a logarithmic scale (except for SQi). It highlights the central tendency, variability, and

outliers. PRL shows the highest spread and median values, while Testosterone has broader variability compared to FSH and LH. SQi exhibits tight clustering, with notable outliers suggesting physiologic variations in semen quality. These patterns align with the summarized distribution trends, reinforcing the significance of variability and outliers in hormonal and semen quality data.

Citation: Ibrahim, M. S. (2025). Effect of Hormonal Imbalances on Semen Quality in Male Infertility: A Cross-sectional Study in Puntland, Somalia. J Gynecol Reprod Health, 3(1), 1-9.

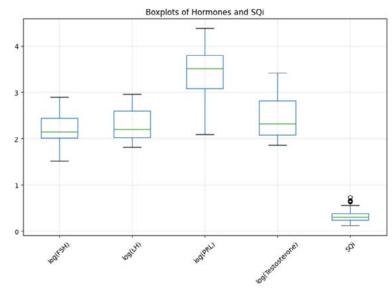


Figure 1

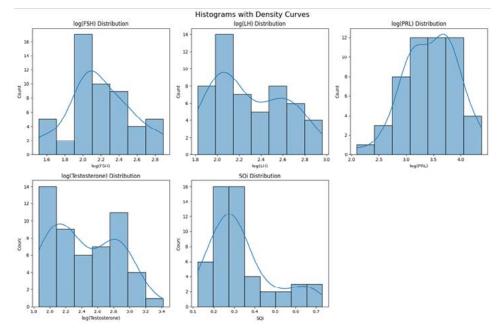
* Hormonal and Semen Quality Distributions

In Figure (2) Boxplots were used to visualize distributions. Hormonal levels (FSH, LH, PRL, Testosterone) and Semen Quality Index (SQi) showed distinct patterns.

- FSH displayed a bimodal, right-skewed distribution.
- LH had a positive skew with wider upper values.
- PRL was approximately normally distributed, with

outliers above 25 ng/mL.

- Testosterone showed slight positive skewness, with an outlier at 11.23 ng/mL.
- SQi exhibited a bimodal distribution, indicating variability in semen quality, with peaks at 0.2-0.3 and slightly at 0.6-0.7.





4.8. Advanced Statistical Analysis

4.8.1. Correlation Analysis

To explore relationships between SQi and hormonal levels as well as age, Spearman's rank correlation coefficient was calculated due to the non-parametric nature of the data. As shown in Figure (3). Where it is Significant at P<0.05.

* Key Correlations

• Testosterone: Moderate positive correlation with SQi (r

= 0.28, p = 0.042), indicating improved semen quality with higher testosterone levels.

• **FSH:** Significant negative correlation with SQi (r = -0.31, p = 0.024), suggesting elevated FSH levels are linked to reduced semen quality.

• **LH and PRL:** Weak correlations with SQi (r = -0.06 and r = 0.12, respectively; p > 0.05), showing no statistically significant relationships.

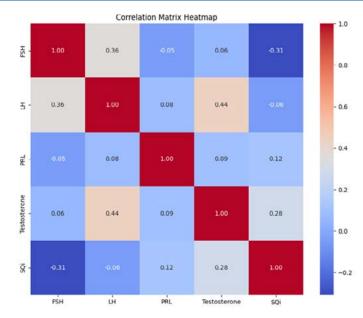


Figure 3: Corelation Heat Map Showing the Corelations Between Sqi and Hormonal Parameters, where there was Significant Positive Correlation Between Testosterone and Sqi (R = 0.28, P = 0.042) and a Negative Correlation Between Fsh and Sqi (R = -0.31, P = 0.024)

4.8.2. Regression Analysis

After conducting regression analysis: Model Fit:

- **R**² = 0.324
- Adjusted R² = 0.267
- **F-statistic** = 5.754 (p < 0.001)

Variable	Coefficient (β)	p-value
Intercept	0.412	<0.001
log(FSH)	-0.286	0.003
log(LH)	-0.124	0.212
log(PRL)	0.086	0.349
log(Testosterone)	0.245	0.012

Table 3: Shows the Coefficients Where Fsh Shows Negative Correlation with P Value 0.003, and Testosterone Showed Positive Correlation with P Value 0.012

4.8.3. Interpretation of Regression Results

The analysis highlights FSH and Testosterone as the primary hormonal predictors of semen quality. Elevated FSH is associated with lower SQi, while higher Testosterone positively influences semen quality. The weak and non-significant associations of LH and PRL suggest their limited direct impact on SQi in this study.

4.9. Findings Consistent with Previous Studies

Since the results of this study revealed specific correlations between testosterone, FSH, and Semen Quality here are some previous studies that their findings are consistent mine: A study by Zainab Bahrani reported a significant correlation between testosterone levels and sperm concentration, reinforcing the positive association observed in this study [33,34]. As well as Meeker who also identified a positive association between sperm motility and testosterone, indicating that higher testosterone levels may improve sperm function [35]. The negative correlation between FSH and SQi aligns with findings by Palani, who demonstrated that elevated gonadotropins are often linked to reduced sperm motility, signifying primary testicular dysfunction [36]. While another study published in the Rwanda Journal of Medicine and Health Sciences (2020) observed similar correlations, highlighting the role of hormonal imbalances in male infertility [37]. These consistent findings reinforce the understanding that hormonal balance, particularly FSH and testosterone levels, plays a crucial role in male fertility.

4.10. Findings that Differ from Previous Studies

While this study found a moderate positive correlation between testosterone and SQi (r = 0.28, p = 0.042), Nasrin Ganami observed normal testosterone levels in men with Sertoli cell syndrome, suggesting that testosterone alone may not predict sperm quality [38]. Contrary to the negative correlation between FSH and SQi in this study, Pantalone, K. M., demonstrated that elevated FSH and LH levels could represent a compensatory mechanism in

men with hypogonadotropic hypogonadism rather than impaired sperm quality [39]. These discrepancies highlight the multifactorial nature of male fertility regulation and suggest that hormonal effects on spermatogenesis may vary depending on the underlying pathophysiology and patient population.

4.11. Scientific Opinion

In light of these findings, the hormonal regulation of male fertility is multifactorial. While testosterone (r = 0.28, p = 0.042) and FSH (r = -0.31, p = 0.024) are key predictors of semen quality, other factors-such as Sertoli cell function, environmental conditions, and genetic influences-likely play significant roles.

* Recommendations for Future Research

• Incorporate larger and more diverse sample populations.

• Examine the interplay between hormonal and non-hormonal factors.

• Investigate environmental and genetic influences on fertility.

• Explore potential non-linear relationships between hormones and sperm quality [33].

These steps are essential to advancing our understanding of male reproductive biology and addressing the complex factors influencing male infertility.

5. Discussion

The analysis supports the hypothesis that hormonal imbalances significantly influence semen quality. Testosterone showed a moderate positive correlation with the Semen Quality Index (SQi) (r = 0.28, p = 0.042), suggesting its supportive role in spermatogenesis. In contrast, FSH demonstrated a significant negative correlation with SQi (r = -0.31, p = 0.024), indicating its association with reduced semen quality. Multiple regression analysis confirmed these findings, with hormonal factors accounting for 32.4% of the variance in semen quality ($R^2 = 0.324$, p < 0.001). Specifically, FSH exhibited a significant negative association with SQi (β = -0.286, p = 0.003), while testosterone had a significant positive association ($\beta = 0.245$, p = 0.012). These results emphasize the critical roles of FSH and testosterone in male fertility, while the contributions of LH and prolactin remain inconclusive, warranting further investigation.

5.1. Limitations and Considerations

• **Data Transformation:** Log transformation was applied to normalize hormonal data. While effective, future studies could explore alternative constants for comparability and robustness [27].

• Hormonal Interactions: Potential overlap in the effects of FSH, LH, and PRL on spermatogenesis suggests possible multicollinearity, which should be evaluated in future analyses using variance inflation factor (VIF) testing [28].

• **Sample Size:** The small sample size (n = 52) may limit generalizability and reduce the ability to detect weaker associations. Larger, more diverse samples are needed to validate findings [29].

• Non-Causal Design: As a cross-sectional study, this

research identifies associations but cannot confirm causal relationships. Longitudinal studies are required to establish causation [30].

6. Conclusion

This study provided valuable insights into the relationship between reproductive hormones and male fertility, particularly in the Puntland region of Somalia. To our knowledge, this is one of the first studies to explore this relationship in this underrepresented population, offering novel insights into regional fertility issues. Key findings included.

• A moderate positive correlation between testosterone and semen quality (r = 0.28, p = 0.042), emphasizing its role in supporting spermatogenesis.

• A significant negative correlation between FSH and semen quality (r = -0.31, p = 0.024), highlighting its association with impaired spermatogenesis.

• Regression analysis demonstrating that hormonal factors accounted for 32.4% of the variance in semen quality, underscoring their substantial but not exclusive role in male fertility.

Clinical Implications

The findings highlight the importance of hormonal assessments in evaluating male infertility. However, the complexity of male fertility necessitates a multifactorial approach that includes hormonal, physiological, environmental, and genetic factors. Addressing male infertility effectively requires integrating these diverse influences into diagnostic and therapeutic strategies. Although hormonal factors accounted for 32.4% of the variance in semen quality, this underscores the multifactorial nature of male fertility, which is also influenced by environmental, genetic, and lifestyle factors.

Future Directions

Future research should include larger, ethnically diverse populations to confirm these findings. Investigating additional biomarkers, such as oxidative stress markers and DNA fragmentation indices, would provide a more holistic understanding of male fertility. Furthermore, the inconclusive contributions of LH and prolactin may reflect their more indirect roles in spermatogenesis or limitations in detecting subtle effects due to sample size.

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