

Original Research

Assessment of Ovarian Response Using FSH/AMH Ratio Across Body Mass Index Categories in Infertile Women: A Retrospective Cross-Sectional Study

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Abstract

Background: Obesity significantly influences female reproductive health; however, its specific impact on hormonal predictors of ovarian response remains uncertain. The follicle-stimulating hormone (FSH)/ anti-Müllerian hormone (AMH) ratio has recently gained attention as a potential marker of ovarian reserve and response to controlled ovarian stimulation. This study aimed to assess the association between the FSH/AMH ratio and oocyte count and to determine whether body mass index (BMI) modifies this relationship. **Methods:** In this retrospective study, 185 women undergoing ovarian stimulation were reviewed, and 92 met predefined clinical and hormonal inclusion criteria. Baseline FSH, luteinizing hormone (LH), AMH, BMI, and oocyte counts were recorded, and the FSH/AMH ratio was calculated. Associations were assessed using Spearman correlation, Kruskal-Wallis tests, and linear regression analysis. **Results:** AMH levels showed a strong positive correlation with oocyte count, while the FSH/AMH ratio demonstrated a strong negative correlation. FSH exhibited a weak negative correlation, and no significant association was observed between BMI and hormonal markers. Neither oocyte count nor the FSH/AMH ratio differed significantly across BMI categories. Linear regression analysis confirmed that the FSH/AMH ratio was an independent predictor of oocyte yield ($p < 0.001$), whereas BMI and its interaction with the ratio were not statistically significant. **Conclusions:** The FSH/AMH ratio is a reliable and BMI-independent predictor of ovarian response. These findings support its clinical utility in fertility assessment and treatment planning, particularly when standard markers are inconclusive.

Keywords: anti-Müllerian hormone; follicle-stimulating hormone; obesity; infertility; assisted reproductive technology

1. Introduction

Obesity represents a major global health issue and is increasingly prevalent among women of reproductive age. It is well-established that excess body weight negatively impacts female reproductive function, contributing to menstrual irregularities, anovulation, impaired oocyte quality, and reduced fertility rates [1–4]. Epidemiological studies indicate that obese women are nearly three times more likely to experience infertility compared to women with a normal body mass index (BMI) [5]. Ovulatory dysfunction—manifesting as oligomenorrhea or anovulation—is more frequently observed in obese women and may occur independently or as part of polycystic ovary syndrome (PCOS), a condition closely linked with obesity [6]. Nevertheless, the precise mechanisms by which obesity impairs reproductive function remain incompletely elucidated.

In addition to reproductive disturbances, obesity is associated with systemic comorbidities such as insulin resistance, type 2 diabetes mellitus, hypertension, cardiovascular disease, and certain cancers, all of which may indirectly compromise reproductive health [7]. Notably, the pattern of fat distribution appears to influence reproductive outcomes. Central (visceral) adiposity, in particular, has been more

strongly associated with ovulatory infertility than peripheral fat distribution [8–10]. Increasing evidence suggests that obesity is not solely a mechanical or metabolic burden but also an endocrine disorder, characterized by the secretion of bioactive molecules known as adipokines [11]. Among these, leptin plays a pivotal role in both energy homeostasis and reproductive regulation [12].

Leptin, primarily secreted by white adipose tissue, serves as a critical signal to the hypothalamic-pituitary-gonadal (HPG) axis and is essential for maintaining reproductive endocrine function. It promotes the secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus, which subsequently stimulates the anterior pituitary to release luteinizing hormone (LH) and follicle-stimulating hormone (FSH) [13]. The importance of leptin in reproductive physiology has been demonstrated in leptin-deficient animal models and in rare human cases of congenital leptin deficiency, both of which exhibit impaired reproductive function that improves with leptin supplementation [14,15]. However, in the context of obesity, chronically elevated leptin levels may induce leptin resistance, impairing its regulatory effects and contributing to reproductive dysfunction [16]. Animal studies have further shown that obesity-induced hyperleptinemia can blunt the hypothala-



mic and ovarian responsiveness to leptin, thereby disrupting folliculogenesis and ovulation [17].

Beyond leptin, the hormones FSH and anti-Müllerian hormone (AMH) are widely recognized as key indicators of ovarian reserve and reproductive capacity [18]. FSH, secreted by the anterior pituitary, stimulates the growth and maturation of ovarian follicles. Elevated basal FSH levels—particularly during the early follicular phase—may reflect diminished ovarian reserve due to decreased negative feedback from estrogen and inhibin β [19]. AMH, in contrast, is produced by granulosa cells of small pre-antral and antral follicles and serves as a reliable marker of the remaining follicular pool. Given its relative stability throughout the menstrual cycle and its decline with advancing age, AMH has become an essential tool in assessing ovarian reserve and predicting response to controlled ovarian stimulation [20].

Obesity has been shown to alter both AMH and FSH levels, although findings in the literature remain inconsistent [21]. Some studies have reported lower AMH levels in obese women, possibly due to impaired granulosa cell function or the inhibitory effect of leptin on *AMH* gene expression [22,23]. Meanwhile, FSH levels in obese women may remain stable or slightly reduced, potentially due to increased peripheral estrogen synthesis via aromatase activity in adipose tissue, which suppresses FSH secretion through negative feedback mechanisms [24].

The hormonal dysregulation associated with obesity—including altered levels of leptin, insulin, estrogen, and androgens—may collectively interfere with normal folliculogenesis, ovulation, and fertility potential [2,25]. To better understand this complex interplay, recent research has investigated composite hormonal indices, particularly the FSH/AMH ratio, as a more integrative marker of ovarian function [26,27]. Since FSH and AMH exhibit opposing trends with declining ovarian reserve—FSH increasing and AMH decreasing—their ratio may more accurately reflect the dynamic hormonal milieu than either marker alone. Several studies have explored the utility of the FSH/AMH ratio in predicting ovarian response in assisted reproductive technology (ART) settings, with some reporting associations between higher FSH/AMH ratios and reduced oocyte yield or diminished ovarian function [28,29].

Given the rising prevalence of obesity among reproductive-aged women and its multifaceted effects on ovarian physiology and endocrine function, further research is needed to clarify the relationships between obesity and reproductive hormone profiles. In the present study, we aimed to evaluate the associations between BMI and key reproductive markers—specifically FSH, AMH, and the FSH/AMH ratio—and to assess their predictive value for oocyte yield in women undergoing controlled ovarian stimulation. We further aimed to investigate whether the FSH/AMH ratio serves as a more sensitive indicator of

ovarian reserve across different BMI categories, thereby enhancing fertility assessment and individualized treatment planning.

2. Materials and Methods

2.1 Study Design and Population

This retrospective cross-sectional study was conducted at the Assisted Reproductive Techniques Center of Acibadem Mehmet Ali Aydınlar University Atakent Hospital between 2015 and 2022. A total of 185 women who underwent controlled ovarian stimulation as part of infertility treatment were initially evaluated. The study received approval from the Institutional Review Board of Acibadem Mehmet Ali Aydınlar University School of Medicine (protocol code 2023-10/427) and was conducted in accordance with the principles outlined in the Declaration of Helsinki.

Inclusion criteria were as follows: women aged 25 to 35 years, with baseline FSH levels ≤ 15 IU/mL, LH levels ≤ 12 IU/mL, AMH levels between 0.5 and 4.5 ng/mL, and ≤ 15 oocytes retrieved following stimulation. The selected age range was intended to encompass women with a relatively preserved ovarian reserve, minimizing the influence of age-related fertility decline, which becomes more pronounced after 35 years. Exclusion criteria included a diagnosis of PCOS, known endocrine disorders, class III obesity (BMI ≥ 40 kg/m²), endometriosis, diminished ovarian reserve (AMH < 0.5 ng/mL), or male factor infertility, in order to reduce potential confounding factors.

2.2 Clinical and Hormonal Assessment

All participants underwent controlled ovarian stimulation using a recombinant gonadotropin protocol. On the second day of the menstrual cycle, patients received recombinant FSH (Gonal-F®, Merck-Serono S.A., Darmstadt, Hesse, Germany) at a dose of 225–300 IU. A GnRH antagonist protocol was employed for all patients. Serum FSH and LH levels were measured on cycle days 2 or 3, and AMH levels were assessed prior to the initiation of stimulation. Hormonal assays were conducted using an electrochemiluminescence immunoassay (ECLIA) on the COBAS 8000 e801 analyzer (Roche Diagnostics GmbH, Mannheim, Germany). Baseline AMH values and post-treatment oocyte counts were recorded for all participants.

Additional parameters, including thyroid-stimulating hormone (TSH) levels and BMI, were also documented. The FSH/AMH ratio was calculated for each patient by dividing the serum FSH level by the AMH concentration. Oocyte retrieval was performed following stimulation, and the total number of retrieved oocytes served as the primary outcome measure. To assess the potential influence of body weight on hormonal profiles and ovarian response, participants were stratified into five BMI categories based on World Health Organization (WHO) classifications: underweight (< 18.50 kg/m²), normal weight (18.50–24.90

kg/m²), overweight (25.00–29.90 kg/m²), class I obesity (30.00–34.90 kg/m²), and class II obesity (35.00–39.90 kg/m²).

2.3 Statistical Analysis

Statistical analyses were performed using GraphPad Prism (version 9.5.1; GraphPad Software Inc., San Diego, CA, USA). Continuous variables were presented as mean $\pm$ standard deviation (SD) for normally distributed data or median with interquartile range (IQRs) for non-normally distributed data. Categorical variables were expressed as frequencies and percentages.

The normality of continuous variables was assessed using the Shapiro-Wilk test, and Levene's test was used to evaluate the homogeneity of variances across BMI groups. For between-group comparisons, one-way analysis of variance (ANOVA) was applied to normally distributed variables (age, BMI, FSH), while the Kruskal-Wallis test was used for non-normally distributed variables (LH, TSH, AMH, FSH/AMH ratio, and oocyte count). Spearman's rank correlation analysis was employed to examine associations between hormonal markers (FSH, AMH, FSH/AMH ratio), BMI, and oocyte yield.

To determine the independent contributions of the FSH/AMH ratio and BMI to oocyte count, a multiple linear regression model was developed. An interaction term (FSH/AMH ratio $\times$ BMI) was included to assess potential effect modification. A secondary model was adjusted for age to control for its potential confounding influence. Model assumptions, including linearity and homoscedasticity, were verified using residual plots and the Breusch-Pagan test. Statistical significance was set at $p < 0.05$.

In addition, a non-parametric bootstrap procedure was conducted to estimate the confidence intervals (CIs) of the mean oocyte count within each BMI category. This method involved 10,000 resampling iterations and was performed to assess the robustness and reliability of subgroup comparisons, particularly for BMI groups with limited sample sizes.

3. Results

A total of 92 women met the predefined inclusion criteria: age between 25 and 35 years, FSH ≤ 15.00 IU/mL, LH ≤ 12.00 IU/mL, AMH between 0.50 and 4.50 ng/mL, and no more than 15 oocytes retrieved following controlled ovarian stimulation. The mean age of participants was 32.70 ± 2.80 years. Mean serum FSH and AMH levels were 7.06 ± 1.67 IU/mL and 1.72 ± 0.89 ng/mL, respectively. The mean FSH/AMH ratio was 5.77 ± 4.35 , with values ranging from 0.88 to 20.44. The mean BMI was 25.20 ± 4.20 kg/m² (range: 17.30–39.20), and the mean number of oocytes retrieved was 8.13 ± 3.33 (range: 2–15).

Table 1 summarizes the baseline characteristics of the study population stratified by BMI category. Across BMI

groups, LH levels differed significantly ($p = 0.050$), while no significant differences were observed for age, FSH, AMH, TSH, or oocyte count (all $p > 0.050$). The BMI distribution confirmed expected differences across the categorized groups ($p < 0.001$).

Spearman's correlation analysis revealed a strong positive association between AMH and oocyte count ($r = 0.63$, $p < 0.001$), and a strong negative association between the FSH/AMH ratio and oocyte count ($r = -0.61$, $p < 0.001$). FSH demonstrated a weak negative correlation with oocyte count ($r = -0.17$, $p = 0.11$), while BMI showed no significant correlation with any ovarian reserve marker ($r = -0.02$, $p = 0.84$).

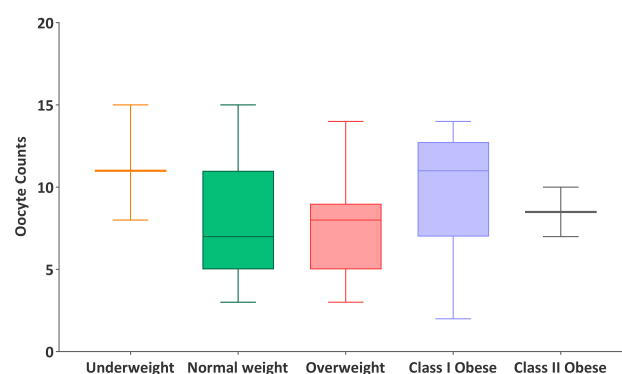


Fig. 1. Distribution of oocyte counts across BMI categories.

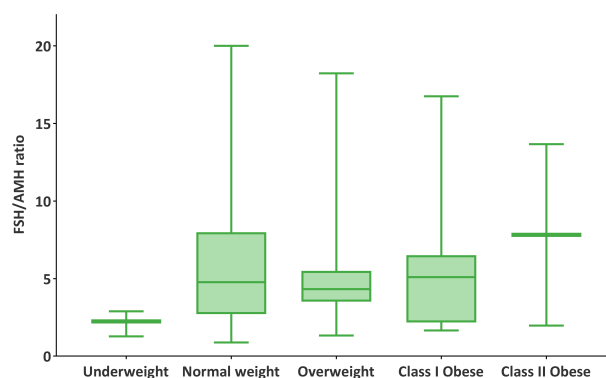


Fig. 2. Distribution of FSH/AMH ratios across BMI categories.

Participants were stratified into five BMI categories based on WHO classifications: underweight ($n = 3$), normal weight ($n = 50$), overweight ($n = 29$), class I obese ($n = 7$), and class II obese ($n = 3$). The highest median oocyte count was observed in the underweight group (11), followed by class II obese (10), class I obese (9), overweight (8), and normal weight (7). Median FSH/AMH ratios were 2.24 (underweight), 4.77 (normal weight), 4.32 (overweight), 5.09 (class I obese), and 9.40 (class II obese), as illustrated in Figs. 1,2. However, the Kruskal-Wallis test revealed no

Table 1. Baseline clinical and hormonal characteristics of participants stratified by BMI category.

	Underweight (<18.50 kg/m ²)	Normal weight (18.50 – 24.90 kg/m ²)	Overweight (25.00 – 29.90 kg/m ²)	Class I Obese (30.00 – 34.90 kg/m ²)	Class II Obese (35.00 – 39.90 kg/m ²)	<i>p</i> -value
Age (years)	33.33 ± 1.53	33.50 [31.25–35.00]	34.00 [32.00–35.00]	34.00 [33.75–35.00]	32.50 ± 2.12	0.890
Oocyte Counts	11.33 ± 3.51	7.00 [5.00–11.00]	7.45 ± 2.78	9.62 ± 4.03	8.50 ± 2.12	0.160
BMI (kg/m ²)	18.00 [17.65–18.00]	22.00 [20.86–23.41]	26.98 ± 1.45	31.98 ± 1.50	36.07 ± 1.03	<0.001
FSH (IU/mL)	6.61 ± 2.67	7.18 ± 1.55	6.50 [5.44–8.75]	6.94 ± 1.44	7.05 ± 1.04	0.780
AMH (ng/mL)	3.09 ± 0.21	1.46 [0.90–2.43]	1.56 ± 0.62	1.76 ± 0.89	1.89 ± 1.87	0.080
LH (IU/mL)	6.50 ± 1.13	5.43 ± 2.15	5.12 ± 2.12	3.69 ± 1.29	5.64 ± 2.20	0.050
TSH (mIU/L)	2.35 ± 0.91	1.87 [1.31–2.52]	1.87 ± 0.81	1.80 ± 0.82	2.48 ± 0.71	0.740

Data are presented as mean $\pm$ standard deviation (SD) for normally distributed variables, and median [interquartile range] for skewed variables. FSH, follicle-stimulating hormone; AMH, anti-Mullerian hormone; LH, luteinizing hormone; TSH, thyroid-stimulating hormone; BMI, body mass index.

statistically significant differences among BMI groups in either oocyte count ($H = 5.35$, $p = 0.253$) or FSH/AMH ratio ($H = 5.58$, $p = 0.134$). These findings suggest that BMI category was not significantly associated with ovarian response or hormonal ratio within this cohort, although observable trends warrant further exploration in larger populations. To further examine the robustness of oocyte count estimates across BMI subgroups, non-parametric bootstrap analyses (10,000 iterations) were performed. The 95% CIs for the mean oocyte counts were as follows: underweight, 6.67–13.67; normal weight, 6.45–8.31; overweight, 6.70–8.28; class I obese, 6.57–10.71; and class II obese, 5.50–10.50. Notably, the widest CIs were observed in the underweight and class II obese groups, consistent with their limited sample sizes ($n = 3$ for each). Despite numerical differences in mean oocyte yield across BMI categories, the overlapping CIs indicate that these variations are likely due to sampling variability rather than statistically robust subgroup effects.

To further assess predictors of ovarian response, linear regression analysis was conducted (Fig. 3). In the initial model, which included both the FSH/AMH ratio and BMI as continuous variables, the FSH/AMH ratio was significantly and negatively associated with oocyte count ($\beta = -0.58$; 95% CI: -0.74 to -0.42 ; $p < 0.001$), explaining 37% of the variance ($R^2 = 0.37$). Conversely, BMI was not significantly associated with oocyte count ($\beta = -0.03$; 95% CI: -0.25 to 0.19 ; $p = 0.754$). An interaction term (FSH/AMH ratio $\times$ BMI) was added to examine effect modification; however, the interaction was not statistically significant ($\beta = 0.00$; $p = 0.763$), indicating that the predictive relationship between the FSH/AMH ratio and oocyte count was not influenced by BMI. A second regression model, adjusted for age, confirmed the robustness of these findings. The FSH/AMH ratio remained a significant independent predictor of oocyte count ($\beta = -0.38$; 95% CI: -0.53 to -0.23 ; $p < 0.001$), whereas BMI ($\beta = -0.02$; 95% CI: -0.17 to 0.13 ; $p = 0.782$) and age ($\beta = 0.01$; 95% CI: -0.19 to 0.21 ; $p = 0.929$) were not significant contributors. This adjusted model accounted for 24.1% of the variance in oocyte count ($R^2 = 0.24$; adjusted $R^2 = 0.22$). Model assumptions regard-

ing linearity and homoscedasticity were verified, and the Breusch–Pagan test showed no evidence of heteroscedasticity ($p = 0.77$), supporting the validity of the regression estimates.

4. Discussion

This study evaluated the predictive value of the FSH/AMH ratio for ovarian response and assessed whether BMI modifies this relationship in infertile women undergoing controlled ovarian stimulation. Our findings demonstrate that the FSH/AMH ratio is a robust and independent predictor of oocyte yield, whereas BMI and its interaction with the ratio were not statistically significant. These results highlight the clinical utility of the FSH/AMH ratio as a composite endocrine marker that integrates both pituitary function and ovarian reserve, providing a more reliable indicator of ovarian responsiveness than either hormone alone, regardless of BMI.

The association between obesity and impaired fertility is well-established and multifaceted. Excess adipose tissue adversely affects ovulation, oocyte quality, and endometrial receptivity, contributing to suboptimal reproductive outcomes in women with elevated BMI [2,30]. Emerging evidence suggests that obesity disrupts the hypothalamic-pituitary-ovarian axis through alterations in insulin signaling, steroidogenesis, and the secretion of bioactive adipokines, thereby impairing ovarian function and responsiveness to gonadotropins. In this study, we observed numerically higher FSH/AMH ratios and lower oocyte yields among women in higher BMI categories, aligning with previous reports of diminished ovarian responsiveness in obese patients [2].

Although BMI is often cited as a determinant of reproductive outcomes, our results suggest that, among women with a preserved ovarian reserve and within a moderate BMI range, BMI alone does not significantly influence AMH, FSH, or their ratio. This finding aligns with prior reports indicating that AMH remains a consistent predictor of ovarian response and is relatively unaffected by BMI [27]. In contrast, FSH tends to exhibit greater variability

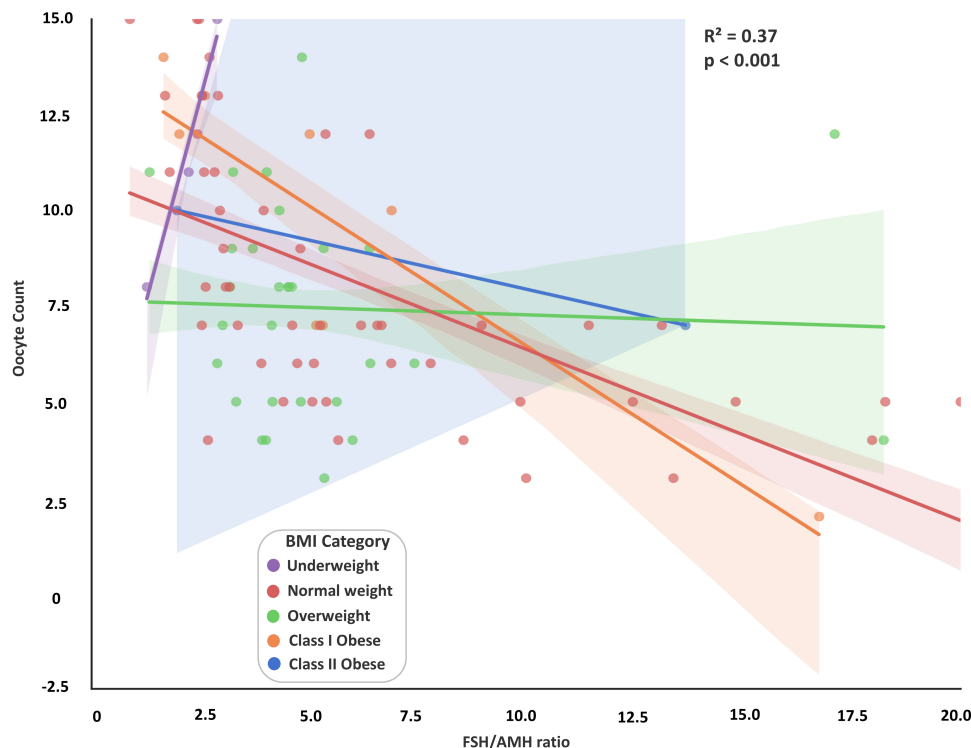


Fig. 3. Relationship between FSH/AMH ratio and oocyte count across BMI categories. A scatterplot illustrates the negative association between the FSH/AMH ratio and the number of retrieved oocytes, stratified by BMI categories: underweight ($<18.50 \text{ kg/m}^2$), normal weight ($18.50\text{--}24.90 \text{ kg/m}^2$), overweight ($25.00\text{--}29.90 \text{ kg/m}^2$), class I obese ($30.00\text{--}34.90 \text{ kg/m}^2$), and class II obese ($35.00\text{--}39.90 \text{ kg/m}^2$). Each dot represents an individual participant, and the shaded areas indicate 95% confidence intervals for the regression lines.

and is less reliable when considered in isolation [18]. The FSH/AMH ratio has emerged as a more integrative marker, combining pituitary activity (FSH) with ovarian follicular output (AMH). In this study, a higher FSH/AMH ratio was strongly correlated with reduced oocyte yield, confirming previous observations that elevated ratios are associated with diminished follicular development and an increased risk of cycle cancellation [28,29].

The FSH/AMH ratio may be particularly valuable in clinical scenarios where individual hormone levels are borderline or contradictory. Prior research has demonstrated that this ratio exhibits higher sensitivity and specificity for predicting poor ovarian response compared to AMH or FSH alone [27,28]. Evidence from animal models of diet-induced obesity supports these findings, showing that elevated FSH/AMH ratios are associated with reduced follicle counts and impaired ovarian function [29]. Similarly, in our cohort, higher ratios were negatively correlated with oocyte yield across all BMI groups, suggesting that this composite index effectively captures subtle disturbances in ovarian function that may not be evident from individual hormone levels.

Although AMH is widely regarded as a marker of ovarian reserve, it is influenced by dynamic intra-ovarian processes. AMH secretion begins during the transition from

primordial to primary follicles and peaks at the antral stage. Its expression is regulated by intra-ovarian factors and may be modulated by FSH [31]. A high FSH/AMH ratio may therefore reflect both a diminished follicular pool and a compensatory increase in pituitary output, indicating accelerated ovarian reserve depletion [18]. Furthermore, obesity has been reported to reduce AMH production through impaired granulosa cell function and decreased FSH receptor expression [22]. In our study, patients with both higher BMI and elevated FSH/AMH ratios exhibited the lowest oocyte counts, suggesting that adiposity may exacerbate hormonal imbalance and compromise ovarian responsiveness.

The majority of evidence supports that women with elevated FSH and low AMH levels have reduced oocyte yield and possibly lower oocyte quality, highlighting the importance of the interplay between these hormones [18,32,33]. Consistent with this, we observed lower oocyte numbers in patients with higher FSH/AMH ratios, regardless of BMI. While no significant interaction between BMI and the ratio was identified, the combination of elevated BMI and high FSH/AMH ratio identified a subgroup at greater risk for poor ovarian response.

Our findings highlight the potential clinical utility of the FSH/AMH ratio as a reliable predictor of ovarian re-

sponse, independent of BMI. This marker may aid clinicians in optimizing patient counseling and stimulation protocols, especially in women with borderline ovarian reserve. Although BMI did not significantly influence hormonal parameters in this study, its potential role in modulating ovarian sensitivity warrants continued consideration during fertility assessment. It is important to note that our cohort included women with BMI values ranging from 17 to 39.9 kg/m², and therefore, the results may not be generalizable to individuals with class III obesity (BMI ≥40 kg/m²).

Limitations

Several limitations must be acknowledged. First, although this study focused on hormonal indicators of ovarian reserve, we recognize that metabolic factors—particularly insulin resistance and adipokine dysregulation—play a critical role in mediating obesity-related reproductive dysfunction. However, key metabolic parameters such as fasting insulin, leptin levels, and homeostasis model assessment of insulin resistance (HOMA-IR) scores were not available in our dataset. This absence restricted our ability to explore the mechanistic pathways underlying the observed associations. Second, the sample size was relatively small, and the representation of women at BMI extremes was uneven. In particular, both the underweight and class II obese groups included only three participants each, which may have contributed to statistical variability and limited our ability to draw definitive conclusions for these subgroups. This imbalance also affects the generalizability of our findings, as the associations observed in mid-range BMI categories may not fully reflect outcomes in populations at the extremes. Interestingly, class II obese women in our study demonstrated the highest median oocyte yield despite having the highest FSH/AMH ratio, a finding likely attributable to sampling variation within the small subgroup. This observation should be interpreted with caution. Lastly, while this study focused on oocyte yield as the primary outcome, we acknowledge that it represents only one component of ART success. Data on additional endpoints such as fertilization rates, embryo quality, and clinical pregnancy outcomes were not consistently available and therefore could not be analyzed.

Future research should include larger, multicenter prospective studies with more diverse populations to validate the predictive value of the FSH/AMH ratio across all BMI categories. Additionally, incorporating BMI-specific thresholds and including key metabolic markers—such as leptin, fasting insulin, and HOMA-IR—could improve the clinical applicability of this ratio. Given the increasing use of the FSH/AMH ratio in fertility decision-making, future studies should also investigate whether BMI-adjusted cutoffs could enhance its predictive performance and support individualized treatment planning. By accounting for both hormonal and metabolic factors, such composite

models may provide a more comprehensive understanding of how adiposity affects reproductive outcomes and help guide more personalized, evidence-based fertility treatment strategies.

5. Conclusions

This study demonstrates that the FSH/AMH ratio is a valuable and BMI-independent predictor of oocyte yield in women undergoing controlled ovarian stimulation. While AMH and FSH are individually informative markers of ovarian reserve, their ratio provides a more integrated assessment of reproductive potential. Our findings confirm that higher FSH/AMH ratios are significantly associated with lower oocyte counts, supporting its clinical utility in identifying patients at risk for suboptimal ovarian response—particularly when standard markers yield inconclusive results.

Although BMI is a well-recognized factor influencing reproductive outcomes, it did not significantly impact hormonal profiles or the FSH/AMH ratio in this study. However, the combination of elevated BMI and a high FSH/AMH ratio was associated with a more pronounced reduction in oocyte yield, highlighting the importance of evaluating both endocrine and metabolic parameters during fertility assessment.

Given the rising prevalence of being overweight or obese among women of reproductive age, the FSH/AMH ratio may serve as a practical and reliable marker across BMI categories. Further prospective studies in larger, more diverse cohorts are warranted to confirm its clinical applicability and to explore whether BMI-adjusted thresholds could enhance its predictive performance.

Abbreviations

AMH, Anti-Müllerian hormone; ART, Assisted Reproductive Technology; AFC, Antral Follicle Count; BMI, Body Mass Index; FSH, Follicle-Stimulating Hormone; GnRH, Gonadotropin-Releasing Hormone; HPG, Hypothalamic-Pituitary-Gonadal; LH, Luteinizing Hormone; PCOS, Polycystic Ovary Syndrome; TSH, Thyroid-Stimulating Hormone; WHO, World Health Organization.

Availability of Data and Materials

The raw data supporting the conclusions of this article will be made available by the authors on request.

Author Contributions

EK and EGA designed the research study, validated the data and results. EK developed the methodology, performed the software work, conducted the investigation, curated the data, and carried out the formal analysis and visualization. EGA provided resources. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final version of the manuscript.

Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board of Acibadem Mehmet Ali Aydinlar University School of Medicine (protocol code 2023-10/427). Written informed consent was obtained from all participants prior to inclusion in the study.

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Conflict of Interest

The authors declare no conflict of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work the authors used ChatGpt-4.0 in order to check spell and grammar. After using this tool, the authors reviewed and edited the content as needed and takes full responsibility for the content of the publication.

References

- [1] Barbouni K, Jotautis V, Metallinou D, Diamanti A, Orovou E, Liepinaitienė A, *et al.* When Weight Matters: How Obesity Impacts Reproductive Health and Pregnancy-A Systematic Review. *Current Obesity Reports*. 2025; 14: 37. <https://doi.org/10.1007/s13679-025-00629-9>.
- [2] Broughton DE, Moley KH. Obesity and female infertility: potential mediators of obesity's impact. *Fertility and Sterility*. 2017; 107: 840–847. <https://doi.org/10.1016/j.fertnstert.2017.01.017>.
- [3] Turner F, Powell SG, Al-Lamee H, Gadhvi A, Palmer E, Drakeley A, *et al.* Impact of BMI on fertility in an otherwise healthy population: a systematic review and meta-analysis. *BMJ Open*. 2024; 14: e082123. <https://doi.org/10.1136/bmjopen-2023-082123>.
- [4] van der Steeg JW, Steures P, Eijkemans MJC, Habbema JDF, Hompes PGA, Burggraaff JM, *et al.* Obesity affects spontaneous pregnancy chances in subfertile, ovulatory women. *Human Reproduction*. 2008; 23: 324–328. <https://doi.org/10.1093/humrep/dem371>.
- [5] Rich-Edwards JW, Spiegelman D, Garland M, Hertzmark E, Hunter DJ, Colditz GA, *et al.* Physical activity, body mass index, and ovulatory disorder infertility. *Epidemiology*. 2002; 13: 184–190. <https://doi.org/10.1097/00001648-200203000-00013>.
- [6] Pasquali R, Patton L, Gambineri A. Obesity and infertility. *Current Opinion in Endocrinology, Diabetes, and Obesity*. 2007; 14: 482–487. <https://doi.org/10.1097/MED.0b013e3282f1d6cb>.
- [7] Zheng L, Yang L, Guo Z, Yao N, Zhang S, Pu P. Obesity and its impact on female reproductive health: Unraveling the connections. *Frontiers in Endocrinology*. 2024; 14: 1326546. <https://doi.org/10.3389/fendo.2023.1326546>.
- [8] Zaadstra BM, Seidell JC, Van Noord PA, te Velde ER, Habbema JD, Vrieswijk B, *et al.* Fat and female fecundity: prospective study of effect of body fat distribution on conception rates. *BMJ (Clinical Research Ed.)*. 1993; 306: 484–487. <https://doi.org/10.1136/bmj.306.6876.484>.
- [9] Lai J, Li X, Liu Z, Liao Y, Xiao Z, Wei Y, *et al.* Association between Waist-Hip Ratio and Female Infertility in the United States: Data from National Health and Nutrition Examination Survey 2017–2020. *Obesity Facts*. 2024; 17: 445–458. <https://doi.org/10.1159/000538974>.
- [10] Morán C, Hernández E, Ruiz JE, Fonseca ME, Bermúdez JA, Zárate A. Upper body obesity and hyperinsulinemia are associated with anovulation. *Gynecologic and Obstetric Investigation*. 1999; 47: 1–5. <https://doi.org/10.1159/000010052>.
- [11] Blüher M. Clinical relevance of adipokines. *Diabetes & Metabolism Journal*. 2012; 36: 317–327. <https://doi.org/10.4093/dmj.2012.36.5.317>.
- [12] Dardeno TA, Chou SH, Moon HS, Chamberland JP, Fiorenza CG, Mantzoros CS. Leptin in human physiology and therapeutics. *Frontiers in Neuroendocrinology*. 2010; 31: 377–393. <https://doi.org/10.1016/j.yfrne.2010.06.002>.
- [13] Moschos S, Chan JL, Mantzoros CS. Leptin and reproduction: A review. *Fertility and Sterility*. 2002; 77: 433–444. [https://doi.org/10.1016/s0015-0282\(01\)03010-2](https://doi.org/10.1016/s0015-0282(01)03010-2).
- [14] Chehab FF, Lim ME, Lu R. Correction of the sterility defect in homozygous obese female mice by treatment with the human recombinant leptin. *Nature Genetics*. 1996; 12: 318–320. <https://doi.org/10.1038/ng0396-318>.
- [15] Licinio J, Caglayan S, Ozata M, Yildiz BO, de Miranda PB, O'Kirwan F, *et al.* Phenotypic effects of leptin replacement on morbid obesity, diabetes mellitus, hypogonadism, and behavior in leptin-deficient adults. *Proceedings of the National Academy of Sciences of the United States of America*. 2004; 101: 4531–4536. <https://doi.org/10.1073/pnas.0308767101>.
- [16] Myers MG, Jr, Leibel RL, Seeley RJ, Schwartz MW. Obesity and leptin resistance: distinguishing cause from effect. *Trends in Endocrinology and Metabolism*. 2010; 21: 643–651. <https://doi.org/10.1016/j.tem.2010.08.002>.
- [17] Ryan NK, Van der Hoek KH, Robertson SA, Norman RJ. Leptin and leptin receptor expression in the rat ovary. *Endocrinology*. 2003; 144: 5006–5013. <https://doi.org/10.1210/en.2003-0584>.
- [18] Broer SL, Broekmans FJM, Laven JSE, Fauser BCJM. Anti-Müllerian hormone: ovarian reserve testing and its potential clinical implications. *Human Reproduction Update*. 2014; 20: 688–701. <https://doi.org/10.1093/humupd/dmu020>.
- [19] Taneja C, Gera S, Kim SM, Iqbal J, Yuen T, Zaidi M. FSH-metabolic circuitry and menopause. *Journal of Molecular Endocrinology*. 2019; 63: R73–R80. <https://doi.org/10.1530/JME-19-0152>.
- [20] Eng PC, Phylactou M, Qayum A, Woods C, Lee H, Aziz S, *et al.* Obesity-Related Hypogonadism in Women. *Endocrine Reviews*. 2024; 45: 171–189. <https://doi.org/10.1210/edrv/bnad027>.
- [21] Moslehi N, Shab-Bidar S, Ramezani Tehrani F, Mirmiran P, Azizi F. Is ovarian reserve associated with body mass index and obesity in reproductive aged women? A meta-analysis. *Menopause*. 2018; 25: 1046–1055. <https://doi.org/10.1097/GME.0000000000001116>.
- [22] Merhi Z, Buyuk E, Berger DS, Zapantis A, Israel DD, Chua S, Jr, *et al.* Leptin suppresses anti-Müllerian hormone gene expression through the JAK2/STAT3 pathway in luteinized granulosa cells of women undergoing IVF. *Human Reproduction*. 2013; 28: 1661–1669. <https://doi.org/10.1093/humrep/det072>.

- [23] Nardo LG, Yates AP, Roberts SA, Pemberton P, Laing I. The relationships between AMH, androgens, insulin resistance and basal ovarian follicular status in non-obese subfertile women with and without polycystic ovary syndrome. *Human Reproduction*. 2009; 24: 2917–2923. <https://doi.org/10.1093/humrep/dep225>.
- [24] De Pergola G, Maldera S, Tartagni M, Pannacciulli N, Loverro G, Giorgino R. Inhibitory effect of obesity on gonadotropin, estradiol, and inhibin B levels in fertile women. *Obesity*. 2006; 14: 1954–1960. <https://doi.org/10.1038/oby.2006.228>.
- [25] Silvestris E, de Pergola G, Rosania R, Loverro G. Obesity as disruptor of the female fertility. *Reproductive Biology and Endocrinology*. 2018; 16: 22. <https://doi.org/10.1186/s12958-018-0336-z>.
- [26] Kim Y, Lee E, Song JY, Kim Y, Lee S. Association between environmental pollutants and the FSH/AMH ratio as a marker of ovarian reserve. *Environmental Analysis, Health and Toxicology*. 2022; 37: e2022029. <https://doi.org/10.5620/eaht.2022029>.
- [27] Buyuk E, Seifer DB, Illions E, Grazi RV, Lieman H, Gindoff P. Elevated basal FSH/AMH ratio is associated with poor ovarian reserve. *Reproductive Biology and Endocrinology*. 2011; 9: 32. <https://doi.org/10.1186/1477-7827-9-32>.
- [28] Gözükar İ, Yılmaz N, Ceran MU, Atalay E, Kahyaoglu İ, Gülerman HC, *et al.* The role of FSH to AMH ratio in poor prognosis patients undergoing ICSI cycle. *Journal of the Turkish German Gynecological Association*. 2022; 23: 184–189. <https://doi.org/10.4274/jtgga.galenos.2022.2021-9-19>.
- [29] Roberts JA, Carpenter RM, Blythe SN, Toporikova N. FSH/AMH Ratio and Adipocyte Size are Linked to Ovarian Dysfunction. *Endocrine Research*. 2020; 45: 174–189. <https://doi.org/10.1080/07435800.2020.1721015>.
- [30] Pasquali R, Pelusi C, Genghini S, Cacciari M, Gambineri A. Obesity and reproductive disorders in women. *Human Reproduction Update*. 2003; 9: 359–372. <https://doi.org/10.1093/humupd/dmg024>.
- [31] Moolhuijsen LME, Visser JA. Anti-Müllerian Hormone and Ovarian Reserve: Update on Assessing Ovarian Function. *The Journal of Clinical Endocrinology and Metabolism*. 2020; 105: 3361–3373. <https://doi.org/10.1210/clinem/dgaa513>.
- [32] Lekamge DN, Barry M, Kolo M, Lane M, Gilchrist RB, Tremellen KP. Anti-Müllerian hormone as a predictor of IVF outcome. *Reproductive Biomedicine Online*. 2007; 14: 602–610. [https://doi.org/10.1016/s1472-6483\(10\)61053-x](https://doi.org/10.1016/s1472-6483(10)61053-x).
- [33] Scantamburlo VM, Linsingen RV, Centa LJR, Toso KFD, Scaraboto D, Araujo Júnior E, *et al.* Association between decreased ovarian reserve and poor oocyte quality. *Obstetrics & Gynecology Science*. 2021; 64: 532–539. <https://doi.org/10.5468/ogs.20168>.