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Adipokine Physiology and its Role in Female Obesity-Related Infertility

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Annotation: Background; Adipokines are bioactive molecules secreted by adipose tissue that play a critical role in regulating metabolic and reproductive functions. In obese females, dysregulated adipokine levels can impair ovarian function, disrupt hormonal balance, and contribute to infertility. **Aims of the study;** To investigate the physiological roles of adipokines and evaluate their impact on hormonal regulation and reproductive function in obese females with infertility. **Methodology;** This case-control study (Jan–May 2024) investigated adipokines in female obesity-related infertility. It included 150 obese infertile women and 50 healthy controls (aged 20–40) from fertility clinics in Nasiriyah, Iraq. Exclusion criteria ruled out PCOS, thyroid disorders, diabetes, and recent hormonal therapy. Blood samples were collected, processed, and stored at –20°C. Serum adipokines (leptin, adiponectin, resistin, visfatin) were measured by ELISA. Reproductive hormones (FSH, LH, estradiol, progesterone) were analyzed using the Cobas e411 analyzer (Roche Diagnostics, Germany). **Result;** The study showed significant differences in BMI, marriage duration, education level, adipokine levels, and reproductive hormones between obese infertile women and healthy controls. Leptin, resistin, and visfatin were elevated, while adiponectin, estradiol, and

progesterone were reduced in patients. Strong correlations were found between adipokines, BMI, and reproductive hormones, suggesting that altered adipokine profiles in obesity negatively affect the hypothalamic-pituitary-ovarian axis, contributing to hormonal imbalance, ovulatory dysfunction, and infertility in women. **Conclusions**; Adipokines significantly impact female obesity-related infertility by altering reproductive hormone levels. Elevated leptin, resistin, and visfatin, alongside reduced adiponectin, disrupt the hypothalamic-pituitary-ovarian axis, impairing ovulation. These imbalances reflect adipose tissue's role as an endocrine disruptor.

Keywords: Adipokines, Obesity, Female Infertility, Reproductive Hormones, Leptin, Adiponectin.

Introduction:

Obesity has become a worldwide health epidemic affecting millions of people, and it has significant implications for female reproductive health. One of the multitudes of effects of obesity is its profound relationship with infertility, a disease that affects around 10–15% of couples worldwide and causes significant psychosocial and financial problems [1]. Obesity-related female infertility is a heterogeneous and multifactorial syndrome involving abnormalities in the endocrine, metabolic, immune and inflammatory pathways. The adipocyte tissue, being an endocrinic organ (the largest endocrinic organ) had also been recognized a few years ago as an important mechanism linking high fat mass to a series of reproductive problems [2,3].

Traditionally, adipose tissue was regarded as a passive storehouse for energy, and however, it is well established that adipose tissue is an active endocrine organ, which generates a number of adipokines such as leptin, adiponectin, resistin, and visfatin [4]. They have significant effects on energy balance, insulin sensitivity, inflammation and reproductive physiology [5]. In obesity, disorganized secretion and circulating levels of adipokines are involved in the development of low-grade inflammation and metabolic dysfunction with potentially negative effects on ovarian function and fertility [6].

Leptin, one of the most well-studied adipokines, is involved in the control of energy balance and reproduction. It reports nutritional status to the hypothalamus and acts to alter the function of the hypothalamic-pituitary-gonadal (HPG) axis to affect gonadotropin secretion and ovulation [7]. On the other hand, leptin is indispensable for pubertal maturation and the onset of reproductive capacity but hyperleptinemia in obesity results in leptin resistance, making its regulatory role ineffective and predisposing to anovulation and infertility [8]. Hyperleptimia has even been reported in obese infertile women, suggesting its involvement in obesity-related reproductive abnormalities [9].

Also, adiponectin is an insulin-sensitizing and anti-inflammatory adipokine which is paradoxically reduced in obesity. Reduced adiponectin levels also appear to contribute to the insulin resistance seen in obesity, which is commonly associated with ovulatory dysfunction, and polycystic ovary

syndrome (PCOS) which is one of the common causes of female infertility [10]. Lower levels of adiponectin could further aggravate ovarian functioning by increasing androgen level and arrest of folliculogenesis [11,12].

Resistin and visfatin, relatively new among the adipokines, have also attracted the attention as regards their relationship to obesity and reproduction. Resistin is involved in inflammation and its relation to insulin resistance might disturb ovarian steroidogenesis and folliculogenesis [13]. Visfatin, also known as nicotinamide phosphoribosyltransferase, affects glucose metabolism and has been linked with disrupted ovarian function and infertility in obese women [14,15].

The intricate interaction between adipokines and reproductive hormones implies that adipose tissue-derived signals may regulate the hypothalamic-pituitary-ovarian axis, ovarian steroidogenesis, and follicular environment, and hence influence fertility. pro-inflammatory adipokines, together with reduced anti-inflammatory adipokines, could cause local ovarian inflammation, oxidative stress and hormonal dysregulation [16,17].

Knowledge about the physiological function of adipokines and their abnormal secretion in obesity is important to deduce therapeutic targets to alleviate obesity-associated infertility. Existing types of treatments, which range from lifestyle change to drug intervention, are effective only in a limited number of cases, demonstrating the importance to find new strategies based on the molecular peculiarities of adipokine action [18,19].

This study purposed to clarify the changes of adipokine profiles in obese infertile women versus their healthy counterparts and to evaluate the relationships between adipokine levels and reproductive hormones. We hope that learning more about the pathology of adipokine physiology and its effects on female reproductive health will aid in the development of better tools and treatments for these women.

Methodology:

This case-control study was conducted from January 2024 to May 2024 to investigate the role of adipokines in female obesity-related infertility. A total of 200 women aged between 20 and 40 years were enrolled, including 150 obese infertile patients and 50 healthy fertile women as controls. Participants were recruited from fertility clinics in Nasiriyah, Iraq. Inclusion criteria involved women aged 20–40 years with a BMI ≥ 30 kg/m² and a diagnosis of primary infertility for at least one year. Exclusion criteria included secondary infertility, presence of polycystic ovary syndrome (PCOS), thyroid disorders, diabetes mellitus, hormonal therapy within the past six months, and smoking or alcohol use. Ethical approval was obtained from the Scientific and Ethical Committee of Mazaya University College (Approval No.: MUC-2024-OBI-FIN-07), and written informed consent was secured from all participants before blood collection. A total of 5 mL of venous blood was collected from each subject using a sterile syringe and transferred into plain tubes. Samples were left to clot at room temperature for 30 minutes, followed by centrifugation at 3000 rpm for 10 minutes. The resulting serum was separated and stored at –20°C until analysis. Serum levels of adipokines, including leptin, adiponectin, resistin, and visfatin, were measured using enzyme-linked immunosorbent assay (ELISA) kits provided by Elabscience Biotechnology Co., Ltd., Wuhan, China, following the manufacturer's instructions. Reproductive hormones—FSH, LH, estradiol, and progesterone—were analyzed using the Cobas e411 automated analyzer (Roche Diagnostics, Germany) based on electrochemiluminescence immunoassay (ECLIA) technology.

Statistical analysis:

Quantitative data were analyzed using SPSS v26 and presented as frequencies and percentages. Normal variables were tested with two-tailed t-tests, while non-normal variables were analyzed using Mann-Whitney U, Wilcoxon, and Chi-square tests. A p-value < 0.05 indicated statistical significance.

Ethical approval:

The study was approved by the Human Ethics Committee of Al-Habboubi Teaching Hospital. All participants were informed about the study and provided written consent. Patient confidentiality was strictly maintained throughout the study.

Results

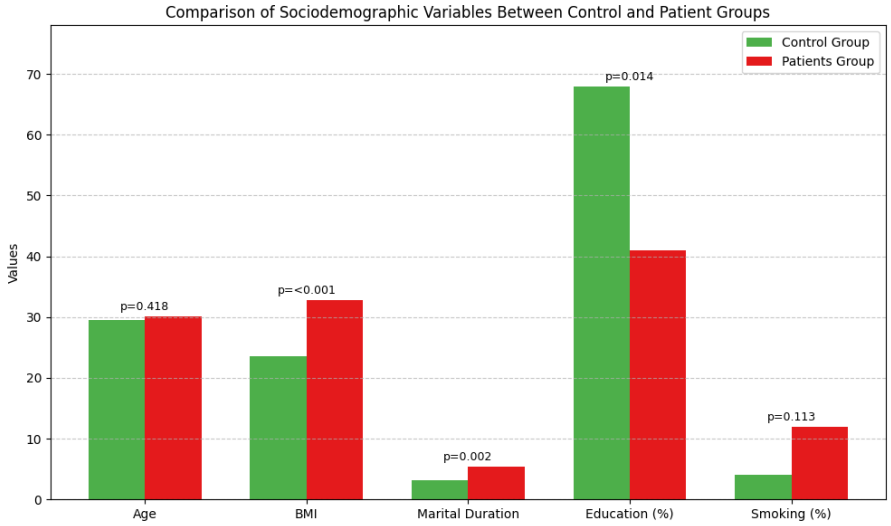
Comparison of Sociodemographic Characteristics Between Obese Infertile Patients and Healthy Controls

The results of the table showed statistically significant differences between women with obesity-related infertility and the control group of healthy women with regard to a number of sociodemographic characteristics. The age of the participants did not show a statistically significant difference between the two groups, as the mean age of the control group was 29.45 ± 4.87 years, compared to 30.12 ± 5.23 years in the patient group (P=0.418). In contrast, a significant increase in the body mass index (BMI) was observed in the patient group, with a mean of 32.78 ± 4.11 kg/m², compared to the control group, which recorded a mean of 23.56 ± 2.91 kg/m² (P<0.001). The results also showed that the duration of marriage was longer in the patient group (5.4 ± 2.3 years) compared to the control group (3.2 ± 1.1 years), with a statistically significant difference (P=0.002). On the other hand, the percentage of higher education was higher in the control group (68%) compared to the patient group (41%), with a statistically significant difference (P=0.014). Regarding smoking, the percentage was higher in the patient group (12%) compared to the control group (4%), but the difference was not statistically significant (P=0.113).

Table 1: Distribution of Age, BMI, Marital Duration, Education Level, and Smoking Status Among Study Groups

Variable	Control Group (n=50)	Patients Group (n=150)	P-value
Age (years) Mean ± SD	29.45 ± 4.87	30.12 ± 5.23	0.418
BMI (kg/m²) Mean ± SD	23.56 ± 2.91	32.78 ± 4.11	<0.001
Marital Duration (years)	3.2 ± 1.1	5.4 ± 2.3	0.002
Education Level – Higher (%)	68%	41%	0.014
Smoking Status – Yes (%)	4%	12%	0.113

P-values < 0.05 were considered statistically significant.

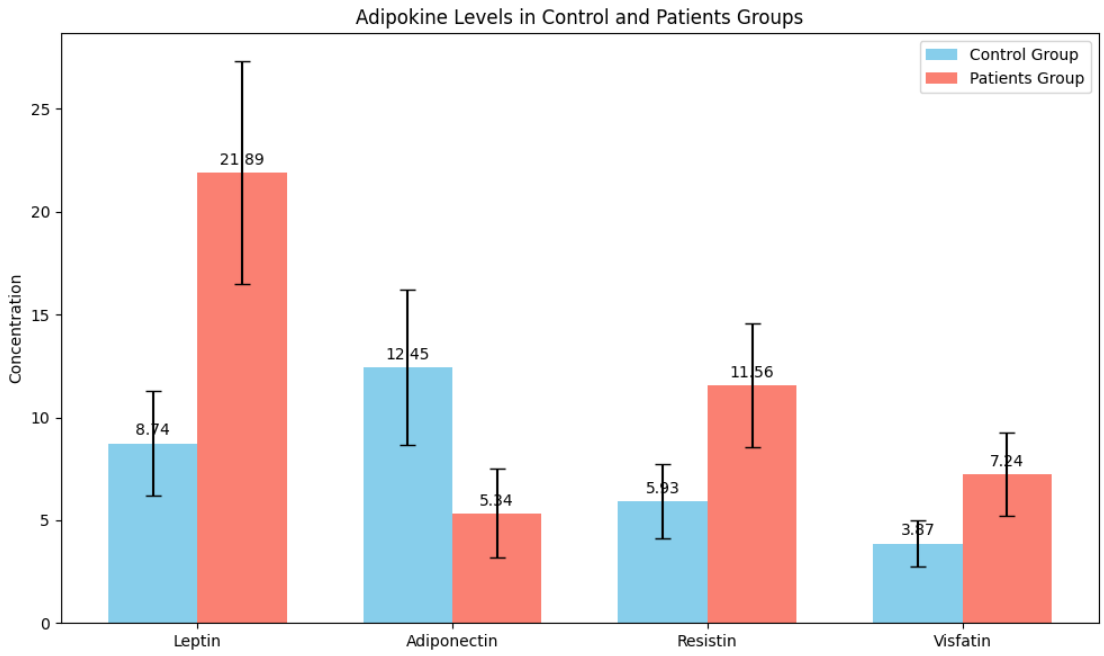


Comparison of Serum Adipokine Levels Between Obese Infertile Women and Healthy Controls

The study results showed highly statistically significant differences in adipokin levels between women with obesity-related infertility and the control group of healthy women. Leptin levels increased significantly in the patient group (21.89 ± 5.42 ng/ml) compared to the control group (8.74 ± 2.53 ng/ml), while adiponectin levels decreased in the patient group (5.34 ± 2.17 µg/ml) compared to (12.45 ± 3.78 µg/ml) in the healthy group. Both differences were highly statistically significant (P<0.001). Similarly, the patient group recorded a significant increase in resistin and visfatin levels, reaching (11.56 ± 3.01) and (7.24 ± 2.03) ng/ml, respectively, compared to (5.93 ± 1.82) and (3.87 ± 1.11) ng/ml in the control group, with clear statistical differences (P<0.001). These results indicate a pivotal role for adipokines in causing or contributing to obesity-related infertility in women.

Table 2: Mean Concentrations of Leptin, Adiponectin, Resistin, and Visfatin in Study Groups

Adipokine	Control Group Mean ± SD	Patients Group Mean ± SD	P-value
Leptin (ng/mL)	8.74 ± 2.53	21.89 ± 5.42	<0.001
Adiponectin (µg/mL)	12.45 ± 3.78	5.34 ± 2.17	<0.001
Resistin (ng/mL)	5.93 ± 1.82	11.56 ± 3.01	<0.001
Visfatin (ng/mL)	3.87 ± 1.11	7.24 ± 2.03	<0.001



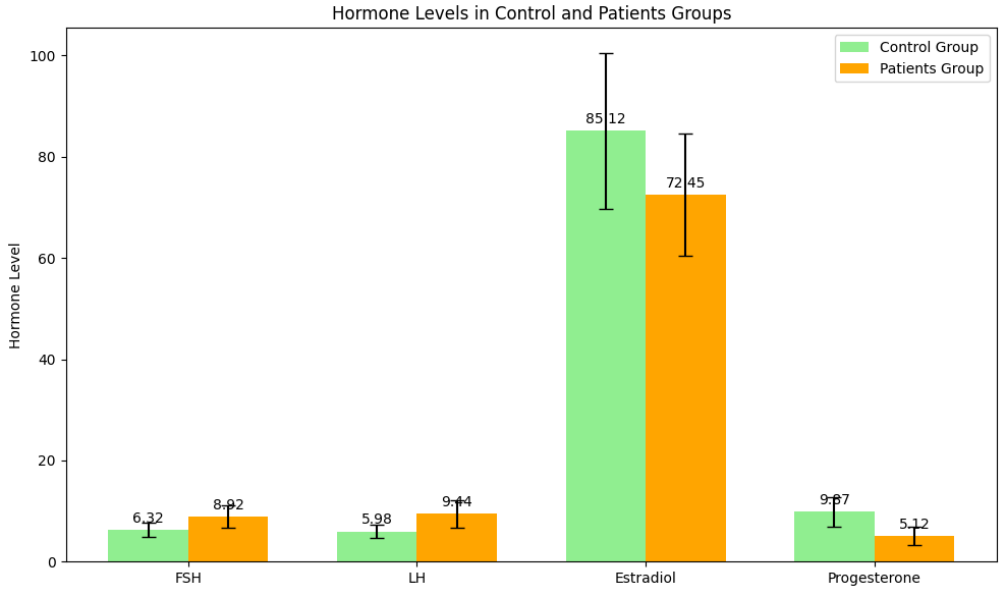
Comparison of Reproductive Hormone Levels Between Obese Infertile Women and Healthy Controls

Results of hormonal analysis showed there were significant differences between women with obesity related infertile and healthy women in the levels of reproductive hormones. Patients manifest more FSH [(8.92 ± 2.21 mIU/mL) than do the controls (6.32 ± 1.45 mIU/mL] and more LH (9.44 ± 2.66 mIU/mL), in contrast to 5.98 ± 1.32 mIU/mL in the control group. These differences were statistically very significant (P<0.001). On the contrary, there was a marked reduction in the levels of estradiol in patient group that decreased to (72.45 ± 12.08 pg/mL) compared to control group which decreased to (85.12 ± 15.32 pg/mL) (P=0.005). Progesterone level was also significantly lower in the patient group (5.12 ± 1.88 ng/mL) compared to control

(9.87 ± 2.91 ng/mL) with highly statistical difference (P<0.001). These findings are suggestive of a role of obesity in disrupting the reproductive hormonal axis, leading to ovulatory dysfunction and infertility in women.

Table 3: Serum Concentrations of FSH, LH, Estradiol, and Progesterone in Study Groups

Hormone	Control Group Mean ± SD	Patients Group Mean ± SD	P-value
FSH (mIU/mL)	6.32 ± 1.45	8.92 ± 2.21	<0.001
LH (mIU/mL)	5.98 ± 1.32	9.44 ± 2.66	<0.001
Estradiol (pg/mL)	85.12 ± 15.32	72.45 ± 12.08	0.005
Progesterone (ng/mL)	9.87 ± 2.91	5.12 ± 1.88	<0.001

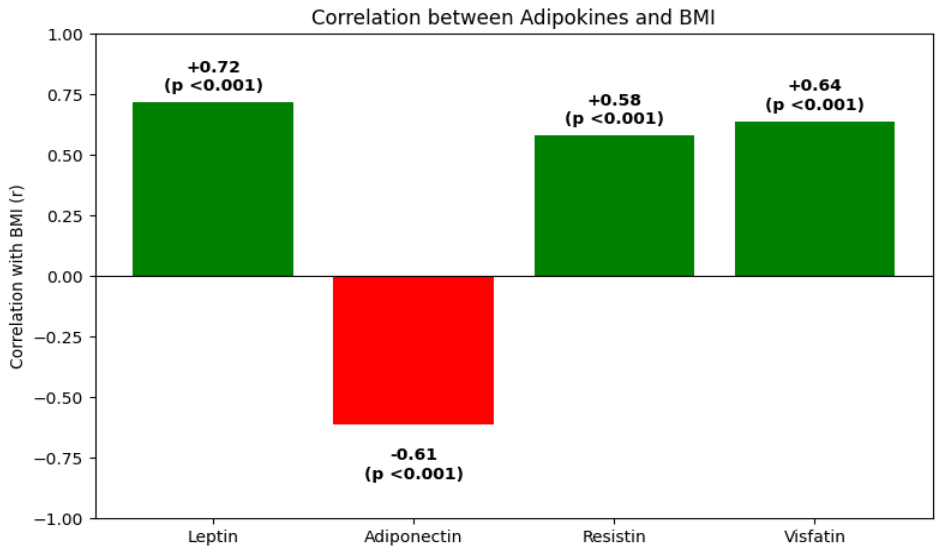


Correlation Between Serum Adipokine Levels and Body Mass Index (BMI) in Obese Infertile Women

The findings of the study of relation of BMI (body mass index) with adipokines indicated that some of the adipokines had strong, significant correlations with obesity extents in infertile women. We found a very significant positive correlation between leptin with BMI($r = +0.72$), ($P < 0.001$) This means that with the increase in obesity there will be a very significant rise in the concentration of this Hormone. Visfatin and resistin close positive associations with BMI were observed, $r = +0.64$ and ($r = +0.58$) respectively; $P < 0.001$. Inversely, adiponectin presented a significant negative association with BMI ($r = –0.61$, $P < 0.001$), indicating that its levels decreased with increasing degrees of obesity. These findings indicate the role of adipokines in the regulation of obesity-associated physiological changes and the reproductive system.

Table 4: Association of Leptin, Adiponectin, Resistin, and Visfatin with BMI

Adipokine	Correlation with BMI (r)	P-value
Leptin	+0.72	<0.001
Adiponectin	-0.61	<0.001
Resistin	+0.58	<0.001
Visfatin	+0.64	<0.001

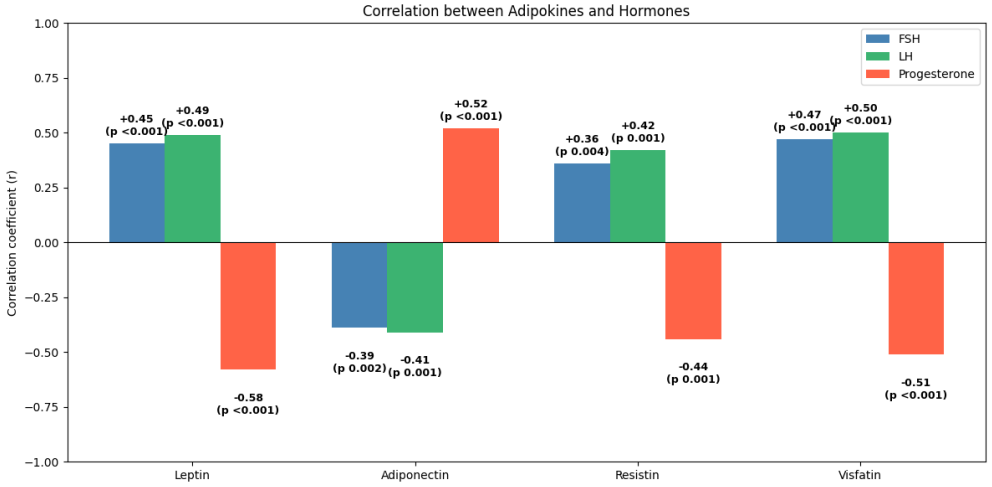


Correlation Between Serum Adipokines and Reproductive Hormones in Obese Infertile Women

Results Statistically significant relationships were found of adipokines levels with reproductive hormones in women with obesity related infertility. Moderate positive correlation was noted between leptin and FSH (r=+0.45, P<0.001) and LH (r=+0.49, P<0.001), and a strong inverse correlation was observed with progesterone (r=–0.58, P<0.001). Adiponectin exhibited a negative correlation with FSH (r=–0.39, P=0.002) and LH (r=–0.41, P=0.001), and a modest positive correlation with progesterone (r=+0.52, P<0.001). Resistin was also weak and moderately correlated with FSH and LH (r=+0.36 and +0.42, respectively, P<0.01), and it displayed an inverse correlation with progesterone (r=–0.44, P=0.001). Also, visfatin was significantly correlated with FSH (r=+0.47, P<0.001) and LH (r=+0.50, P<0.001) and moderately negatively correlated with progesterone levels (r=−0.51, P<0.001). These associations are indicative of the possible involvement of adipokines in the control of the hypothalamic-pituitary-ovarian axis and their adverse effect on ovarian function in obese women.

Table 5: Associations of Leptin, Adiponectin, Resistin, and Visfatin with FSH, LH, and Progesterone Levels

Adipokine	FSH (r)	LH (r)	Progesterone (r)	P-value (FSH)	P-value (LH)	P-value (Prog)
Leptin	+0.45	+0.49	-0.58	<0.001	<0.001	<0.001
Adiponectin	-0.39	-0.41	+0.52	0.002	0.001	<0.001
Resistin	+0.36	+0.42	-0.44	0.004	0.001	0.001
Visfatin	+0.47	+0.50	-0.51	<0.001	<0.001	<0.001



Discussion:

The present study investigated the role of adipokines in female obesity-related infertility by comparing serum levels of leptin, adiponectin, resistin, and visfatin, alongside reproductive hormone profiles, between obese infertile women and healthy controls. Our findings demonstrated significant alterations in adipokine levels and reproductive hormones in obese infertile women, corroborating the hypothesis that adipokines play a pivotal role in obesity-associated reproductive dysfunction.

Firstly, the markedly elevated leptin levels observed in obese infertile women compared to controls align with several previous studies. Leptin, primarily produced by adipose tissue, acts as a key regulator of energy balance and reproductive function. Elevated leptin levels have been linked to leptin resistance in obesity, which may disrupt hypothalamic-pituitary-ovarian (HPO) axis signaling and impair ovulation [20]. The findings of our study were consistent with an earlier study that also found higher levels of leptin in obese infertile females [21]. Others studies, however, have failed to find significant differences in leptin levels, possibly due to differences in population or assay [21]. These contradictions emphasize the complexity of the action of leptin and the effect of genetic background and the level of obesity.

On the contrary, the level of adiponectin was significantly lower in patient. Adiponectin, which has been inversely correlated with obesity, is an insulin-sensitizing and anti-inflammatory protein [22]. Lower adiponectin can lead to insulin resistance, associated with several reproductive problems including PCOS [23]. Our results agree with [23] who described lower adiponectin levels among obese infertile women and indicated that decreased production of adiponectin may worsen ovarian function. However, other studies have reported infertility status does not have an impact on adiponectin levels, this may be explained by varying BMI or metabolic comorbidity [24].

The observed elevations in resistin and visfatin levels among patients also support a growing body of evidence that implicates these adipokines in metabolic and reproductive abnormalities. Resistin, implicated in inflammation and insulin resistance, may further disrupt follicular development through pro-inflammatory pathways [25]. Visfatin, similarly, has been associated with metabolic dysregulation and altered ovarian function [8]. Our results agree with the findings of [26], who documented higher serum resistin and visfatin in obese infertile women. However, contradictory reports exist, where visfatin levels showed no correlation with infertility or BMI. Such inconsistencies may stem from sample size variations or differences in assay sensitivity [27].

Regarding reproductive hormones, the increased levels of FSH and LH and decreased progesterone and estradiol levels in obese infertile women are indicative of disrupted ovarian function and impaired luteal phase, consistent with the literature [28]. Elevated FSH and LH suggest altered gonadotropin regulation possibly mediated by adipokine-induced HPO axis dysfunction. The negative correlation between leptin and progesterone aligns with reports that leptin excess can impair corpus luteum function and progesterone synthesis [29]. Decreased estradiol levels may reflect follicular arrest or diminished aromatase activity in granulosa cells, a phenomenon observed in obesity [29]. Our findings concur with those of [30], but some studies report variable hormone profiles depending on infertility etiology and obesity severity [31].

Correlations between adipokines and BMI were consistent with the expected physiological trends. The strong positive correlation of leptin, resistin, and visfatin with BMI supports their role as obesity-related mediators of metabolic and reproductive dysfunction. The inverse correlation of adiponectin with BMI further confirms its protective role [32]. These relationships have been previously documented in both infertile and general obese populations [33]. The correlations between adipokines and reproductive hormones highlight the complex interplay influencing fertility; leptin’s positive correlation with gonadotropins and negative correlation with progesterone suggest a dual effect in stimulating gonadotropin secretion but impairing luteal function. Adiponectin’s inverse relationship with FSH and LH and positive association with progesterone support its role in promoting normal ovarian steroidogenesis [34].

Conclusion:

In Conclusion, our study adds to the growing evidence that adipokines are key modulators linking obesity to female infertility by altering metabolic and hormonal homeostasis. While the results largely agree with previous research, variations in adipokine profiles across studies highlight the influence of demographic, metabolic, and methodological factors. Future studies should focus on longitudinal designs and mechanistic investigations to better elucidate causality and therapeutic targets.

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