

Original article

Association of Body Mass Index with Inflammatory Biomarkers Among Women with Infertility

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ABSTRACT

Infertility affects a growing number of women worldwide, and obesity-associated chronic low-grade inflammation is increasingly recognized as a contributor to impaired female reproductive function. This study aimed to compare body mass index (BMI) and serum concentrations of the pro-inflammatory cytokines interleukin-6 (IL-6), interleukin-1 β (IL-1 β) and tumor necrosis factor-alpha (TNF- α) between obese women with secondary infertility and healthy fertile women. A case-control study was conducted at Al-Sadr Teaching Hospital and the Infertility Center Laboratories, Najaf Governorate, Iraq, from January to June 2025. Ninety-five married women aged 20–45 years were enrolled: 55 obese women (BMI ≥ 30 kg/m²) diagnosed with secondary infertility and 40 healthy fertile women of normal body weight (BMI 18.5–24.9 kg/m²) as controls. Women with pregnancy, acute infection, chronic inflammatory or autoimmune disease, malignancy, or use of medications affecting immune markers were excluded. Height and weight were measured with calibrated instruments to calculate BMI, and venous blood was collected under sterile conditions; serum IL-6, IL-1 β and TNF- α were quantified by enzyme-linked immunosorbent assay (ELISA). Groups were compared with the independent-samples t-test. Mean BMI was significantly higher in infertile women than in controls (31.2 ± 4.6 vs. 23.4 ± 2.8 kg/m²; $P < 0.001$), as expected from the selection criteria. Serum IL-6 (14.09 ± 4.00 vs. 0.82 ± 0.43 pg/mL), TNF- α (12.65 ± 4.33 vs. 0.26 ± 0.12 pg/mL) and IL-1 β (23.34 ± 5.58 vs. 2.06 ± 1.19 pg/mL) were all markedly elevated in the infertile group (all $P < 0.001$). Obesity-associated infertility is therefore accompanied by a pronounced systemic pro-inflammatory profile, supporting the hypothesis that chronic low-grade inflammation contributes to the pathophysiology of female infertility. Because the two groups differed in both adiposity and fertility status, the independent contribution of each factor could not be separated; nevertheless, measuring inflammatory biomarkers alongside BMI may add useful information to the clinical evaluation of infertile women, and improving metabolic health may help to improve reproductive outcomes.

Keywords: Body mass index; Female infertility; Inflammation; Interleukin-1 β ; Interleukin-6; Obesity; Tumor necrosis factor- α

1-INTRODUCTION

The prevalence of infertility has increased in recent years, affecting approximately 8–12% of couples of reproductive age worldwide (Vander Borgh & Wyns, 2018). The World Health Organization (WHO) defines infertility as the failure of a sexually active couple to achieve pregnancy after one year of regular unprotected intercourse (Zegers-Hochschild et al., 2009). Infertility affects not only women's reproductive health but also the psychological well-being of couples and their families (Inhorn & Patrizio, 2015), and roughly 40% of cases are attributable to female factors, including hormonal imbalance, anatomical abnormalities, genetic predisposition and lifestyle-related factors (Vander Borgh & Wyns, 2018). Increasing evidence indicates that several fertility-related disorders are associated with chronic inflammatory conditions (Ameho & Klutstein, 2025).

Obesity is projected to affect one in five women worldwide by 2030, making it one of the most important global public-health challenges. Beyond increasing the risk of chronic disease, obesity adversely affects female reproductive health and has been associated with menstrual irregularity, impaired or absent ovulation, early pregnancy loss and a higher incidence of pregnancy and childbirth complications (Wang et al., 2024; Zheng et al., 2023). Body mass index (BMI) is the most widely used anthropometric indicator of obesity, defined by the WHO as a BMI ≥ 30 kg/m² (Venkatesh et al., 2022). Elevated BMI is associated with cardiovascular disease, type 2 diabetes, metabolic disorders, several cancers and infertility; however, BMI alone may not fully capture the effect of obesity on fertility, because women with similar BMI values may differ substantially in metabolic status, body composition and fat distribution (Phillips, 2013; Jaswa et al., 2020).

The link between obesity and reproductive dysfunction is largely mediated by adipose tissue, which functions as an active endocrine and immune organ. With excess adiposity, particularly visceral fat, adipocyte hypertrophy, tis-

Due to hypoxia and infiltration of immune cells alter the local microenvironment, and adipose tissue becomes a chronic source of pro-inflammatory mediators (Lin et al., 2024). Adipocytes and infiltrating classically activated (M1) macrophages secrete increased amounts of tumor necrosis factor- α (TNF- α), interleukin-6 (IL-6) and interleukin-1 β (IL-1 β), while secretion of the anti-inflammatory, insulin-sensitizing adipokine adiponectin falls; this imbalance is considered a principal mechanism of obesity-related metabolic complications (Uribe-Querol & Rosales, 2022; Silvestris et al., 2018).

Chronic low-grade inflammation impairs the female reproductive system at several levels. Within the ovary, pro-inflammatory cytokines alter immune-cell activity, disturb follicular development and ovulation, and promote a pro-inflammatory ovarian microenvironment that is particularly evident in obese women and in polycystic ovary syndrome (PCOS), where inflammation is also linked to hyperandrogenism (He et al., 2020; Deng et al., 2024). TNF- α , IL-1 β and IL-6 act directly on granulosa cells, impairing their function and promoting apoptosis, thereby inhibiting follicular growth and maturation and disrupting ovulation (Chen et al., 2023; Duffy et al., 2019). IL-1 β and TNF- α also suppress gonadal steroidogenesis and interfere with gonadotropin-receptor signaling, and TNF- α concentrations in follicular fluid are higher in infertile than in fertile women (Akoum et al., 2008). IL-1 β further contributes to endometrial disorders, particularly endometriosis, where it is associated with nerve growth factor expression and dyspareunia (Peng et al., 2020), and elevated peritoneal IL-6 suppresses natural killer cell activity, facilitating the persistence of ectopic endometrial lesions (Velasco et al., 2010).

Obesity-associated inflammation also acts centrally. Adipose-tissue macrophages amplify systemic inflammation (Duan et al., 2023), and obesity increases blood–brain barrier permeability, allowing peripheral immune cells to reach the hypothalamus, where resident microglia are activated through Toll-like receptor 4 (TLR4) and nuclear factor-kappa B (NF- κ B) signaling in response to elevated circulating saturated fatty acids (Douglass et al., 2017; Cena et al., 2020). Obesity-induced hypothalamic inflammation contributes to menstrual and ovulatory dysfunction and has been implicated in PCOS, one of the leading causes of female infertility. Gonadotropin-releasing hormone (GnRH)-secreting neurons and kisspeptin neurons express receptors for interleukins, prostaglandins and TNF- α , and pro-inflammatory mediators, including TNF- α and bacterial lipopolysaccharide, reduce GnRH secretion and disrupt the hypothalamic–pituitary–ovarian (HPO) axis (Figure 1) (Ameho & Klutstein, 2025; Douglass et al., 2017).

Thus, excess adipose tissue promotes the continuous release of IL-6, TNF- α and IL-1 β , producing chronic low-grade systemic inflammation that affects ovarian function, oocyte maturation, implantation and central neuroendocrine control of reproduction. Integrating inflammatory assessment with metabolic and clinical evaluation may therefore improve understanding of the relationship between obesity and female infertility. The present study compared BMI and serum IL-6, IL-1 β and TNF- α between obese women with secondary infertility and healthy fertile women in Najaf, Iraq.

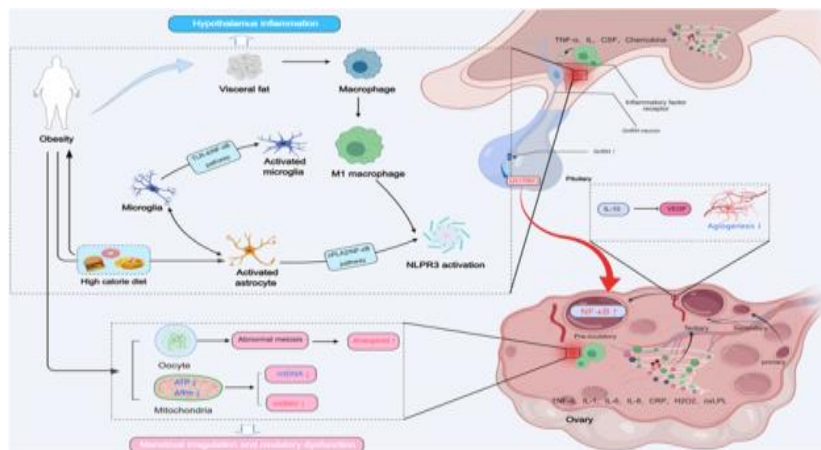


Figure 1: Obesity-associated inflammation and female infertility. Activation of inflammatory pathways in the hypothalamus impairs the function of GnRH-secreting neurons, while cytokines released by macrophages in visceral adipose tissue adversely affect the ovarian microenvironment, oocyte maturation, mitochondrial function and angiogenesis, reducing reproductive efficiency (adapted from Ameho & Klutstein, 2025).

2-MATERIALS AND METHODS

Study design and participants

This case-control study was conducted at Al-Sadr Teaching Hospital and the Infertility Center Laboratories in Najaf Governorate, Iraq, from January to June 2025. Ninety-five married women aged 20–45 years were enrolled. The patient group comprised 55 obese women ($\text{BMI} \geq 30 \text{ kg/m}^2$) with secondary infertility, defined as inability to conceive for at least 12 months of regular unprotected intercourse after a previous pregnancy and diagnosed by the

attending specialist at the Infertility Center. The control group comprised 40 healthy fertile married women of normal body weight (BMI 18.5–24.9 kg/m²) and comparable age. Women who were pregnant or had acute infection, chronic inflammatory disease, malignancy, confirmed autoimmune disease, or were receiving medications that could influence inflammatory or immunological markers were excluded.

Ethical approval

The study was approved by the ethics committee of the Infertility Center, Al-Sadr Teaching Hospital, Najaf, Iraq (approval No. [to be inserted], dated [to be inserted]). Written informed consent was obtained from all participants before sample collection, and all personal information was kept strictly confidential.

Measurement of body mass index

Body weight was measured to the nearest 0.1 kg on a calibrated electronic scale with participants in light clothing and without shoes, standing upright with weight evenly distributed on both feet. Height was measured with a calibrated stadiometer with participants barefoot, upright and looking straight ahead. BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m²); a BMI $\geq$ 30 kg/m² was classified as obese and 18.5–24.9 kg/m² as normal weight.

Sample collection and laboratory analysis

Approximately 5 mL of venous blood was collected from each participant under sterile conditions. Samples were centrifuged at 3000 rpm for 10 min, and the separated serum was stored at –20 °C until analysis, which was completed within three months of collection; samples were thawed only once. Serum IL-6, IL-1 β and TNF- α were measured with commercial ELISA kits (Elabscience Biotechnology Co., Ltd., Wuhan, China; catalogue numbers [to be inserted]) according to the manufacturer's instructions, using a microplate reader ([manufacturer, model, country]). Assay sensitivities and intra- and inter-assay coefficients of variation were as stated by the manufacturer. Laboratory personnel were not informed of the case or control status of the samples.

Statistical analysis

Data were analyzed with IBM SPSS Statistics version 28 (IBM Corp., Armonk, NY, USA). Continuous variables are expressed as mean $\pm$ standard deviation (SD). Normality was assessed with the Shapiro–Wilk test, and differences between the patient and control groups were analyzed with the independent-samples t-test. A P value < 0.05 was considered statistically significant.

3-RESULTS

Body mass index in fertile and infertile women

Mean BMI was significantly higher in infertile women (31.2 ± 4.6 kg/m²) than in the control group (23.4 ± 2.8 kg/m²; $P < 0.001$; Table 1). This difference reflects the selection of obese women for the patient group and of normal-weight women as controls, and confirms that the two groups were clearly separated with respect to adiposity.

Inflammatory biomarkers in fertile and infertile women

All three cytokines were markedly elevated in infertile women compared with controls (all $P < 0.001$; Table 1). Serum IL-6 rose from 0.82 ± 0.43 pg/mL in controls to 14.09 ± 4.00 pg/mL in infertile women, TNF- α from 0.26 ± 0.12 to 12.65 ± 4.33 pg/mL, and IL-1 β from 2.06 ± 1.19 to 23.34 ± 5.58 pg/mL. The coefficient of variation was highest for TNF- α (34.2%) and lowest for BMI (14.7%).

Table 1: Body mass index and serum inflammatory markers in healthy fertile women and obese women with secondary infertility.

Parameter	Control (n = 40) Mean $\pm$ SD	Patients (n = 55) Mean $\pm$ SD	CV%	P value
BMI (kg/m ²)	23.4 ± 2.8	31.2 ± 4.6	14.74	< 0.001**
IL-6 (pg/mL)	0.82 ± 0.43	14.09 ± 4.00	28.39	< 0.001**
TNF- α (pg/mL)	0.26 ± 0.12	12.65 ± 4.33	34.23	< 0.001**
IL-1 β (pg/mL)	2.06 ± 1.19	23.34 ± 5.58	23.91	< 0.001**

BMI = body mass index; IL-6 = interleukin-6; TNF- α = tumor necrosis factor-alpha; IL-1 β = interleukin-1 β ; CV% = coefficient of variation; SD = standard deviation. Independent-samples t-test; **highly significant difference at $P < 0.01$.

4-DISCUSSION

Body mass index and female infertility

The present findings agree with numerous studies reporting a positive association between obesity and infertility. Obesity adversely affects multiple physiological systems, including the female reproductive system, and reduces

reproductive potential (Silvestris et al., 2018). Obesity is highly prevalent among infertile women and is associated with menstrual irregularity, oligo-ovulation and anovulation, and overweight and obese women experience lower pregnancy rates, higher miscarriage rates and more pregnancy-related complications than women of normal weight (Mikhael et al., 2019). Women with a BMI above 29 kg/m² are at increased risk of menstrual disturbance and infertility (Turner et al., 2024), and obesity impairs fertility through impaired follicular development, reduced oocyte quality and quantity, suboptimal fertilization, and defective embryo development and implantation (Ennab & Atiomo, 2023).

Several hormonal and metabolic mechanisms underlie this relationship. Increased adipose tissue enhances peripheral conversion of androgens to estrogens, disrupting gonadotropin secretion, while obesity-associated insulin resistance and hyperinsulinemia raise circulating androgens and reduce sex hormone-binding globulin, growth hormone and insulin-like growth factor-binding proteins. Elevated leptin further disturbs the HPO axis and impairs the function of granulosa and theca cells (Pavli et al., 2024; Silvestris et al., 2018). Even obese women with regular cycles may have undetected ovulatory dysfunction, and hyperandrogenism adversely affects both endometrial receptivity and oocyte maturation, so that obesity is a major contributor to female infertility, particularly that associated with ovulatory dysfunction.

Inflammatory biomarkers in obesity-associated infertility

The more than ten-fold elevation of IL-6, TNF- α and IL-1 β in obese infertile women indicates a pronounced chronic low-grade inflammatory state. This agrees with the cross-sectional study of Duan et al. (2023), who reported higher inflammatory markers in infertile than in fertile women, and with experimental evidence that obesity-induced inflammation causes granulosa-cell apoptosis and abnormal folliculogenesis (Chen et al., 2023). Persistent inflammatory activation disturbs folliculogenesis, oocyte maturation, hormonal regulation and immune signaling within the reproductive tract, and ovulation itself shares many features with an inflammatory process that is disrupted when inflammation becomes chronic (Duffy et al., 2019). Circulating IL-6 rises with BMI (Straub et al., 2000), and excess lipid accumulation in adipose tissue increases IL-6 and TNF- α production while reducing adiponectin, thereby enhancing oxidative stress and sustaining systemic inflammation.

Adipose tissue is now recognized as an active endocrine organ that secretes adiponectin, leptin, resistin, chemokines and pro-inflammatory cytokines such as IL-6, TNF- α and monocyte chemoattractant protein-1. Tissue hypoxia, adipocyte hypertrophy and macrophage and neutrophil infiltration all stimulate the production of these mediators, so that inflammation within adipose tissue drives systemic inflammation and increases the risk of obesity-related metabolic and reproductive disorders (Uribe-Querol & Rosales, 2022; Lin et al., 2024). Activation of Toll-like receptors by obesity-associated hyperlipidemia further amplifies IL-6 and TNF- α production and maintains chronic inflammation. PCOS, one of the most common obesity-associated reproductive disorders, is characterized by exactly this chronic low-grade inflammation, which disrupts ovarian physiology and endocrine regulation (Deng et al., 2024; He et al., 2020).

The pronounced difference in inflammatory biomarkers observed here supports the hypothesis that chronic inflammation is a major biological mechanism linking obesity to impaired female fertility. Evaluating inflammatory biomarkers alongside BMI may therefore improve the clinical assessment of infertile women and help to identify those who might benefit from weight reduction and anti-inflammatory lifestyle interventions, which have been shown to improve fertility outcomes after medical weight loss (Pavli et al., 2024).

Study limitations

Several limitations should be acknowledged. First, the patient group differed from the control group in two factors simultaneously, obesity and secondary infertility; without a normal-weight infertile group and an obese fertile group, the design cannot separate the contribution of adiposity from that of infertility to the elevated cytokine levels. Second, the specific causes of infertility in the patient group (for example PCOS, tubal factor or unexplained infertility) were not recorded, although PCOS in particular is strongly associated with both obesity and inflammation. Third, the analysis compared group means only; correlation between BMI and individual cytokine concentrations within the groups was not examined, so the findings describe an association at the group level rather than a dose-response relationship. Fourth, the sample size was modest and no a priori power calculation was performed. Finally, the cross-sectional design precludes causal inference. Larger prospective studies with appropriate comparison groups and correlation analysis are needed to clarify the causal relationship between obesity, chronic inflammation and female infertility.

CONCLUSION

Obese women with secondary infertility showed markedly higher serum IL-6, TNF- α and IL-1 β than healthy fertile women of normal weight, indicating that obesity-associated infertility is accompanied by chronic low-grade systemic inflammation. These findings support the hypothesis that inflammatory mechanisms disrupting hormonal reg-

ulation, ovarian function and ovulation contribute to the pathophysiology of female infertility. Assessing inflammatory biomarkers in combination with BMI may provide useful clinical information in the evaluation of infertile women, and improving metabolic health and reducing inflammation may help to improve reproductive outcomes. Further large-scale prospective studies with appropriate comparison groups are recommended to establish causality and identify therapeutic targets.

REFERENCES

- Akoum, A., Al-Akoum, M., Lemay, A., Maheux, R., & Leboeuf, M. (2008). Imbalance in the peritoneal levels of interleukin-1 and its decoy inhibitory receptor type II in women with endometriosis, infertility, and pelvic pain. *Fertility and Sterility*, 89(6), 1618–1624. <https://doi.org/10.1016/j.fertnstert.2007.06.019>
- Ameho, S., & Klutstein, M. (2025). The effect of chronic inflammation on female fertility. *Reproduction*, 169, e240197. <https://doi.org/10.1530/REP-24-0197>
- Cena, H., Chiovato, L., & Nappi, R. E. (2020). Obesity, polycystic ovary syndrome, and infertility: A new avenue for GLP-1 receptor agonists. *Journal of Clinical Endocrinology & Metabolism*, 105(8), e2695–e2709. <https://doi.org/10.1210/clinem/dgaa285>
- Chen, R., Wu, X., Qiu, H., et al. (2023). Obesity-induced inflammatory miR-133a mediates apoptosis of granulosa cells and causes abnormal folliculogenesis. *Acta Biochimica et Biophysica Sinica*, 55, 1234–1246. <https://doi.org/10.3724/abbs.2023089>
- Deng, H., Chen, Y., Xing, J., et al. (2024). Systematic low-grade chronic inflammation and intrinsic mechanisms in polycystic ovary syndrome. *Frontiers in Immunology*, 15, 1470283. <https://doi.org/10.3389/fimmu.2024.1470283>
- Douglass, J. D., Dorfman, M. D., Fasnacht, R., Shaffer, L. D., & Thaler, J. P. (2017). Astrocyte IKK β /NF- κ B signaling is required for diet-induced obesity and hypothalamic inflammation. *Molecular Metabolism*, 6(4), 366–373. <https://doi.org/10.1016/j.molmet.2017.01.010>
- Duan, Y., Zhou, Y., Peng, Y., Shi, X., & Peng, C. (2023). Inflammatory markers in women with infertility: A cross-sectional study. *International Journal of General Medicine*, 16, 1113–1121. <https://doi.org/10.2147/IJGM.S405793>
- Duffy, D. M., Ko, C., Jo, M., Brannstrom, M., & Curry, T. E. (2019). Ovulation: Parallels with inflammatory processes. *Endocrine Reviews*, 40, 369–416. <https://doi.org/10.1210/er.2018-00075>
- Ennab, F., & Atiomo, W. (2023). Obesity and female infertility. *Best Practice & Research Clinical Obstetrics & Gynaecology*, 89, 102336. <https://doi.org/10.1016/j.bpobgyn.2023.102336>
- He, S., Mao, X., Lei, H., et al. (2020). Peripheral blood inflammatory-immune cells as predictors of infertility in women with polycystic ovary syndrome. *Journal of Inflammation Research*, 13, 441–450. <https://doi.org/10.2147/JIR.S260770>
- Inhorn, M. C., & Patrizio, P. (2015). Infertility around the globe: New thinking on gender, reproductive technologies and global movements in the 21st century. *Human Reproduction Update*, 21, 411–426. <https://doi.org/10.1093/humupd/dmv016>
- Jaswa, E. G., Rios, J. S., Cedars, M. I., Santoro, N. F., Pavone, M. E. G., Legro, R. S., et al. (2020). Increased body mass index is associated with a nondilutional reduction in anti-Müllerian hormone. *Journal of Clinical Endocrinology & Metabolism*, 105, 3234–3242. <https://doi.org/10.1210/clinem/dgaa513>
- Lin, Y., Gao, R., Jing, D., Liu, Y., Da, H., Birnbaumer, L., et al. (2024). TRPC absence induces pro-inflammatory macrophage polarization to promote obesity and exacerbate colorectal cancer. *Frontiers in Pharmacology*, 15, 1392328. <https://doi.org/10.3389/fphar.2024.1392328>
- Mikhael, S., Punjala-Patel, A., & Gavrilova-Jordan, L. (2019). Hypothalamic-pituitary-ovarian axis disorders impacting female fertility. *Biomedicines*, 7(1), 5. <https://doi.org/10.3390/biomedicines7010005>
- Pavli, P., Triantafyllidou, O., Kapantais, E., Vlahos, N. F., & Valsamakis, G. (2024). Infertility improvement after medical weight loss in women and men: A review of the literature. *International Journal of Molecular Sciences*, 25(3), 1909. <https://doi.org/10.3390/ijms25031909>
- Peng, B., Alotaibi, F. T., Sediqi, S., Bedaiwy, M. A., & Yong, P. J. (2020). Role of interleukin-1 β in nerve growth factor expression, neurogenesis, and deep dyspareunia in endometriosis. *Human Reproduction*, 35(4), 901–912. <https://doi.org/10.1093/humrep/deaa017>
- Phillips, C. M. (2013). Metabolically healthy obesity: Definitions, determinants and clinical implications. *Reviews in Endocrine and Metabolic Disorders*, 14, 219–227. <https://doi.org/10.1007/s11154-013-9252-x>
- Silvestris, E., de Pergola, G., Rosania, R., & Loverro, G. (2018). Obesity as disruptor of the female fertility. *Reproductive Biology and Endocrinology*, 16(1), 22. <https://doi.org/10.1186/s12958-018-0336-z>
- Straub, R. H., Hense, H. W., Andus, T., Schölmerich, J., Riegger, G. A., & Schunkert, H. (2000). Hormone replacement therapy and the interrelation between serum interleukin-6 and body mass index in postmenopausal women: A population-based study. *Journal of Clinical Endocrinology & Metabolism*, 85, 1340–1344. <https://doi.org/10.1210/jcem.85.3.6355>

- Turner, F., Powell, S. G., Al-Lamee, H., et al. (2024). Impact of body mass index on fertility in an otherwise healthy population: A systematic review and meta-analysis. *BMJ Open*, 14, e082123. <https://doi.org/10.1136/bmjopen-2023-082123>
- Uribe-Querol, E., & Rosales, C. (2022). Neutrophils actively contribute to obesity-associated inflammation and pathological complications. *Cells*, 11, 1883. <https://doi.org/10.3390/cells11121883>
- Vander Borgh, M., & Wyns, C. (2018). Fertility and infertility: Definition and epidemiology. *Clinical Biochemistry*, 62, 2–10. <https://doi.org/10.1016/j.clinbiochem.2018.03.012>
- Velasco, I., Ación, P., Campos, A., Ación, M. I., & Ruiz-Maciá, E. (2010). Interleukin-6 and other soluble factors in peritoneal fluid and endometriomas and their relation to pain and aromatase expression. *Journal of Reproductive Immunology*, 84(2), 199–205. <https://doi.org/10.1016/j.jri.2009.11.004>
- Venkatesh, S. S., Ferreira, T., Benonisdottir, S., Rahmioglu, N., Becker, C. M., Granne, I., et al. (2022). Obesity and risk of female reproductive conditions: A Mendelian randomisation study. *PLoS Medicine*, 19, e1003679. <https://doi.org/10.1371/journal.pmed.1003679>
- Wang, W., Hou, S., Wang, K., Ling, B., & Yu, H. (2024). Association of body roundness index with female infertility: 2013–2018 NHANES. *Frontiers in Nutrition*, 11, 1416637. <https://doi.org/10.3389/fnut.2024.1416637>
- Zegers-Hochschild, F., Adamson, G. D., de Mouzon, J., Ishihara, O., Mansour, R., Nygren, K., et al. (2009). International Committee for Monitoring Assisted Reproductive Technology (ICMART) and the World Health Organization (WHO) revised glossary of ART terminology, 2009. *Fertility and Sterility*, 92, 1520–1524. <https://doi.org/10.1016/j.fertnstert.2009.09.009>
- Zheng, L., Yang, L., Guo, Z., Yao, N., Zhang, S., & Pu, P. (2023). Obesity and its impact on female reproductive health: Unraveling the connections. *Frontiers in Endocrinology*, 14, 1326546. <https://doi.org/10.3389/fendo.2023.1326546>

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Data availability: The data supporting the findings of this study are available from the corresponding author on reasonable request.

Ethical approval: The study was approved by the ethics committee of the Infertility Center, Al-Sadr Teaching Hospital, Najaf, Iraq (approval No. [to be inserted]), and written informed consent was obtained from all participants.

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