NARRATIVE REVIEW

Female infertility – risk factors

Saira Furqan, Sarah Saba Yasin, Muhammad Saad, Muhammad Salik

Abstract

Female infertility is a complex health concern with a multitude of risk factors and aetiologies. This current narrative review was planned to highlight the various significant contributors to female infertility, including age-related decline in fertility, ovulatory dysfunctions, endometriosis, uterine abnormalities, pelvic and tubal adhesions, and lifestyle factors, such as smoking, alcohol consumption, caffeine intake, exercise, psychological stress and drug abuse. While advanced age and some medical conditions undeniably lower reproductive potential, modifiable lifestyle choices also play a vital role. Early diagnosis, prompt and appropriate management, and awareness of these factors are imperative for optimising fertility outcomes. Public health strategies should emphasise education and awareness of lifestyle choices that impact fertility, alongside advocating timely interventions for those at risk. There is a critical need for robust research to augment the present understanding of female infertility and its complex interplay with these factors.

Keywords: Female infertility, Risk factors, Ovulatory disorders, Endometriosis, Lifestyle factors, Pelvic adhesions, Tubal blockage, Uterine abnormalities, Age-related decline, Nutrition and fertility.

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Introduction

Infertility is a complicated and emotionally challenging condition affecting couples worldwide. While both male and female factors contribute to infertility, female infertility encompasses a broad spectrum of causes, requiring a comprehensive understanding of the underlying risk factors. (Figure 1).

The aetiology of female infertility is related to a variety of genetic, physiological, lifestyle and environmental factors. Ovulatory disorders are the most common identifiable factors of female infertility, accounting for 25% of the known causes.[1] Endometriosis is another leading contributor to female infertility, with 30-50% of women with endometriosis expected to experience infertility.[2] Structural abnormalities, including uterine anomalies and tubal blockages, are responsible for 11% of the known causes of female infertility.[1] Infections, such as pelvic inflammatory disease, may lead to scarring, and subsequent infertility. Moreover, lifestyle factors, such as obesity, smoking, excessive alcohol consumption, and illicit drug use, can negatively impact fertility.

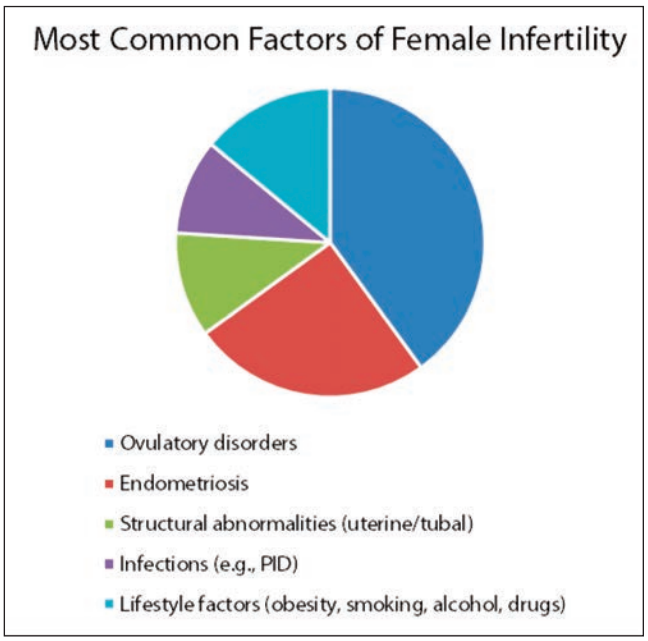

Figure-1: Most common identifiable factors of female infertility.

Recognising the importance of addressing this global health concern, the current narrative review was planned to explore the intricate interplay of risk factors associated with female infertility. A deeper understanding of the risk factors is crucial to optimising fertility outcomes and improving the overall reproductive health of women globally.

Aetiology

Age-related fertility decline

Age-related fertility decline significantly impacts female reproductive health. With aging, there is a decline of ovarian reserve and oocyte quality. As aging occurs, not only does the number of oocytes decrease, but the oocytes remaining are more prone to genetic abnormalities,

Department of Medicine, Aga Khan University Hospital, Karachi, Pakistan.
Correspondence: Saira Furqan. e-mail: saira.furqan@aku.edu
ORCID ID: 0000-0002-5526-0673

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contributing to reduced fertility.[3]

Advanced maternal age is also linked to increased pregnancy complications, such as gestational diabetes mellitus (GDM), hypertension (HTN) and caesarean section (CS).[4]

Nutritional factors

Dietary factors, like unhealthy diets, micronutrient deficiencies, obesity, undernutrition, and chronic stress are linked to nutrition-related infertility.

A diet high in processed foods, sugary drinks and saturated fats, combined with insufficient fruits, vegetables and whole grains, contributes to infertility. Obesity, a major global issue, disrupts hormonal balance, impairs ovulation, and increases the risk of polycystic ovary syndrome (PCOS), with infertility rates in overweight women being three times higher than in those of normal weight.[5]

Malnutrition and suboptimal nutrient intake affect 48.6% of women of reproductive age. Deficiencies in micronutrients, like iron, zinc, foliate and vitamin D, are linked to increased infertility risk.[6]

Disordered eating or excessive exercise also causes hormonal imbalances, menstrual irregularities, and ovulatory dysfunction.[7] Chronic stress worsens fertility issues by disrupting the hypothalamic-pituitary-adrenal (HPA) axis.

Addressing these issues through targeted nutritional interventions and preconception care can improve reproductive outcomes and overall health. Nutritional assessments, individualised dietary plans, and supplementation can correct deficiencies and boost fertility.

Ovulation disorders

Ovulatory disorders account for around 25% of female infertility cases, making their understanding crucial for effective management and better fertility outcomes.[1]

Oligo-ovulation (irregular ovulation) and anovulation (absent ovulation) prevent the release of mature oocytes, leading to infertility. These disorders emphasise the critical role of ovulation in reproduction. The World Health Organisation (WHO) has classified various ovulatory disorders.[7-10]

Hypothalamic Amenorrhea: This condition, caused by stress, eating disorders, excessive exercise, or genetic mutations, leads to the suppression of the hypothalamic-pituitary-ovarian axis. Reduced Gonadotropin-releasing hormone (GnRH) secretion, high cortisol and abnormal follicle growth result in anovulation and low oestrogen.[7] Rare genetic mutations may also contribute to this condition.[8]

PCOS: PCOS is the most common type of ovulatory disorders, affecting many reproductive-age women. It involves menstrual irregularities, infertility and insulin resistance. The interaction between insulin resistance, hyperinsulinaemia, and hyperandrogenism, alongside hypothalamic-pituitary dysfunction, leads to ovarian dysfunction and anovulation. Regional disparities in the diagnosis of PCOS shed light on the importance of socioeconomic and healthcare differences in women's reproductive health. A study found that women from lower-income regions were more likely to be diagnosed with PCOS based on ultrasound findings, even when they had regular cycles and no hormonal abnormalities. Therefore, while warning that limited access to a complete endocrine evaluation may lead to overdiagnosis or misclassification, the study stressed the need to manage infertility regionally.[9] The study further found that diagnoses of polycystic ovaries were more common in women from the Middle East, North Africa and South Asia. In these regions, there was a greater reliance on ultrasound imaging rather than a complete clinical and hormonal assessment. This inconsistency in diagnostic approach contributes to regional differences in how PCOS is reported, and its role in infertility in certain populations.[9]

Premature Ovarian Failure: Premature ovarian failure, marked by decreased ovarian function before age 40, results from accelerated follicle depletion or reduced steroid production. Genetic disorders like Turner Syndrome and Fragile X Syndrome are common causes.[10] Autoimmune conditions and toxins, including chemotherapy and radiation, also lead to ovarian failure.

Pituitary Adenoma: Pituitary adenomas can affect infertility in various ways. A prolactinoma can disrupt GnRH secretion, lower Luteinizing hormone (LH) levels, and lead to anovulation. Pituitary macroadenoma may lead to structural damage to gonadotropin-producing cells causing amenorrhea and infertility. Patients with secondary hypothyroidism may also have infertility. Adrenocorticotropic hormone (ACTH) producing pituitary tumours may cause inhibition of GnRH, leading to infertility.

Medical disorders

Diabetes Mellitus: Menstrual irregularities and infertility may indicate type 1 or type 2 diabetes as contributing factors. Diabetic women often have a shorter reproductive span due to delayed menstruation onset and early menopause.[11] Complications like microvascular or cardiovascular issues have also been linked to significantly

lower fertility rates in these cases. Studies show that better glycaemic control and prevention of diabetes-related complications can improve menstrual irregularities and fertility, bringing fertility rates closer to those of the general population.[12]

Hypothyroidism: Hypothyroidism affects 2-4% of women of reproductive age.[13] Undiagnosed thyroid disorders can contribute to infertility and subfertility. Even subclinical hypothyroidism can cause anovulation directly or by raising prolactin levels. Many women with hypothyroidism-related infertility also experience hyperprolactinemia due to increased Thyrotropin-releasing hormone (TRH) production,[9] highlighting the complex link between thyroid function and reproductive health, requiring careful management to improve fertility.

Cushing's Syndrome: Cushing's Syndrome, characterised by excess cortisol, disrupts GnRH secretion and increases infertility risk. A study of 45 women with newly diagnosed Cushing's disease showed that 80% of females suffered from menstrual irregularities. Of these, 31% experienced oligomenorrhoea, 33% had amenorrhoea, and others faced irregular or excessive bleeding.[14] Despite these challenges, if a female conceived, there was a very high risk of GDM, HTN, eclampsia, intrauterine growth retardation, abortion, stillbirth or premature delivery.[14]

Acromegaly: Women with acromegaly often experience menstrual irregularities, anovulation, and infertility due to disruptions in the hypothalamus-pituitary-gonadal axis caused by excess Growth hormone (GH) and Insulin-like growth factor 1 (IGF-1). Hyperprolactinaemia and impaired gonadotropin secretion, possibly due to tumour effects, also contribute to infertility.[15] Effective treatment for acromegaly, whether surgical or medical, improves fertility outcomes. Recent registry data show that over 70% of women with acromegaly were able to conceive spontaneously after treatment, and only a small proportion required somatostatin analogues or dopamine agonists during pregnancy.[16] These findings indicate that achieving biochemical control of acromegaly before conception significantly enhances reproductive potential and allows for generally safe maternal and fetal outcomes.

Structural and reproductive system

Endometriosis: Affecting 10-15% of reproductive-age women, it is characterised by the presence of endometrial tissue outside the uterus, most commonly in the pelvic region, but sometimes elsewhere in the abdomen. It is associated with symptoms such as pelvic pain and infertility, with infertility rates among women with endometriosis ranging 30-50%.[12] Endometriosis is classified into four stages by the American Society of Reproductive Medicine (ASRM) (Table). In the early stages (I and II), infertility is thought to result from inflammation caused by increased production of prostaglandins, cytokines, and immune cells, which impair ovarian and tubal function, leading to issues with follicle formation, fertilisation and implantation. In more advanced stages (III and IV), pelvic adhesions and anatomical changes distort the pelvic structures, disrupting the movement of gametes and interfering with the fertilization process. The altered pelvic environment creates physical barriers that further complicate conception. Additionally, advanced endometriosis may impair folliculogenesis due to the presence of endometriotic lesions and cysts, which can negatively affect follicular growth and maturation, reducing both the quantity and quality of available oocytes. The precise mechanisms of this disruption are still under investigation.

Table: Stages of endometriosis as per the American Society of Reproductive Medicine (ASRM).

Endometriosis Stage	Presentation of the Condition
Stage I	Minimal Few superficial implants
Stage II	Mild More and deeper implants
Stage III	Moderate Many deep implants Small cysts on one or both ovaries Presence of filmy adhesions
Stage IV	Severe Many deep implants Large cysts on one or both ovaries Many dense adhesions

Uterine Fibroids: These are common benign tumours that can distort the uterine cavity, obstruct fallopian tubes, and impair gamete transport, leading to lower pregnancy and implantation rates. They are present in 5-10% of infertile patients, and are the primary cause of infertility in 1-2.4% cases.[17] Studies show improved pregnancy rates after fibroid removal, with a 2024 meta-analysis demonstrating a significant increase in live birth rates post-myomectomy for women with otherwise unexplained infertility.[18] Treatment options include medical therapy, surgery and uterine artery embolisation (UAE). For women seeking to conceive, myomectomy is the preferred option, with treatment decisions depending on the fibroid's size, location and number, as well as the patient's fertility goals.

Pelvic and Tubal Adhesions: Along with uterine and tubal abnormalities, these are significant contributors to female infertility. Effective management requires understanding their prevalence, pathophysiology and impact on fertility. Pelvic inflammatory disease (PID), often caused by

pathogens, like neisseria (N.) gonorrhoeae, chlamydia (C.) trachomatis and mycoplasma (M.) genitalium, is a leading cause of adhesions. About 15% of women with N. gonorrhoeae infections develop PID, with research indicating a fivefold increase in infertility among those with a history of PID.[19,20] The number and severity of PID episodes directly affect infertility risk, highlighting the importance of addressing PID-related tubal factor infertility.

Hydrosalpinges: Characterised by fluid accumulation in the fallopian tubes, they impair fertility. Chronic inflammation and damage lead to tubal obstruction and disrupted fluid distribution, creating a hostile environment for implantation. Studies have shown a reduction in pregnancy rates during in-vitro fertilisation (IVF) when hydrosalpinx is present.[21] Retrograde flow of toxins and prostaglandins from the damaged fallopian tube into the endometrium further diminishes endometrial receptivity. The pathophysiology of pelvic and tubal adhesions involves chronic inflammation and tissue damage. In PID, C. trachomatis triggers an inflammatory response that leads to adhesions. Chronic inflammation induces the production of prostaglandins and cytokines and recruits immune cells like macrophages and natural killer cells, impairing ovarian and tubal function. This inflammation affects follicular development, fertilisation and implantation. In hydrosalpinges, acute and chronic inflammations damage the fallopian tubes' structural integrity, leading to obstruction and fluid buildup, further complicating fertility.

Congenital Uterine Anomalies: These result from developmental defects, such as failure of the Müllerian duct to develop, leading to conditions like unicornuate or bicornuate uterus.[22] Complex cases may involve mesonephric anomalies, often linked to uterine malformations, hemivagina, and renal issues, such as renal agenesis and duplicate collecting systems. These anomalies are thought to arise from genetic, environmental or pharmacological factors, including exposure to ionising radiation, infections (e.g., rubella), or teratogenic drugs (e.g., thalidomide) during early pregnancy.

Studies suggest that women with mild Mullerian anomalies are three times more likely to experience oligomenorrhea or amenorrhea compared to those with normal uterine cavities (58.6% vs. 19.6%).[22] This has led to the theory that defects in steroid receptor proteins in deformed uteri could cause eugonadotropic amenorrhea. Additionally, polycystic ovaries are more prevalent in infertile women with Mullerian anomalies (29.9% vs. 20.1%), especially those with septate or bicornuate uteri.[23]

Although research is limited, recent literature shows a higher prevalence of septate uterus in infertile populations compared to the general population, suggesting a possible link to infertility.[24] Further studies are needed to fully understand these associations.

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Lifestyle

Smoking: A well-established factor in female infertility, smoking negatively impacts ovarian function by reducing ovarian reserve, increasing follicular depletion, and impairing oocyte quality. Nicotine and other toxins in cigarettes disrupt hormonal balance, cause oxidative stress, and accelerate follicular aging. It also raises the risk of tubal factor infertility, pregnancy loss, delayed conception, and reduced response to fertility treatments like IVF.[25]

Excessive Alcohol Intake: It disrupts the hypothalamic-pituitary-ovarian axis, causing hormonal imbalances and irregular menstrual cycles.[26] It also impairs oocyte quality, lowers implantation rates, and increases miscarriage risk. Alcohol-induced liver dysfunction further disrupts oestrogen metabolism, which affects reproductive function. Women trying to conceive are advised to limit or avoid alcohol due to its potential negative impact on fertility and foetal development.

Excessive Caffeine Intake: It is associated with delayed conception and an increased risk of miscarriage.[27] It can interfere with the hormonal balance required for optimal reproductive function and negatively affect oocyte quality. Moderate intake (<200mg per day) is generally safe.

Excessive Exercise and Low Body Mass Index (BMI): Exercise is beneficial for overall health, but excessive physical activity can disrupt hormonal balance, leading to menstrual irregularities and anovulation.[28] It causes hypothalamic amenorrhea, which is linked to infertility. On the other hand, a sedentary lifestyle and obesity can also impair fertility. Moderate physical activity is recommended to maintain reproductive health.

Stress and Anxiety: It impact fertility by disrupting the hypothalamic-pituitary-ovarian axis, leading to hormonal imbalances and menstrual irregularities. This may also reduce the success of fertility treatments. Stress-reduction techniques, such as mindfulness, relaxation exercises and emotional support, are important for women facing infertility.

Drug Abuse: It poses significant risks to reproductive health. Opioid abuse disrupts the hypothalamic-pituitary-gonadal axis, leading to hormonal imbalances, menstrual irregularities and reduced ovarian reserves.[29] Cocaine impairs ovarian function, reduces oocyte quality and

quantity, and increases the risk of tubal damage and ectopic pregnancy.[30] Cannabis use disrupts hormonal balance and menstrual cycles, potentially affecting ovulation and early pregnancy.[30] Methamphetamine use causes menstrual irregularities, anovulation and diminished ovarian reserves, with stimulant properties reducing blood flow to the uterus, impacting fertility.[31] Other drugs, like amphetamines, hallucinogens and anabolic steroids, can also impair ovulation and overall reproductive health. Healthcare providers should address drug abuse in patients with infertility, offering support for addiction recovery and promoting awareness of its detrimental effects on fertility.

Discussion

While the current narrative review provides a comprehensive overview of female infertility risk factors, a few limitations should be noted. The study is based on secondary data from existing literature, which limits the ability to provide new experimental findings. However, this approach allows for a broader synthesis of current knowledge. Additionally, some variation exists in the methodologies and populations of the studies reviewed, making it challenging to generalise conclusions to all demographic groups. Nevertheless, these studies offer valuable insights across different contexts. Another consideration is the variability in diagnostic criteria for conditions such as PCOS and endometriosis, which can complicate direct comparisons. The global prevalence of endometriosis is estimated to be approximately 6-13% of women. Variations in reported prevalence are notable, with studies indicating rates of 2-11% among asymptomatic women, 5-50% among infertile women, and 5-21% among women hospitalised for pelvic pain.[32] Accurately measuring the prevalence of endometriosis poses significant challenges. Diagnosis often relies on invasive procedures, such as surgical visualisation or advanced imaging techniques, given the absence of reliable non-invasive biomarkers. Furthermore, historical neglect in public health policy and research has contributed to widespread under-recognition of the disease. Symptoms such as chronic pelvic pain and fatigue are frequently normalised or dismissed, leading to stigmatisation and delayed care. In some regions, access to diagnostic tools like laparoscopy is predominantly reserved for fertility evaluations, which may only be available to married women, further limiting diagnostic opportunities. The wide variability in clinical presentations, symptom severity, and patient experiences adds another layer of complexity to timely and accurate diagnosis. Also, limited access to healthcare services in low- and middle-income countries (LMICs) contributes to global disparities in diagnosis, management and treatment of endometriosis.[32]

Continued research is essential to understand the complex relationship between endometriosis and infertility, and PCOS and infertility. Investigating the molecular and cellular mechanisms that contribute to endometriosis-related infertility could uncover new therapeutic targets. Moreover, advances in diagnostic techniques, such as transvaginal ultrasound and magnetic resonance imaging (MRI), may offer non-invasive ways to diagnose infertility more accurately.

Ongoing studies into novel therapeutic approaches, including the role of epigenetics and genetics, hold promise for improving fertility outcomes in women. Additionally, targetted therapies may offer new opportunities for preserving and restoring fertility in affected individuals. Addressing this standardisation in future research may improve the clarity of findings. Lastly, while existing research provides valuable data on risk factors and interventions, a lack of long-term follow-up in some cases limits the understanding about how these factors influence fertility outcomes over time. Despite these considerations, the current narrative review presents a strong foundation for future research and practical recommendations for managing female infertility.

Conclusion

Female infertility is a complex issue influenced by various risk factors. Understanding these risk factors is crucial for effective prevention, diagnosis and management of infertility. Significant risk factors contributing to female infertility include age-related decline, ovulatory disorders, endometriosis, pelvic/tubal adhesions, and lifestyle factors. These risk factors must be addressed and managed adequately to improve the chances of successful reproductive outcomes in women. Public health should focus on educating women regarding the impact of their lifestyle and healthy behaviours on improving fertility outcomes. Women who may be at risk of infertility due to age or medical conditions should be counselled and encouraged to undergo timely fertility preservation and management. Prompt diagnosis and appropriate management of certain ovulatory disorders, endometriosis and pelvic/tubal adhesions are essential for optimising fertility potential. There is a dire need for further research to deepen human knowledge and understanding of the complex relationship between female infertility and its risk factors. With greater knowledge, healthcare professionals can effectively identify and address these risk factors to provide individuals and couples with comprehensive support, and help them adequately achieve their reproductive goals.

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References

1. Walker MH, Tobler KJ. Female Infertility. Treasure Island, FL: StatPearls Publishing; 2025.
2. Nezhat C, Khoyloo F, Tsuei A, Armani E, Page B, Rduch T, et al. The prevalence of endometriosis in patients with unexplained infertility. J Clin Med. 2024;13:444. doi: 10.3390/jcm13020444.
3. Seifer DB, Feinberg EC, Hsu AL. Ovarian aging and fertility. JAMA. 2024;332:1750–1. doi: 10.1001/jama.2024.18207.
4. Glick I, Kadish E, Rottenstreich M. Management of pregnancy in women of advanced maternal age: improving outcomes for mother and baby. Int J Womens Health. 2021;13:751–9. doi: 10.2147/IJWH.S283216.
5. Liu D, Luo X, Zhou K. Association between current relative fat mass and history of female infertility based on the NHANES survey. Sci Rep. 2025;15:6294. doi: 10.1038/s41598-025-89417-y.
6. Skoracka K, Ratajczak AE, Rychter AM, Dobrowolska A, Krela-Kaźmierczak I. Female fertility and the nutritional approach: the most essential aspects. Adv Nutr. 2021;12:2372–86. doi: 10.1093/advances/nmab068.
7. Vigil P, Meléndez J, Soto H, Petkovic G, Bernal YA, Molina S. Chronic stress and ovulatory dysfunction: implications in times of COVID-19. Front Glob Womens Health. 2022;3:e866104. doi: 10.3389/fgwh.2022.866104.
8. Liang Y, Huang J, Zhao Q, Mo H, Su Z, Feng S, et al. Global, regional and national prevalence and trends of infertility among individuals of reproductive age (15–49 years) from 1990 to 2021, with projections to 2040. Hum Reprod. 2025;40:529–44. doi: 10.1093/humrep/deae292.
9. Bernardi LA, Afeiche M, Schliep KC, Mumford SL. Regional variation in polycystic ovary morphology and associations with reproductive hormones and clinical features in a multicenter cohort. Hum Reprod 2024;40:529–40.
10. Fernando WD, Vincent A, Magraith K. Premature ovarian insufficiency and infertility. Aust J Gen Pract 2023;52:32-8. doi: 10.31128/AJGP-08-22-6531
11. Christou MA, Kalpatsanidis A, Kolibianakis EM. Diabetes Mellitus and Infertility. In: Goulis DG, eds. Comprehensive Clinical Approach to Diabetes During Pregnancy. Cham, Switzerland: Springer Nature Switzerland AG, 2022; pp 377-93. Doi: 10.1007/978-3-030-89243-2_20
12. Shang Y, Zhou H, He R, Lu W. Dietary modification for reproductive health in women with polycystic ovary syndrome: a systematic review and meta-analysis. Front Endocrinol (Lausanne). 2021;12:e735954. doi: 10.3389/fendo.2021.735954.
13. Chen L, Hu R, Zhang R, Xie J, Qin Y, Zheng W, et al. Impact of thyroid hormone treatment on maternal pregnancy outcomes in women with subclinical hypothyroidism without TPOAb: a retrospective cross-sectional study. Thyroid Res. 2022;15:9.
14. Nieman LK. Recent updates on the diagnosis and management of Cushing's syndrome. Endocrinol Metab (Seoul). 2018;33:139–46. doi: 10.3803/EnM.2018.33.2.139.
15. Jiao R, Ju J, Wang L, Yang H, Yao Y, Deng K, et al. Safety of pregnancy in acromegaly patients and maternal and infant outcomes after pregnancy: single-center experience from China and review of the literature. BMC Endocr Disord. 2023;23:104. doi: 10.1186/s12902-023-01341-2.
16. Tönjes A, Würfel M, Quinkler M, Knappe UJ, Honegger J, Krause-Joppig N, et al. Pregnancy and acromegaly: clinical outcomes of retrospectively analysed data from the German acromegaly registry. Reprod Biol Endocrinol 2024;22:48. doi: 10.1186/s12958-024-01207-9.
17. Freytag D, Günther V, Maass N, Alkatout I. Uterine fibroids and infertility. Diagnostics (Basel) 2021;11:1455. Doi: 10.3390/diagnostics11081455
18. Donnez J, Donnez O, Ève Thys-Jelgersma G, Dolmans M-M, Gyselaers W. Uterine fibroid-related infertility: mechanisms and impact of treatment. Fertil Steril. 2024;122:345–59. Doi: 10.1016/j.fertnstert.2024.02.049
19. Russell MW. Immune Responses to Neisseria gonorrhoeae: Challenges and Opportunities With Respect to Pelvic Inflammatory Disease. J Infect Dis 2021;224(Suppl 2):s96-102. doi: 10.1093/infdis/jiaa766.
20. Jenkins SM, Vadakekut ES. Pelvic Inflammatory Disease. Treasure Island, FL: StatPearls Publishing; 2025.
21. Pérez-Milán F, Caballero-Campo M, Carrera-Roig M, Moratalla-Bartolomé E, Domínguez-Arroyo JA, Alcázar-Zambrano JL, et al. Hydrosalpinx treatment before in-vitro fertilization: systematic review and network meta-analysis. Ultrasound Obstet Gynecol 2025;65:414-26. doi: 10.1002/uog.27697.
22. Feghali E, Etrusco A, Haydamous J, Ayed A, Laganà AS, Chiantera V, et al. Concurrent diagnosis of adenomyosis and congenital uterine anomalies: a review. J Pers Med 2023;13:716. Doi: 10.3390/jpm13050716
23. Sarma DK, Verma V, Marotta F, Kumar M. Polycystic ovary syndrome: etiology, current management, and future therapeutics. J Clin Med. 2023;12:1454. Doi: 10.3390/jcm12041454
24. Practice Committee of the American Society for Reproductive Medicine. Electronic address: asrm@asrm.org. Evidence-based diagnosis and treatment for uterine septum: a guideline. Fertil Steril 2024;122:251-65. doi: 10.1016/j.fertnstert.2024.02.033.
25. He S, Wan L. Associations between smoking status and infertility: a cross-sectional analysis among USA women aged 18-45 years. Front Endocrinol (Lausanne) 2023;14:1140739. doi: 10.3389/fendo.2023.1140739.
26. Weinland C, Mühle C, von Zimmermann C, Kornhuber J, Lenz B. Sulphated dehydroepiandrosterone serum levels are reduced in women with alcohol use disorder and correlate negatively with craving: a sex-separated cross-sectional and longitudinal study. Addict Biol 2022;27:e13135. Doi: 10.1111/adb.13135
27. Emokpae MA, Brown SI. Effects of lifestyle factors on fertility: practical recommendations for modification. Reprod Fertil 2021;2:r13–26. Doi: 10.1530/raf-20-0046
28. Logue DM, Madigan SM, Melin A, Delahunt E, Heinen M, Donnell SM, Corish CA. Low energy availability in athletes 2020: an updated narrative review. Nutrients 2020;12:835. Doi: 10.3390/nu12030835
29. Turner S, Allen VM, Graves L, Tanguay R, Green CR, Cook JL. Guideline No. 443a: Opioid use throughout women's lifespan: fertility, contraception, chronic pain, and menopause. J Obstet Gynaecol Can 2023;45:102143.

30. Rosati L, Chianese T, Mileo A, De Falco M, Capaldo A. Cocaine Effects on Reproductive Behavior and Fertility: An Overview. Vet Sci 2023;10:484. doi: 10.3390/vetsci10080484.

31. Chen Q, Wang L, Zhang J, Cheng J, Lin J, Peng Y, et al. Toxic Effects of Methamphetamine on Reproductive Systems, Embryo Development, and Newborns: An Evidence-based Review. Curr Med Chem 2025;32:6216-30. doi: 10.2174/0109298673299759240603091021.

32. Rahmioglu N, Zondervan KT. Endometriosis: disease mechanisms and health disparities. Bull World Health Organ 2024;102:919-21. doi: 10.2471/BLT.24.292660.

Author Contribution:

SF, SSY, MS & MS: Concept, design, data acquisition, analysis, interpretation, drafting, revision, final approval and agreement to be accountable for all aspects of the work.