JOURNAL OF EDUCATION, HEALTH AND SPORT
eISSN 2391-8306 · Open Access · Peer-reviewed
apcz.umk.pl/JEHS · Nicolaus Copernicus University in Toruń

Cite as: PIERZYŃSKA, Klaudia, GODYŃ-MÜLLER, Kacper, BRZEZIŃSKI, Michał, ZEIGNER, Martin, BŁASZCZYK, Joanna, GRZEŚKOWIAK, Michał, KASPRZAK, Mateusz, PELANT, Hanna, KULAK, Bartosz and MAJCHRZAK, Zuzanna. Psychotropic Drugs and Reproductive Health in Women and Men: A Review of Current Evidence. Journal of Education, Health and Sport. 2026;93:72549. https://doi.org/10.12775/JEHS.2026.93.72549

ARTICLE TIMELINE
Received: 25.05.2026 Revised: 20.06.2026
Accepted: 20.06.2026 Published: 23.06.2026

INDEXING & EVALUATION
MEiN points: 40 Unique ID: 201159
Disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

The journal has been awarded 40 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32318). Unique Journal Identifier: 201159. Scientific disciplines: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne z 2019 – aktualny rok 40 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu). © The Authors 2026.

Psychotropic Drugs and Reproductive Health in Women and Men: A Review of Current Evidence

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ABSTRACT

Background: Fertility disorders are becoming an increasingly important health problem, particularly in highly developed countries. In addition to lifestyle and environmental factors, mental disorders and their pharmacological treatment may negatively affect reproductive function. Psychotropic medications, including antidepressants, antipsychotics, benzodiazepines, mood stabilizers, and methylphenidate, have been associated with hormonal disturbances, sexual dysfunction, and impaired fertility parameters in both women and men.

Aim: To evaluate the impact of psychotropic medications on male and female fertility, with particular emphasis on reproductive dysfunctions associated with antidepressants, antipsychotics, benzodiazepines, mood stabilizers, and methylphenidate.

Material and Methods: A literature review was conducted using PubMed and Google Scholar databases. Articles concerning fertility, psychotropic drugs, prolactin, sperm quality, libido, PCOS, and depression were analyzed. The review included clinical studies, review

articles, animal studies, and case reports evaluating the effects of psychotropic medications on reproductive health.

Results: Available evidence suggests that psychotropic medications may negatively affect reproductive function through multiple mechanisms, including hormonal dysregulation, hyperprolactinemia, impaired spermatogenesis, sexual dysfunction, oxidative stress, and disturbances of the hypothalamic–pituitary–gonadal axis. SSRIs and TCAs were associated mainly with impaired semen quality and sexual dysfunction, antipsychotics with hyperprolactinemia-related reproductive disorders, and benzodiazepines with erectile dysfunction and decreased libido. Mood stabilizers, particularly valproic acid and carbamazepine, demonstrated potential adverse effects on both male and female fertility, whereas findings regarding methylphenidate remained inconsistent. Available studies indicate important differences between men and women in the manifestation of reproductive adverse effects.

Conclusions: The relationship between psychotropic medications and fertility remains insufficiently understood. Existing studies are limited, frequently outdated, and often provide inconclusive results. Furthermore, it remains difficult to distinguish the effects of psychotropic medications from the impact of underlying psychiatric disorders, chronic stress, anxiety, and depression. Further high-quality contemporary studies are necessary to better evaluate the effects of psychotropic drugs on reproductive health in both women and men.

Keywords: Psychotropic drugs; Fertility; Infertility; SSRI; Antipsychotics; Benzodiazepines; Mood stabilizers; Methylphenidate; Hyperprolactinemia; Sexual dysfunction.

Introduction

Fertility is defined as the ability to conceive and achieve pregnancy. Fertility disorders are becoming an increasingly significant problem, particularly in highly developed countries. Factors contributing to reduced fertility include lifestyle habits, such as substance use, environmental conditions, and, most importantly, advanced maternal age. In developed countries, women statistically begin attempting to conceive at a later age [1]. Infertility, in turn, is defined as the inability to achieve pregnancy after 12 months or longer of regular unprotected sexual intercourse. In women, fertility markers are considered to include sex hormone levels and ovarian function assessment, whereas in men, semen quality represents a key reproductive parameter [2].

Contemporary studies indicate that lifestyle-related and environmental factors play a significant role in the pathogenesis of infertility in both women and men. Chronic stress, obesity, improper nutrition, smoking, alcohol consumption, and exposure to environmental pollutants may negatively affect hormonal balance, ovulation, and semen quality, thereby reducing reproductive potential [3].

Studies indicate that semen quality in men has deteriorated over recent decades. This phenomenon is primarily attributed to lifestyle factors and exposure to substances that disrupt endocrine function in males. An increasing prevalence of testicular dysgenesis syndrome (TDS) has also been observed. Among environmental factors, the greatest significance is attributed to smoking (including marijuana use), alcohol consumption, obesity, stress, endocrine disruptors, and even the use of mobile phones and wireless internet-connected laptops [4]. In women, particular attention has been paid to the influence of diet, including high intake of trans fats and sugars, obesity, stress, intensive physical activity, menstrual disorders, and endometriosis [5]. Therefore, stress appears to be a common factor contributing to an increased risk of infertility.

Mental disorders, including depression, are also strongly associated with stress. Depression assessed using the Beck Depression Inventory (BDI) has been shown to correlate with poorer semen quality, including lower semen volume, reduced sperm concentration, decreased total sperm count, and impaired total sperm motility, compared with healthy men [6].

As demonstrated, fertility is influenced by numerous factors, ranging from lifestyle habits, substance use, and environmental exposures to mental health disorders. These conditions themselves may contribute to impaired fertility, while the treatment of psychiatric disorders, discussed in this review, may additionally affect both fertility and libido.

Materials and methods

Data for this article were collected using the PubMed and Google Scholar databases. The literature was selected according to the overall topic of fertility, with particular emphasis on the specific effects of psychotropic drugs on reproductive function. Articles were searched using keywords such as fertility; psychotropic drugs; prolactin; sperm quality; libido; PCOS; depression.

SSRI

Selective serotonin reuptake inhibitors (SSRIs) are commonly used in the treatment of depression. Due to delayed childbearing among women, the number of patients undergoing infertility diagnostics has increased. Psychological stress may disrupt the regulation of the hypothalamic–pituitary–gonadal axis, potentially leading to sexual dysfunction, decreased libido, and reduced self-esteem. Depression associated with stress related to potential infertility is frequently treated with antidepressants. However, SSRIs themselves may also contribute to sexual dysfunction. They may interfere with hormonal balance and can lead to anovulatory cycles in women as well as disturbances of spermatogenesis in men. Although numerous review articles have examined the effects of SSRIs on pregnancy, relatively few studies specifically evaluate their impact on fertility itself [7].

Antidepressants alone have not been shown to reduce the probability of achieving pregnancy. Interestingly, benzodiazepines used in women with anxiety disorders were associated with reduced fertility. Nevertheless, it remains difficult to determine whether impaired fertility results from the medication itself or from the underlying psychiatric condition [8]. Another important group included women undergoing in vitro fertilization (IVF). No significant differences were found between women receiving SSRIs and those not receiving SSRIs in terms of pregnancy rates or live birth rates. However, women treated with SSRIs more frequently demonstrated a poorer ovarian response to stimulation therapy [7]. In a Swedish population study including 23,557 women, SSRI use was not associated with differences in pregnancy rates or the mean number of live births. The lowest likelihood of successful IVF was observed among women receiving antidepressants other than SSRIs, in whom psychotic disorders and treatment failure occurred more frequently. Clinical studies have therefore not conclusively confirmed a negative effect of antidepressants on IVF outcomes. Moreover, compared with untreated women, patients receiving treatment for anxiety and depression with SSRIs demonstrated a lower risk of miscarriage [9].

Considerably more data are available regarding the male aspect of infertility. Interestingly, serotonin itself does not appear to negatively affect semen quality, whereas SSRIs do. Men treated with SSRIs were found to present reduced sperm motility, lower total sperm count, sperm DNA damage, and a higher proportion of morphologically abnormal spermatozoa. Additionally, combined treatment with SSRIs and other psychotropic medications was associated with further deterioration in sperm motility, suggesting a clinically significant effect [7]. One of the most important findings concerned paroxetine and its influence on sperm DNA fragmentation. During paroxetine treatment, the mean DNA fragmentation index

increased from 13.8% to 30.3%. This may negatively affect proper embryo development in both natural conception and IVF settings. Although the study was not specifically designed to evaluate fertility outcomes, the findings may indicate a potential increase in the risk of fertility impairment compared with the general population. Furthermore, treatment was associated with decreased serum testosterone and estradiol concentrations. Importantly, the abnormalities induced by paroxetine were not reflected in standard semen analysis parameters. Following discontinuation of treatment, these changes normalized, leading to the hypothesis that paroxetine may impair sperm transport rather than sperm production; however, this mechanism has not yet been fully elucidated [7,10].

Tricyclic antidepressants

Particular attention among tricyclic antidepressants (TCAs) should be paid to amitriptyline. The drug was approved for clinical use in 1961 as a derivative of imipramine. Although it is still used in the treatment of depression, it is not considered a first-line therapy due to its higher incidence of adverse effects and greater risk of overdose compared with SSRIs or SNRIs [11]. Amitriptyline has also been investigated for its potential impact on reproductive health. One study evaluating the influence of antidepressants on female fertility included patients treated with TCAs. No abnormalities in menstrual cycle length or variability were observed; however, the study demonstrated a statistically significant reduction in female fertility. Analysis of individual ovulatory cycles revealed that antidepressant use during a given cycle was associated with a lower probability of conception in that particular cycle [12].

Due to the limited human data available, other authors investigated the effects of amitriptyline on reproductive hormones in adult female rats. The study demonstrated increased levels of malondialdehyde (MDA), a marker of oxidative stress associated with DNA damage and disruption of proteins involved in steroidogenesis. Increased oxidative stress in women is also associated with a higher risk of endometriosis, polycystic ovary syndrome (PCOS), luteal phase defects, fallopian tube obstruction, and abnormal placental implantation, all of which may contribute to infertility. The study further demonstrated impaired oxidative balance, with elevated MDA levels accompanied by decreased antioxidant enzyme activity, which remained low even after treatment discontinuation, potentially suggesting irreversible changes. In addition, significant reductions in estrogen and progesterone levels, particularly estrogen, were observed, which may indicate an effect of amitriptyline on the hypothalamic–pituitary–gonadal axis [13].

In men, the role of TCAs in infertility appears to be even more pronounced than in women. One study assessed the effect of amitriptyline on sperm motility using a membrane migration method and compared several substances. Amitriptyline, together with another TCA, imipramine, demonstrated the strongest inhibitory effect on sperm motility. As lipophilic compounds, these drugs are thought to act through adsorption to lipid structures within the cell membrane [14]. Another study demonstrated a reversible inhibitory effect on spermatogenesis. Several potential mechanisms have been proposed. One of them involved increased synthesis of fibroblast growth factor 2 (FGF-2), a signaling pathway involved in the early stages of spermatogenesis. However, the exact mechanism of TCA action within the testes remains incompletely understood. Another proposed mechanism is disruption of the oxidative–reductive balance. Spermatogenic tissues are particularly sensitive to disturbances in reactive oxygen and nitrogen species (ROS/RNS) levels and lipid peroxidation, which may result in sperm DNA damage. Additional mechanisms include disturbances in metabolic and intracellular signaling pathways, such as selective activation of ERK kinase, increased synthesis of the transcription factor Egr-1, and enhanced NF-κB transcriptional activity. Antidepressants may also impair spindle formation, organelle segregation, and mitochondrial function. Collectively, these mechanisms may contribute to the negative effects of TCAs on male fertility [15].

Trazodone

As early as 1989, an association between trazodone use in the treatment of depression and the occurrence of priapism in men was reported. Ischemic priapism is a urological emergency that requires rapid diagnosis and treatment in order to preserve erectile function. Prolonged ischemia may lead to fibrosis of the corpora cavernosa and permanent erectile dysfunction. These complications may secondarily impair male fertility by hindering or preventing normal sexual intercourse [16].

Studies indicate that trazodone is among the medications most commonly associated with drug-induced priapism, primarily due to antagonism of α1-adrenergic receptors leading to impaired detumescence. As a consequence, ischemic priapism may develop and result in long-term complications, including erectile dysfunction and the need for urological intervention. Prolonged priapism may impair fertility not only through mechanical dysfunction of the penis, but also through psychological consequences, such as feelings of

rejection and difficulties in maintaining interpersonal relationships, which may further contribute to infertility [17].

Priapism associated with trazodone most commonly occurs at the initiation of therapy or following dose escalation. A history of previous prolonged erections appears to increase the risk of developing this complication. Polypharmacy, particularly the combination of lithium with antipsychotic medications, has also been associated with a higher risk. Chelsea Eisenach, MD, and Sean Lynch, MD, described a patient who experienced recurrent episodes of priapism after subsequent exposure to antipsychotic medications following an initial trazodone-induced episode [18].

Antipsychotic drugs

Antipsychotic drugs represent one of the main groups of medications associated with reproductive dysfunction, primarily through mechanisms related to increased prolactin levels. Normal prolactin concentrations are considered to be <25 ng/mL in women and <20 ng/mL in men. Physiological prolactin levels are necessary for normal lactation, regulation of sexual function, and control of pancreatic β-cell proliferation. Physiological increases in prolactin occur during pregnancy, the postpartum period, stress, and physical exertion. Drug-induced hyperprolactinemia occurs in approximately 15–45% of cases and is most commonly associated with three groups of medications: antipsychotics, tricyclic antidepressants, and prokinetic agents such as metoclopramide [19].

In women, hyperprolactinemia may occur outside physiological conditions such as pregnancy and lactation. It has been observed significantly more frequently in women over 20 years of age and represents one of the most common causes of secondary amenorrhea. In contrast, elevated prolactin levels are a relatively rare cause of secondary amenorrhea in adolescent girls [20]. Increased prolactin concentrations may lead to ovulatory dysfunction, irregular menstrual cycles, oligomenorrhea, or secondary amenorrhea. Women may also experience galactorrhea, decreased libido, hypogonadism, difficulties conceiving, and infertility [21]. Hyperprolactinemia is present in approximately 15–20% of infertile women and is considered one of the most common endocrine causes of reproductive dysfunction. Elevated prolactin disrupts the hypothalamic–pituitary–ovarian axis, leading to suppression of gonadotropin secretion and ovulatory disturbances. Consequently, anovulatory cycles and luteal phase defects may occur, contributing to impaired fertility. Treatment is primarily based on dopamine agonists such as bromocriptine and cabergoline, which effectively reduce prolactin

levels and restore ovulation in most patients. In cases of drug-induced hyperprolactinemia, switching to medications that do not elevate prolactin levels should be considered whenever possible. Cabergoline demonstrates high efficacy and good tolerability, and currently available data do not indicate an increased risk of congenital malformations or pregnancy complications associated with its use. Moreover, long-term treatment before pregnancy and lower prolactin concentrations reduce the risk of prolactinoma enlargement during pregnancy [22].

Hyperprolactinemia is also observed in up to 11% of infertile men. Elevated prolactin levels impair the hypothalamic–gonadal axis by suppressing gonadotropin-releasing hormone (GnRH) secretion, which secondarily decreases LH, FSH, and testosterone production. These hormonal disturbances may impair spermatogenesis, ranging from deterioration of semen parameters to complete suppression of sperm production. Clinically, this may manifest as secondary hypogonadism and male infertility. Additionally, prolactin is believed to exert direct effects on the male reproductive system. Prolactin receptors have been identified in Leydig cells, Sertoli cells, and the epithelium of the efferent ducts, suggesting a role in the regulation of steroid hormone production, sperm maturation, and secretory and transport functions within the male reproductive tract. Most men with antipsychotic-induced hyperprolactinemia remain asymptomatic, although some may develop decreased libido and erectile dysfunction. The greatest increases in prolactin levels, often exceeding 200 μg/L, have been reported with metoclopramide, risperidone, and phenothiazines. Available studies indicate that elevated prolactin levels are more common in men with azoospermia or oligospermia compared with men presenting normal semen parameters. Hyperprolactinemia may also be associated with varying degrees of spermatogenic impairment, ranging from reduced sperm production to complete absence of germ cells. Furthermore, elevated prolactin levels may negatively affect male sexual function, particularly through ejaculatory disorders such as delayed ejaculation, although the relationship between hyperprolactinemia and erectile dysfunction has not been conclusively established [23].

After confirmation of hyperprolactinemia, further diagnostic evaluation should be performed to determine its underlying cause. In women planning pregnancy, all confirmed cases of hyperprolactinemia should be treated according to etiology. Cases unrelated to neuroleptic treatment are most commonly managed with cabergoline at the lowest effective dose, which restores normal fertility in the majority of patients. There are also reports suggesting that even mild or transient hyperprolactinemia may be associated with recurrent miscarriage. Although

data regarding men remain less conclusive, chronic hyperprolactinemia may also negatively affect male fertility through both hormonal disturbances and impaired reproductive function, and therefore requires appropriate treatment and further evaluation [24].

Benzodiazepines

Through their action on the central nervous system, benzodiazepines interact with $GABA_A$ receptors, producing sedative, anxiolytic, hypnotic, muscle relaxant, and anticonvulsant effects. Their use during pregnancy has been associated with an increased risk of fetal malformations [25]. The available literature more commonly focuses on the effects of benzodiazepines on the fetus rather than on fertility itself.

Benzodiazepines may adversely affect sexual function, although the exact mechanisms remain incompletely understood and study results are partly inconsistent. Available data suggest that the use of these drugs may be associated with erectile dysfunction, decreased libido, and ejaculatory disorders, including delayed ejaculation. Increased GABAergic activity within the central nervous system is believed to play a significant role by inhibiting pathways responsible for sexual arousal and erection. In addition, the sedative properties of benzodiazepines may reduce sexual desire and overall sexual activity. The literature also emphasizes that sexual dysfunction occurs more frequently when benzodiazepines are combined with other psychotropic medications, particularly antidepressants and lithium. Combination therapy has been shown to significantly increase the risk of sexual dysfunction compared with monotherapy. Patients using benzodiazepines have reported both erectile dysfunction and reduced sexual satisfaction, which may negatively affect quality of life, self-esteem, and interpersonal relationships. Importantly, some studies have demonstrated improvement in sexual function following benzodiazepine discontinuation, further supporting a potential association between these drugs and sexual dysfunction.

The effects of diazepam, alprazolam, lorazepam, and clonazepam on sexual function have also been investigated. Diazepam may impair reproductive function through inhibition of the hypothalamic–pituitary–gonadal axis, reduction of testosterone production, and disturbances in steroidogenesis. It may additionally affect calcium channels, DNA synthesis, and cell proliferation, potentially leading to impaired spermatogenesis. Studies have also demonstrated degeneration of seminiferous tubules and a reduction in the number of spermatogenic epithelial cells, which may contribute to decreased fertility, reduced libido, and impaired sexual function. Alprazolam may negatively affect sexual function, particularly at high doses

or when combined with other psychotropic medications. It has been associated with erectile dysfunction, decreased libido, and difficulty achieving orgasm. Studies suggest that benzodiazepines may increase the risk of erectile dysfunction by approximately twofold, especially during combination therapy with antidepressants or lithium. In contrast, lorazepam has been associated with increased sexual drive, potentially related to reduced impulse control and decreased sexual inhibition. Clonazepam therapy, however, has been linked to significant erectile dysfunction in men, and among patients with PTSD, erectile dysfunction was one of the most commonly reported adverse effects [26].

Another study demonstrated that sleep disturbances combined with benzodiazepine use were associated with a twofold increase in erectile dysfunction among men over 65 years of age. Similar to previous findings, particular attention was drawn to clonazepam, which was associated with decreased libido, erectile dysfunction, delayed ejaculation, and anorgasmia [27].

Apart from their effects on sexual dysfunction and hormonal dysregulation, benzodiazepines have also been shown to affect gametes. Diazepam negatively influenced reproductive processes in *Paracentrotus lividus*. The drug impaired gamete quality, reduced fertilization success, and disrupted embryonic development. Diazepam primarily acted on mitochondria, microtubules, and TSPO and GABA receptors, inducing oxidative stress and impairing sperm function, cell division, and spindle organization. Following fertilization, disturbances in embryogenesis were also observed, including abnormalities in gastrointestinal tract development [28].

Mood stabilizers

One of the most important studies investigating the effects of valproic acid on male fertility was conducted by Nishimura et al. in a rat model. The authors observed reduced testicular and epididymal weight, degeneration of seminiferous tubules, decreased fertility, and impaired sperm motility following chronic treatment. Administration of 500 mg of valproic acid resulted in decreased epididymal weight, disturbances in spermatogenesis associated with retention of stage 19 spermatids during stages IX–XI, as well as degenerative changes within seminiferous tubules and loss and exfoliation of spermatogenic cells observed histopathologically. Similar retention of spermatids in stages IX–XI has also been described with compounds such as cisplatin, cadmium, 2,5-hexanedione, and nitrofuran, whose effects were associated with frequent Sertoli cell damage. In this study, the percentage of motile

spermatozoa was identified as the most sensitive parameter, suggesting that sperm motility assessment and histopathological examination are particularly important in evaluating the toxic effects of valproic acid. Although the study did not directly demonstrate impaired fertility, the authors highlighted the drug's adverse effects on male reproductive organs, which may contribute to fertility disturbances [29].

A fertility study conducted in southern India demonstrated that approximately 38% of women with epilepsy experience infertility. It has been suggested that treatment with valproic acid in women with epilepsy may induce symptoms of polycystic ovary syndrome (PCOS) through metabolic effects such as weight gain, insulin resistance, and enhanced androgenic activity, ultimately contributing to PCOS development. A pooled analysis of data from 11 independent studies demonstrated that women with epilepsy treated with valproic acid developed PCOS significantly more frequently than those receiving other antiepileptic drugs, with an almost twofold increased risk observed in the valproic acid group. However, findings regarding the association between valproic acid and PCOS remain inconclusive [30,31].

In summary, the use of valproic acid in women of reproductive age remains a significant clinical challenge requiring an individualized therapeutic approach and careful decision-making. Despite the documented risk of adverse reproductive and teratogenic effects, valproic acid may still represent the most effective therapeutic option for seizure control in selected patients. Therefore, a thorough risk–benefit assessment is essential, taking into account disease severity, the effectiveness of alternative therapies, reproductive plans, and patient preferences [31].

Another mood stabilizer, carbamazepine, has also been investigated for its effects on reproductive dysfunction since the 20th century. Current evidence is mainly based on an Iranian study conducted in a relatively small group of men. Antiepileptic drugs, particularly hepatic enzyme inducers such as phenytoin and carbamazepine, are thought to contribute to hormonal disturbances. These agents increase serum sex hormone-binding globulin (SHBG) concentrations, thereby reducing testosterone bioavailability and potentially impairing fertility. The study demonstrated significant deterioration in semen quality among patients treated with carbamazepine, suggesting a direct negative effect on male fertility [32].

In contrast to antipsychotic medications, lithium used in bipolar disorder does not usually induce hyperprolactinemia. Nevertheless, sexual dysfunction has been reported in approximately one-third of patients receiving lithium therapy. Although lithium remains one

of the most effective mood stabilizers, available data regarding its effects on male reproductive function remain insufficient. Interestingly, one study demonstrated that acetylsalicylic acid administered at a dose of 240 mg/day significantly reduced the severity of sexual dysfunction and improved erectile function in patients treated with lithium [33].

Methylphenidate

Animal studies have demonstrated that administration of methylphenidate to rats was associated with increased cortisol levels and decreased concentrations of total glutathione, superoxide dismutase, and catalase. No changes in prolactin levels were observed. However, methylphenidate administration was considered to increase oxidative stress, which may contribute to oxidative DNA damage. Oxidative stress within the ovaries was associated with infertility despite the absence of inflammatory markers [34].

These findings remain inconclusive, however, as another study demonstrated different results. In this experiment, Wistar rats received methylphenidate at doses of 2.5 mg/kg or 5.0 mg/kg, or water via intragastric administration, from postnatal day 21 to day 60. Subsequently, estrous cycle patterns, estradiol levels, reproductive organ weights, and sexual behavior were assessed. No significant differences were identified in the analyzed parameters. The authors concluded that methylphenidate treatment during periods corresponding to childhood and early adulthood did not adversely affect reproductive function in adult female rats [35].

In men, studies have shown significantly lower FSH levels in methylphenidate users compared with non-users. Total and progressive sperm motility were significantly associated with current methylphenidate use, whereas total sperm count was associated with both current and past use of the drug. In contrast to many previous studies based on animal models, this study, which analyzed more than 9,000 semen samples, demonstrated improved semen parameters among methylphenidate users. Current users achieved the highest values of the assessed semen parameters, while former users demonstrated intermediate results that still remained more favorable than those observed in men who had never used methylphenidate. This association persisted after adjustment for potential confounding factors such as age and smoking status. The authors suggested that one possible mechanism may involve reduction of oxidative stress and enhancement of antioxidant defense mechanisms, thereby supporting normal sperm function [36].

As previously mentioned, animal studies remain contradictory. Chronic methylphenidate exposure has also been shown to negatively affect the male reproductive system. In animal

models, a reduced number of Leydig cells and significantly decreased testosterone concentrations were observed, potentially leading to impaired spermatogenesis and testicular dysfunction. In addition, methylphenidate-treated groups demonstrated reduced fertility and decreased sperm motility, suggesting adverse effects on reproductive parameters. The authors emphasized that similar disturbances in spermatogenesis and fertility have also been described in studies investigating cocaine exposure. The study further highlighted the importance of carefully weighing the benefits and risks of methylphenidate treatment in ADHD, particularly in young individuals [37].

Conclusions

The impact of psychotropic medications on fertility remains insufficiently understood, and the available studies are limited, often outdated, and inconclusive. An additional challenge is distinguishing whether reduced fertility results directly from the medications themselves or from the underlying psychiatric disorders, chronic stress, anxiety, and other mental health conditions. Current evidence suggests that psychotropic drugs may affect women and men differently — women more commonly experience hormonal disturbances, ovulatory dysfunction, and hyperprolactinemia, whereas men more frequently present impaired semen parameters, erectile dysfunction, and disturbances of spermatogenesis. SSRIs, TCAs, benzodiazepines, antipsychotics, and mood stabilizers may all negatively affect reproductive function, although the exact mechanisms remain incompletely understood. Further contemporary studies investigating the effects of psychotropic medications on male and female reproductive health are therefore urgently needed.

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All authors have read and agreed with the published version of manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: The authors confirm that the data supporting the findings of this study are available within the article's bibliography.

Conflict of interest: None declared.

Declaration of AI Use: During the preparation of this work, the authors used large language models (e.g. Claude by Anthropic) in order to improve English language, readability, and text formatting. After using these tools/services, the authors thoroughly reviewed and edited the content as needed and take full and sole responsibility for the final content of the publication.

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