The New Journal of Urology
eISSN 3023-6940
doi: https://doi.org/10.33719/nju1797653

REVIEW ARTICLE

The Effects of Paternal Selective Serotonin Reuptake Inhibitor Use on Male Fertility and Pregnancy Outcomes: A Narrative Review

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Submitted: *2025–10–06*
Accepted: *2026–02–18*

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Abstract

Objective : This narrative review evaluates current evidence regarding the impact of paternal selective serotonin reuptake inhibitor (SSRI) use on male fertility parameters and neonatal outcomes.

Material and Methods: A comprehensive search of PubMed/MEDLINE and Web of Science databases was conducted for studies published up to December 2025. Out of 132 screened records, 13 studies investigating common SSRIs (e.g., sertraline, citalopram, escitalopram, fluoxetine, paroxetine, fluvoxamine) and newer agents (vortioxetine, vilazodone) were included in the narrative synthesis.

Results: Evidence indicates that SSRIs may cause transient impairments in sperm motility, morphology, and DNA integrity, which are generally reversible following treatment discontinuation. Large-scale cohort studies demonstrate no significant association between paternal SSRI use and major congenital malformations, low Apgar scores, or small-for-gestational-age (SGA) births. While a modest increase in preterm birth risk was noted, this is likely attributable to confounding by the underlying paternal psychiatric condition rather than direct pharmacological exposure. Similarly, associations with neurodevelopmental outcomes, such as Autism Spectrum Disorder (ASD) and Attention Deficit/Hyperactivity Disorder (ADHD), are more likely explained by paternal psychiatric conditions rather than direct drug exposure.

Conclusion: Paternal SSRI use does not appear to pose a significant risk to clinical fertility or offspring health. Given the transient nature of semen abnormalities, treatment should be tailored individually to balance psychiatric stability and reproductive goals.

Keywords: infertility, male, paternal exposure, pregnancy, selective serotonin reuptake inhibitors, teratogens

Cite; Bakirli DC, Horoz E, Yilmaz I, Ilbey YO. The Effects of Paternal Selective Serotonin Reuptake Inhibitor Use on Male Fertility and Pregnancy Outcomes: A Narrative Review. New J Urol. 2026;21(2):105–112. **doi:** https://doi.org/10.33719/nju1797653

INTRODUCTION

Depression and anxiety disorders represent a significant global health issue affecting millions of individuals and necessitating widespread pharmacological intervention (1,2). Selective serotonin reuptake inhibitors (SSRIs) have emerged as the first-line treatment for these conditions due to their proven efficacy and relatively favorable safety profile compared to older antidepressants (1,3). Their therapeutic action primarily involves inhibition of the presynaptic serotonin transporter (SERT), which prevents the reuptake of serotonin into the presynaptic neuron and increases extracellular serotonin levels at the postsynaptic membrane (1,4). This enhancement of serotonergic signaling is believed to underlie the antidepressant and anxiolytic effects of the drug class (5). Beyond their primary psychiatric indications, the clinical utility of SSRIs has expanded to include off-label applications for conditions such as fibromyalgia and neurocardiogenic syncope (4). Furthermore, owing to their well-documented effect of delaying ejaculation, SSRIs are frequently prescribed for the treatment of premature ejaculation, which further increases their use among men of reproductive age (1,6). Consequently, the consumption of SSRIs has surged dramatically in recent decades, particularly in Western countries. This trend has made SSRIs one of the most frequently prescribed classes of medication globally (3,7). Notably, epidemiological data show increasing SSRI use among men of reproductive age. For example, a population-based study in Denmark reported a threefold increase in paternal SSRI use over a twenty-year period (3,8).

Male factor infertility is a major contributor to reproductive failure, accounting for about half of all infertility cases worldwide (6). The causes of male infertility are multifactorial. Medication-induced reproductive toxicity is an increasingly recognized concern (6,9). While many studies have examined the impact of maternal SSRI exposure on pregnancy outcomes and fetal development, the potential reproductive consequences of paternal exposure remain underdefined and less studied (2). This gap in the literature is critical. Serotonin (5-hydroxytryptamine) receptors are present in human spermatozoa and testicular tissue, suggesting a functional role for serotonin in spermatogenesis, sperm motility, and the acrosome reaction (1,10). Emerging preclinical and clinical evidence suggests that SSRIs may disrupt male reproductive function through various mechanisms. These include alteration of hormonal homeostasis, induction of sperm DNA fragmentation, and inhibition of sperm transport (10–13).

Given the increasing overlap between the peak age of antidepressant use and the prime reproductive years, clarifying the safety profile of these drugs is essential for informed clinical counseling (2,3). Furthermore, concerns have been raised regarding the potential teratogenic effects of paternal exposure and its long-term implications for offspring neurodevelopment.

This narrative review evaluates current evidence on the impact of paternal SSRI use on male fertility and pregnancy outcomes. We focus on commonly prescribed agents, including sertraline, citalopram, escitalopram, fluoxetine, paroxetine, and fluvoxamine. We also examine newer agents, such as vortioxetine and vilazodone. We review their effects on semen parameters, sperm DNA integrity, and neonatal health to provide a guide for clinicians managing men of reproductive age.

MATERIAL AND METHODS

Search Strategy and Data Sources

A comprehensive literature search was conducted using electronic databases, including PubMed/MEDLINE and Web of Science. We looked for studies published up to December 2025. For MEDLINE, we used the following Medical Subject Headings (MeSH) terms and keywords: ("Serotonin Reuptake Inhibitors" OR "SSRIs" OR "Vortioxetine" OR "Vilazodone" OR "Fluvoxamine" OR "Sertraline" OR "Fluoxetine" OR "Paroxetine" OR "Citalopram" OR "Escitalopram") AND ("Infertility, Male" OR "Semen Analysis" OR "Spermatozoa" OR "Paternal Exposure" OR "DNA Fragmentation" OR "CatSper" OR "Caspases").

The search strategy for Web of Science was defined as:TI=("selective serotonin reuptake inhibitor" OR SSRI OR vortioxetine OR vilazodone OR fluoxetine OR paroxetine OR citalopram OR escitalopram OR sertraline OR fluvoxamine) AND TS=("male fertility" OR "semen quality" OR "sperm quality" OR "paternal exposure" OR "paternal use" OR spermatozoa OR testis).

To ensure maximum coverage and minimize the risk of missing relevant data, additional specialized sources were screened. These included the subscription-based reproductive toxicology database Reprotox®, MotherToBaby (OTIS), and UpToDate®. These sources were screened for background/context and did not contribute to the PRISMA record counts.

Study Selection and Inclusion Criteria

Using the defined search strategies, 80 studies were identified in MEDLINE and 78 in Web of Science. After removing 26 duplicates, 132 titles and abstracts were screened, of which 91 were excluded. Subsequently, full texts were sought for 41 reports, but 14 could not be retrieved. Ultimately, of the 27 full texts assessed, 13 were included in the narrative synthesis (Figure 1).

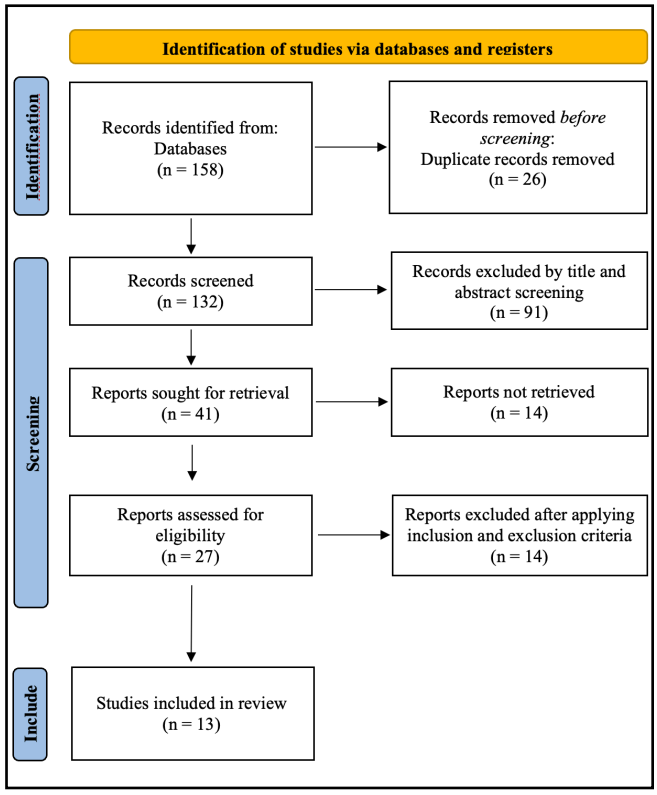

Figure 1. PRISMA diagram showing the study selection process

The inclusion criteria targeted studies involving men receiving SSRI therapy, regardless of indication, and their offspring. Additionally, preclinical research in animal models and in vitro human sperm studies assessing toxicity and mechanisms of action were also considered. The agents evaluated included sertraline, fluoxetine, paroxetine, citalopram, escitalopram, fluvoxamine, vilazodone, and vortioxetine.

Outcome measures were classified into three categories:

1. Conventional Semen Parameters: Concentration, motility, and morphology.
2. Molecular Markers: DNA damage/fragmentation, oxidative stress, and ion channel inhibition (e.g., CatSper).
3. Clinical Neonatal Outcomes: Preterm birth, small-for-gestational-age (SGA), major congenital malformations, Apgar score, and neurodevelopmental outcomes (e.g., Autism Spectrum Disorder (ASD) and Attention Deficit/Hyperactivity Disorder (ADHD), intellectual disability).

Eligible study designs for review included randomized controlled trials, cohort and case-control studies, animal experiments (limited to rat and mouse models), in vitro semen studies, and literature reviews.

Exclusion Criteria

Studies were excluded if they:

1. Investigated other antidepressant groups (e.g., Tricyclics, monoamine oxidase inhibitors) in isolation.
2. Focused exclusively on maternal exposure without distinguishing paternal effects.
3. Lacked full-text access, were retracted, or were subject to an "expression of concern" to ensure data reliability.

RESULTS

Thirteen studies were included; findings on sperm quality are presented in Table 1, and neonatal outcomes are presented in Table 2.

Regarding conventional semen parameters, meta-analytic data demonstrated statistically significant impairments in sperm morphology, concentration, motility and DNA integrity among SSRI users, with these changes being most pronounced within the first three months of exposure (14). Specifically, a prospective study on the use of escitalopram (10 mg/day) showed significant declines in these parameters after 12 weeks compared to baseline values (12). Conversely, a cohort study of 8,861 men found no significant associations between SSRI use and sperm morphology, concentration, or motility after adjusting for confounders (15).

Table 1. Effects of SSRIs on Male Fertility and Semen Quality (Preclinical and Clinical Evidence)

Study Author (Year)	Study Design	SSRI Type	Key Findings
Rahban et al. (2021) (10)	Human Sperm (*in vitro*)	Sertraline	Sertraline potently inhibits CatSper channels. Impairs acrosome reaction and egg penetration.
Roostaee et al. (2025) (13)	Human Sperm (*in vitro*)	Fluoxetine	Induction of oxidative stress (ROS), increased DNA fragmentation, and apoptosis.
Ilgin et al. (2017) (11)	In vivo Animal (Rats)	Citalopram	Reduced sperm concentration, increased DNA damage and testicular tissue damage. Linked to oxidative stress (low Glutathione) and hormonal imbalance.
Erkilinc et al. (2025) (17)	Animal Depression Model	Fluoxetine, Vortioxetine	Depression caused testis damage. Antidepressants mitigated damage and reduced inflammatory markers (IL-6, Cas-8).
Pham et al. (2022) (15)	Retrospective Cohort (n=8.861)	Mixed SSRIs	No statistically significant difference in semen parameters (sperm concentration, motility, or morphology) between SSRI users and non-users.
Alsabhan et al. (2024) (21)	Retrospective Cohort Study (n=299)	Mixed SSRIs	No significant difference in sperm count (p=0.069), motility, or viscosity between users and controls.
Tanrikut et al. (2010) (16)	Prospective Clinical Study (n=35)	Paroxetine	Sperm DNA fragmentation increased from 13.8% to 30.3% ; standard semen parameters remained largely normal.
Koyuncu et al. (2011) (12)	Prospective Clinical Study (n=25)	Escitalopram	Significant reduction in sperm concentration, motility, and morphology after 3 months of treatment.
Xu et al. (2022) (14)	Meta-analysis	Mixed SSRIs	Significant decrease in morphology (p=0.002), concentration (p=0.02), and motility (p=0.04). DNA damage (DFI) increased (p=0.0002).

Abbreviations: Cas-8, caspase 8; DFI, DNA fragmentation index; IL-6, interleukin 6; ROS, reactive oxygen species; SSRI, selective serotonin reuptake inhibitor.

Table 2. Paternal SSRI Exposure and Pregnancy/Offspring Outcomes

Study (Year)	Data Source (n)	SSRI Type	Outcome Measured	Key Findings & Statistical Significance
Garvik et al. (2025) (3)	Nationwide Cohort Study (n=13.547)	Mixed SSRIs	Preterm birth, SGA, malformations	Preterm birth risk increased with Citalopram, Escitalopram (OR: 1.15). No significant association with SGA, malformations, or APGAR scores.
Viktorin et al. (2018) (2)	Prospective Cohort (n=170.508)	Mixed SSRIs	Preterm birth, ASD, intellectual disability	No significant risk for preterm birth (OR: 0.91), malformations (OR: 1.06), or ASD (HR: 1.13).
Yang et al. (2018) (18)	National Cohort (n=781.470)	Mixed SSRIs	ADHD risk in offspring	Slight ADHD risk increase (HR: 1.26). Association likely due to underlying psychiatric condition, not drug effect.
Yang et al. (2017) (19)	Population-based cohort study (n=669.922 children)	Mixed SSRIs	ASD risk in offspring	1.62-fold risk attenuated after adjusting for paternal history. Sibling analysis confirmed no drug-related risk.

Abbreviations: ADHD, attention-deficit/hyperactivity disorder; ASD, autism spectrum disorder; HR, hazard ratio; OR, odds ratio; SGA, small for gestational age; SSRI, selective serotonin reuptake inhibitor.

Molecular evaluations indicate that SSRIs may impair sperm function through oxidative stress and DNA damage. Research involving paroxetine demonstrated an increase in the sperm DNA fragmentation index from %13.8 to %30.3, even when standard semen parameters remained normal (16). Meta-analytic data further support a significant overall increase in the DNA fragmentation index among SSRI users (14). In vitro studies on fluoxetine-exposed human sperm revealed elevated levels of reactive oxygen species (ROS) and malondialdehyde (MDA), alongside

reduced total antioxidant capacity (TAC) and the activation of apoptotic pathways through the upregulation of Caspase 8, Caspase 9, and BAX (13). Furthermore, sertraline has been identified as a potent inhibitor of the sperm-specific CatSper calcium channel, which is essential for fertilization (10).

Animal models provide additional insight into testicular structural changes. Citalopram exposure in rats resulted in reduced sperm counts and structural damage to testicular tissue (11). However, an experimental depression model showed that while untreated stress caused significant testicular damage, treatment with fluoxetine or vortioxetine mitigated this damage and reduced inflammatory markers such as IL-6 and Caspase 8 (17).

Epidemiological data from large-scale cohorts indicate that paternal SSRI use is not associated with major adverse neonatal outcomes. A nationwide cohort study of 13.547 children found no significant associations between paternal SSRI exposure and major congenital malformations, low Apgar scores, or SGA births. However, a modest increase in the risk of preterm birth was identified (OR 1.15; 95% CI 1.06-1.23), particularly with citalopram and escitalopram (3). Another prospective cohort study of 170.508 individuals found no significant risk for preterm birth (aOR 0.91; 95% CI 0.79-1.04), malformations (OR 1.06; %95 CI 0.90-1.26), or ASD (aHR 1.13; %95 CI 0.84-1.53) (2).

Long-term neurodevelopmental studies have shown a slight increase in the risk of ADHD associated with paternal SSRI exposure (HR: 1.26; 95% CI: 1.06–1.51) (18). In another study regarding ASD, initial findings indicated an increased risk (HR 1,62; 95% CI: 1.33–1.96). However, this association was significantly attenuated after adjusting for paternal psychiatric history (aHR: 1.43; 95% CI: 1.18–1.74). In addition, the risk disappeared in sibling analyses (19).

DISCUSSION

Impact on Semen Parameters and Testicular Histopathology

Multiple studies indicate that SSRI use may negatively affect semen parameters. However, findings across studies are inconsistent. A systematic review and meta-analysis by Xu et al. demonstrated statistically significant impairments in sperm morphology, concentration, and motility among SSRI users. No effect was found on semen volume. These changes were most pronounced within the first three months of exposure, suggesting a time-dependent effect (14). Similarly, Oliveira et al. reported reductions in serum testosterone levels, decreased sperm production, diminished sperm reserves, and prolonged epididymal transit time (20). Long-term SSRI exposure (at least 6 months), particularly with agents such as citalopram and sertraline, has also been associated with reduced total sperm count and motility compared with healthy controls (6).

Prospective clinical data further support these observations. Specifically, treatment with escitalopram at a dose of 10 mg/day was associated with significant declines in sperm concentration, motility, and morphology after 12 weeks compared with baseline values (12). Similarly, fluoxetine has been highlighted as a potential contributor to reproductive toxicity, including reduced reproductive organ weight and sperm concentration (9). In addition, experimental studies have shown that citalopram exposure induces reproductive toxicity in animal models, including reduced sperm count and structural damage to testicular tissue (11).

In contrast, several large observational studies have reported no significant associations between SSRI use and semen parameters. For example, a cohort study of 8,861 men undergoing fertility evaluation found no differences in semen volume, concentration, motility, or morphology between SSRI users and non-users after adjustment for confounders (15). Likewise, a retrospective analysis of 299 men attending an infertility clinic found no significant differences between SSRI users and non-users in sperm liquefaction, motility, viscosity, or sperm count (21). Furthermore, available evidence suggests that SSRI-associated changes in semen parameters may be reversible following discontinuation of treatment, indicating a potential transient effect (9). Overall, these inconsistencies highlight the variability of clinical findings and suggest that SSRI-associated changes in semen parameters may not be uniform across all populations. Additionally, the potential confounding effect of underlying psychiatric conditions has also been emphasized.

In an experimental model of chronic stress–induced depression, untreated stress was associated with marked testicular damage and elevated inflammatory markers. In contrast, treatment with fluoxetine or vortioxetine improved

testicular histopathology and reduced inflammatory and apoptotic markers compared to untreated stressed animals (17). These findings suggest that the reproductive effects observed with SSRI use may, in part, reflect the influence of depression or chronic stress, rather than a direct toxic effect of the medication itself.

Molecular Mechanisms: DNA Integrity, Oxidative Stress, and Ion Channels

Research indicates that SSRIs may impair sperm function at a molecular level, even when routine semen parameters appear normal. Studies involving sertraline and paroxetine have demonstrated significantly higher rates of DNA fragmentation compared with behavioral therapy or untreated controls (6). A study on healthy volunteers treated with paroxetine revealed a significant increase in sperm DNA fragmentation (from 13.8% to 30.3%). This increase occurred despite normal standard semen analysis, suggesting subclinical reproductive toxicity (16). These findings are supported by meta-analytic data and narrative reviews. These reviews indicate a significant increase in the sperm DNA fragmentation index among SSRI users ($p = 0.0002$) (9,14).

Two principal mechanisms have been proposed to explain these molecular effects: oxidative stress and ion channel inhibition. In vivo studies in male rats have shown that citalopram exposure reduces testicular glutathione levels and increases oxidative stress, accompanied by sperm DNA damage and histopathological alterations (11). In parallel, in vitro studies using human sperm exposed to fluoxetine have demonstrated elevated levels of ROS and MDA. These findings also include a reduction in TAC and activation of apoptotic pathways, shown by upregulation of Caspase-8, Caspase-9, and Bax, along with downregulation of the anti-apoptotic BCL-2 gene (13).

In addition to oxidative mechanisms, inhibition of sperm-specific ion channels has also emerged as a relevant pathway. For example, several SSRIs, particularly sertraline, have been identified as potent inhibitors of the CatSper calcium channel. Given that CatSper-mediated calcium influx is essential for sperm hyperactivation and the acrosome reaction, its inhibition may impair fertilization capacity independently of standard semen parameters (10).

Pregnancy and Neonatal Outcomes

Epidemiological data from large population-based cohorts are generally reassuring regarding the safety of paternal SSRI use in relation to pregnancy and neonatal outcomes (22,23). For instance, a nationwide cohort study including 13,547 children exposed to paternal SSRI use during the three months prior to conception found no significant associations with SGA birth, low Apgar scores, major congenital malformations, or infection risk (3). However, a modest but statistically significant increase in the risk of preterm birth was observed (OR: 1.15). This was particularly associated with paternal citalopram and escitalopram use (3). Nevertheless, these findings should be interpreted with caution due to potential confounding by indication. The underlying paternal psychiatric condition could independently influence pregnancy outcomes.

Regarding long-term neurodevelopmental risks, studies have largely pointed towards non-causal associations. Yang et al. investigated the relationship between paternal SSRI use and ADHD and found a statistically significant increase in risk. However, a similar risk was observed in children whose fathers had discontinued treatment long before conception. These findings suggest that the association stems from confounding factors, such as the genetic transmission of psychopathology or shared environmental factors, rather than direct pharmacological effects (18). Similarly, in an analysis of ASD, Yang et al. reported that while initial data suggested an increased risk, further stratification revealed this was likely driven by confounding by indication. The risk persisted among offspring of 'former users'. Limitations of this study included reliance on dispensing data and the inability to adjust for diagnoses managed exclusively in primary care settings (19). In a similar vein, another large nationwide cohort study by Viktorin et al. examined the impact of paternal antidepressant use on preterm birth, malformations, ASD, and intellectual disability. This study found no significant associations and concluded that paternal use is generally safe (2).

Several methodological limitations should be considered when interpreting the findings of this review. First, the reviewed literature is partly based on animal studies, primarily involving rats and mice. The drug doses used in these experiments are often higher than therapeutic levels,

and physiological differences between species may limit their relevance to human clinical settings. Second, most large and high-quality epidemiological data originate from Nordic national registries, particularly those of Sweden and Denmark. This geographic clustering may limit the external validity and generalizability of the results to populations with different ethnic, genetic, and healthcare characteristics. Third, because of confounding by indication, it is difficult to determine whether the reported neonatal or neurodevelopmental outcomes result from SSRI exposure itself or from the underlying paternal psychiatric disorder. Prescription-based analyses cannot verify actual medication use or exposure timing, and missing data on key lifestyle factors such as smoking, alcohol intake, and body mass index may have contributed to residual confounding. Finally, the limited clinical evidence available for newer agents, such as vortioxetine and vilazodone, combined with small sample sizes in some studies, makes it difficult to draw firm conclusions about the reproductive safety of the entire SSRI class.

CONCLUSIONS

Current evidence shows that paternal SSRI exposure can temporarily impair sperm parameters and DNA integrity, but these effects are reversible and do not consistently impact clinical fertility outcomes. Large-scale epidemiological data show that paternal SSRI use does not raise the risk of major congenital anomalies or neurodevelopmental disorders, including autism and ADHD, in offspring. The small increase in preterm birth risk likely results from confounding paternal factors, not a direct drug effect.

As a conclusion of this review, paternal SSRI use does not appear to pose a significant teratogenic risk. Clinically, current data do not justify the routine discontinuation of SSRIs in men trying to conceive, especially when psychiatric stability is essential. Instead, an individualized approach is recommended, focusing on using the lowest effective dose and preferring agents with fewer reproductive side effects, such as sertraline. It may be beneficial to reassure patients that semen alterations appear to be largely reversible after stopping medication (2,9,12). Further prospective studies are warranted to clarify dose dependency, reversibility, and potential long-term effects on offspring.

Conflict of Interest: No conflict of interest was declared by the authors.

Funding: No financial support has been received.

Author Contributions:

Concept and Design: D.C.B, I.Y, E.H
Supervision: I.Y, Y.O.I.
Data Collection and/or Analysis: D.C.B, E.H
Analysis and/or Interpretation: I.Y., E.H., D.C.B
Literature Search: D.C.B, E.H
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Critical Review: I.Y., Y.O.I.

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