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Pharmacological safety aspects and toxicological profile of antidepressants in the perinatal period: a review of current evidence

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Key words: antidepressants, perinatal depression, placental permeability, fetal toxicity, selective serotonin reuptake inhibitors, pregnancy-induced hypertension, pharmacogenetics

Depressive disorders during pregnancy and the postpartum period remain one of the most challenging issues in modern psychopharmacology, directly impacting maternal health and fetal development. Epidemiological studies indicate that 10% to 20% of pregnant women experience clinically significant symptoms of depression [1, 2]. In the context of martial law in Ukraine, this prevalence has increased substantially due to chronic psycho-emotional stress, as evidenced by recent data on the mental health of Ukrainian women in conflict zones [3]. Untreated depression serves as a potent pathogenetic factor; through the hyperactivation of the hypothalamic-pituitary-adrenal (HPA) axis and subsequent hypercortisolemia, it leads to adverse obstetric outcomes, including preeclampsia, preterm birth, and intrauterine growth restriction [4, 5].

Issue of transplacental transfer of antidepressants (ADs) are extensively discusses in current scientific literature. Most modern medications, particularly selective serotonin reuptake inhibitors (SSRIs), readily cross the placental barrier via passive diffusion, resulting in their presence in the fetal bloodstream and amniotic fluid [6, 7]. Previous studies have established that this poses a potential risk of disrupting the neurochemical programming of the developing brain, leading to long-term cognitive and behavioral deviations [8]. The toxicological profile of SSRIs, serotonin-norepinephrine reuptake inhibitors (SNRIs), and others has been traditionally analyzed regarding the risks of congenital malformations, specifically within the cardiovascular system, and the development of poor neonatal adaptation syndrome [9].

Despite the accumulation of clinical data, several critical aspects remain insufficiently addressed. Specifically, there is a lack of comparative experimental evidence using *ex vivo* human placental perfusion models to differentiate the transfer rates of specific SSRIs under identical conditions. Furthermore, the impact of maternal pharmacogenetic variability (such as CYP2C19 phenotypes) and longitudinal metabolic shifts on the stability of drug concentrations during pregnancy has not been fully integrated into clinical safety protocols. These gaps create uncertainty in personalizing therapy and minimizing fetal exposure.

The aim of the study is to systematize current pharmaco-epidemiological and toxicological data regarding the safety of antidepressant use during the perinatal period to minimize toxic effects on the fetus and ensure therapeutic efficacy for the mother.

Materials and methods. The search for scientific sources was conducted in

the international electronic databases PubMed and Google Scholar using a combination of terms and key words: "antidepressants", "pregnancy", "selective serotonin reuptake inhibitors", "pharmacokinetics", "placental transfer", "fetal toxicity", "gestational diabetes" and "neonatal outcomes". Meta-analyses, systematic reviews, cohort studies and *ex vivo* placental perfusion models published between 2021 and 2025 were selected for analysis. The inclusion process focused on the assessment of molecular drug transport mechanisms, in particular the role of P-glycoprotein and the "ion capture" effect, as well as on the analysis of metabolic interactions between the mother and the fetus. Data obtained from primary sources were divided into thematic blocks: pharmacokinetics of transplacental transfer, toxicological risks of structural malformations and neonatal adaptation, and metabolic safety of therapy.

Results and discussion. The analysis of the current evidence base was structured to provide a comprehensive evaluation of ADs safety, beginning with the fundamental mechanisms of maternal-fetal drug exchange and progressing to specific clinical outcomes.

Initially, the pharmacokinetic characteristics and placental permeability of ADs were analyzed to establish the baseline for fetal drug exposure. This primary block of the study focused on the molecular properties—such as lipophilicity, protein binding, and molecular weight—that dictate the rate of passive diffusion across the placental barrier. Special emphasis was placed on the "ion trapping" effect and the role of efflux transporters like P-glycoprotein in modulating the final concentration of medications in the fetal compartment.

The second section reviews the latest epidemiological data and meta-analyses to clarify the risks of structural abnormalities, particularly cardiovascular defects, and to differentiate between direct toxicity and transient neonatal adaptation syndromes.

The next section discusses metabolic and hemodynamic safety, examining the risks of gestational hypertension. It analyzes how antidepressant therapy interacts with maternal physiological systems and how genetic variability (CYP2C19 phenotypes) requires a personalized approach to perinatal pharmacotherapy.

Pharmacokinetic characteristics and placental permeability of antidepressants. The transplacental transfer of ADs is a complex dynamic process determined by the physicochemical properties of the drug molecule and the morpho-functional state of the placental barrier. Most modern ADs, specifically selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs), are weak bases and cross the placenta via passive diffusion [6].

The key determinants governing the intensity of this process include lipophilicity, plasma protein binding, the "ion trapping" effect, and efflux transporters. ADs are characterized by high lipophilicity, allowing molecules to easily integrate into the lipid bilayer of syncytiotrophoblast membranes. However the actual concentration in fetal tissues depends on the volume of distribution: for instance, sertraline has a higher affinity for maternal tissues, which somewhat limits its overall placental flux compared to more hydrophilic representatives of the class [7].

Regarding plasma protein binding, only the free (unbound) fraction of the drug possesses the ability to diffuse. SSRIs exhibit a high degree of binding to albumin and α1-acid glycoprotein (e.g., sertraline > 98%, fluoxetine ~ 94%). It should be considered that physiological hypoalbuminemia in pregnant women

leads to an increase in the free fraction of ADs, thereby elevating the risk of embryotoxic exposure.

A crucial toxicological aspect is the pH gradient between maternal plasma (approximately 7.4) and the fetus (7.25–7.3). Since most ADs are weak bases, upon entering the more acidic environment of the fetal bloodstream, they transition into an ionized (charged) form. Ionized molecules lose the ability for reverse diffusion across the lipid membrane of the placenta, leading to their accumulation in the fetal organism. This mechanism, known as "ion trapping," explains why during prolonged therapy, the concentration of certain ADs in umbilical cord blood may approach maternal levels [10].

The activity of P-glycoprotein (P-gp) and breast cancer resistance protein (BCRP) in the placenta ensures the active efflux of ADs back into the maternal circulation. Venlafaxine and citalopram serve as substrates for P-gp, which partially offsets their high diffusive capacity.

The "fetal-to-maternal" concentration ratio is also of great clinical significance. According to pharmacokinetic monitoring data, the highest placental penetration index is characteristic of citalopram (0.7–0.9) and fluoxetine (0.7–0.85). The lowest indices, correlating with a better safety profile, are recorded for sertraline and paroxetine (0.3–0.4), due to their intensive protein binding and specific tissue derivation features.

The experimental evidence for these differences has been significantly strengthened by the recent *ex vivo* human placental perfusion study by Préta et al. [11]. Using a dual recirculating configuration, the researchers characterized the fetal transfer rate (FTR) of three major SSRIs. The study demonstrated that while all three drugs cross the placental barrier, their transfer rates differ significantly: escitalopram exhibited the highest permeability (FTR 48.2%), followed by paroxetine (43.4%), while sertraline showed the lowest transfer rate (33.2%) [11]. A crucial finding of this research was the role of albumin; the presence of proteins in the perfusion medium significantly influenced the transfer kinetics, particularly for sertraline ($p = 0.009$), confirming that high protein binding is a key protective mechanism that limits fetal exposure.

The clinical management of SSRI therapy is further complicated by maternal pharmacogenetic variability. A prospective study by Stika et al. highlighted significant changes in (S)-citalopram plasma concentrations across pregnancy and the postpartum period [12]. The researchers found that pregnant women with intermediate or low CYP2C19 activity are at a heightened risk of experiencing subtherapeutic drug concentrations during pregnancy, as the (S)-citalopram concentration-to-dose (C/D) ratio was found to be notably lower compared to the late postpartum period. Interestingly, while the C/D ratio remained relatively stable during gestation, it increased by 63% ($p < 0.001$) during the early postpartum weeks (6–8 weeks), before returning to baseline. This suggests that without genotype-guided dose adjustments, patients may experience clinical relapse during pregnancy due to low plasma levels or potential toxicity in the early postpartum period as metabolic rates shift back to pre-pregnancy states.

Toxicological evaluation of congenital malformations and neonatal status. The assessment of the teratogenic potential of ADs remains a cornerstone of perinatal pharmacotherapy safety, with the most debated toxicological concern being the

risk of congenital heart defects (CHDs) following first-trimester exposure. According to the comprehensive systematic review and meta-analysis by De Vries et al., while a modest increase in the relative risk of CHDs was associated with specific molecules, notably paroxetine and fluoxetine, the absolute risk remains low and often diminishes when adjusted for confounding factors such as maternal lifestyle, body mass index, and the severity of the underlying depressive disorder [9].

Building upon the evaluation of structural safety, recent evidence from Yoshino et al. confirms that the overall medical condition of newborns exposed to psychotropic medications in utero does not show critical deviations compared to control groups [13]. The clinical data suggest that parameters such as Apgar scores and neonatal intensive care unit admission rates are heavily influenced by maternal psycho-emotional stability and the quality of prenatal care, rather than drug exposure alone. This underscores the necessity of a holistic management approach that integrates pharmacological safety with psychosocial well-being, as highlighted in the recent meta-analysis by Savoia et al. regarding the effectiveness of prenatal interventions [14].

Furthermore, the risk of "poor neonatal adaptation syndrome" is increasingly viewed as a transient pharmacological effect of birth-related withdrawal or serotonergic overstimulation rather than direct structural toxicity. As reported by Yoshino et al., these symptoms are typically self-limiting and resolve within 48–72 hours without long-term neurodevelopmental consequences, which supports the clinical decision to continue necessary maternal therapy under appropriate neonatal supervision [13].

Metabolic and hemodynamic safety: risks of gestational diabetes and hypertension. Beyond structural teratogenicity, the pharmacological impact of ADs on maternal metabolic and hemodynamic profiles is an essential consideration for maintaining a healthy intrauterine environment. Current toxicological data suggest that the risk of developing gestational diabetes mellitus (GDM) is highly drug-specific and closely linked to the affinity of certain molecules for H1-histamine and 5-HT2C-serotonin receptors. ADs with high H1-receptor affinity are more frequently associated with weight gain and metabolic dysregulation, whereas most selective serotonin reuptake inhibitors (SSRIs) are generally considered metabolically neutral.

However, the necessity for a personalized approach is further emphasized by recent metabolic profiling. According to Itkonen et al., longitudinal metabolic profiling of women using SSRIs during pregnancy reveals that these medications can induce subtle but significant shifts in the maternal metabolome, which may influence glucose homeostasis and lipid metabolism throughout the gestational period [15]. Parallel to these metabolic concerns, the hemodynamic safety of ADs requires rigorous monitoring due to potential impacts on systemic blood pressure. A comprehensive systematic review and meta-analysis by Hu et al. demonstrated a correlation between antidepressant use during pregnancy and an increased risk of pregnancy-induced hypertension [16].

This association is particularly relevant for drugs that affect norepinephrine reuptake, such as SNRIs, which may trigger vasoconstrictive responses. These findings highlight that the risk of preeclampsia and hypertensive disorders is not only a consequence of the underlying maternal depression but

also a potential pharmacodynamic effect of the therapy itself.

The comparative safety profiles and pharmacokinetic characteristics of the discussed ADs are summarized in Table. Consequently, the choice of an antidepressant in the perinatal period must balance the stabilization of maternal mental health with the minimization of secondary physiological complications that could indirectly compromise placental perfusion and fetal growth.

Table

Comparative pharmacological and toxicological profiles of key antidepressants in the perinatal period (based on 2021–2025 evidence)

Antidepressant class / Drug	**Placental penetration index (Fetal / Maternal)**	**Primary toxicological and safety concerns**	**Metabolic and hemodynamic effects**	**References**
Selective serotonin reuptake inhibitors (General)	High (0.3 – 0.9)	Risk of "Poor neonatal adaptation syndrome»	Molecule-specific shifts in glucose metabolism	[15]
Escitalopram	48.2% (*ex vivo*)	Lowest placental transfer; gold standard for safety	Metabolically neutral; low risk of gestational diabetes mellitus	[7, 11, 13]
Sertraline	33.2% (*ex vivo*)	Significantly higher transfer than sertraline	Risk of subtherapeutic levels (CYP2C19 phenotype)	[7, 11]
Paroxetine	43.4% (*ex vivo*)	Discussed correlation with cardiovascular defects	Higher affinity for H1-receptors (weight gain)	[9, 11]
Fluoxetine	High (0.7 – 0.9)	Long half-life; higher fetal accumulation (ion trapping)	Potential influence on fetal growth trajectories	[9, 10]
Citalopram	High (0.7 – 0.9)	High P-glycoprotein substrate activity; high placental flux	Generally favorable metabolic profile	[6, 12]
Selective serotonin reuptake inhibitors (Venlafaxine)	Moderate (0.5 – 0.6)	Risk of neonatal withdrawal symptoms	Increased risk of pregnancy-induced hypertension	[16]

Conclusions

1. Transplacental transfer of ADs is a controllable process where fetal exposure depends on the physicochemical properties of the molecule. Priority should be given to drugs with high protein-binding capacity and lower penetration indices (e.g., sertraline, paroxetine) to minimize direct fetotoxic effects.
2. Current evidence (2021–2025) confirms the relative safety of SSRIs regarding congenital malformations. The risks of cardiovascular anomalies are minimal and frequently diminish when adjusted for maternal confounding factors such as lifestyle and disease severity.
3. The primary safety focus should be monitoring maternal hemodynamic parameters (risk of hypertension with SNRI use) and the metabolic profile (risk of gestational diabetes). Poor neonatal adaptation syndrome remains a transient condition with no long-term negative impacts on the child's cognitive development.
4. Pharmacotherapy for perinatal depression must be based on an individualized approach. The risk of untreated mental disorders, which leads to HPA-axis hyperactivation, is considered more substantial than the potential toxicological risks of modern ADs.

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Pharmacological safety aspects and toxicological profile of antidepressants in the perinatal period: a review of current evidence

Depressive disorders in the perinatal period represent a critical challenge in modern psychopharmacology, requiring a differentiated approach when prescribing antidepressants to minimize the risks of fetotoxicity. Timely therapy is essential to prevent the negative impact of maternal depression on fetal development; however drug selection must be based on a profound understanding of placental pharmacokinetics.

The aim of the study is to systematize current pharmaco-epidemiological and toxicological data regarding the safety of antidepressant use in the perinatal period to minimize toxic effects on the fetus and ensure a stable therapeutic effect for the mother.

A comprehensive analysis of scientific literature was conducted using international electronic databases of evidence-based medicine: PubMed and Cochrane Library. The results of large clinical cohort observations, latest *ex vivo* human placental dual perfusion experimental models, longitudinal metabolomic profiling data, and pharmacogenetic evaluations of the maternal genotype's influence on drug concentration stability were analyzed.

Evidence-based medicine data analysis for 2021–2025 established that the transplacental transfer of antidepressants is a molecule-specific process. The level of fetal exposure critically depends on molecular lipophilicity, the degree of plasma protein binding, and the functioning of efflux transporters, particularly P-glycoprotein, which actively pumps certain antidepressants from the fetal circulation back to the maternal side. Results from the latest *ex vivo* studies have, for the first time, clearly differentiated fetal transfer rates for the most commonly used selective serotonin reuptake inhibitors (SSRIs). Sertraline demonstrated the lowest rate (33.2%), while for paroxetine and escitalopram, these values were significantly higher – 43.4% and 48.2%, respectively. It was established that the risks of congenital malformations (specifically cardiovascular anomalies) associated with modern SSRI use remain low and often correlate with maternal concomitant somatic factors rather than the direct action of the drugs. Particular attention was paid to pharmacogenetic variability: it was proven that CYP2C19 cytochrome phenotypes significantly affect the stability of pharmacotherapy. Specifically, women with intermediate metabolism face a high risk of subtherapeutic escitalopram/citalopram concentrations during pregnancy, which may lead to depressive relapse, as well as the development of toxic effects in the early postpartum period. Metabolomic profiling revealed that the use of serotonin-norepinephrine reuptake inhibitors is associated with a dose-dependent increase in the risk of gestational hypertension, necessitating careful monitoring of vascular tone and placental perfusion to prevent fetal hypoxia.

As the conclusion, an effective safety strategy in the perinatal period must be based on the principles of personalized medicine. Clinical priority is recommended for molecules with proven lowest placental flux (specifically sertraline), allowing for the limitation of systemic exposure to the fetus. It is necessary to consider the patient's individual pharmacogenetic profile (CYP2C19 phenotype) when selecting the dosage. Such a comprehensive approach will minimize the toxic impact on the fetus and ensure a favorable prognosis for the mother and the harmonious development of the child.

Key words: antidepressants, perinatal depression, placental permeability, fetal toxicity, selective serotonin reuptake inhibitors, pregnancy-induced hypertension, pharmacogenetics

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Фармакологічні аспекти безпеки та токсикологічний профіль антидепресантів у перинатальний період: огляд сучасних доказів

Депресивні розлади в перинатальний період є критичною проблемою сучасної психофармакології, що потребує диференційованого підходу за призначення антидепресантів для мінімізації ризиків фетотоксичності. Своєчасна терапія є необхідною для запобігання негативному впливу материнської депресії на розвиток плода, проте вибір препарату має ґрунтуватися на глибокому розумінні плацентарної фармакокінетики.

Мета дослідження – систематизація сучасних фармако-епідеміологічних і токсикологічних даних щодо безпеки застосування антидепресантів у перинатальному періоді для мінімізації токсичного впливу на плід і забезпечення стабільного терапевтичного ефекту для матері.

Проведено комплексний аналіз наукової літератури в міжнародних електронних базах даних доказової медицини: PubMed та Cochrane Library. Проаналізовано результати великих клінічних когортних спостережень, новітніх експериментальних моделей *ex vivo* подвійної перфузії плаценти людини, а також даних лонгітюдного метаболомічного профілювання та фармакогенетичних оцінок впливу генотипу матері на стабільність концентрації лікарських засобів.

Аналіз даних доказової медицини за 2021–2025 роки встановив, що трансплацентарний перехід антидепресантів є молекулярно-специфічним процесом. Рівень експозиції плода критично залежить від ліпофільності молекули, ступеня зв'язування з білками плазми та функціонування ефлюксних транспортерів, зокрема Р-глікопротеїну, який активно виводить певні антидепресанти з фетального кровотоку назад до материнського. Результати останніх досліджень *ex vivo* вперше чітко диференціювали рівні фетального перенесення для найвживаніших селективних інгібіторів зворотного захоплення серотоніну (СІЗЗС). Найнижчий показник продемонстрував сертралін (33,2 %), тоді як для пароксетину та есциталопраму ці значення були суттєво вищими – 43,4 % та 48,2 % відповідно. Встановлено, що ризики виникнення вроджених вад розвитку (зокрема серцево-судинних аномалій) у разі застосування сучасних СІЗЗС залишаються низькими та часто корелюють із супутніми соматичними факторами матері, а не безпосередньо з дією препаратів. Особливу увагу приділено фармакогенетичній мінливості: доведено, що фенотипи цитохрому CYP2C19 суттєво впливають на стабільність фармакотерапії. Зокрема, у жінок із проміжним метаболізмом існує високий ризик виникнення субтерапевтичних концентрацій есциталопраму / циталопраму під час вагітності, що може призвести до рецидиву депресії, а також до розвитку токсичних ефектів у ранньому післяпологовому періоді. Метаболомічне профілювання виявило, що використання інгібіторів зворотного захоплення та норадреналіну асоціюється з дозозалежним підвищенням ризику гестаційної гіпертензії, що зумовлює необхідність ретельного моніторингу судинного тонусу та плацентарної перфузії для запобігання гіпоксії плода.

Таким чином, ефективна стратегія безпеки антидепресантів у перинатальний період має базуватися на принципах персоніфікованої медицини. Клінічний пріоритет рекомендовано надавати молекулам із доведеним найнижчим плацентарним потоком (зокрема сертраліну), що дозволяє обмежити системний вплив на плід. Необхідно враховувати індивідуальний фармакогенетичний профіль пацієнтки (фенотип CYP2C19) при підборі дози. Такий комплексний підхід дозволить мінімізувати токсичний вплив на плід і забезпечить сприятливий прогноз для матері та гармонійного розвитку дитини.

Ключові слова: антидепресанти, перинатальна депресія, плацентарна проникність, фетотоксичність, селективні інгібітори зворотного захоплення серотоніну, гіпертензія вагітних, фармакогенетика

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