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REVISION

Epilepsy in pregnancy: Antiseizure medication selection and fetal risk

Epilepsia na gestação: Escolha dos medicamentos anticrise e risco fetal

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Abstract

Epilepsy management during pregnancy requires effective prevention of maternal seizures while limiting avoidable fetal exposure to medications with structural and neurodevelopmental toxicity. Treatment decisions depend on the epilepsy syndrome, previous treatment response, medication dose, gestational timing, and the risks of modifying an effective treatment regimen. This narrative

review examines comparative fetal risks, preconception planning, pharmacokinetic monitoring, folate supplementation, and care after delivery. Targeted PubMed searches and November 2026 guidance identified prospective pregnancy registries, developmental cohorts, pharmacokinetic investigations, population-based studies, and multidisciplinary recommendations. Evidence was synthesized descriptively without a new meta-analysis. The 2024 EURAP analysis reported major congenital malformations in 9.9% (153/1,549) of valproate-exposed pregnancies, compared with 3.1% (110/3,584) for lamotrigine, 2.9% (13/443) for oxcarbazepine, and 2.5% (33/1,325) for levetiracetam, within a nonrandomized monotherapy cohort. Valproate also has the most consistent association with adverse cognitive and developmental outcomes. Findings for topiramate vary by outcome and study design, with established concerns about oral clefts and fetal growth and uncertainty about neurodevelopmental effects. Lower observed malformation rates do not establish complete safety, and information remains limited for several newer agents. Pregnancy-related concentration declines require individualized monitoring, while postpartum normalization of clearance creates a risk of toxicity if increased doses are continued unchanged. Folate should be prescribed before conception and during pregnancy, although the optimal dose remains unsettled. An effective medication should not be abruptly discontinued after pregnancy recognition. Care combines syndrome-specific prescribing, informed discussion of absolute risks, planned monitoring, appropriate fetal assessment, and coordinated neurological and obstetric follow-up.
Keywords: Epilepsy; Pregnancy; Anticonvulsants; Congenital Abnormalities.

Resumo

O manejo da epilepsia durante a gestação exige prevenir crises maternas e limitar a exposição fetal evitável a medicamentos associados a toxicidade estrutural e do neurodesenvolvimento. A escolha terapêutica depende da síndrome epiléptica, da resposta anterior, da dose, do período gestacional e dos riscos de alterar um tratamento eficaz. Esta revisão narrativa analisa riscos fetais comparativos, planejamento pré-concepcional, monitorização farmacocinética, suplementação de folato e cuidados após o parto. Buscas direcionadas no PubMed e recomendações profissionais de novembro de 2026 forneceram estudos de registros gestacionais, coortes de desenvolvimento, investigações farmacocinéticas e estudos populacionais. A síntese foi descritiva, sem nova metanálise. No registro EURAP de 2024, a prevalência de malformações congênitas maiores foi de 9,9% (153/1.549) com valproato, 3,1% (110/3.584) com lamotrigina, 2,9% (13/443) com oxcarbazepina e 2,5% (33/1.325) com levetiracetam, em uma coorte não randomizada de monoterapia. O valproato também apresenta associação consistente com desfechos cognitivos e do desenvolvimento desfavoráveis. Para o topiramato, existem preocupações com fissuras orais e crescimento fetal, além de incertezas sobre efeitos específicos no neurodesenvolvimento. Menores taxas observadas de malformações não comprovam segurança absoluta, e os dados para fármacos novos permanecem limitados. Alterações das concentrações durante a gestação demandam monitorização individualizada, enquanto a normalização pós-parto da depuração exige reavaliação de doses. Recomenda-se suplementação de folato antes e durante a gravidez, embora a dose ideal permaneça incerta. Medicamentos eficazes não devem ser interrompidos abruptamente após o

reconhecimento da gravidez. A conduta integra escolha conforme a síndrome, discussão dos riscos absolutos, avaliação fetal e acompanhamento neurológico e obstétrico.
Palavras-chave: Epilepsia; Gravidez; Anticonvulsivantes; Anormalidades Congênitas.

Introduction

Pregnancy in a patient with epilepsy introduces competing concerns about convulsive seizures and fetal medication exposure that require coordinated neurological and obstetric care. Seizures can produce injury and physiological compromise, whereas treatment decisions made without attention to reproductive safety may expose the fetus to preventable harm. A management plan should therefore be established before conception whenever possible and should remain responsive to changes in seizure control, drug exposure, and the patient's priorities throughout pregnancy [1].

Fetal risk includes structural malformations, impaired growth, and developmental outcomes that may become evident only years after birth. The Neurodevelopmental Effects of Antiepileptic Drugs (NEAD) study demonstrated clinically important differences in childhood cognition after exposure to different monotherapies, establishing why an anatomically normal newborn does not complete the assessment of medication-associated risk. These outcomes require separate discussion because a reassuring finding for one endpoint cannot establish safety for every other endpoint [2].

The dose that controlled seizures before conception may not produce the same circulating concentration during pregnancy. Prospective pharmacokinetic evidence shows substantial exposure changes for several commonly used medications, making fixed-dose continuation an incomplete strategy for some patients. This review examines how comparative fetal evidence, syndrome-specific treatment, concentration monitoring, and reproductive counseling can be integrated without destabilizing effective seizure control [3].

Methods

Review design and information sources

This article is a narrative review based on targeted PubMed searches and relevant professional guidance consulted in November 2026. Search concepts included epilepsy, pregnancy, antiseizure medication, congenital malformation, neurodevelopment, fetal growth, valproate, lamotrigine, levetiracetam, oxcarbazepine, topiramate, therapeutic drug monitoring, folic acid, and breastfeeding. Original studies were prioritized for quantitative results, and multidisciplinary guidance was used to contextualize clinical decisions.

For this revision, PubMed searches were last verified on 14 November 2026 using "epilepsy AND pregnancy AND ("antiseizure medication" OR "antiepileptic drugs") AND ("congenital malformations" OR neurodevelopment)"; "epilepsy AND pregnancy AND ("therapeutic drug monitoring" OR "folic acid" OR breastfeeding)"; "epilepsy AND pregnancy AND (guideline

[Publication Type] OR consensus)". No publication-date or language filters were applied to these verification searches. Guidelines and consensus reports were identified through PubMed and selected for pregnancy-specific recommendations with a stated evidence-review or expert-consensus basis.

Publications were selected for direct relevance to medication choice, maternal seizure control, fetal outcomes, or management across pregnancy and the postpartum period. Recent studies were prioritized, with older prospective investigations retained when they established important developmental or pharmacokinetic findings, and overlapping registry populations were recognized as related evidence rather than independent replication. Selection was clinically focused rather than exhaustive, and no registered protocol, duplicate independent screening, formal risk-of-bias assessment, or new patient data collection was undertaken.

Evidence synthesis and interpretation

Statistical synthesis was descriptive, preserving each study's population, exposure window, outcome definition, denominator, and follow-up period. Reported proportions, adjusted effect estimates, confidence intervals, and cognitive scores were interpreted within the original design, with attention to confounding by indication, dose, treatment changes, missing follow-up, and multiple comparisons. No new pooled risk, comparative effect, heterogeneity statistic, publication-bias test, or inferential analysis was calculated because the selected studies addressed different endpoints and were not assembled through a systematic quantitative-synthesis protocol.

Results

Professional guidance on clinical priorities and treatment selection

The 2024 guideline from the American Academy of Neurology, American Epilepsy Society, and Society for Maternal-Fetal Medicine prioritizes prevention of convulsive seizures while recommending consideration of lamotrigine, levetiracetam, or oxcarbazepine when appropriate for the epilepsy syndrome and comorbidities. It advises avoiding valproate when clinically feasible because of congenital malformation and neurodevelopmental risks, while warning against casually removing or replacing treatment that is effectively controlling convulsive seizures during an established pregnancy. These recommendations support individualized selection rather than an assumption that the medication with the lowest reported fetal risk will necessarily control every patient's epilepsy [4].

Preconception review should verify the diagnosis and seizure classification, previous responses and failures, current adherence, and the practical consequences of a breakthrough convulsive seizure. A transition to another medication, when appropriate, is best completed with specialist supervision before conception, allowing time to establish effectiveness and tolerability rather than exposing pregnancy to an untested regimen. Counseling should also address interactions with contraception, a plan for pregnancy recognition, and a documented approach to monitoring and rescue treatment [1].

These paragraphs summarize professional recommendations; the subsequent subsections report findings from primary studies.

Seizure control during pregnancy

The Maternal Outcomes and Neurodevelopmental Effects of Antiepileptic Drugs study, abbreviated MONEAD, enrolled 351 pregnant women and 109 nonpregnant controls with epilepsy to distinguish pregnancy-associated changes from background variation. Among participants with the relevant seizure history and complete epoch data, seizures impairing awareness increased in 70/299 pregnant participants (23%) and 23/93 controls (25%; odds ratio 0.93, 95% confidence interval [CI] 0.54–1.60), while medication-dose changes occurred in 74% and 31%, respectively. This finding is reassuring about outcomes under active specialist management, but it does not show that pregnancy has no pharmacological consequences or that the observed stability would persist without individualized dose adjustment [5].

The same comparison illustrates why seizure outcomes must be interpreted together with management intensity rather than as an isolated natural-history observation. More frequent changes in medication doses were part of the clinical context in which pregnant participants achieved seizure outcomes similar to those of controls. A patient with increasing seizures, vomiting, interrupted medication access, or altered adherence therefore requires reassessment rather than reassurance based solely on average cohort results [5].

Valproate withdrawal after conception

An observational analysis from the International Registry of Antiepileptic Drugs and Pregnancy (EURAP) compared pregnancies in which valproate was withdrawn, replaced, or maintained during the first trimester. Generalized tonic-clonic seizures occurred in 33% of the withdrawal group and 29% of the switch group, compared with 16% among those maintaining treatment, although treatment selection and disease severity prevent a causal interpretation. The results reinforce the distinction between reducing avoidable valproate exposure through preconception planning and making an abrupt change after conception that may introduce additional maternal and fetal danger without reversing exposure that has already occurred [6].

Comparative congenital malformation risk

The 2024 EURAP prospective analysis included 9,840 pregnancies exposed to eight frequently used monotherapies, with major congenital malformations assessed through one year after birth. Reported prevalences were 9.9% (153/1,549; 95% CI 8.5–11.5%) for valproate, 6.3% (9/142; 95% CI 3.4–11.6%) for phenytoin, 6.2% (21/338; 95% CI 4.1–9.3%) for phenobarbital, 5.4% (121/2,255; 95% CI 4.5–6.4%) for carbamazepine, 4.9% (10/204; 95% CI 2.7–8.8%) for topiramate, 3.1% (110/3,584; 95% CI 2.5–3.7%) for lamotrigine, 2.9% (13/443; 95% CI 1.7–5.0%) for oxcarbazepine, and 2.5% (33/1,325; 95% CI 1.8–3.5%) for levetiracetam. These absolute proportions are clinically informative, but different group sizes, nonrandomized prescribing, and residual differences in patients and doses preclude treating them as precise head-to-head rankings for an individual pregnancy [7].

EURAP's earlier comparison similarly found important between-drug differences and demonstrated that dose at conception influenced malformation risk for several medications. A dose-dependent association supports using the lowest dose that reliably controls the relevant seizures, but does not establish a universally harmless dose or justify reducing treatment below an effective exposure. The clinical target is an effective lower-risk regimen, with dose reduction considered in relation to seizure history and fetal evidence rather than as an independent numerical objective [8].

The 2018 and 2024 EURAP publications belong to the same continuing registry and should not be described as wholly independent confirmations from unrelated populations. Their value lies partly in the expansion of exposure information and the assessment of changing prescribing patterns, while limitations inherent to observational pregnancy registration remain. Comparisons should retain the definition of major congenital malformation and the duration of infant follow-up, because ascertainment restricted to birth would not necessarily produce the same estimates [8].

Topiramate and specific structural risks

A population-based cohort of more than 1.3 million pregnancies found oral clefts in 4.1 per 1,000 topiramate-exposed live births compared with 1.1 per 1,000 unexposed births, with an adjusted risk ratio of 2.90 and a 95% confidence interval of 1.56–5.40. The association was stronger among patients treated for epilepsy and in higher-dose analyses, although indication and dose were closely related in the observed population. This illustrates why a medication's aggregate malformation prevalence should be supplemented by information on particular defects, exposure timing, and the clinical dose range relevant to the patient [9].

Fetal growth outcomes

The North American pregnancy registry compared singleton infants exposed to topiramate or zonisamide monotherapy with those exposed to lamotrigine. Small-for-gestational-age birth occurred in 17.9%, 12.2%, and 6.8%, respectively, with a significant adjusted association for topiramate and a less precise estimate for zonisamide. These observations raise a separate growth concern that is not resolved by a normal anatomical scan and should not be equated with a proven increase in every adverse perinatal outcome or with a causal estimate applicable to all treatment combinations [10].

Valproate and cognitive development

In the prospective Neurodevelopmental Effects of Antiepileptic Drugs study, 305 mothers and 311 children contributed to the primary analysis, and 224 children completed six-year follow-up. Adjusted mean intelligence quotient was 97 after valproate exposure, compared with 105 after carbamazepine and 108 after lamotrigine or phenytoin exposure, with a dose-dependent valproate association across several cognitive domains. Although observational allocation and incomplete follow-up limit causal precision, the consistency and clinical relevance of these findings distinguish valproate from medications whose long-term risks remain largely uncertain because exposure samples are small [2].

Autism and the interpretation of conflicting findings

A 2024 United States cohort assessed medication exposure during the second half of pregnancy and reported an adjusted autism hazard ratio of 2.67 for valproate compared with no medication exposure among children of mothers with epilepsy. In the same analysis, adjusted estimates were 0.96 for topiramate and 1.00 for lamotrigine, with confidence intervals that included the null. The crude group differences therefore require adjustment for the underlying indication and related characteristics, and the relatively reassuring topiramate result for autism does not negate its established structural and growth concerns [11].

The Nordic register-based study of antiepileptic drugs in pregnancy (SCAN-AED) reported increased autism and intellectual-disability risks after prenatal exposure to topiramate or valproate, with higher estimates in some dose analyses. Differences from the United States findings include exposure definitions, population characteristics,

outcomes, follow-up, and potential residual confounding, which prevent a simple declaration that one study invalidates the other. Counseling should acknowledge the conflicting topiramate neurodevelopmental evidence while recognizing that the valproate signal is more consistent across different designs and developmental endpoints [12].

A separate United Kingdom and Swedish cohort of more than three million children also reported associations between valproate exposure and several developmental outcomes, with selected signals for other medications. Topiramate exposure was associated with intellectual disability, but confidence intervals were wide, and combined-treatment analyses were particularly susceptible to differences in epilepsy severity. Large administrative datasets improve statistical reach for uncommon outcomes, yet their size does not remove exposure misclassification, unmeasured familial influences, or the limitations of prescription dispensing as a measure of actual ingestion [13].

A 2026 United States cohort broadened developmental assessment beyond autism and again identified associations for valproate, alongside findings requiring further evaluation for zonisamide. Signals involving other drugs were generally modest or based on uncommon outcomes and multiple comparisons, with several intellectual-disability estimates remaining imprecise. Because this investigation and earlier United States analyses used related healthcare databases and overlapping calendar periods, their findings should be interpreted as an extension of the evidence rather than automatically counted as fully independent population replication [14].

Developmental outcomes with contemporary treatment

MONEAD's three-year assessment found no difference in its primary verbal outcome between children of women with epilepsy and children of women without epilepsy. The cohort predominantly reflected contemporary prescribing, making it useful for counseling about commonly used newer regimens while providing limited information about agents with few exposures. A similar group mean cannot exclude effects within a specific drug or concentration range, and early childhood assessment does not capture every learning or behavioral difficulty that may emerge at school age [15].

At six years, the MONEAD analysis compared 298 children of women with epilepsy and 89 children of healthy women and found no significant difference in the primary Verbal Index Score. Lamotrigine or levetiracetam, alone or in combination, accounted for 78% of treated maternal exposures in the analyzed epilepsy group, and some outcomes varied with medication exposure. The results support cautious reassurance for well-managed contemporary treatment while underscoring that group-level findings cannot establish complete developmental safety or be generalized to poorly represented drugs [16].

Newer medications and incomplete safety information

A lacosamide pharmacovigilance analysis reported 202 prospective maternal-exposure cases with known outcomes, including 44 monotherapy cases and 158 polytherapy cases; two twin pregnancies in the polytherapy group accounted for 160 polytherapy outcomes and 204 outcomes overall. Malformations were reported in one of 44 monotherapy pregnancy outcomes and 11 of 160 polytherapy outcomes, but the small monotherapy sample, selective reporting, different treatment contexts, and incomplete outcome availability limit comparison with established registries. These preliminary findings should not be converted into a claim that

lacosamide is safer than better-characterized alternatives, nor should polytherapy and monotherapy percentages be treated as an unbiased estimate of the effect of adding medication [17].

Discussion

Pharmacokinetics and individualized monitoring

MONEAD documented pregnancy-related reductions in dose-normalized concentrations of up to 56.1% for lamotrigine and 36.8% for levetiracetam, with relevant changes also observed for oxcarbazepine and several other agents. A useful monitoring plan relates measured concentrations to the patient's own effective baseline and interprets them together with seizure frequency, adherence, tolerability, and sampling conditions. These concentration changes demonstrate altered exposure, but do not justify applying one automatic dose increase to every patient or escalating medication solely to match a population laboratory reference interval [3].

A prospective lamotrigine study found increased clearance throughout pregnancy and an association between lower ratios to an individual target concentration and seizure worsening. A ratio below 0.65 was predictive in that specific cohort, while an empirical postpartum taper was associated with reduced maternal toxicity. The finding supports baseline-oriented monitoring and a documented reduction plan after delivery, with the numerical threshold treated as study-specific evidence rather than a universally validated cutoff for all medications or epilepsy syndromes [18].

Postpartum dose review is especially important when treatment was escalated during gestation, because clearance can move toward the nonpregnant state while sleep disruption and missed doses simultaneously threaten seizure control. The lamotrigine cohort demonstrates that preventing toxicity and preserving seizure control are linked tasks requiring a planned transition rather than abrupt cessation or indefinite continuation of the final gestational dose. Clinical reassessment should account for symptoms of excessive exposure and the patient's individual trajectory instead of assuming that every patient returns to baseline on the same day [18].

Folate supplementation and uncertainty about dose

The 2024 multidisciplinary guideline recommends at least 0.4 mg of folic acid daily before conception and during pregnancy for patients receiving antiseizure medication. This minimum should not be confused with proof that one dose is optimal for every clinical situation, and higher-dose prescribing should reflect individual indications and applicable guidance. Appropriate fetal structural assessment and growth surveillance when indicated remain necessary because folate supplementation does not eliminate medication-associated malformation or developmental risk [4].

A reanalysis of the Neurodevelopmental Effects of Antiepileptic Drugs cohort associated periconceptional folate with better cognitive outcomes at three and six years. The association persisted after adjustment for several measured characteristics, but supplementation was not randomly assigned and may still mark differences in pregnancy planning, access to care, and other health behaviors. Its contribution is supportive evidence for timely supplementation rather than a randomized demonstration of the best dose or a guarantee that folate prevents the developmental effects of valproate exposure [19].

A Nordic registry investigation associated prescribed high-dose folic acid with childhood cancer among offspring of mothers with epilepsy, based on 18 exposed and 29 unexposed cases, with estimated cumulative risks to age 20 years of 1.4% and 0.6%, respectively. The small event count, wide confidence interval, prescription-based exposure measure, and possible confounding by indication prevent a causal conclusion and should be considered alongside studies reporting developmental benefits of supplementation. This unresolved signal supports research and individualized high-dose decisions, while providing no basis for advising patients to abandon the recommended routine folate supplementation [20].

Breastfeeding and continuity of care

A MONEAD breastfeeding investigation obtained 164 matched infant–mother concentration pairs from 138 infants and found that approximately half of measured infant drug concentrations were below quantification limits. Infant exposure varied by medication, and maternal concentrations were related to infant lamotrigine concentrations, supporting attention to the specific regimen rather than a blanket assumption that all transfer is negligible. The evidence supports breastfeeding for many treated patients while retaining individualized infant observation, maternal dose review, and specialist advice when sedation, poor feeding, or other concerns arise [21].

Evidence limitations and clinical application

Fetal endpoints require separate interpretation because normal anatomy does not establish normal later development. Large observational cohorts remain vulnerable to confounding, exposure misclassification, and imprecise estimates for rare outcomes. Counseling should distinguish established risks, comparative reassurance, and unresolved uncertainty [14].

This review is limited by its targeted PubMed search and nonexhaustive publication selection. No formal assessment of the certainty of the evidence was performed. These features may restrict coverage and the strength and generalizability of the narrative conclusions.

Table 1 - *Original studies informing the review Primary study reports are listed; guidance is cited in the text. Shared-cohort reports are separate analyses, not independent populations.*

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
Meador et al (2013).[2]	Prospective NEAD cohort; 305 mothers and 311 children; 224 completed six-year assessment.	Lower adjusted intelligence quotient after valproate exposure.	Dose-related cognitive concerns; observational treatment≈assignment.
Pennell et al (2022).[3]	MONEAD prospective pharmacokinetic cohort; pregnant and nonpregnant epilepsy groups.	Substantial gestational concentration declines, especially lamotrigine and levetiracetam.	Supports individualized monitoring.
Pennell et al (2020).[5]	MONEAD; 351 pregnant women and 109 epilepsy controls.	Similar seizure-worsening proportions; more dose changes during pregnancy.	Outcomes occurred under active management.
Tomson et al (2016).[6]	EURAP; withdrawal 93, switch 38, maintained valproate 1,588 pregnancies.	Convulsive seizures more frequent after withdrawal or switching.	Nonrandomized changes cannot≈establish causality.
Battino et al (2024).[7]	EURAP prospective monotherapy analysis; 9,840≈pregnancies involving eight medications.	Malformation prevalence varied substantially between drugs.	Lower observed prevalence with levetiracetam, oxcarbazepine, and lamotrigine.
Tomson et al (2018).[8]	Earlier EURAP prospective comparison of eight monotherapies.	Drug-specific and dose-related malformation differences.	Overlaps the continuing EURAP registry.

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
Hernandez-Diaz et al (2018).[9]	Population-based pregnancy cohort; first-trimester topiramate exposure.	Oral clefts 4.1 versus 1.1 per 1,000 exposed and unexposed births.	Dose and indication affect interpretation.
Hernández-Díaz et al (2014).[10]	Registry; topiramate 347, zonisamide 98, and lamotrigine 1,581 singleton neonates.	Higher small-for-gestational-age prevalence with topiramate.	Growth is a distinct fetal endpoint.
Hernández-Díaz et al (2024).[11]	United States cohort; second-half gestational exposure and autism follow-up.	Adjusted valproate association persisted; topiramate association attenuated.	Indication adjustment matters.
Bjørk et al (2022).[12]	Nordic SCAN-AED cohort; medication-exposed and unexposed children.	Topiramate and valproate developmental associations.	Exposure definitions differ across studies.
Madley-Dowd et al (2024).[13]	United Kingdom and Swedish cohort; more than three million children.	Valproate and selected other drug associations with developmental diagnoses.	Residual confounding remains possible.
Straub et al (2026).[14]	United States cohort; multiple developmental endpoints and individual drugs.	Valproate associations and additional signals requiring confirmation.	Rare outcomes and multiple comparisons limit certainty.

Reference	Population / Intervention / Comparison	Outcomes	Main conclusions
Meador et al (2023).[15]	MONEAD offspring assessment at three years.	No primary verbal-outcome difference between maternal groups.	Limited information for less-used drugs.
Meador et al (2025).[16]	MONEAD at six years; 298 children of women with epilepsy and 89 controls.	No significant primary verbal-score difference.	Predominantly lamotrigine and levetiracetam exposures.
Perucca et al (2024).[17]	Lacosamide pharmacovigilance; 202 prospective maternal-exposure cases: 44 monotherapy and 158 polytherapy, including two twin pregnancies; 204 outcomes overall.	One malformation among 44 monotherapy outcomes and 11 among 160 polytherapy outcomes.	Small, selected sample; comparative safety unestablished.
Pennell et al (2008).[18]	Prospective lamotrigine study; 53 pregnancies.	Clearance increased; individualized concentration related to seizure risk.	Postpartum taper reduced toxicity likelihood.
Meador et al (2020).[19]	NEAD reanalysis of the same developmental cohort.	Periconceptional folate associated with better cognition.	Not an independent cohort or≈randomized dose trial.
Vegrim et al (2022).[20]	Nordic registry study; 27,784 children of mothers with epilepsy.	High-dose folate association with childhood cancer; small event counts.	Causality and optimal dosing remain unresolved.
Birnbaum et al (2020).[21]	MONEAD breastfeeding analysis; 164 matched concentration pairs from 138 infants.	Infant concentrations generally below maternal concentrations.	Transfer varies by medication.

Legend: EURAP, International Registry of Antiepileptic Drugs and Pregnancy; NEAD, Neurodevelopmental Effects of Antiepileptic Drugs; MONEAD, Maternal Outcomes and Neurodevelopmental Effects of Antiepileptic Drugs; SCAN-AED, Nordic register-based study of antiepileptic drugs in pregnancy.

Conclusion

Antiseizure medication selection in pregnancy should preserve effective seizure control while minimizing avoidable fetal risk through planning before conception whenever possible. Valproate has the most consistent structural and neurodevelopmental concerns, while lamotrigine, levetiracetam, and oxcarbazepine are important alternatives when appropriate for the epilepsy syndrome and supported by the available outcome-specific evidence. Individualized concentration monitoring, folate supplementation, coordinated fetal assessment, breastfeeding counseling, and postpartum dose review complete a management strategy that avoids both undertreatment and unsupported claims of absolute safety.

Declaration of Artificial Intelligence Use

OpenEvidence was used to support literature searches, the organization of scientific evidence, and the preparation and revision of the manuscript. The tool was not credited with authorship and was not used to fabricate data, results, or references. All scientific content, references, and interpretations were critically reviewed and validated by the authors, who approved the final submitted version and assume full responsibility for the integrity and accuracy of the work.

Conflicts of Interest

The authors declare no conflicts of interest.

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Author Contributions

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