MedCrave
Step into the World of Research

Journal of Psychology & Clinical Psychiatry

Review article

Open Access

CrossMark

Developing tapering protocols for psychotropic medications in adolescents with fetal alcohol spectrum disorder: incorporating relational, environmental, and cultural aspects of care

Abstract

Psychotropic medications are commonly prescribed to adolescents with Fetal Alcohol Spectrum Disorder (FASD), despite limited disorder-specific evidence and the increased vulnerability of this group to adverse and paradoxical reactions. Inspired by presentations and debates at the International Conference on FASD, organized by the Santa Casa- Health Department of the Pontifical Delegation of Loreto-Italy (Dipartimento Sanitario "Santa Casa" della Delegazione Pontificia di Loreto), this article emphasizes the need for clearer, ethically grounded tapering strategies. Patterns of polypharmacy, inconsistent clinical responses, and iatrogenic effects underscore the urgency of creating structured tapering guidelines and protocols based on developmental, relational, and cultural factors.

Drawing on clinical experience in Italy and the United States—including work within Native American communities where structural inequities amplify the consequences of pharmacological overreliance—the article presents a tapering model that integrates neurodevelopmental vulnerability, environmental stability, relational continuity, and cultural context. Tapering is reframed as a therapeutic process aimed at restoring autonomy, reducing exposure to unnecessary medications, and strengthening the individual's relational and cultural resources.

The discussion emphasizes the importance of understanding behavioral changes from a neurodevelopmental perspective, fostering collaboration among caregivers, educators, and community resources, and addressing systemic pressures that contribute to medication overuse. The article concludes by stressing the need for long-term pilot studies to evaluate the practicality and impact of this framework in different settings. Such research will be essential for refining tapering strategies that are aligned with current neurodevelopmental stages, ethical standards, and community-based psychosocial and recovery models.

Keywords: fetal alcohol spectrum disorder (FASD), psychotropic medication tapering, relational and culturally informed care, neurodevelopmental vulnerability

Volume 17 Issue 1 - 2026

Marcello Maviglia,[1] Mauro Ceccanti,[2] Fiorenzo Mignini,[3] Norman Cooeyate,[4] Donald Hume,[5] Mario Vitali,[6] Marisa Patrizia Messina[7]

[1]Clinical Professor, Family and Community Medicine/ Core Faculty Member at CNAH (Center for Native American Health)/ University Of New Mexico, USA
[2]Faculty of Medicine, Sapienza University of Rome, Italy
[3]Director of the Santa Casa – Health Department of the Pontifical Delegation of Loreto, Italy
[4]Department of Family and Community Medicine, University of New Mexico Health Sciences Center/ Core Faculty, Center for Native American Health (CNAH), University of New Mexico, USA
[5]Senior Peer Specialist, Presbyterian Healthcare Services, USA
[6]Psychiatrist, AST4 Fermo, Italy
[7]Department of Maternal–Infant Health and Urology, Sapienza University of Rome, Italy

Correspondence: Marcello Maviglia, MD, MPH, Clinical Professor, Family and Community Medicine/ Core Faculty Member at CNAH (Center For Native American Health) / University of New Mexico, Albuquerque, USA

Received: December 26, 2025 | **Published:** January 15, 2026

Introduction

Psychotropic medications remain a central component of treatment for many adolescents with Fetal Alcohol Spectrum Disorder (FASD), even though no disorder-specific evidence supports their use.[1–5] The American Academy of Pediatrics notes that pharmacological interventions for FASD are largely extrapolated from other neurodevelopmental conditions and lack rigorous validation.[1] Epidemiological studies consistently document high rates of prescribing and polypharmacy, with variable therapeutic benefit and frequent adverse effects.[3,4,6,7]

In Italy, recent national guidelines highlight prevention, personalized care, and non-drug interventions as primary strategies.[8] These themes were reinforced at the International Conference on FASD in Loreto, Italy, where clinicians emphasized the risks associated with early and prolonged psychiatric medications use in vulnerable populations.[9–11]

Parallel clinical experience in the United States, particularly within Native American communities, reinforces these concerns. Historical trauma, structural inequities, and intergenerational adversity intersect with high rates of prenatal alcohol exposure, creating contexts in which standardized pharmacological approaches often offer limited benefit and may contribute to iatrogenic complications.[10,12,13] Collaborative work at the Center for Native American Health has highlighted the importance of culturally grounded, relational, and narrative approaches that attend to ecological and symbolic dimensions of care.[12–14]

At the same time, algorithmic prescribing frameworks—while useful—do not fully account for the heterogeneity of neurocognitive functioning, environmental instability, or the risk of misinterpreting iatrogenic reactions as deterioration of the primary condition.[5,6,15]

Neurobiological research further underscores the need for caution. Structural and functional imaging studies reveal widespread alterations in connectivity, cortical organization, and network efficiency in children with prenatal alcohol exposure.[3,16–19] These findings suggest that the neurobiological substrates targeted by psychotropic medications may differ substantially from those in other populations, increasing the likelihood of unpredictable or exaggerated pharmacodynamic responses.

Taken together, these observations point to a persistent gap between prescribing practices and the current evidence base. The reliance on psychotropic medications in FASD reflects broader systemic tendencies toward medicalization rather than a robust therapeutic rationale.[20–22] This context underscores the need for structured, ethically grounded tapering protocols that integrate neurobiological,

relational, and cultural considerations and support more sustainable pathways of care.

Psychotropic medications and FASD: evidence gaps and iatrogenic risks

Psychotropic medications are often prescribed to people with Fetal Alcohol Spectrum Disorder (FASD) despite limited evidence of their effectiveness.[1,3,5,6] These treatments are frequently adapted from those used for ADHD, mood disorders, or disruptive behavior disorders, even though the neurobehavioral profile of FASD is unique and not directly comparable to these conditions.[1,2,5]

Epidemiological studies consistently reveal high rates of psychotropic prescription, polypharmacy, and inconsistent clinical responses.[3,6,7] Adverse effects—including sedation, cognitive dulling, irritability, and paradoxical activation—are common and often mistaken for worsening of the underlying condition.[6,7,23] This misunderstanding can lead to increased doses or additional medications, creating a cycle of pharmacological dependence.

Current clinical guidance on using medications for disruptive behaviors in FASD highlights the limited evidence and increased risk of adverse reactions, emphasizing the need for careful and individualized prescribing.[24] The risk of iatrogenic harm is further magnified by the neurodevelopmental vulnerabilities associated with prenatal alcohol exposure. Here, "iatrogenic" not only refers to direct clinical effects—such as sedation, activation, cognitive dulling, or paradoxical reactions—but also to broader social and cultural effects that occur when medication use unintentionally disrupts functioning or relationships. Clinically, iatrogenic harm might manifest as new or worsening symptoms caused by the medication; socially, it may appear as side effects that hinder school participation or daily routines; and culturally, it can happen when pharmacological treatments overshadow community-based practices or undermine relational supports vital for well-being in many cultural settings.[8,10–12]

Structural and functional brain changes linked to prenatal alcohol exposure increase sensitivity to medication side effects and make therapeutic responses less predictable.[3,16,17,24] Neuroimaging studies showing widespread alterations in connectivity, cortical organization, and network efficiency highlight the need for careful consideration when prescribing psychotropic medications to this group.[16–19] When these neurobiological vulnerabilities combine with the clinical, social, and cultural aspects of iatrogenic risk, the potential for harm becomes significant, underscoring the importance of cautious prescribing and developing tapering protocols that prioritize relational and ecological stability.

Beyond neurodevelopmental factors, critiques of contemporary psychopharmacology highlight the potential for medications to induce abnormal brain states, conceal underlying psychosocial causes of distress, and result in long-term dependence.[20–22] These concerns are particularly relevant in FASD, where environmental instability, trauma histories, and relational disruptions frequently influence behavioral presentation.

Overall, the literature shows a consistent gap between prescribing practices and the existing evidence. Psychotropic medications are often used to manage behaviors resulting from neurodevelopmental vulnerabilities or environmental stressors rather than clearly defined psychiatric conditions.[1,2,5,8] This context highlights the need for tapering protocols that emphasize relational stability, environmental predictability, and culturally appropriate supports.

Neurobiological vulnerability and pharmacokinetic instability in FASD

Prenatal alcohol exposure disrupts neuronal proliferation, migration, and synaptogenesis, leading to widespread changes in both cortical and subcortical organization.[16,17] Structural MRI studies consistently show reductions in white matter integrity, abnormalities in corpus callosum shape, and region-specific volume differences, especially in the frontal lobes, basal ganglia, cerebellum, and other areas involved in executive function and motor coordination.[16,18] Functional imaging further shows decreased connectivity within networks responsible for attention, executive function, and emotional regulation.[3,17]

Recent work using resting-state functional connectivity—a method that examines how different brain regions communicate with one another when a person is not engaged in a specific task—has helped clarify the extent of network instability in FASD. These studies consistently show weaker and less coordinated activity in the frontoparietal networks and in the neural architecture that supports self-reflection and baseline cognitive organization. Such patterns are closely linked to difficulties in attention, executive functioning, and the integration of sensory and emotional information.[3] Taken together, these findings indicate that the neural systems responsible for organizing and coordinating higher-order cognitive processes operate less efficiently in individuals with FASD.

Beyond these structural and functional differences, prenatal alcohol exposure also changes the biophysical properties of neuronal membranes. Variations in lipid composition, membrane fluidity, and receptor distribution affect how psychotropic medications are absorbed, distributed, and bound at the cellular level.[25,26] Advances in mass spectrometry and metabolomics have clarified how these lipidomic and metabolic changes contribute to pharmacokinetic variability and heightened sensitivity to adverse effects.[26,27]

These lipidomic disruptions also connect with cytochrome P450 (CYP450) pathways that control the metabolism of many psychotropic drugs. Prenatal alcohol exposure can alter the expression and function of several CYP450 isoenzymes, and shifts in membrane lipid composition can affect the microenvironment in which these enzymes operate. Variations in membrane fluidity and lipid–protein interactions may influence enzyme shape and substrate binding, thus impacting metabolic clearance. In individuals with FASD—who already face metabolic and neurodevelopmental vulnerabilities—these combined effects help explain their increased sensitivity to standard pediatric doses and the unpredictable pharmacodynamic responses often seen in practice.[3,16,17,24] In this context, lipidomic abnormalities are not just structural issues but part of a broader metabolic instability that influences how psychotropic medications are metabolized, highlighting the importance of cautious, personalized tapering approaches.

Experimental models using human-induced pluripotent stem cell–derived cerebral organoids—three-dimensional clusters of human neural tissue that replicate key features of early brain development—demonstrate that alcohol exposure disrupts neurodevelopmental pathways from the earliest stages. In these models, alcohol interferes with processes such as neuronal differentiation (the maturation of precursor cells into specialized neurons), synaptic maturation (the development of functional communication points between neurons), and the initial formation of neural circuits that support coordinated network activity.[28] These early disruptions lead to long-lasting changes in how neurons organize and connect, providing a mechanistic explanation for the unpredictable or exaggerated responses to psychotropic medications often seen clinically in individuals with FASD.

Citation: Maviglia M, Ceccanti M, Mignini F, et al. Developing tapering protocols for psychotropic medications in adolescents with fetal alcohol spectrum disorder: incorporating relational, environmental, and cultural aspects of care. *J Psychol Clin Psychiatry.* 2026;17(1):11–17. DOI: 10.15406/jpcpy.2026.17.00846

These neurobiological findings highlight the importance of cautious, personalized, and ethically based tapering protocols. Variations in brain structure and function, changes at the membrane level (such as lipid composition and receptor distribution), and metabolic vulnerabilities all affect individual responses to medication. These vulnerabilities emphasize the need for environmental stability, ongoing relational support, and culturally appropriate assistance during the tapering process.

Principles guiding a tapering protocol in FASD

Developing a tapering plan for adolescents with FASD requires an approach that integrates neurobiological sensitivity, environmental stability, relational continuity, and cultural context. The goal is not simply to reduce or discontinue medications, but to create conditions in which the individual can function more independently and with less risk of treatment-related harm.

A first principle is recognizing that neurodevelopmental instability amplifies the impact of environmental fluctuations. Executive functioning deficits, impaired stress regulation, and reduced adaptive capacity mean that even minor changes in routine can influence behavior.[16,17,24] Tapering should therefore occur only when the individual's environment is predictable, with consistent caregiving, stable routines, and access to supportive relationships.[8,12,14]

A second principle emphasizes the importance of a comprehensive, multidimensional assessment before reducing any medication. This involves examining developmental history, trauma exposure, educational background, cultural context, and relational networks.[8,12,14] It also requires a thorough evaluation of the current medication plan, including the original reason for prescribing, the presence of polypharmacy, and any documented adverse reactions.[3,6,7]

A third principle is shared decision-making. Adolescents with FASD often have limited control over their treatment paths, and involving them meaningfully in the tapering process can boost engagement and ease anxiety.[12,14] Caregivers, educators, and cultural or community supports should also be included, especially when relational networks are central to daily functioning.[10–12]

A fourth principle is gradualism. Given the pharmacodynamic variability associated with FASD, dose reductions should be made gradually, allowing time to observe behavioral, cognitive, and emotional changes.[5,23]

Rapid tapering increases the risk of withdrawal-like reactions, behavioral instability, and misinterpreted transient adjustment symptoms.[20–22] A slow, deliberate approach helps clinicians distinguish between short-term fluctuations and more persistent changes that may require intervention.

Although general tapering frameworks in child psychiatry often recommend reducing medications by approximately 5–10% every 2–4 weeks, with slower schedules for antipsychotics and mood stabilizers, applying such percentage-based rules to adolescents with FASD is considerably more complex. Neurodevelopmental instability, altered pharmacokinetics, and a history of prolonged or multi-drug exposure frequently make these individuals more sensitive to even minor dose adjustments.[3,16,17,23] For this reason, percentage-based reductions should be viewed only as broad orientation points rather than prescriptive rules. In practice, tapering in FASD often requires even more minor decrements, longer intervals between reductions, and extended periods of stabilization before proceeding to the next step. Until more disorder-specific evidence becomes available, clinicians should adopt a cautious, individualized withdrawal schedule that emphasizes consistency, slow pacing, and close monitoring of relational and environmental factors. This conservative approach aligns with the broader developmental and neurobiological vulnerabilities described in FASD and reduces the likelihood of misinterpreting predictable therapeutic adjustments as relapse or deterioration.[20–22]

A fifth principle involves integrating non-pharmacological supports throughout the tapering process. Behavioral interventions, educational accommodations, sensory regulation strategies, and culturally rooted practices help stabilize functioning and decrease dependence on medications.[8,12,29–33] These supports are crucial for individuals whose neurodevelopmental profiles make them especially sensitive to environmental and relational changes.[34–40]

Clinical experience with individuals who present multiple comorbid behavioral and health challenges further illustrates that withdrawal processes require structured supervision and careful environmental coordination to prevent destabilization.[40]

Finally, tapering should be understood as a long-term process. Monitoring must continue beyond medication discontinuation, with attention to school performance, emotional regulation, social relationships, and cultural or community engagement.[12,14,34–36] Sustained follow-up helps ensure that progress is maintained and that new challenges are addressed without defaulting to medication.

Because tapering occurs within complex relational and institutional systems, disagreements among stakeholders are common. Parents, school staff, behavioral specialists, and clinicians may have different expectations about the role of medication, often shaped by their daily experiences with the adolescent and by the structural pressures within each setting. These tensions can become especially evident when school staffs focus on managing the classroom, while caregivers emphasize long-term developmental goals or reducing iatrogenic exposure. In such cases, a structured conflict-resolution process is crucial. This involves clarifying the rationale for tapering based on the adolescent's neurocognitive profile and environmental needs, grounding discussions in these facts,[16,17,24] and re-establishing shared goals that prioritize relational stability and functional adaptation over immediate behavioral control.[8,12,14] When disagreements are addressed through open communication and by returning to the core principles of the tapering protocol, the team is better positioned to stay coherent and prevent premature medication reinstatement caused by predictable adjustment reactions.[20–22] Thus, conflict resolution is a vital part of the long-term monitoring process, ensuring decisions stay aligned with the adolescent's developmental path and environmental context.

Structure of the proposed tapering protocol

The tapering protocol is divided into three interconnected stages: multidimensional assessment, integrated intervention with gradual dose reduction, and ongoing monitoring. This structure reflects that tapering in FASD is not a straightforward pharmacological adjustment but a complex clinical process influenced by neurodevelopmental vulnerability, environmental stability, relational continuity, and cultural context.[8,12,14,34]

Each stage builds on the previous one, ensuring that dose reductions happen only when the individual's developmental, relational, and ecological conditions can support the change. This approach acknowledges that medication effects in FASD are rooted in broader systems of meaning, caregiving, and daily functioning, so tapering must be approached with careful planning, flexibility, and ongoing collaboration. Since the psychological and relational aspects of FASD are central to functioning, tapering should be considered a developmental intervention rather than merely a pharmacological adjustment.

Phase one: multidimensional assessment

This initial phase sets the foundation for all later decisions. It involves a thorough, clinically based review of developmental history, trauma history, school performance, cultural background, and social connections.[8,12,14]

The aim is to understand the individual not just through diagnostic labels but through the lived experiences where behaviors develop and are understood.

A detailed evaluation of the current medication regimen is essential. This includes clarifying the original rationale for prescribing, identifying patterns of polypharmacy, and documenting any adverse reactions or paradoxical responses.[3,6,7] In many cases, medications were introduced during periods of crisis or instability, and their continued use may reflect inertia rather than ongoing benefit.

A central task in this phase is distinguishing behaviors that arise from neurodevelopmental vulnerabilities (e.g., executive dysfunction, sensory dysregulation), environmental stressors (e.g., inconsistent caregiving, school demands), and medication-related effects (e.g., activation, sedation, irritability).[16,17,23] This differentiation is crucial: tapering is most successful when clinicians can identify which behaviors are likely to improve with dose reduction and which require environmental or relational interventions. This phase often reveals that what appears to be "worsening behavior" is, in fact, a medication side effect or a response to instability in the caregiving environment.

Phase two: integrated intervention and gradual tapering

This phase combines non-pharmacological supports with carefully paced dose reductions.[5,23] The focus is on establishing a stable ecological and relational environment where the individual can tolerate and adapt to medication changes.

Behavioral strategies, educational accommodations, sensory regulation techniques, and culturally grounded practices provide the necessary support to maintain functioning during tapering.[8,12,29–33] These interventions are not secondary; they are essential to the tapering process. They help decrease dependence on medications by addressing the underlying neurodevelopmental and environmental factors contributing to distress.

Dose reductions should be gradual, intentional, and tailored to the individual's developmental profile. Temporary changes in behavior, sleep, or emotional regulation are normal and should not be mistaken for clinical decline.[20–22] Open communication with caregivers, educators, and community supports is crucial to avoid premature medication reinstatement or unnecessary escalation. This process often involves repeated adjustments, with clinicians observing patterns over several weeks rather than days.

Importantly, tapering is not just a pharmacological action but a relational process. The individual's sense of safety, predictability, and control must be maintained throughout. When tapering is seen as a collaborative effort rather than a withdrawal of support, adolescents are more likely to handle temporary discomfort and engage meaningfully in the process.

Phase three: longitudinal monitoring and relational consolidation

Monitoring goes well beyond stopping medication. This phase highlights long-term adaptation, focusing on school performance, emotional regulation, social relationships, and cultural or community involvement.[12,14,34–36]

The aim is to ensure that progress made during tapering continues and that new challenges are managed through relational and environmental supports instead of reverting to medication.

Relational consolidation—predictable routines, consistent caregiving, and stable support networks—is vital for maintaining progress.[8,12,14] Adolescents with FASD depend heavily on relational anchors to manage daily demands, and tapering is most effective when these anchors stay strong. This reliance reflects the broader neurodevelopmental profile of FASD, where challenges with stress regulation, executive functioning, and adaptive flexibility make relational stability a key organizing force in daily life.[16,17,24] When caregiving settings are predictable and emotionally supportive, adolescents are better able to withstand the temporary fluctuations that accompany dose reductions and are less likely to interpret internal changes as signs of threat or instability. Conversely, when relational or ecological conditions are inconsistent, even minor medication adjustments can heighten dysregulation and lead to misinterpretation of taper-related reactions as clinical worsening.[20–22]

Therefore, relational stability is not just an addition to tapering but a fundamental therapeutic requirement, shaping the individual's ability to incorporate medication changes into a coherent developmental path.

In communities where identity is central to well-being, ongoing engagement with cultural practices strengthens resilience and provides a framework for emotional regulation.[10–12]

Cultural continuity can serve as a protective factor, buffering the stress of developmental transitions and supporting long-term recovery.

This final phase acknowledges that tapering is not an endpoint but part of a wider move toward developmentally informed, culturally grounded, and relationally supported care.

Table 1 translates the principles outlined above into a structured tapering protocol. It is intended as a practical guide that integrates neurodevelopmental understanding with relational stability and cultural grounding, supporting clinicians and caregivers in implementing medication reduction safely and thoughtfully.

Discussion

The tapering protocol outlined here reflects a broader shift in FASD care: a move away from symptom-driven pharmacology toward approaches rooted in neurodevelopment, relational continuity, and cultural context.[12,14,34]

This change is vital because the psychological aspects of FASD—stress regulation, attachment patterns, trauma histories, and identity formation—cannot be effectively addressed through medication alone. Instead, they need interventions that acknowledge the developmental and relational foundations of behavior.

In the treatment of FASD, polypharmacy is especially problematic because many behaviors prompting medication—such as emotional lability, impulsivity, sensory dysregulation, and difficulty with transitions—stem from neurodevelopmental instability rather than specific psychiatric disorders.[1,2,5,6] When medications are used to manage these developmentally based behaviors, clinicians often face inconsistent or paradoxical responses, increased sensitivity to side effects, and the need for complex medication regimens that provide limited benefits.[3,6,7,23] This situation highlights a fundamental mismatch between the neurobiological characteristics of FASD and the pharmacological approaches commonly used in child psychiatry.

Because the psychological and relational dimensions of FASD are central to functioning, tapering must be approached as a developmental intervention rather than a pharmacological experiment. The tapering protocol addresses this mismatch by embedding medication reduction within a broader system of environmental stabilization, relational support, and culturally grounded interventions.[8,12,29–33] This integrated approach acknowledges that psychological functioning in FASD is inseparable from the ecological and relational contexts in which the individual lives. Tapering becomes not merely a medication adjustment but a process that requires predictable routines, emotional availability, and cultural continuity.

A key aspect of tapering in FASD is understanding behavioral fluctuations. These changes should be viewed through a neurodevelopmental perspective.[16,17,24] In many adolescents with FASD, behavior shifts during tapering often result from withdrawal effects and the individual's baseline neurodevelopmental vulnerabilities. Without this understanding, caregivers and clinicians might mistake predictable, short-term adjustment reactions for relapse or decline, which can lead to premature medication reinstatement or escalation.[20–22] Such reactions can reinforce cycles of dependence and obscure the adolescent's actual psychological needs. Therefore, a nuanced understanding of these fluctuations is crucial to differentiate withdrawal-related instability from true clinical deterioration.

Caregivers, educators, and community supports play essential roles in this process.[12,14,34–36] Their ability to provide consistent routines, emotional containment, and coordinated expectations directly affects tapering outcomes. In cultural contexts where identity, belonging, and relational ties are vital to well-being—such as Native American communities—integrating cultural practices enhances resilience and encourages psychological integration.[10–12] These practices provide grounding, continuity, and meaning, all of which are crucial during periods of neurobiological and emotional change.

The protocol also questions broader systemic trends toward medicalization.[20–22] Overreliance on medication often results from structural constraints—such as limited behavioral services, fragmented care systems, and insufficient caregiver support—rather than therapeutic need.[10–12,37–39] These issues disproportionately impact communities already affected by historical trauma, social inequalities, and limited access to culturally responsive services. Recognizing these systemic pressures is essential for developing tapering strategies that are both clinically effective and ethically responsible.

Emerging research on mixed withdrawal processes further illustrates how biological vulnerability, social instability, and recovery challenges intersect. These findings reinforce the need for tapering models that extend beyond pharmacological considerations to include psychological, relational, and cultural dimensions of care. In this sense, tapering becomes part of a broader therapeutic reorientation—one that prioritizes developmental trajectories, relational stability, and cultural identity.

Finally, the protocol emphasizes the importance of long-term research on tapering outcomes across different cultural and environmental settings.[3,16–19,28] International collaboration is crucial for creating evidence-based guidelines that reflect the complexity of FASD and the psychological realities of individuals living with it. Such research will help improve tapering strategies that respect neurodevelopmental vulnerabilities, cultural identity, and long-term well-being.

A necessary next step is the development of a longitudinal pilot study designed to evaluate the feasibility and clinical impact of the tapering framework outlined in Table 1. Such a study would require a multidisciplinary structure, integrating pharmacological monitoring, neurodevelopmental assessment, and systematic documentation of relational, environmental, and cultural factors. A small cohort of adolescents with FASD—recruited from settings where stable caregiving and consistent educational supports are present—would undergo tapering guided by the three-phase protocol. Pharmacological data would include dose-reduction trajectories, adverse-effect profiles, and patterns of behavioral fluctuation over time. Neurodevelopmental measures would track changes in attention, executive functioning, emotional regulation, and adaptive behavior, using tools already familiar to clinicians working in FASD.[16,17,24] Parallel documentation of contextual variables—caregiver stability, school coordination, cultural engagement, and exposure to environmental stressors—would allow investigators to examine how relational and ecological conditions shape tapering outcomes.[8,12,14,34–36]

Table 1 Structured tapering protocol for adolescents with FASD

Phase	Core clinical tasks	Relational / environmental requirements	Cultural / community supports	Clinical cautions
Multidimensional assessment	(i) Review developmental history, trauma exposure, school functioning, cultural background, and relational networks.[8,12,14] (ii) Evaluate current medications: rationale, polypharmacy, adverse or paradoxical effects.[3,6,7] (iii) Distinguish neurodevelopmental vulnerabilities, environmental stressors, and medication-related symptoms.[16,17,23]	(i) Stable caregiving and predictable routines. (ii) Capacity for consistent observation and communication.	(i) Consultation with cultural leaders or community representatives when relevant.	(i) Do not initiate tapering in unstable environments. (ii) Avoid misattributing neurodevelopmental instability to psychiatric relapse.

Citation: Maviglia M, Ceccanti M, Mignini F, et al. Developing tapering protocols for psychotropic medications in adolescents with fetal alcohol spectrum disorder: incorporating relational, environmental, and cultural aspects of care. *J Psychol Clin Psychiatry.* 2026;17(1):11–17. DOI: 10.15406/jpcpy.2026.17.00846

Table 1 Continued....

Integrated intervention + Gradual tapering	(i) Implement behavioral, educational, sensory-regulation, and culturally grounded supports.[8,12,29–33] (ii) Reduce medication slowly, in small increments.[5,23] (iii) Monitor cognitive, emotional, and behavioral changes; adjust pace as needed.[20–22]	(i) Coordinated routines across home and school. (ii) Clear communication about expected fluctuations.	(i) Integrate culturally meaningful practices that support regulation and identity.	(ii) Expect transient dysregulation; avoid rapid dose reductions. (iii) Do not escalate medications in response to predictable adjustment effects.
Longitudinal monitoring + Relational consolidation	(i) Continue monitoring after discontinuation: school performance, emotional regulation, social functioning.[12,14,34–36] (ii) Reinforce adaptive strategies and address new stressors early.	(i) Sustained relational continuity and stable caregiving. (ii) Ongoing collaboration among caregivers, educators, and clinicians.	(i) Maintain engagement with cultural practices and community networks.[10–12]	(i) Avoid reflexive reintroduction of medications. (ii) Prioritize relational and environmental interventions before pharmacological changes.

The study would follow participants for at least 12–18 months, capturing both the tapering period and the post-discontinuation phase, when relational consolidation and environmental predictability become especially important. Qualitative input from caregivers, educators, and cultural or community representatives would complement quantitative measures, ensuring that the evaluation reflects the lived experience of those supporting the adolescent. The primary aim would not be to produce definitive efficacy data but to determine whether the protocol is implementable across settings, to identify points of vulnerability within the tapering process, and to clarify which relational or contextual factors most strongly influence stability. Findings from such a pilot study would provide the empirical foundation needed to refine the protocol and guide larger, multisite investigations capable of informing future clinical guidelines.

Conclusion

Managing FASD requires approaches that go beyond symptom-focused medication.[1,2,5,6] Individuals with FASD are particularly vulnerable to adverse and unpredictable reactions, so tapering methods must be informed by neurodevelopmental insights, environmental stability, and relational support.[3,16–19,23] These elements create the psychological and developmental foundation crucial for effective adaptation.

The proposed protocol reframes tapering as a therapeutic process rather than a technical maneuver. By integrating multidimensional assessment, gradual dose reduction, and long-term monitoring, the model aligns pharmacological decisions with developmental history, cultural context, and the lived realities of daily functioning.[8,12,14,29–36] This approach recognizes that psychological well-being in FASD emerges from the interplay of neurobiology, relationships, identity, and environment — and that medication changes must occur within a framework that supports these interconnected domains.

Implementing such protocols requires interdisciplinary collaboration, caregiver engagement, and sustained attention to relational and ecological stability.[12,14,34–36] It also calls for a shift in clinical culture: one that acknowledges the limits of pharmacological solutions and places greater emphasis on autonomy, cultural identity, and stable developmental trajectories.[20–22] This shift is not merely a matter of technique but of ethics — a commitment to practices that honor the complexity of the young person's developmental and psychological world.[39–42]

The protocol presented here offers a framework to support this transition. By integrating neurodevelopmental science, cultural knowledge, and relational practice, it promotes more sustainable and ethically grounded pathways of care for adolescents with FASD. It also establishes the foundation for the kind of longitudinal pilot studies needed to validate and refine this model in real-world settings. Such studies — grounded in multidisciplinary assessment, careful pharmacological monitoring, and systematic attention to relational, environmental, and cultural conditions — will be essential for determining how tapering protocols can be adapted across diverse communities and systems of care.

Effective treatment in FASD requires us to see beyond symptoms and toward the developmental, relational, and cultural worlds in which young people grow. The work ahead involves not only improving clinical practice but also building the empirical evidence that will allow tapering approaches to evolve in ways that honor the lived experience, vulnerability, and resilience of those affected by prenatal alcohol exposure.

Acknowledgments

None.

Funding

None.

Conflicts of interest

The author declares that there are no conflicts of interest.

References

1. American Academy of Pediatrics. *Pharmacologic interventions in fetal alcohol spectrum disorders*. 2021.
2. Wozniak JR, Riley EP, Charness ME. Clinical presentation, diagnosis, and management of fetal alcohol spectrum disorder. *Lancet Neurol*. 2019;18(8):760–770.

3. Fan J, Woods KJ, Jacobson JL, et al. Lower resting state functional connectivity partially mediates adverse effects of prenatal alcohol exposure on arithmetic performance in children. *Alcohol Clin Exp Res*. 2024;48(6):1050–1062.
4. Popova S, Charness ME, Burd L, et al. Fetal alcohol spectrum disorders. *Nat Rev Dis Primers*. 2023;9(1):11.
5. Durr MR, Petryk S, Mela M, et al. Utilization of psychotropic medications in children with FASD: a retrospective review. *BMC Pediatr*. 2021;21:512.
6. Ritfeld GJ, Kable JA, Holton JE, Coles CD. Psychopharmacological treatments in children with fetal alcohol spectrum disorders: a review. *Child Psychiatry Hum Dev*. 2022;53(2):268–277.
7. Senturias Y, Ali MM, West K. Psychotropic medication utilization among children diagnosed with fetal alcohol spectrum disorder. *Pediatrics*. 2022;150(4):e2022056797.
8. Fiorentino D, Coriale G, Tarani L, et al. Italian guidelines for the diagnosis and treatment of fetal alcohol spectrum disorders: prevention and health promotion. *Riv Psichiatr*. 2024;59(5):250–258.
9. Maviglia M. *Il lato oscuro della terapia farmacologica nel FASD*. Presented at: Congresso Internazionale sul FASD; 2025; Loreto, Italy.
10. Ceccanti M, Vitali M. *Epidemiologia e clinica del FASD in Italia*. Presented at: Congresso Internazionale sul FASD; 2025; Loreto, Italy.
11. Mignini F. *Tavola rotonda: Linee guida per la dismissione farmacologica nel FASD*. Presented at: Congresso Internazionale sul FASD; 2025; Loreto, Italy.
12. Gillespie B, Romero R, DeCora Guimaraes V, et al. *Addressing FASD: Working with and learning from indigenous communities*. Presented at the 9th International Research Conference on FASD; Seattle, WA. 2025.
13. Gone JP. The red road to wellness: cultural reclamation in a Native First Nations community treatment center. *Am J Community Psychol*. 2011;47(1–2):187–202.
14. Cooeyate NJ, Maviglia M, Hume D. The supervision gap in peer support workforce: implications for developing effective peer support programs in Native American communities. *J Psychol Clin Psychiatry*. 2024;15(3):211–217.
15. Mela M, Hanlon-Dearman A, Ahmed AG, et al. Treatment algorithm for the use of psychopharmacological agents in individuals prenatally exposed to alcohol and/or with diagnosis of fetal alcohol spectrum disorder (FASD). *J Popul Ther Clin Pharmacol*. 2020;27(3):e1–e13.
16. Rockhold MN, Holman PJ. Structural MRI and mental health associations in children with and without fetal alcohol spectrum disorder: Translational insights. *Alcohol Clin Exp Res*. 2024;48(2):241–245.
17. Donald KA, Eastman E, Howells FM, et al. Neuroimaging effects of prenatal alcohol exposure on the developing human brain: a magnetic resonance imaging review. *Acta Neuropsychiatr.* 2015;27(5):251–269.
18. Lebel C, Roussotte F, Sowell ER. Imaging the impact of prenatal alcohol exposure on the structure of the developing human brain. *Neuropsychol Rev.* 2011;21(2):102–118.
19. Marquardt K, Brigman JL. The impact of prenatal alcohol exposure on social, cognitive and affective behavioral domains: Insights from rodent models. *Alcohol*. 2016;51:1–15.
20. Moncrieff J, Cohen D. Do antidepressants cure or create abnormal brain states? *PLoS Med*. 2006;3(7):e240.
21. Healy D. *The antidepressant era: false hopes and real risks*. Harvard University Press. 1999.
22. Breggin PR. *Psychiatric drug withdrawal: a guide for prescribers, therapists, patients, and their families*. New York: Springer; 2013.
23. Wrath AJ, Mela M, Le T, et al. Psychotropic medication usage in individuals with fetal alcohol Spectrum disorder (FASD) and psychiatric co-morbidities in Canada. *J Fetal Alcohol Spec Risk Prev*. 2022;4(SP1):e70–e90.
24. Petrelli B, Weinberg J, Hicks GG. Effects of prenatal alcohol exposure (PAE): insights into FASD using mouse models of PAE. *Biochem Cell Biol.* 2018;96(2):131–147.
25. Lauwers E, Goodchild RE, Skibo G, et al. Membrane lipids in presynaptic function. *Neuron*. 2016;90(1):11–25.
26. Burghardt KJ, Goodrich JM, Dolan CV, et al. Metabolomics, lipidomics, and antipsychotics. *Biomedicines*. 2023;11(12):3295.
27. Ma X, Fernández FM. Advances in mass spectrometry imaging for spatial cancer metabolomics. *Mass Spectrom Rev*. 2024;43(2):235–268.
28. Arzua T, Yan Y, Jiang C, et al. Modeling alcohol-induced neurotoxicity using human induced pluripotent stem cell-derived three-dimensional cerebral organoids. *Transl Psychiatry*. 2020;10:347.
29. Coles CD, Kable JA, Taddeo E, et al. A metacognitive strategy for reducing disruptive behavior in children with fetal alcohol spectrum disorders: GoFAR pilot. *Alcohol Clin Exp Res*. 2015;39(11):2224–2233.
30. American Academy of Pediatrics. *Behavioral interventions in Fetal Alcohol Spectrum Disorders*. American Academy of Pediatrics; 2025.
31. Do2Learn. *GoFAR Overview*. Do2Learn; 2025.
32. Alert Program. *Enhancing lives with Fetal Alcohol Spectrum Disorders.* Alert Program; 2025.
33. Coles CD, Kable JA, Taddeo E, et al. Community translation of the Math Interactive Learning Experience Program for children with FASD. *Res Dev Disabil*. 2015;39:1–11.
34. Grant TM, Ernst CC, Streissguth AP. Intervention with high-risk alcohol and drug-abusing mothers: II. Three-year findings from the Seattle model of paraprofessional advocacy. *J Community Psychol*. 1999;27(1):19–38.
35. Mariani A. *Affettività e relazione educativa efficace*. Firenze: IUL Press; 2021.
36. Ceccanti M, Fiorentino D, Coriale G, et al. Maternal risk factors for fetal alcohol spectrum disorders in a province in Italy. *Drug Alcohol Depend*. 2014;145:201–208.
37. Di Vittorio G, Gori R. Comunità terapeutiche. *Salute Soc*. 2015;14(2):45–60.
38. Caritas Italiana. *Osservatorio sulle Povertà e le Risorse.* Caritas Italiana; 2022.
39. De Leonardis O, Mauri D, Rotelli F. Deinstitutionalisation, another way. *Health Promotion*. 1996;1:151–165.
40. Maviglia M, Cooeyate NJ. Implementing psychiatric drug withdrawal practices: challenges with individuals with multiple comorbid behavioral and health problems. *J Psychol Clin Psychiatry*. 2024;15(2):79–85.
41. Maviglia M, Cooeyate NJ, Hume D. Beyond tapering: understanding the biological, social, and recovery challenges of mixed withdrawal from psychiatric medications and opioids. *J Psychol Clin Psychiatry*. 2025;16(4):122–133.
42. Ozsarfati J, Koren G. Medications used in the treatment of disruptive behavior in children with FASD --a guide. *J Popul Ther Clin Pharmacol*. 2015;22(1):e59–e67.

Citation: Maviglia M, Ceccanti M, Mignini F, et al. Developing tapering protocols for psychotropic medications in adolescents with fetal alcohol spectrum disorder: incorporating relational, environmental, and cultural aspects of care. *J Psychol Clin Psychiatry.* 2026;17(1):11–17. DOI: 10.15406/jpcpy.2026.17.00846