Journal of Advances in Medicine and Pharmaceutical Sciences
p-ISSN: 2964-268X, e-ISSN: 2963-4253

Case Report Paper

Exploratory Clinical Study on Controlled *álak* Microdosing for Post-Detoxification Recovery

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Article History
Received:
26.08.2025

Revised:
11.08.2025

Accepted:
30.08.2025

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Abstract: Post-detoxification phases of methamphetamine dependence are often characterized by neurophysiological instability, including tremors, insomnia, and anxiety, due to suppressed GABAergic activity and hyperdopaminergic rebound. Traditional Filipino distilled spirit, *álak* or *lambanog*, contains trace levels of ethanol and minor fusel alcohols which may exert mild GABA-A receptor agonism when microdosed under medical supervision. This exploratory clinical study investigates the potential of controlled álak microdosing as an adjunct for stabilizing central nervous system function in post-detoxified methamphetamine patients. Twelve post-detoxification patients (n=12) from rehabilitation centers in Quezon and Batangas were administered álak microdoses (≤10% ethanol, 5–10 ml/day) for 14 days under medical supervision. Physiological and psychological parameters including sleep quality, tremor frequency, and anxiety, were measured using the Clinical Opiate Withdrawal Scale (COWS) adapted for stimulant recovery and the Beck Anxiety Inventory (BAI). Data were analyzed descriptively and statistically using paired t-tests. Eight out of twelve participants (66.7%) demonstrated marked improvement in sleep continuity ($p<0.05$), tremor reduction ($p<0.05$), and decreased anxiety scores (mean reduction 23.4%, $p<0.05$). No signs of ethanol intoxication, craving substitution, or hepatic distress were observed. Mild headache was reported in one participant but resolved spontaneously. Controlled álak microdosing exhibits promising GABAergic modulatory effects that may facilitate neurophysiological stabilization in methamphetamine recovery. While preliminary, these findings support further biochemical validation and integration of ethnopharmacological knowledge into harm-reduction frameworks.

Keywords: GABA-A Receptor, *álak* or *lambanog*, Methamphetamine Dependence, Neurophysiological Stabilization, Ethnopharmacology.

Journal of Advances in Medicine and Pharmaceutical Sciences, vol. 4, no. 2, pp. 64-73, November 2025. **DOI: 10.36079/lamintang.jamaps.922**

1. Introduction

Methamphetamine dependence remains one of the most challenging forms of substance use disorder worldwide, with high relapse rates and profound neurochemical dysregulation. The stimulant's neurotoxicity induces long-term alterations in dopaminergic and GABAergic pathways, which continue to affect patients even after detoxification. In Southeast Asia, particularly in the Philippines, methamphetamine (locally known as *shabu*) constitutes a major drug of concern due to its accessibility and severe withdrawal sequelae [1] [2].

The post-detoxification period is often marked by neurophysiological instability characterized by tremors, anxiety, dysphoria, and sleep disturbance. These symptoms stem from disrupted inhibitory neurotransmission following the cessation of chronic stimulant use. Current pharmacological interventions such as benzodiazepines or non-benzodiazepine anxiolytics offer symptomatic relief but are limited by dependency risk, high cost, and restricted access within rehabilitation centers. Neuropharmacological studies have shown that methamphetamine withdrawal involves downregulation of GABA-A receptor activity and impaired inhibitory tone in the central nervous system (CNS) [3]. The resulting hyperexcitability manifests as physiological tremor and psychological restlessness. Restoration of GABAergic equilibrium is thus a crucial therapeutic goal in the post-detoxification phase [4] [5].

Ethanol, in low concentrations, acts as a partial agonist at the GABA-A receptor complex, enhancing chloride ion influx and neuronal hyperpolarization. While excessive ethanol exposure is neurotoxic, controlled microdosing may transiently restore inhibitory neurotransmission without inducing dependency. This paradoxical role of ethanol underlies its potential as a modulated neurostabilizer when used within pharmacological boundaries.

In various rural regions of the Philippines, traditional distilled spirits such as *álak* or *lambanog* have long been used in ritual and medicinal contexts, often to relieve muscle fatigue, induce relaxation, or reduce post-illness tremor. These traditional practices embody a form of community-based pharmacology that aligns with the principles of harm reduction and cultural healing. However, scientific evaluation of such practices remains limited [6].

Microdosing defined as the administration of substances in sub-therapeutic quantities to elicit physiological modulation without intoxication has gained attention in neuropsychopharmacology. Controlled *álak* microdosing represents an ethnopharmacological variant of this concept, focusing on gentle GABAergic activation through naturally fermented ethanol compounds under medical oversight.

Despite increasing literature on ethnopharmacological alcohol derivatives, no systematic clinical exploration has yet examined the physiological potential of traditional spirits in controlled recovery contexts. The intersection between cultural pharmacology and neurochemical stabilization remains underexplored in Southeast Asian clinical settings. Given the addictive potential of ethanol, any exploration into its controlled therapeutic use demands strict ethical framing and medical supervision. The purpose is not substitution but modulation supporting neurochemical homeostasis during vulnerable recovery phases. Thus, this study operates within a harm-reduction and non-addictive paradigm.

Integrating culturally familiar yet clinically regulated agents such as *álak* may enhance therapeutic adherence, reduce anxiety-related relapse triggers, and build a bridge between local tradition and biomedical practice. Such integration aligns with the Philippine Department of Health's advocacy for culturally adaptive mental health and rehabilitation frameworks [7].

This exploratory clinical study aims to evaluate the physiological effects of controlled *álak* microdosing on neurostabilization among post-detoxified methamphetamine patients. The working hypothesis posits that low-dose ethanol exposure can activate GABA-A receptors sufficiently to reduce tremor, anxiety, and sleep disruption without inducing dependency or intoxication.

2. Literatur Review

2.1 Neurochemical Basis of Post-Detoxification Instability

Post-detoxification in methamphetamine dependence is often marked by a paradoxical neurological state dopaminergic exhaustion alongside GABAergic inhibition. The absence of methamphetamine's dopamine-releasing stimulus leaves patients in a neurochemical vacuum characterized by anxiety, tremors, insomnia, and dysphoria. Research in neuropharmacology identifies that GABA-A receptor downregulation persists weeks after detoxification, delaying neurostabilization [8]. This physiological

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deficit explains why many relapse episodes occur not due to craving per se, but due to the discomfort of neurochemical disequilibrium [9].

Clinical studies on benzodiazepine-assisted detoxification show that GABA-A modulation can ease this transitional instability, yet prolonged benzodiazepine use carries its own dependence risks [10] [11]. Hence, safer GABAergic interventions are sought particularly those offering mild tonic modulation rather than strong receptor binding. Low-dose ethanol has been shown to transiently enhance GABAergic tone and reduce hyperexcitability in animal models [12], suggesting a potential neurochemical rationale for controlled ethanol microdosing in recovery contexts.

Importantly, the efficacy of such interventions depends on dosage precision and contextual supervision. Ethanol, in pharmacological quantities, is neurotoxic; but at microdoses (typically <0.02 g/kg), studies show increased alpha-wave activity and parasympathetic balance without intoxicating effects [13] [14]. The hypothesis underpinning this study using traditional *álak* in microdoses therefore aligns with the concept of "subthreshold pharmacodynamics," where a compound operates as a neuromodulator rather than a psychoactive agent [15].

In the Philippines, where methamphetamine misuse ("shabu") remains a public health concern, understanding neurochemical stabilization through accessible means could have significant social and clinical implications. *Álak*, as a distilled spirit, contains trace ethanol capable of mild GABAergic activation when carefully titrated. However, what differentiates this approach from mere alcohol use lies in intentionality and medical supervision transforming a culturally embedded substance into a pharmacologically controlled adjunct.

1.1. Ethnopharmacological Role of Álak in Filipino Healing Traditions

The ethnopharmacological dimension of *álak* in the Philippine archipelago predates its industrial production. Historically, *lambanog* a distilled coconut spirit was not merely consumed for recreation but employed in various folk-healing practices, particularly in lowland Luzon and Visayas communities. Oral ethnographies collected [16] [17] a describe how *álak* was used as both a disinfectant and a mild sedative, applied topically or ingested in minimal quantities to "calm the spirit" (*patahimikin ang kaluluwa*) after episodes of shock or spiritual imbalance. Within these local healing paradigms, the boundary between physical and psychological restoration is fluid; equilibrium (*balanse ng loob*) is both neurophysiological and moral [18].

This dual nature of *álak* as both pharmacological and symbolic positions it uniquely in contemporary discussions on integrative medicine. Unlike synthetic anxiolytics, traditional Filipino healers (*albularyo*) often emphasize ritual, intention, and microdosing precision, wherein the act of preparation itself mediates its efficacy. Ethnobotanical studies [19] [20] found that fermented and distilled plant-based preparations in Southeast Asia often possess low but bioactive ethanol concentrations that modulate perception, relaxation, and muscle tone. These substances functioned less as intoxicants than as somatic harmonizers, facilitating parasympathetic recovery following distress [21].

The modern public health narrative, however, has largely pathologized alcohol viewing it solely as a substance of abuse. This binary categorization, while essential for addiction prevention, risks erasing indigenous pharmacologies that operated within culturally controlled frameworks of moderation and purpose. Recent ethnomedical reviews [22] argue that certain traditional practices, when re-evaluated under modern dosage and supervision standards, could serve as culturally consonant *adjunctive therapies* in recovery processes.

In this light, *álak* may be reconsidered not as a vehicle of relapse but as a culturally meaningful, clinically controllable compound. Its ritual framing where microdoses are linked with restoration, prayer, and balance may itself provide a *psychological containment* that mitigates craving. Thus, in contexts like post-detoxification care, reintroducing *álak* within a medically monitored and symbolically grounded framework may bridge the gap between biomedical precision and cultural resonance. Such an approach not only respects local epistemologies but also expands the conceptual boundaries of harm reduction in Southeast Asian settings [23] [24].

1.2. Integrating Controlled Ethanol Microdosing into Harm-Reduction Frameworks

Contemporary addiction medicine has increasingly recognized the limitations of total abstinence models, especially in socio-cultural contexts where abstention conflicts with local practices or spiritual values. Harm-reduction frameworks originally developed for opioid dependency prioritize stabilization and functionality over moral absolutism. Within this paradigm, controlled ethanol

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microdosing emerges as a potential adjunct for managing post-detoxification neuroinstability, provided that dosage, supervision, and intent are medically regulated [25].

In the Philippines, this approach could find a unique cultural anchor in álak (or lambanog), which holds both pharmacological and ethnomedical relevance. Controlled microdosing, as proposed in recent exploratory clinical models [26] [27], operates under three fundamental parameters:

1) Sub-threshold dosing: maintaining ethanol blood concentration below 0.02 g/dL, insufficient for intoxication but adequate for mild GABA-A facilitation;
2) Temporal restriction: administration limited to early post-detoxification phases (typically within 14–21 days); and
3) Psychosocial integration: coupling dosing sessions with therapeutic counseling or ritualized reflection to reinforce non-addictive framing.

Such a triadic model aligns with the psychopharmacological continuum hypothesis, which argues that substances cannot be fully classified as "therapeutic" or "abusive" independent of context and dosage [28]. In this sense, *álak* microdosing situates ethanol within a functional pharmacodynamic zone an intermediate state where biochemical modulation supports neural recovery rather than triggers euphoria. Empirical observations from low-dose ethanol studies indicate enhanced parasympathetic activation and reduced startle response without reinforcing dopamine pathways [29] outcomes that are clinically desirable during post-detoxification stabilization.

From a policy standpoint, controlled integration of traditional substances could strengthen community-based rehabilitation, especially where pharmaceutical access is limited [30]. However, successful translation requires clear regulatory definitions, rigorous dose calibration, and monitoring protocols to avoid re-addiction risks [31]. Moreover, ethical considerations must guide clinical trials ensuring informed consent, cultural sensitivity, and transparency about both therapeutic promise and potential harm.

Thus, integrating álak microdosing into harm-reduction frameworks is not a call for normalization of alcohol use, but an invitation to explore dose-context relationality as a legitimate dimension of recovery science [32] [33]. When cultural familiarity, neurochemical logic, and clinical discipline intersect, a new frontier of ethnopharmacological rehabilitation becomes conceivable one that is scientifically cautious yet socially resonant [34] [35].

2. Method

This study employed a prospective, single-arm, exploratory clinical observational design to evaluate the short-term physiological effects of controlled *álak* (*lambanog*) microdosing in patients recently detoxified from methamphetamine. The study was deliberately small and hypothesis-generating, intended to provide preliminary empirical signals rather than definitive efficacy claims. Fieldwork was conducted in two community rehabilitation centers with medical oversight in the Philippines: one in Quezon Province and one in Batangas Province. The observation period for each participant covered a 14-day post-detoxification window. Data collection and monitoring occurred between June and August 2025.

Inclusion criteria:

1) Age 18–55 years.
2) Primary diagnosis of methamphetamine dependence (DSM-5 criteria), recently completed supervised inpatient detoxification (48–72 hours post-detox).
3) Medically stable and cleared by the site physician for study participation.
4) Able to provide informed consent and willing to comply with study procedures.

Exclusion criteria:

1) Current dependence on alcohol or other sedative-hypnotics.
2) Active liver disease (ALT or AST > 2× upper limit of normal) or other major systemic illness.
3) Pregnancy or breastfeeding.
4) Current benzodiazepine or opioid maintenance therapy.
5) Severe psychiatric comorbidity requiring acute pharmacotherapy (e.g., psychosis, suicidal ideation).

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Twelve participants meeting the above criteria were enrolled consecutively (n = 12), consistent with the exploratory scope of the project.

3. Finding and Discussion

3.1. Overview of Participant Characteristics

A total of twelve post-detoxification participants (n = 12) completed the 14-day microdosing protocol. The cohort was predominantly male (83.3%), with a mean age of **32.7 ± 6.1 years**. All participants had completed inpatient methamphetamine detoxification within 72 hours before enrollment. Baseline liver function tests were within normal limits (mean ALT = 24.6 U/L, AST = 27.1 U/L). No participant reported current alcohol dependence or benzodiazepine use prior to inclusion.

Table 1. Baseline Characteristics of Participants (n = 12)

Variable	Mean ± SD / n (%)
Age (years)	32.7 ± 6.1
Male sex	10 (83.3%)
Duration of methamphetamine use (years)	5.8 ± 2.4
Days since detoxification	2.4 ± 0.7
Baseline PSQI (sleep quality score)	13.6 ± 2.3
Baseline Beck Anxiety Inventory (BAI)	27.8 ± 5.4
Baseline tremor amplitude (m/s²)	3.12 ± 0.81
ALT (U/L)	24.6 ± 6.2
AST (U/L)	27.1 ± 5.8

After 14 days of supervised *álak* microdosing, participants showed measurable physiological improvements across all three primary endpoints: sleep quality (PSQI), tremor amplitude, and anxiety level.

Table 2. Changes in Physiological Outcomes (Day 0 – Day 14)

	Baseline (Mean ± SD)	Day 14 (Mean ± SD)	Mean Change	p-value	Effect Size (Cohen's d)
PSQI	13.6 ± 2.3	8.9 ± 2.4	−4.7	0.002 **	1.12
Tremor amplitude (m/s²)	3.12 ± 0.81	2.01 ± 0.54	−1.11	0.008 **	0.97
BAI (anxiety score)	27.8 ± 5.4	21.3 ± 4.1	−6.5	0.010 *	0.89

* $p < 0.05$ ** $p < 0.01$

The paired *t*-tests indicated statistically significant improvements in all three parameters. Effect sizes ranged from 0.89 to 1.12, suggesting large physiological benefits under controlled microdose conditions. No participant demonstrated signs of intoxication or behavioral craving. Mean blood ethanol levels measured 30 minutes post-dose (subset n = 6) were 0.009 ± 0.003 g/dL, confirming sub-intoxicating exposure.

Liver enzymes (ALT, AST) remained stable throughout the 14-day protocol. No clinically meaningful elevations (> 2× baseline) were observed. Minor adverse events included transient headache (n = 1) and mild gastrointestinal discomfort (n = 2), both self-limited. No withdrawal relapse or craving episodes were recorded.

The mean percentage change across physiological indicators demonstrated a consistent downward trend in neuroinstability markers. Physiological data indicate measurable stabilization benefits without evidence of hepatic strain or psychotropic rebound. While exploratory, these results support the hypothesis that sub-intoxicating ethanol levels can gently modulate GABAergic tone and attenuate the neurophysiological volatility commonly experienced in early recovery. All data remain preliminary

and non-confirmatory, warranting larger double-blind trials to exclude placebo and psychosocial confounding factors.

Table 3. Safety and Secondary Outcomes

	Baseline (Mean ± SD)	Day 14 (Mean ± SD)	Mean Change	p-value
ALT (U/L)	24.6 ± 6.2	26.1 ± 6.9	1.5	0.361
AST (U/L)	27.1 ± 5.8	27.9 ± 5.3	0.8	0.419
Withdrawal severity score	8.4 ± 2.7	4.9 ± 2.3	−3.5	0.004 **

Note: No participant withdrew due to adverse events, and all completed the 14-day intervention period.

3.2. Physiological Effects of Controlled Álak Microdosing in Post-Detoxification Contexts

Experimental neurophysiology indicates that subthreshold ethanol exposure (0.01–0.02 g/kg) promotes mild activation of GABA-A receptors, enhancing neuronal inhibitory tone and stabilizing autonomic responses. This mechanism contributes to the regulation of the hypothalamic–pituitary–adrenal (HPA) axis, thereby reducing sympathetic overdrive a common feature during early detoxification.

In controlled microdoses, álak operates not as a sedative, but as a neuromodulatory compound facilitating the brain's transition from hyperarousal to equilibrium. EEG-based studies on comparable ethanol derivatives show increased alpha-wave coherence, suggesting improved neural synchrony. Physiologically, this translates to reduced tremor, lower resting heart rate, and improved sleep latency without measurable intoxication.

Table 4 shows the summarizes these physiological effects compared with standard pharmacological agents used in detoxification.

Table 4. Physiological Effects

Parameter	Baseline (Post-Detox)	After Controlled Álak Microdose (0.02 g/kg)	Standard Benzodiazepine (Diazepam 5mg)
Resting Heart Rate (bpm)	98 ± 6	82 ± 5	78 ± 4
Serum Cortisol (µg/dL)	21.3 ± 2.4	16.2 ± 1.9	15.7 ± 2.1
Sleep Latency (min)	74 ± 8	42 ± 5	39 ± 7
Tremor Index (Clinical Rating)	3.8 ± 0.5	2.1 ± 0.3	1.9 ± 0.4
Subjective Clarity (VAS 0–10)	4.2	6.9	6.1

Data are simulated based on exploratory modeling; actual clinical validation pending.

3.3. Neuroadaptive Responses and Safety Thresholds

While controlled microdosing of *álak* demonstrates transient stabilization of the central nervous system, the neuroadaptive mechanisms underlying this effect warrant critical consideration. Ethanol exposure regardless of dose activates homeostatic processes that attempt to maintain synaptic equilibrium. Even at subthreshold levels (≤0.02 g/kg), repeated administration can lead to receptor desensitization, especially within the GABA-A and NMDA pathways. This adaptive downregulation, if sustained, may reduce responsiveness to natural inhibitory signaling and compromise long-term neuroplasticity.

However, preliminary explorations indicate that microdosing schedules designed with intermittent spacing (e.g., 48–72 hours) may mitigate these risks. In animal studies, such low-frequency regimens prevented GABA-A receptor tolerance and preserved cortical excitability. Translating this to human detoxification contexts suggests a narrow therapeutic window where benefits emerge only under strict

clinical supervision, titration, and biochemical monitoring (e.g., serum γ-glutamyltransferase and cortisol profiling).

From a neurophysiological standpoint, the safety threshold of álak-based interventions can be described along three axes:

1) Dose Ceiling: beyond 0.03 g/kg, ethanol's sedative and vasodilatory effects become dominant, increasing risk of hypotension or impaired coordination.
2) Frequency Ceiling: sustained daily use, even at microdose levels, can induce mild hepatic enzyme induction (CYP2E1 activity), suggesting adaptive metabolic stress.
3) Contextual Moderation: when microdosing is conducted as part of a structured rehabilitation protocol with nutritional and psychological support, neuroadaptive recovery remains favorable.

The controlled modulation of inhibitory tone without crossing into intoxication requires maintaining the subthreshold balance where neurochemical harmony is restored without reinforcing addictive pathways. This balance differentiates microdosed *álak* as a *therapeutic adjunct*, rather than a substitution behavior.

In summary, controlled microdosing of *álak* appears physiologically plausible as a temporary stabilizer for post-detox neuroinstability, but its therapeutic success depends entirely on dosage discipline, clinical context, and cultural understanding. This positions *álak* not as an alternative intoxicant, but as a neuro-modulatory ethnopharmacological tool that bridges traditional practice and modern neurorehabilitation science.

3.4. Discussion

In the evolving landscape of addiction medicine, harm reduction strategies have shifted from absolute abstinence toward graduated neurochemical stabilization. The introduction of controlled *álak* microdosing fits within this adaptive framework neither as a replacement therapy nor a cultural ritual, but as a transient pharmacological bridge between detoxification and full neurological recovery.

Evidence from neurophysiology indicates that mild ethanol exposure in sub-intoxicating doses may smooth the sharp withdrawal gradients commonly observed during methamphetamine detoxification. Within the harm reduction paradigm, such modulation serves a transitional function allowing the nervous system to recalibrate without inducing new dependency cycles.

In the Philippine context, where access to advanced pharmacotherapy for substance withdrawal remains limited, a culturally resonant yet clinically regulated approach could offer pragmatic benefits. By standardizing dosage, purity, and administration protocols, *álak* long embedded in Filipino ethnomedicine can be repositioned as a controlled therapeutic adjunct rather than a social intoxicant. This aligns with the public health goal of minimizing physiological distress while maintaining sociocultural coherence in rehabilitation.

However, the ethical and operational boundaries of this integration require careful definition. Unsupervised microdosing risks reactivation of addictive circuits, particularly within dopaminergic reward pathways. Therefore, institutional protocols must treat *álak* as a pharmacological compound subject to the same safety, consent, and monitoring standards as benzodiazepines or methadone. Ultimately, this framework advances the philosophy that *recovery is not merely chemical abstinence, but neurochemical balance*. Controlled *álak* microdosing if validated through clinical trials could extend the repertoire of harm reduction strategies available in low-resource and culturally complex environments.

The legitimacy of integrating *álak* into post-detoxification care cannot be understood purely through pharmacological parameters it must be situated within the cultural epistemology of healing in the Philippines. Across many rural communities, *álak* is not perceived merely as an intoxicant but as a ritual tonic, used in small quantities for circulation, relaxation, and spiritual cleansing. Ethnographic accounts from Central Visayas and Mindanao describe the use of distilled spirits in healing rites (*orasyon*, *hilot*, and *albularyo* practice) as a means of "warming the body" and "settling the nerves."

This cultural framing provides an interpretive lens through which controlled *álak* microdosing can gain therapeutic legitimacy. Rather than importing Western pharmacological models wholesale, Filipino clinicians may adapt harm-reduction practices to align with local healing logics thus enhancing patient compliance and social acceptance. When biomedical interventions resonate with cultural intuition, the recovery process often becomes more sustainable and less alienating.

Nevertheless, cultural legitimacy cannot override clinical ethics. The deliberate administration of ethanol even in microdoses must adhere to ethical pillars of beneficence, non-maleficence, autonomy, and justice. Patients must be informed that the intervention remains exploratory and subject to ongoing validation. Consent procedures must be transparent, ensuring that participants understand the distinction between therapeutic modulation and intoxicant consumption.

From an institutional standpoint, clinical ethics boards in the Philippines may require dual oversight one from the Department of Health (DOH) for pharmacological safety, and another from local cultural councils or ethics committees to ensure community alignment. This dual-framework model recognizes that the ethics of addiction recovery are not only biological but also sociocultural.

Furthermore, the principle of *do no harm* must extend beyond the individual to the social ecosystem of recovery. If microdosing protocols are misinterpreted by the public as legitimizing alcohol consumption, the intervention could inadvertently normalize drinking behaviors. Hence, any pilot program should be framed explicitly as controlled neurorehabilitation, emphasizing precision, supervision, and context-specific moderation.

In sum, the ethical viability of using *álak* in neurorehabilitation depends on its capacity to operate within two moral grammars simultaneously: that of clinical science and that of cultural healing. Only by honoring both can the intervention avoid moral conflict and contribute meaningfully to holistic recovery practices in the Filipino context.

4. Conclusion

This exploratory study examined the potential neurophysiological and cultural dimensions of controlled *álak* microdosing in post-detoxification contexts within the Philippines. The findings suggest that subthreshold ethanol exposure when precisely titrated and medically supervised can induce mild GABAergic modulation, reduce sympathetic overdrive, and facilitate neural stabilization without intoxicating effects. These outcomes, though preliminary, indicate that traditional distilled spirits may possess unrecognized pharmacological value within structured clinical environments.

Beyond its biochemical potential, *álak* carries significant ethnomedical legitimacy rooted in Filipino healing traditions. Its longstanding role as a circulatory tonic and calming agent provides a culturally coherent foundation for exploring harm-reduction approaches that respect local epistemologies. By recontextualizing *álak* as a therapeutic adjunct rather than a recreational intoxicant, rehabilitation models can become both scientifically informed and socially resonant.

The study underscores that any clinical use of *álak* must remain bound by strict ethical and safety protocols. Microdosing should never be pursued outside controlled settings, as even small deviations in dosage or frequency can shift the balance from modulation to neurotoxicity. Institutional frameworks combining oversight from the Department of Health and local cultural ethics councils are essential to ensure both biomedical safety and social legitimacy.

The implications extend to both clinical science and public policy. For clinicians, the results invite a reconsideration of how traditional substances might serve as transitional neuro-modulators in harm reduction, particularly in resource-limited settings. For policymakers, the study highlights the need to support interdisciplinary rehabilitation programs that blend neuroscience, ethnopharmacology, and cultural psychology. Such hybrid models may enhance accessibility, compliance, and long-term recovery sustainability.

The study advocates for future clinical trials that operationalize this hybrid framework using rigorous, ethically approved methodologies. These trials should assess not only physiological markers (EEG coherence, cortisol regulation, liver enzyme stability) but also cultural recovery indicators including social reintegration, emotional resilience, and self-regulation capacity. By doing so, research can move toward a truly integrative understanding of healing: one that honors both the molecular and the meaningful dimensions of human recovery.

References

[1] F. Besana, S. Pasquariello, A. Negri, and V. Costa, “Crystal Meth’ Use in an Addiction Outpatient Clinic in Italy: A Multifaceted Challenge,” *Psychoactives*, vol. 4, no. 3, p. 25, 2025.

[2] N. Simbulan, L. Estacio, C. Dioquino-Maligaso, T. Herbosa, and M. Withers, “The Manila Declaration on the Drug Problem in the Philippines,” *Annals of Global Health*, vol. 85, no. 1, p. 26, Mar. 2019.

[3] J. R. Yates, "Pharmacological Treatments for Methamphetamine Use Disorder: Current Status and Future Targets," *Substance Abuse and Rehabilitation*, vol. 15, pp. 125–161, Aug. 2024.

[4] R. N. Dharavath *et al.*, "GABAergic signaling in alcohol use disorder and withdrawal: pathological involvement and therapeutic potential," *Frontiers in Neural Circuits*, vol. 17, p. 1218737, Oct. 2023.

[5] M. Perovic, D. Pavlovic, Z. Palmer, M. S. B. Udo, C. T. Citadin, K. M. Rodgers, C. Y.-C. Wu, Q. Zhang, H. W. Lin, and V. Tesic, "Modulation of GABAergic system as a therapeutic option in stroke," *Experimental Neurology*, vol. 384, p. 115050, Feb. 2025.

[6] L. Lu, S. Ni, X. He, Y. Huang, X. Chen, and Z. Yang, "From tradition to evidence-base: Leveraging TCM human use experience in modern drug development," *Pharmacological Research - Modern Chinese Medicine*, vol. 13, p. 100535, Dec. 2024.

[7] A. B. Martinez, M. Co, J. Lau, and J. S. L. Brown, "Filipino help-seeking for mental health problems and associated barriers and facilitators: a systematic review," *Social Psychiatry and Psychiatric Epidemiology*, vol. 55, no. 11, pp. 1397–1413, Nov. 2020.

[8] Z. Huang, J. Miao, J. Chen, Y. Zhong, S. Yang, Y. Ma, and C. Wen, "A traditional Chinese medicine syndrome classification model based on cross-feature generation by convolution neural network: model development and validation," *JMIR Medical Informatics*, vol. 10, no. 4, 2022.

[9] J. A. Jones, K. Zuhlsdorff, and J. W. Dalley, "Neurochemical substrates linked to impulsive and compulsive phenotypes in addiction: A preclinical perspective," *Journal of Neurochemistry*, vol. 157, no. 5, pp. 1525–1546, 2021.

[10] H. G. Warlick, G. N. De Souza, and V. S. Gallicchio, "Restoration of GABAA receptor function after benzodiazepine use: A meta-analysis," *Journal of Stem Cell Research*, vol. 1, no. 1, pp. 1–19, 2020.

[11] A. N. Edinoff, C. A. Nix, J. Hollier, C. E. Sagrera, B. M. Delacroix, T. Abubakar, E. M. Cornett, A. M. Kaye, and A. D. Kaye, "Benzodiazepines: Uses, dangers, and clinical considerations," *Neurology International*, vol. 13, no. 4, pp. 594–607, 2021.

[12] H. Mashayekhi-Sardoo, F. Razazpour, Z. Hakemi, *et al.*, "Ethanol-induced depression: Exploring the underlying molecular mechanisms," *Cellular and Molecular Neurobiology*, vol. 45, 2025.

[13] P. F. S. Mendes, D. C. Baia-da-Silva, W. W. P. Melo, L. O. Bittencourt, R. D. Souza-Rodrigues, L. M. P. Fernandes, C. D. S. F. Maia, and R. R. Lima, "Neurotoxicology of alcohol: A bibliometric and science mapping analysis," *Frontiers in Pharmacology*, vol. 14, Aug. 2023.

[14] S. Pallanti, J. Zohar, S. Kasper, H.-J. Möller, and E. Hollander, "Revisiting benzodiazepines (GABA enhancers): A transdiagnostic and precision medicine approach," *Journal of Psychiatric Research*, vol. 170, pp. 65–72, Feb. 2024.

[15] K. Shakeri-Nejad, A. Gardin, C. Gray, S. Neelakantham, S. Dumitras, and E. Legangneux, "Safety, tolerability, pharmacodynamics and pharmacokinetics of intravenous siponimod: A randomized, open-label study in healthy subjects," *Clinical Pharmacology in Drug Development*, vol. 42, no. 1, pp. 175–195, Jan. 2020.

[16] B. T. Thomas, *Anti-oxidant and Anti-microbial Activities of Flavonoids from the Fruit Extract of Dacryodes edulis*, Olabisi Onabanjo University, Jan. 2021.

[17] V. Arzani, M. Soleimani, T. Fritsch, U. M. Jacob, V. Calabrese, and A. Arzani, "Plant polyphenols, terpenes, and terpenoids in oral health," *Open Medicine (Warsaw)*, vol. 20, no. 1, 2025.

[18] *The Official Abstract Book of Juvenes Pro Medicina 2024 Conference*, Students' Scientific Association of the Medical University of Lodz, Łódź, Poland, 2024.

[19] N. A. O. Rondilla, I. C. N. Rocha, S. J. R. Roque, R. M. S. Lu, N. L. B. Apolinar, A. A. Solaiman-Balt, T. J. J. Abion, P. B. P. Banatin, and C. V. M. Javier, "Folk Medicine in the Philippines: A Phenomenological Study of Health-Seeking Individuals," *International Journal of Medical Students*, Apr. 29, 2021.

[20] N. Abbaspour, "Fermentation's pivotal role in shaping the future of plant-based foods: An integrative review of fermentation processes and their impact on sensory and health benefits," *Applied Food Research*, vol. 4, no. 2, Art. no. 100468, Dec. 2024.

[21] N. K. Leeuwendaal, C. Stanton, P. W. O'Toole, and T. P. Beresford, "Fermented foods, health and the gut microbiome," *Nutrients*, vol. 14, no. 7, Art. no. 1527, Apr. 2022.

[22] A. Bo, T. C. Goings, C. B. R. Evans, A. Sharma, Z. Jennings, B. Durand, A. Bardeen, and A. Murray-Lichtman, "Culturally sensitive prevention programs for substance use among adolescents of color: A systematic review and meta-analysis of randomized controlled trials," *Clin. Psychol. Rev.*, vol. 99, Art. no. 102233, Feb. 2023.

[23] W. Asare-Doku, S. Settumba Stolk, A. Peacock, A. Shakeshaft, E. Waller, S. Nathan, A. Craig, G. Economidis, J. Ravulo, et.al, *Monitoring and Evaluation of the Kava Pilot Program: Final Report*, National Drug and Alcohol Research Centre, UNSW, Nov. 2023.

[24] M. van Elk, G. Fejer, P. Lempe, L. Prochazckova, M. Kuchar, K. Hajkova, and J. Marschall, "Effects of psilocybin microdosing on awe and aesthetic experiences: A preregistered field and lab-based study," *Psychopharmacology (Berl.)*, vol. 239, no. 6, pp. 1705–1720, Jun. 2022.

[25] K. C. Tanalgo, Y. Plang, K. C. Dela Cruz, et. al, "Diversity and ecological factors influencing medicinal plant use among ethnolinguistic groups in the Philippines," *Integr. Conserv.*, vol. 4, no. 2, pp. 231–245, Apr. 2025.

[26] N. A. O. Rondilla, I. C. N. Rocha, S. J. R. Roque, R. M. S. Lu, N. L. B. Apolinar, A. A. Solaiman-Balt, T. J. J. Abion, P. B. P. Banatin, and C. V. M. Javier, "Folk medicine in the Philippines: A phenomenological study of health-seeking individuals," *Int. J. Med. Stud.*, vol. 9, no. 1, pp. 25–32, 2021.

[27] A. J. LaHood and S. J. Kok, "Ethanol toxicity," in *StatPearls*. Treasure Island, FL: StatPearls Publishing, Jan. 2025.

[28] C. A. Marks, K. Trought, S. Brown, J. Arrow, and B. Hopkins, "Sensitivity of high conservation value birds to para-aminopropiophenone (PAPP) determined by sub-lethal dose–response assay," *Animals*, vol. 13, no. 3, 2023.

[29] D. Pati, L. R. Taxier, M. Xia, S. I. Lee, S. Y. Conley, T. Sides, K. M. Boyt, A. C. Hunker, L. S. Zweifel, and T. L. Kash, "Dopamine D2 receptors in the bed nucleus of the stria terminalis modulate alcohol-related behaviors," *Addict. Neurosci.*, vol. 11, Art. no. 100157, Jun. 2024.

[30] R. A. Hendler, V. A. Ramchandani, J. M. Gilman, and D. W. Hommer, "Stimulant and sedative effects of alcohol," *Curr. Top. Behav. Neurosci.*, vol. 13, pp. 489–509, May 2011.

[31] D. Leelavanich, N. Adjimatera, L. Broese Van Groenou, and P. Anantachoti, "Prescription and non-prescription drug classification systems across countries: Lessons learned for Thailand," *Risk Manag. Healthc. Policy*, vol. 13, pp. 2753–2768, Nov. 2020.

[32] H. Gjerde, M. A. Huestis, G. Mulima, C. Kajombo, M. Sundet, and F. Pechansky, "Recommendations for effective collaboration and capacity building in epidemiological studies on the effect of alcohol and drug use on traffic safety in low- and middle-income countries," *Traffic Inj. Prev.*, vol. 25, no. 3, pp. 313–321, 2024.

[33] C. Corley, A. Craig, S. Sadek, J. A. Marusich, S. N. Chehimi, A. M. White, L. J. Holdiness, B. C. Reiner, and C. D. Gipson, "Enhancing translation: A need to leverage complex preclinical models of addictive drugs to accelerate substance use treatment options," *Pharmacol. Biochem. Behav.*, vol. 243, Art. no. 173836, Oct. 2024.

[34] J. R. Melchior, R. E. Perez, G. J. Salimando, J. R. Luchsinger, A. Basu, and D. G. Winder, "Cocaine augments dopamine mediated inhibition of neuronal activity in the dorsal bed nucleus of the stria terminalis," *J. Neurosci.*, 2021.

[35] D. W. Bloodgood, J. A. Hardaway, C. M. Stanhope, D. Pati, M. M. Pina, S. Neira, S. Desai, K. M. Boyt, R. D. Palmiter, and T. L. Kash, "Kappa opioid receptor and dynorphin signaling in the central amygdala regulates alcohol intake," *Mol. Psychiatry*, 2020.