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The Efficacy and Safety of Ibogaine Treatment for Substance Use Disorders – A Narrative Review

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ABSTRACT

Introduction and aim of the study. Substance use disorders, including opioid, cocaine, and alcohol dependence, represent a significant global health challenge with high relapse rates despite existing treatments. Ibogaine, a naturally occurring indole alkaloid derived from Tabernanthe iboga, has attracted increasing scientific interest due to its reported ability to reduce withdrawal symptoms and cravings. Our study aims to evaluate the efficacy and safety of ibogaine in the treatment of substance use disorders and to determine its therapeutic potential.
Materials and methods. The paper is based on an analysis of studies available in databases such as PubMed, Google Scholar, and other scientific databases. Observational studies, retrospective surveys, controlled trials, and review papers on the use of ibogaine in the context of opioid, cocaine, and alcohol dependence were used.
Conclusions. Ibogaine, due to its modulation of serotonergic, dopaminergic, and glutamatergic neurotransmitter systems, shows promising potential in the treatment of substance use disorders.

However, serious safety concerns exist, particularly cardiotoxicity manifesting as QT prolongation and potentially fatal arrhythmias. It is advisable to conduct further large-scale randomized controlled trials aimed at a more thorough evaluation of the efficacy and safety of ibogaine in the treatment of addiction.
Keywords: ibogaine, substance use disorders, opioid dependence, cocaine dependence, alcohol dependence, addiction treatment

1. Introduction

Ibogaine is one of the most potent naturally occurring psychoactive substances known to humankind. It is an indole alkaloid derived from the root bark of *Tabernanthe iboga*, a plant which grows in several countries of West Central Africa [1][2]. For centuries, it has been used by practitioners of the Bwiti religion in ceremonial rites to connect with the ancestors [3] and attain spiritual knowledge, as well as for its stimulant effects in suppressing hunger and fatigue [4].

Over the past few decades, ibogaine has attracted increasing scientific interest for its potential therapeutic applications, particularly in the treatment of substance use disorders. A growing body of preliminary research suggests that ibogaine may be effective in reducing cravings and alleviating withdrawal symptoms, most notably in individuals with opioid dependence. These effects are hypothesized to arise from ibogaine's unique and multifaceted interactions with multiple neurotransmitter systems [3].

Despite the encouraging findings, ibogaine has also been associated with potentially serious or even life-threatening adverse effects, including cardiotoxicity and possible neurotoxicity, which raise substantial safety concerns and makes the clinical use of ibogaine highly controversial. Its legal status varies widely across countries, and often is classified as a controlled substance, which significantly limits formal clinical research [5].

This study constitutes a narrative review of the existing literature on ibogaine, synthesizing current knowledge regarding its pharmacological profile, possible mechanisms of action, therapeutic potential, and associated risks. The primary aim of this review is to critically evaluate the possible efficacy of ibogaine in the treatment of substance use disorders, while carefully weighing these potential benefits against the documented safety concerns and limitations surrounding its use.

2. Mechanisms of Action

The pharmacological mechanisms of ibogaine are highly complex and remain incompletely understood. Studies indicate that ibogaine simultaneously modulates several neurotransmitter systems, most notably the serotonergic and dopaminergic systems [6]. Ibogaine has been shown to noncompetitively inhibit the serotonin transporter (SERT), exhibiting high potency as a serotonin reuptake inhibitor, and to competitively block the dopamine transporter (DAT) [3][7][8]. In addition to its effects on monoamine transporters, ibogaine demonstrates agonistic activity at serotonin 5-HT_{2}A receptors, κ-opioid receptors and sigma-2 receptors [3][9].

Ibogaine has also been identified as an antagonist of the α3β4 nicotinic acetylcholine receptor, which has been associated with the upregulation of glial cell line-derived neurotrophic factor (GDNF) in the ventral tegmental area (VTA) [10], a midbrain structure engaged in reward processing. The VTA is a primary target of addictive substances, which typically enhances dopamine release and contributes to compulsive drug use and habit formation [11].

Furthermore, ibogaine acts as an antagonist of the N-methyl-D-aspartate (NMDA) receptor, a receptor critically involved in synaptic plasticity and the neural mechanisms underlying drug-induced behaviors, including craving and relapse [3].

Collectively, these diverse pharmacological actions may explain ibogaine's reported effects on addiction-related processes.

3. Substance Use Disorders (SUDs)

Drugs of abuse elevate the level of extracellular dopamine in the striatum, a brain region responsible for incentive-based learning and reward evaluation. Its activity is also associated with pathological risk-taking and the development of addictive behaviors [13].

Increased dopamine signaling in the dorsal striatum has been associated with the motivation to seek and obtain drugs when individuals with addiction are exposed to drug-associated cues (conditioned stimuli), whereas chronic drug use is associated with a progressive decline in dopaminergic function, reflected in reduced dopamine D2 receptor availability and diminished dopamine release in the striatum of addicted individuals [14].

3.1. Opioid Use Disorder

The most extensive clinical data for ibogaine treatment derive from opioid use disorder populations. Preclinical evidence suggests that NMDA receptor antagonism contributes to the suppression of opioid withdrawal symptoms [15], providing a mechanistic rationale for ibogaine's observed clinical effects.

3.1.1. Acute Efficacy

A recent observational study [6] enrolled 191 treatment-seeking opioid- and cocaine-dependent individuals (144 men, 47 women) who met DSM-IV criteria and demonstrated active use at program entry. Participants received oral ibogaine HCl (8-12 mg/kg) under open-label conditions with comprehensive safety monitoring including 12-lead ECG and continuous telemetry. Opioid-dependent patients were transitioned to short-acting morphine sulfate prior to ibogaine administration. The cohort represented a treatment-resistant population with an average of 5.5 ± 7.2 prior treatment admissions, 19.2 ± 13.0 days of opioid use in the preceding month, and 11.2 ± 8.6 years of lifetime use.

Participants demonstrated significantly decreased craving across all five Heroin Caving Questionnaire-29 (HCQ-29) domains at both program discharge and one-month follow-up compared to baseline ($p < 0.0001$). The magnitude of craving reduction was consistent across all measured dimensions, suggesting comprehensive interruption of the psychological drivers of compulsive use.

Patient surveys corroborate these clinical findings: in a retrospective survey of 88 patients treated for opioid dependence, 80% reported that ibogaine either eliminated or drastically reduced their withdrawal symptoms, and 50% reported a reduction in opioid cravings-with 25% indicating that craving reduction persisted for three months or longer [16].

3.1.2. Longitudinal Abstinence Outcomes

While the immediate effects of ibogaine on craving and withdrawal are well-documented, the question of long-term efficacy is critical for evaluating its therapeutic potential. Several observational studies have tracked opioid-dependent individuals for periods extending up to 12 months following ibogaine treatment.

A prospective observational study conducted in Mexico (n=30) tracked participants using the Addiction Severity Index (ASI) over 12 months. At one-month follow-up, 50% of participants reported total abstinence from opioids. Subsequent assessments revealed abstinence rates of 33% at 3 months, 20% at 6 months, 37% at 9 months, and 23% at 12 months [17].

More robust findings emerged from a legally regulated observational study in New Zealand (n=14), which employed laboratory verification through urine screening. This study reported sustained negative results for opioids in 87.5% of participants at 3 months, 85.7% at 6 months, and 75% at 12 months [18]. Notably, the study also documented highly significant reductions in ASI drug use composite scores at 12 months ($p=0.002$).

A retrospective survey of 88 patients treated in Mexico between 2011 and 2015 found that 30% of participants reported never returning to opioid use after their initial treatment. Among these sustained abstainers, 54% had remained sober for at least one year and 31% for at least two years. At the time of survey, 41% of all respondents reported sustained abstinence exceeding six months [17]. A Brazilian retrospective study (n=75) focusing on polydrug users reported that 61% remained abstinent during follow-up, with median abstinence durations of 5.5 months for single-treatment recipients and 8.4 months for those receiving multiple treatments [19].

3.1.3. Comparative Efficacy

These outcomes gain particular significance when compared to conventional treatment approaches. Standard success rates for maintaining abstinence eight weeks after short-term buprenorphine-naloxone taper have been recorded as low as 6.6% [20]. A comparative analysis from UC Davis utilizing abstinence rates at one-month post-treatment follow-up found that 50% of patients in the ibogaine cohort displayed no opioid use, compared to only 18% abstinence in the buprenorphine group [21]. Critically, while medication-assisted treatments such as methadone and buprenorphine require daily administration to maintain stability, ibogaine is typically administered as a single "flood dose" intended to facilitate rapid detoxification and provide a window of opportunity for behavioral change. When asked to compare ibogaine to other detoxification methods they had previously attempted, approximately 71% of surveyed patients rated ibogaine treatment as "much better," and 85% stated they would make the same decision to seek treatment again [17].

3.1.4. Harm Reduction Outcomes

Even for individuals who do not achieve complete long-term abstinence, ibogaine treatment demonstrates significant harm reduction potential. In Davis et al. study, approximately 48% of those who relapsed following treatment reported that their post-treatment drug use substantially decreased in both frequency and volume compared to pre-treatment levels. Furthermore, participants frequently demonstrated durable improvements in legal status, family and social relationships, and psychiatric well-being for up to 12 months, regardless of whether they achieved total cessation [16]. These findings suggest that ibogaine's therapeutic value extends beyond binary abstinence outcomes to encompass broader measures of recovery and quality of life.

It must be noted that these results should be interpreted with caution due to methodological limitations. Most data derive from small convenience samples and rely predominantly on self-reporting rather than consistent laboratory verification. Rigorous, large-scale randomized controlled trials remain lacking to definitively validate these success rates.

3.2. Cocaine Use Disorder

Cocaine Use Disorder (CUD) is characterized by the compulsive use of cocaine despite the consequences it brings to an individual's mental health, physical well-being and behavioral functioning [22].

CUD, like other substance use disorders, is a chronic condition marked by high relapse rates, which often requires multiple treatment approaches to sustain the recovery. Currently, the gold-standard practice for treating CUD are psychosocial treatments. However, most individuals never receive proper care due to limited availability of effective and accessible treatment programmes [22].

The results from the 2019 National Survey on Drug Use and Health showed that 5.5 million people in the population aged 12 and older used cocaine past year, thus it is considered a global public health problem. It is estimated that about one in 16 to 20 cocaine users will become dependent within the first year after the initial use [23].

The global prevalence of cocaine use and the absence of a consistently effective treatment, emphasizes a critical need to explore novel therapeutic approaches. In this context, the investigation of ibogaine has drawn increasing scientific interest as a potential therapeutic option for cocaine dependence.

Ibogaine's anti-addictive effects in cocaine use disorder may involve the neuropeptide dynorphin A. Following cocaine exposure, dynorphin A is released in the striatum and produces dysphoric effects by activating κ-opioid receptors-essentially counteracting cocaine's rewarding properties [24]. Preclinical studies in rats found that while ibogaine alone did not increase striatal dynorphin A levels but pretreatment with ibogaine markedly amplified the dynorphin A release triggered by cocaine [25].

These findings suggest that ibogaine may enhance the brain's natural aversive response to cocaine, making the drug experience less rewarding. If confirmed in humans, this mechanism could partly explain ibogaine's effectiveness in reducing cocaine use, though further investigation is needed [25][26].

3.2.1. Acute Efficacy

A recent open-label study with human volunteers seeking to detoxify from cocaine (n = 191) reported statistically significant reductions in craving the drug supported by measures both at discharge and at one-month follow-up [10]. Specifically, the improvements were observed across multiple validated scales, including Cocaine Craving Questionnaire (CCQ) and Minnesota Cocaine Craving Scale (MCCS). Assessment using the Cocaine Craving Questionnaire (CCQ-29) indicated that ibogaine-assisted detoxification was associated with a marked reduction in cocaine craving ($p < 0.0001$). Consistent findings were obtained with the Minnesota Cocaine Craving Scale, which demonstrated significant decreases across the three core dimensions of craving (intensity, frequency and duration) at the time of program discharge

($p < 0.0001$). Follow-up evaluations conducted approximately one month after discharge suggested sustained beneficial effects of ibogaine detoxification on drug craving in cocaine-dependent individuals.

3.2.2. Controlled Trial Evidence

While large-scale controlled trials for cocaine use disorder are lacking, a pilot randomized, placebo-controlled study (n=20) conducted in Brazil provides the most methodologically rigorous evidence currently available [27]. This trial enrolled male participants diagnosed with cocaine dependence according to DSM-IV criteria. The experimental group (n=10) received a single oral dose of 1800 mg of encapsulated ibogaine extract (75% purity), while the control group (n=10) received placebo capsules containing sugar powder. Participants were followed for 24 weeks with biweekly visits during which urine samples were collected to detect cocaine use.

The ibogaine group demonstrated statistically significant reductions in cocaine craving as measured by the Minnesota Cocaine Craving Scale (MCCS). Craving intensity scores dropped from a baseline of 7.4 to 1.3 at 72 hours post-administration and remained suppressed at 2.6 after 24 weeks. In contrast, no such improvement was observed in the placebo group. Analysis of urine toxicology samples revealed a lower rate of cocaine-positive results in the ibogaine-treated group over the 24-week follow-up period, suggesting reduced relapse frequency. Importantly, no serious adverse reactions or cardiovascular events were reported, and electrocardiograms remained normal for both groups throughout the study.

Despite these encouraging findings, methodological limitations warrant cautious interpretation. The small sample size limits statistical power and generalizability. Additionally, the study utilized 75% pure ibogaine extract rather than pharmaceutical-grade ibogaine hydrochloride, complicating comparison with other investigations.

Researchers at the Institute of Psychiatry at the University of São Paulo (USP) are preparing a larger Phase IIb randomized, placebo-controlled trial (NCT03380728) with 80 cocaine and crack-dependent participants to more rigorously establish safety and efficacy parameters [28]. Such larger-scale investigations are essential to validate the promising preliminary findings and advance ibogaine toward potential clinical application for cocaine use disorder.

Collectively, the available evidence suggests that ibogaine may produce rapid and sustained reductions in cocaine craving and use. The immediate onset of therapeutic effects distinguishes it from conventional approaches and positions ibogaine as a potentially transformative intervention for a condition that currently lacks effective pharmacological treatments.

3.3. Alcohol Use Disorder

Clinical evidence for alcohol use disorder is more limited than for opioid and cocaine dependence, though the GDNF mechanism provides a compelling rationale for ibogaine's potential efficacy.

3.3.1. The GDNF Mechanism

A cornerstone hypothesis regarding ibogaine's impact on reducing alcohol consumption centers on its ability to upregulate GDNF expression within the ventral tegmental area (VTA), a critical node in the mesocorticolimbic reward pathway [29]. Chronic ethanol consumption inhibits the excitability of VTA neurons [30], leading to reduced firing rates of dopaminergic cells during early abstinence [31]. GDNF, by contrast, enhances the excitability of midbrain dopaminergic neurons through inhibition of A-type potassium channels [32], effectively counteracting the neuroadaptations induced by chronic alcohol exposure.

Preclinical studies have provided direct evidence for GDNF as a primary mediator of ibogaine's effects on lowering alcohol consumption. Microinjection of ibogaine into the VTA precipitously decreases ethanol consumption in rodent models, while co-administration of anti-GDNF neutralizing antibodies into the VTA successfully blocks this effect [29].

A particularly intriguing finding is that a single dose of ibogaine appears to initiate an autoregulatory positive feedback loop. The initial induction of GDNF protein triggers signaling pathways that lead to further transcription of the GDNF gene, potentially explaining why the drug's effects can persist for weeks or months after it has been cleared from the body [29].

3.3.2. Comparative Pharmacotherapy

Ibogaine's proposed mechanism represents a fundamentally different approach compared to conventional AUD pharmacotherapies. Naltrexone reduces the rewarding effects of alcohol through opioid receptor antagonism, while acamprosate helps maintain abstinence by modulating glutamatergic neurotransmission; however, both require chronic daily dosing and do not address long-term neuroadaptations in the restorative manner proposed for ibogaine [33]. Ibogaine, by contrast, is theorized to promote a state of enhanced neuroplasticity, creating a finite "window of opportunity" during the post-treatment period where behavioral changes and new habits can be more easily established.

3.3.3. Clinical Evidence

Human clinical data specifically for AUD remain limited but are beginning to emerge. A retrospective analysis of 75 polydrug users in Brazil, which included individuals dependent on alcohol, found that 61% remained abstinent at follow-up [19]. Patients who underwent a single ibogaine treatment reported a median abstinence duration of 5.5 months, while those receiving multiple treatments extended their median abstinence to 8.4 months.

3.4. The Therapeutic Experience

Beyond objective measures of abstinence and craving, the subjective dimensions of ibogaine treatment provide critical insight into its therapeutic mechanisms. A significant portion of ibogaine's perceived therapeutic value is attributed to its "oneirophrenic" (waking dream) - dream-like introspective experience that facilitates deep psychological insight and may provide additional therapeutic benefit as an adjuvant to subsequent psychotherapy [6][16][34].

In the Mash et al. observational study (n=191) [6], 67% of patients reported gaining insightful knowledge about themselves during the ibogaine experience, and 43% gained specific insights into the root causes of their addiction. The majority of participants (88%) rated their experience as one of the five most spiritually significant events of their lives.

Importantly, data suggest a correlation between subjective experience quality and treatment outcomes. "Treatment responders"-defined as those who achieved long-term abstinence or significant use reduction-rated their sessions as significantly more spiritually meaningful and insightful than non-responders. Subjects frequently described feeling "cleansed" or reborn (50%) and being given a second chance at life (40%). Some reported seeing images of their own death and gaining an impending awareness of self-destruction if they failed to achieve abstinence (18.3%). These profound subjective experiences may contribute to the motivational and behavioral changes necessary for sustained recovery. Patients also frequently report lasting changes in personality and outlook, including increased gratitude, authenticity, and appreciation for life. These qualitative shifts suggest that ibogaine's therapeutic effects extend beyond neurochemical recalibration to encompass meaningful psychological transformation.

4. Safety Profile

4.1. Acute Tolerability

Data from Mash et al. [6] provide insight into the tolerability profile under controlled conditions. Ibogaine was generally well tolerated, with nausea, vomiting, and ataxia as the most common side effects observed shortly after administration. Perceptual changes during the absorption phase typically subsided within 4-6 hours. No serious adverse events occurred in this cohort. Orthostatic hypotension was observed in 5% of subjects, with approximately 2% experiencing moderate-severity adverse events. Notably, orthostatic hypotension and bradycardia occurred more frequently in cocaine-dependent subjects, likely attributable to volume depletion; routine administration of intravenous fluids one hour prior to dosing effectively normalized these symptoms. Survey data corroborate these findings: approximately 74% of patients reported significant physical discomfort during the acute phase, including nausea, ataxia, and tremors [19]. Many patients reported residual effects lasting up to 72 hours, including exhaustion and insomnia, which typically required a "grey day" of rest before mood and energy normalized.

4.2. Cardiotoxicity

The primary safety concern with ibogaine treatment is sudden cardiac death and related complications, ranging from transient, self-limiting bradycardia to life-threatening prolonged QT interval with polymorphic ventricular tachycardia and cardiac arrest. The blockade of hERG (human ether-à-go-go-related gene) potassium channels plays a crucial role in ibogaine's cardiotoxicity, as it delays cardiac repolarization, resulting in QT prolongation and, subsequently, ventricular tachycardias including Torsades de Pointes arrhythmias. Both direct cardiotoxicity and modulation of myocardial ion channels contribute to the prodysrhythmic effects of ibogaine. Electrolyte imbalances further amplify this risk: hypokalemia and hypomagnesemia were detectable in 100% of analyzed fatality cases where such testing was performed, as these imbalances potentiate hERG channel blockade and further delay cardiac repolarization. [35] In the majority of reported cases, patients who experienced life-threatening cardiac events-including ventricular arrhythmias and cardiac arrest triggered by QTc prolongation-improved with supportive management, although a small number experienced post-arrest neurologic deficits due to hypoxia [26].

Importantly, case reports indicate that ibogaine can cause ventricular tachyarrhythmias and QT prolongation even in individuals without pre-existing cardiovascular conditions or family history, underscoring that cardiac risk is not limited to those with obvious predisposing factors [36].

These findings highlight that while ibogaine carries inherent cardiac risk, many adverse outcomes may be preventable through rigorous patient selection and medical supervision.

4.3. Neurotoxicity

Animal studies have demonstrated neurodegenerative processes associated with ibogaine administration, specifically excitotoxic effects on Purkinje cells in the cerebellum [37]. As tremors in rodents are associated with stimulation of the inferior olive, concerns have been raised about potential neurotoxicity in humans at therapeutic dosages [26]. However, it remains unclear whether the olivo-cerebellar organization in humans is comparable to that of rodents.

Some evidence suggests ibogaine may be less neurotoxic in humans. An autopsy performed on a woman who had received four doses of ibogaine (10-30 mg/kg) over 15 months revealed no signs of cerebellar damage and normal Purkinje cells [38]. In clinical studies, early nausea and mild tremors were frequently reported but resolved without sequelae. However, case reports document more severe neurological symptoms including ataxia, muscle spasms, tonic-clonic seizures, and severe nausea [36][39][40][41][42][43].

In isolated cases, permanent cognitive deficits and vision loss persisted for weeks following hospitalization [43]; these deficits were attributed to hypoxia during ibogaine-induced respiratory depression rather than direct neurotoxicity.

4.4. Mortality and Risk Factors

A critical question for evaluating ibogaine's clinical viability is the actual incidence of treatment-related mortality. From 1990 until 2020, approximately 33 deaths temporally associated with ibogaine ingestion have been documented in the peer-reviewed literature [44].

4.4.1. Outcomes in Supervised Medical Settings

Evidence from well-controlled clinical environments demonstrates markedly lower mortality when rigorous protocols are followed. A systematic review by Köck et al. [5], analyzing 24 studies encompassing 705 individuals, identified only two fatalities within those research cohorts. Similarly, the Mash et al. study [6] and Brazilian retrospective data [19] reported no fatalities under optimal conditions-comprehensive screening, continuous monitoring, and immediate access to cardiac intervention. While QT prolongation was observed at higher plasma concentrations, no subjects in the Mash et al. study developed clinically significant arrhythmias.

These data suggest that ibogaine-related mortality can be significantly reduced through systematic screening, exclusion of high-risk patients, and continuous monitoring.

By contrast, the New Zealand fatality involving "Mrs. A" illustrates the consequences of inadequate protocol adherence. The Health and Disability Commissioner investigation found that monitoring was not maintained to medical standards, the dose administered was unusually high, and medication interactions were not properly managed [18]. This case reinforced that physicians assuming responsibility for ibogaine treatment must maintain non-delegable medical supervision throughout the critical 24-48 hour post-administration period.

5. Conclusions

Despite significant advances in the pharmacological and psychosocial treatment of substance use disorders, current therapeutic approaches remain insufficient for a substantial proportion of individuals struggling with addiction. High relapse rates and limited effectiveness in certain populations determine the urgent need for the exploration of novel and alternative treatment strategies. The emerging evidence on ibogaine suggests potential benefits, particularly in the management of withdrawal symptoms and the reduction of substance craving, warranting further scientific attention.

Conceptually, ibogaine appears to function as a neurochemical "recalibration" - providing a window of opportunity for behavioral change by interrupting the physical dependency cycle-rather than as a cure requiring ongoing administration. This distinguishes it mechanistically from conventional maintenance-based approaches. However, long-term success remains highly dependent on post-treatment integration and psychosocial support; without these elements, the risk of returning to established behavioral patterns persists.

Although ibogaine is associated with serious and potentially life-threatening adverse effects, including cardiotoxicity, these risks should not categorically prevent its investigation as a therapeutic agent if the clinical benefits outweigh the associated harms. Rather, they emphasize the necessity for rigorous research protocols and comprehensive risk-benefit assessments. Any continued investigation or clinical application of ibogaine should be confined to strictly controlled medical settings, incorporating continuous physiological monitoring and thorough pre-treatment screening. The establishment of standardized treatment guidelines, clear exclusion criteria, and safety protocols is essential to minimize the likelihood of fatal outcomes. Patients must be fully informed of the potential risks, including the possibility of sudden cardiac death, as part of an ethical and transparent informed consent process.

The critical gap in the current evidence base is the absence of large-scale, randomized controlled trials. Until such trials are conducted with adequate sample sizes, standardized protocols, and long-term follow-up, the true efficacy and optimal treatment parameters will remain uncertain. The planned Phase IIb trial for cocaine dependence at the University of São Paulo represents a step toward addressing this deficit, but similar efforts are urgently needed for other substance dependences.

Finally, further research should also focus on the development and evaluation of ibogaine analogues or ibogaine-like compounds that may retain therapeutic efficacy while offering improved safety profiles. Such efforts could contribute to expanding the range of viable treatment options for addiction and ultimately improve outcomes for individuals for whom existing therapies have proven inadequate.

Author's Contribution:

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Formal analysis: ML,ZC,DP,KD,AC,BC,MW

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