INNOVATION > HEALTHCARE

Ibogaine's Impact Lasted A Year: How Should We Interpret It?

By Robert Glatter, MD, Contributor. Robert Glatter is a New York-based...

Follow Author

Published Sep 01, 2026, 02:15pm EDT

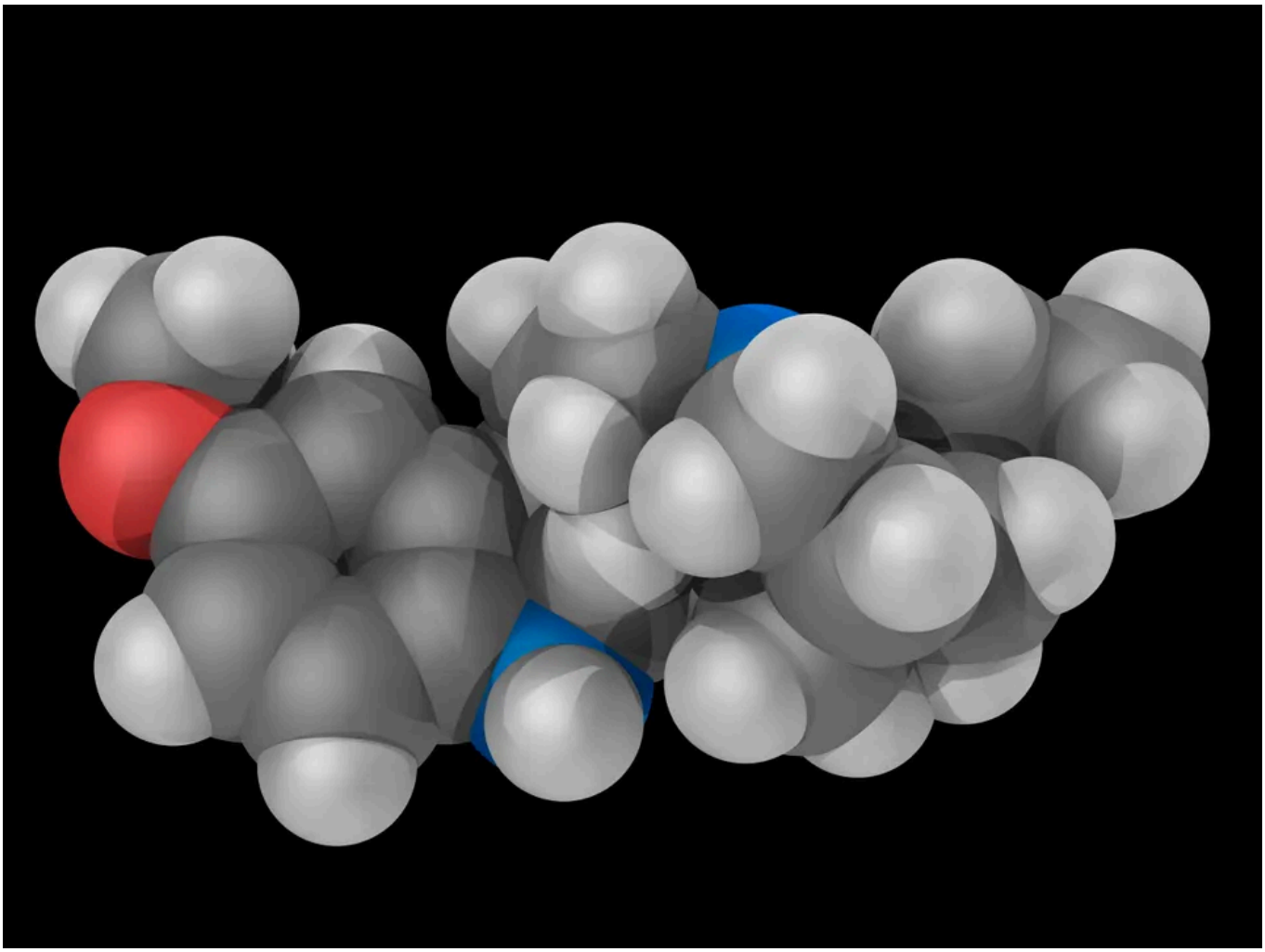
Add Us On Google

0:00 / 16:47

This voice experience is generated by AI. Learn more.

Summary

Show More



Ibogaine, molecular model. Naturally occurring psychoactive substance found in a number of plants. Atoms are represented as spheres and are colour-coded: carbon (grey), hydrogen (white), nitrogen (blue) and oxygen (red).
GETTY

In 2024, a Stanford team reported a finding that drew immediate attention across psychiatry, traumatic brain injury research, and psychedelic medicine.

Thirty former U.S. Special Operations service members, all living with repeated traumatic brain injuries (TBIs) from combat and blast exposure, had traveled to Mexico for treatment with ibogaine, a psychoactive alkaloid from the West African shrub Tabernanthe iboga. Afterward, they showed striking reductions in post-traumatic stress, depression, anxiety, and functional disability .

The obvious question was whether those improvements would last. A new 12-month follow-up from the Magnesium–Ibogaine: Stanford Traumatic Injury to the CNS (MISTIC) study suggests much of it did. But the finding is both remarkable and considerably more complicated than “one treatment lasted a year” implies — and the complication may matter more than the headline.

The paper also carries a legacy. Nolan Williams, MD, who directed the Stanford Brain Stimulation Laboratory, conceived and led MISTIC as principal investigator; he died in October 2025, before the follow-up manuscript was submitted, and the paper is dedicated to him. Cammie Rolle, PhD, Clinical Assistant Professor of Psychiatry and Behavioral Sciences at Stanford University School of Medicine, assumed corresponding author responsibilities.

What follows is a tribute to Nolan's life and work--of a remarkable and inspiring young investigator who had insights that will shape and transform the trajectory of how a unique medicine, ibogaine, can transform the lives of those suffering from TBI, leading to depression, anxiety and PTSD.

BENAVIDES, TEXAS - MAY 27: Jesus Bocanegra, 24, walks in uniform to a Memorial Day weekend service May 27, 2006 in Benavides, Texas. Bocanegra has been diagnosed with Post-Traumatic Stress Disorder, or PTSD, a result of his se ... More
GETTY IMAGES

What happened Over The Following Year

25 of the original 30 participants completed assessments at roughly 12 months, with evaluations at three, six, and nine months as well. PTSD, depression, and anxiety symptoms, along with self-reported functional disability, remained substantially below baseline at one year, with large effect sizes.

Among those who entered remission immediately after treatment, the estimated probability of remaining in remission at 12 months was 84% for PTSD, 66% for depression, and 61% for anxiety "In practical terms, most of those whose symptoms had resolved right after treatment were still doing well a year later, with PTSD holding up best," Rolle explained.

Symptoms were rated with clinician-administered instruments rather than self-report alone--a meaningful strength in a small observational study. Durability matters here because these were not patients recovering from recent concussions. Participants averaged roughly 39 lifetime TBIs, predominantly mild, with about 15 years since the most severe injury; 21 had received formal mental health treatment and 19 had used psychotropic medication. These were chronically symptomatic people, not people in the acute window after an injury," Rolle emphasized. That chronicity makes spontaneous recovery a less compelling explanation for the change. It does not establish that ibogaine caused it.

Does This Mean Ibogaine Treats PTSD Or Depression Generally?

No--and this is perhaps the study's most important limitation. "Every one of the 30 participants had a history of traumatic brain injury, so strictly speaking the study cannot speak to people without one," Rolle said.

At baseline, 23 participants met criteria for PTSD, 15 for major depressive disorder, and 14 for an anxiety disorder; sensitivity analyses restricted to those subgroups preserved the effects.

Investigators also examined participants with mild TBI alone, and separately those who pursued no psychotherapy, psychiatric medication, or other psychedelics during follow-up. Those analyses increase confidence that the signal was not created by a few unusual participants, but they do not answer the more fundamental question. "What none of these analyses can do is separate a TBI-specific effect from a more general effect," Rolle explained, "because there is no comparison group without TBI and no control condition of any kind."

While the unrecognizeable people share during group therapy, the emotional support animal sits in the circle.
GETTY

MISTIC was open-label, observational, and naturalistic in design. It cannot establish that someone with PTSD after childhood abuse, assault, or a motor vehicle collision — without a formal diagnosis of a TBI — would respond similarly. The limitation is even stronger for treatment-resistant depression: treatment resistance was not an enrollment requirement, and having taken a psychiatric medication is not the same as having failed multiple adequate trials.

As Rolle emphasizes, "the findings have not been validated for PTSD or treatment-resistant depression in the absence of TBI."

Older observational work does suggest mood symptoms may improve after ibogaine in substance use disorder populations That is a biologically interesting hypothesis, not a substitute for trials in patients without TBI — and those case series remain vulnerable to selection effects, expectancy, variable dosing, and concurrent psychosocial care.

The Limitation Nobody Should Ignore

The largest interpretive problem is what happened after the initial treatment. Most participants used another psychedelic during the following year. Some underwent additional psychotherapy, and some participants received ibogaine again. This is therefore not a controlled experiment showing what one ibogaine exposure does for 12 months. It is a longitudinal observation of what happened to veterans whose year began with magnesium-ibogaine.

Psychiatrist Owen Muir, M.D.,DFAACAP, FCTMSS, Chief Scientific Officer and Co-Founder at Radial Health, and newly appointed Chief Medical Officer at Neurolief, who was not involved in the MISTIC study, puts it plainly: "Everyone in the study had a traumatic brain injury, so it tells us nothing about cases of PTSD or depression without a TBI." Nor, he argues, can the design isolate the drug from everything that followed it. "The study design does not allow us to isolate what ibogaine alone did versus the year of psychedelic retreats that followed. These are remarkable results, but we also have to move forward with the scientific method to understand which component of the healing was due to which intervention." Rolle agrees, calling the

subsequent psychedelic use “a genuine confound for any claim about durability and one we state plainly in the paper.”

It is reasonable to say patients who improved after magnesium-ibogaine frequently remained stable and with improvement a year later. It is not defensible to conclude that a single ibogaine exposure, by itself, produced a year of remission.

An Unusual Place In The Psychedelic Pipeline

Ibogaine also differs pharmacologically from psilocybin and other classic serotonergic psychedelics. Rolle describes ibogaine as an oneirogen: producing dream-like subjective effects and an unusually broad pharmacologic profile rather than predominant 5-HT2A agonism. It is metabolized to noribogaine, and both compounds interact with opioid, serotonergic, dopaminergic, sigma, and glutamatergic NMDA targets while differing in pharmacokinetics and electrophysiologic effects, which frustrates any attempt to credit one mechanism.

The Stanford group has now produced neurobiological observations from the same cohort. A multimodal imaging study reported gradual increases in regional cerebral blood flow across cortical, limbic, and striatal regions, with increases in the left insula and cingulate tracking improvement in TBI-related disability. A structural analysis found increased cortical thickness in 11 regions, subcortical volumetric expansion in eight, and predicted brain age reduced by roughly 1.3 years at one month.

These represent intriguing potential biomarkers of neuroplastic change. They remain observational and cannot establish that ibogaine repaired brain injury. Muir describes ibogaine as among the most promising and most risky compounds in the field; Rolle is more cautious about characterizing ibogaine’s potential. “I would not describe it as ahead of psilocybin or MDMA,” she said, noting other compounds have progressed much further through controlled and closely monitored development.

Enthusiasm Tempered With Restraint

The primary reason such enthusiasm requires restraint is safety. “The dominant concern is cardiac,” Rolle said. Ibogaine inhibits hERG potassium channels, prolongs

ventricular repolarization and the QT interval, and has been associated with torsades de pointes (a deadly cardiac arrhythmia); serious events and deaths appear throughout the literature. In one supervised detoxification study, half of participants exceeded a corrected QT interval of 500 ms, raising the risk of developing torsades de pointes.

MISTIC was built around that safety principle: medical screening, magnesium around treatment, staged dosing, continuous cardiac monitoring, and trained medical personnel on site. Treatment took place at Ambio Life Sciences in Mexico; Stanford's role was research and assessment, not administration.

The original study reported no serious or unexpected adverse events; participants developed transient cerebellar findings, and headache and nausea were common. That is not proof of safety. Patients with significant cardiac, hepatic, renal, neurologic, or psychotic disorders were excluded, and 30 selected participants are far too few to characterize rare but catastrophic complications. "These results describe ibogaine delivered with magnesium, screening, monitoring, and trained staff," Rolle emphasized. "Importantly, this is NOT evidence that it is safe to take casually, from an unvetted provider, or without a cardiac workup." That may be the most important take-away for patients reading about these results.

WASHINGTON, DC - APRIL 18: U.S. President Donald Trump speaks in the Oval Office as he signed an Executive Order April 18, 2026 in Washington, DC. The executive order directs the Food and Drug Administration to issue new gu ... More

GETTY IMAGES

Why The Federal Government And States Are Paying Attention

Ibogaine remains a Schedule I controlled substance and is not FDA approved. But the landscape is moving faster than it was in 2024. An April 18, 2026, executive order directed HHS to accelerate access to treatments for serious mental illness, and days later the FDA announced supporting actions for serotonin-2A agonists, including national priority vouchers for three psychedelic programs.

The agency also allowed an early-phase study of noribogaine hydrochloride — ibogaine's principal metabolite — to proceed under an Investigational New Drug application, the first U.S. study of an ibogaine derivative it has permitted; the sponsor, DemeRx NB, is investigating it for alcohol use disorder (AUD). The FDA cautioned that allowing a study to proceed does not mean a drug has been approved or found safe or effective, and the action applies to noribogaine, not ibogaine.

States have moved forward too. Texas Senate Bill 2308, signed in June 2025, committed $50 million in matching funds toward FDA-directed ibogaine trials, with the award going to a UTHealth Houston–led consortium ; more than a dozen other states have introduced or funded related measures. None of that lowers the evidentiary standard, and Rolle declines to predict when — or whether — ibogaine will reach approval. “The honest answer is that no one knows,” she said.

A Clinician’s Perspective

“In 2015, I treated two patients who had traveled to Mexico for ibogaine treatment for PTSD associated with opioid addiction, said Dr. Glen Brooks, anesthesiologist, expert faculty, ASKP3, and the founder of New York Ketamine, based in New York City. “Both described an intense five-day experience involving hallucinations and depersonalization that they found unpleasant. They reported some benefit, but later came to my clinic seeking ketamine treatment for persistent PTSD symptoms and felt they experienced greater improvement.”

“Ketamine research has taught us that metabolites may contribute importantly to the biological effects of the parent compound. Hydroxynorketamine, or HNK, has generated particular interest because of its potential role in AMPA signaling and neuroplasticity. Whether a similar relationship exists between ibogaine and its active metabolite, noribogaine, remains an important research question," offered Brooks.

“But enthusiasm has to be balanced by patient safety. Ibogaine--and potentially noribogaine--can affect cardiac repolarization and has been associated with QT prolongation, dangerous ventricular arrhythmias and reported deaths. Any future therapeutic development needs rigorous clinical trials, careful patient selection and appropriate cardiac monitoring. The goal should not simply be to find what works, but to determine whether we can preserve therapeutic benefit while reducing toxicity," he added.

What Subsequent Research Still Needs To Demonstrate

A rigorous trial or set of trials could randomize participants to a control group, prospectively define concomitant psychotherapy and later psychedelic exposure, characterize cardiac and other adverse events systematically, and enroll enough participants to generate meaningful safety data. It would also separate ibogaine from the environment in which it is delivered — preparation, therapeutic support, expectancy, dosing, monitoring, integration — and treat psychotherapy or psychedelic use during follow-up as data, not background.

Trials enrolling both TBI and non-TBI populations could establish whether the benefit is injury-specific or broader; treatment-resistant depression requires its own studies. So does diversity. This cohort was entirely male Special Operations veterans, and earlier reports of psychedelic benefit in that same population underscore how much of the signal may be population-specific.

Muir argues the preliminary evidence creates an obligation: “If we think we have an approach that can get veterans with TBI and PTSD well, which we now have every reason to suspect, we have to study it with more rigor.”

That is where the MISTIC findings should be positioned. The paper does not show that ibogaine cures PTSD, that one dose produces a year of recovery, that it works without TBI, or that it is safe outside intensive screening and cardiac monitoring.

What it does show is hard to dismiss: chronically symptomatic veterans with extraordinary cumulative TBI exposure improved substantially after magnesium-ibogaine treatment. Much of that improvement was still evident a year later, the effect survived several sensitivity analyses, and parallel imaging has identified biological changes worth investigating. The cohort was also small, entirely male, self-selected, unblinded, treated outside the U.S., and heavily exposed to additional psychedelics during follow-up.

Both realities can coexist. Ibogaine may prove to be an important neuropsychiatric treatment, potentially useful only in narrower populations, or may be emblematic of previously published data that belongs to the therapeutic ecosystem that followed it.

Only controlled trials can tell those apart—which is also the most meaningful continuation, and a tribute to Dr. Nolan Williams's work: running the key trials capable of answering the missing questions.

Find Robert Glatter, MD on LinkedIn and X. Visit Robert's website.