Ibogaine and the Antidepressant-Damaged Brain

How a Single Treatment May Restore What SSRIs Took Away

A Guide for Those Still Waiting to Wake Up

Introduction: The Problem No One Warned You About

The constellation of symptoms that persists after antidepressant discontinuation goes by several names. Post-SSRI Sexual Dysfunction (PSSD) describes the genital numbness, loss of libido, and pleasure-less orgasms that can persist indefinitely. Anhedonia, the inability to experience pleasure, extends far beyond sexual function into every domain of life. Emotional blunting, cognitive fog, depersonalization, and a pervasive sense of being disconnected from your own identity round out the picture. The European Medicines Agency formally recognized PSSD as a legitimate medical condition, and researchers have proposed that the underlying mechanisms involve persistent changes in serotonin receptor sensitivity, dopamine suppression, and possibly epigenetic alterations in gene expression.

The purpose of this document is to explain, in plain language backed by published research, how ibogaine may offer a path toward recovery for people trapped in this neurochemical prison. Ibogaine is not a magic bullet, and it carries real risks that must be carefully managed under medical supervision. But its unique pharmacological profile acts on the exact systems that antidepressants damage, in ways that no other known compound can replicate.

What Antidepressants Actually Do to the Brain

To understand why ibogaine may help, you first need to understand what went wrong. The damage antidepressants cause is not random. It follows a predictable neurochemical pattern that explains virtually every symptom you are experiencing.

Serotonin Receptor Downregulation

SSRIs work by blocking the serotonin transporter (SERT), which prevents serotonin from being recycled back into the neuron after it is released. This artificially increases serotonin levels in the synaptic cleft. The brain, always seeking equilibrium, responds by reducing the number and sensitivity of serotonin receptors. This is called downregulation. The 5-HT1A autoreceptors, which act as the brain's thermostat for serotonin release, become desensitized. Research in rats has demonstrated that this desensitization of 5-HT1A receptors can persist long after SSRI discontinuation, potentially accounting for the emotional blunting and anhedonia that characterize the post-SSRI state.

Dopamine Suppression

Serotonin and dopamine exist in a reciprocal relationship. When serotonin levels are chronically elevated, dopamine output is suppressed. This is not a side effect of SSRIs; it is a direct pharmacological consequence. Dopamine is the neurotransmitter responsible for motivation, reward, pleasure, sexual arousal, and the felt sense that life is worth living. When dopamine signaling in the mesolimbic reward pathway (the circuit connecting the Ventral Tegmental Area, or VTA, to the Nucleus Accumbens and Prefrontal Cortex) is chronically suppressed, you lose the capacity to feel pleasure from activities that once brought joy. This is the neurological basis of anhedonia. The dopamine neurons themselves may not be dead, but they are functionally offline, trapped in a state of reduced firing and diminished receptor sensitivity.

Neurotrophic Factor Depletion

The brain maintains and repairs itself through proteins called neurotrophic factors. The most important of these include BDNF (Brain-Derived Neurotrophic Factor), GDNF (Glial Cell Line-Derived Neurotrophic Factor), and NGF (Nerve Growth Factor). Chronic SSRI exposure and the neurochemical chaos of withdrawal can dysregulate the production of these proteins. Without adequate neurotrophic support, damaged neural circuits cannot repair themselves, new connections cannot form, and the brain essentially becomes stuck. It is like trying to rebuild a house without lumber, nails, or workers. The blueprint for recovery may exist, but the raw materials are missing.

Potential Demyelination and White Matter Changes

Emerging research is beginning to explore whether chronic serotonergic disruption may also affect the myelin sheath, the insulation coating around nerve fibers that allows electrical signals to travel efficiently. Disrupted myelination can slow neural communication, contributing to the cognitive fog, slow processing speed, and general sense of mental heaviness that many post-SSRI sufferers describe. While this area of research is still developing, it provides another piece of the puzzle for why recovery can be so slow and incomplete.

What Is Ibogaine?

Ibogaine is a naturally occurring psychoactive alkaloid found in the root bark of the Tabernanthe iboga plant, which grows in the rainforests of Central West Africa. It has been used for centuries in the spiritual traditions of the Bwiti people of Gabon as a sacramental medicine for initiation rituals and healing ceremonies.

In the Western world, ibogaine first attracted scientific attention in the 1960s when a young heroin addict named Howard Lotsof accidentally discovered that a single dose eliminated his withdrawal symptoms and cravings. Since then, thousands of people have used ibogaine to interrupt addiction to opioids, alcohol, stimulants, and other substances. Clinical research, while still in earlier stages due to ibogaine's legal status in many countries, has increasingly validated what anecdotal reports have long suggested: ibogaine does something to the brain that no other compound does.

When ingested, ibogaine is metabolized in the liver by the enzyme CYP2D6 into its primary active metabolite, noribogaine (also called 12-hydroxyibogamine). While ibogaine itself has a half-life of roughly 4 to 8 hours, noribogaine persists much longer, with a documented half-life of 28 to 49 hours. This is critical because many of ibogaine's most important therapeutic effects, including the sustained neuroplasticity window, the GDNF autoregulatory loop, and the extended modulation of serotonin and dopamine transporters, are driven primarily by noribogaine.

What makes ibogaine unique among all known psychoactive substances is the sheer breadth of its receptor pharmacology. It does not act on one system. It acts on nearly every system that antidepressants damage, simultaneously and in a complementary fashion. This is not a shotgun approach; it is a precisely coordinated multi-system intervention that addresses the root causes of post-SSRI neurological damage at their source.

How Ibogaine Works: The Multi-Receptor Reset

Understanding ibogaine's potential for post-antidepressant recovery requires walking through each of the receptor systems it engages. What follows is a system-by-system explanation of how ibogaine addresses the specific damage that SSRIs and SNRIs leave behind.

Serotonin System: A Different Kind of Reuptake Inhibition

Ibogaine and noribogaine are both potent serotonin reuptake inhibitors, but they interact with the serotonin transporter (SERT) in a fundamentally different way than SSRIs. While SSRIs stabilize SERT in its outward-facing conformation (blocking reuptake from outside the cell), ibogaine and noribogaine stabilize the inward-open conformation of SERT. This distinction is not trivial. The inward-open conformation is associated with serotonin efflux, meaning ibogaine may actually cause serotonin to be released through the transporter rather than simply blocking its reuptake. This mechanism more closely resembles what MDMA does to SERT than what Prozac does. The therapeutic significance is that ibogaine's serotonergic action may help recalibrate desensitized serotonin receptors through a fundamentally different mechanism than the one that caused the damage in the first place.

Dopamine System: Restarting the Reward Circuit

Ibogaine has direct affinity for the dopamine transporter (DAT) and produces biphasic effects on dopamine levels. This means it can modulate dopamine signaling in both directions depending on the state of the system. For someone whose dopamine circuits have been chronically suppressed by serotonin excess, this bidirectional modulation may help nudge the system back toward its natural set point.

But ibogaine's most powerful effect on dopamine is indirect, operating through the upregulation of GDNF in the Ventral Tegmental Area (VTA). More on this in the next section, but the key point is that ibogaine does not merely boost dopamine levels temporarily the way a stimulant would. It supports the actual survival and repair of the dopamine neurons themselves, addressing the root cause of anhedonia rather than masking it.

NMDA Receptors: Quieting the Noise

Ibogaine is a non-competitive antagonist of NMDA glutamate receptors. This is the same mechanism of action that makes ketamine an effective rapid-acting antidepressant. NMDA antagonism reduces excitotoxicity (the process by which overactive glutamate signaling damages neurons) and opens a window of neuroplasticity. In a brain that has been destabilized by SSRI withdrawal, excitotoxicity may be contributing to ongoing neural damage. By calming this overactivity, ibogaine creates a more favorable environment for repair and reorganization to take place.

Kappa Opioid Receptors: The Anti-Dysphoria Switch

Ibogaine and noribogaine act as partial agonists at kappa opioid receptors. The kappa opioid system is deeply involved in mood regulation, and its dysregulation has been implicated in dysphoria, anhedonia, and the subjective sense of emotional suffering that goes beyond simple sadness. Full kappa agonists tend to produce dysphoria, but partial agonists like noribogaine can modulate the system without triggering that negative emotional response. The kappa opioid receptor also plays a critical role in remyelination. It regulates the differentiation of oligodendrocyte precursor cells (OPCs) into mature oligodendrocytes, the cells responsible for producing myelin. This means ibogaine's kappa opioid activity may directly support the repair of damaged white matter.

Mu Opioid Receptors: Resensitizing the Pleasure System

Ibogaine acts as a weak antagonist or partial agonist at mu opioid receptors. The mu opioid system is central to the experience of physical pleasure and emotional warmth. Chronic SSRI use can alter opioid receptor sensitivity, contributing to the emotional numbness and physical anhedonia (including loss of orgasmic pleasure) that characterize PSSD. By gently modulating mu opioid receptors, ibogaine may help resensitize a pleasure system that has been pharmacologically dulled.

Sigma Receptors: Neuroprotection and Repair

Ibogaine has affinity for both sigma-1 and sigma-2 receptors. Sigma receptors are involved in neuroprotection, modulation of inflammatory responses, and cellular repair processes. Sigma-2 receptor activation has been linked to oligodendrogenesis and white matter recovery. In the context of post-antidepressant damage, sigma receptor modulation may contribute to reducing neuroinflammation and supporting the structural repair of damaged neural tissue.

Nicotinic Acetylcholine Receptors

Ibogaine is a non-competitive antagonist at nicotinic acetylcholine receptors, particularly the alpha-3-beta-4 subtype. While this receptor's role in post-SSRI recovery is less directly established, the cholinergic system interacts extensively with both dopamine and serotonin circuits. Modulation of nicotinic receptors may contribute to the overall rebalancing of neurotransmitter systems that ibogaine facilitates.

The GDNF Effect: Ibogaine's Most Important Mechanism

If you take only one thing away from this document, let it be this: ibogaine is one of the only known substances that can orally stimulate the production of Glial Cell Line-Derived Neurotrophic Factor (GDNF) in deep brain regions, specifically in the Ventral Tegmental Area (VTA), the origin point of the dopamine reward pathway.

GDNF is a survival factor for dopaminergic neurons. It binds to the GFR-alpha-1/RET receptor complex and activates signaling cascades that promote neuron survival, dendritic growth, and synaptic remodeling. In plain language, GDNF is the protein that keeps dopamine neurons alive, healthy, and capable of forming new connections. It is the construction crew that rebuilds the reward circuit.

Research published in Frontiers in Pharmacology (Marton et al., 2019) demonstrated that a single administration of ibogaine at 40 mg/kg simultaneously increased the expression of three neurotrophic factors, GDNF, BDNF, and NGF, across multiple brain regions involved in dopamine signaling, including the VTA, Nucleus Accumbens, Prefrontal Cortex, and Substantia Nigra. The increase in GDNF protein levels was selective to the VTA at the 40 mg/kg dose, which corresponds to the effective dose found in previous behavioral studies. This selectivity is significant because the VTA is the exact brain region where dopamine neurons that drive motivation, pleasure, and emotional connection originate.

Perhaps even more remarkable is what happens after the initial GDNF increase. Research by He and Ron (2006) demonstrated that ibogaine triggers a self-sustaining autoregulatory loop in which GDNF positively regulates its own expression. Once ibogaine triggers the initial increase in GDNF, the protein continues to promote its own production long after ibogaine and noribogaine have been cleared from the body. This autocrine feedback loop provides a mechanistic explanation for why a single ibogaine treatment can produce effects that last weeks, months, or even permanently. The GDNF loop essentially restarts the brain's own repair machinery, and once that machinery is running, it can sustain itself.

For someone whose dopamine system has been suppressed by years of SSRI exposure, this GDNF effect is the closest thing to a neurological reset that currently exists. It does not just temporarily increase dopamine levels. It supports the repair and regrowth of the very neurons that produce dopamine, potentially restoring the brain's capacity for pleasure, motivation, and emotional connection from the ground up.

Neuroplasticity and Structural Brain Repair

Dendritic Spine Growth: Rebuilding Neural Connections

Noribogaine has been classified as a psychoplastogen, a compound that can rapidly promote measurable changes in brain structure within 24 to 72 hours of a single administration. Research published in Cell Reports (Ly et al., 2018) demonstrated that noribogaine robustly increases dendritic spine density in cortical neurons, with potency comparable to ketamine. Dendritic spines are the tiny protrusions on neurons where synapses form. They are the physical connection points between brain cells. When spines are lost, circuits go silent. When spines grow, circuits come back online.

Depression, chronic stress, and chronic SSRI exposure are all associated with spine loss in the prefrontal cortex. The ability of noribogaine to promote rapid spine growth means it can physically rebuild connections that have been lost or weakened. This is not metaphorical. Fluorescence microscopy studies show actual structural changes in neurons within 24 hours of treatment. The clinical significance is that this spine growth may underlie the rapid improvements in cognitive clarity, emotional range, and sense of self that some people report after ibogaine treatment.

It is also worth noting that this psychoplastogenic effect appears to be mediated through the 5-HT2A receptor and the TrkB/mTOR signaling pathway, the same pathway through which ketamine and classical psychedelics promote plasticity. However, ibogaine's simultaneous upregulation of GDNF, BDNF, and NGF provides a broader foundation of neurotrophic support that classical psychedelics and ketamine do not offer to the same degree.

Remyelination: Restoring Signal Speed

One of the most exciting areas of ibogaine research involves its potential to promote remyelination, the repair of the myelin sheath that insulates nerve fibers. Research published in Frontiers in Neuroscience (Govender et al., 2024) demonstrated that ibogaine upregulates two key myelination markers, CNPase and Myelin Basic Protein (MBP), at both the gene and protein levels. These effects were observed 72 hours after administration, suggesting they are driven primarily by noribogaine through its kappa opioid receptor activity.

The kappa opioid receptor is an important regulator of oligodendrocyte differentiation. Oligodendrocytes are the cells that produce myelin in the central nervous system. By activating kappa opioid receptors on oligodendrocyte precursor cells, noribogaine may stimulate these cells to mature and begin producing new myelin. A 2025 case report published in Frontiers in Immunology documented dramatic lesion shrinkage and decreased ADC values (suggesting remyelination and reduced inflammation) in a multiple sclerosis patient following ibogaine treatment, providing early clinical evidence that these preclinical findings translate to human neuroimaging outcomes.

For post-SSRI sufferers, the implications are significant. If chronic serotonergic disruption has contributed to subtle white matter changes (and the cognitive fog, slow processing, and general mental heaviness would be consistent with this), then ibogaine's remyelination potential offers a mechanism for restoration that no conventional treatment currently provides.

The Plasticity Window: Why Timing Matters

Ibogaine does not permanently rewire the brain in a single session. What it does is open a transient window of enhanced neuroplasticity, a period during which the brain is unusually receptive to forming new connections and strengthening existing ones. GDNF upregulation creates this window. Dendritic spine growth builds new structural scaffolding within it. And the visionary or oneirogenic experience that accompanies ibogaine's psychoactive phase may facilitate emotional processing and cognitive updating that takes advantage of this heightened plasticity.

This is why integration after ibogaine treatment is so critical. The window opens, but you have to walk through it. Healthy behaviors, emotional processing, physical exercise, social connection, and continued self-care during the weeks and months following treatment can help consolidate the neural changes that ibogaine initiates. Ibogaine provides the raw materials and the construction window. What you build during that window determines the long-term outcome.

Noribogaine: The Silent Workhorse

Much of what makes ibogaine therapeutically valuable is actually driven by noribogaine, its primary metabolite. When you ingest ibogaine, the liver enzyme CYP2D6 converts it into noribogaine, which then circulates in the body for days. This extended presence is critical because many of ibogaine's most important effects, including SERT modulation, kappa opioid receptor activation, dendritic spine growth, and GDNF sustenance, are either initiated or maintained by noribogaine.

Noribogaine is a potent serotonin reuptake inhibitor, but as discussed earlier, it interacts with SERT in a fundamentally different way than SSRIs. It is a moderate kappa opioid receptor agonist with higher affinity than ibogaine itself, meaning the kappa-mediated effects on mood regulation and remyelination intensify as ibogaine is metabolized. It is classified as a psychoplastogen capable of increasing dendritic arbor complexity in cortical neurons. And its extended half-life (28 to 49 hours in CYP2D6 extensive metabolizers) means that the neuroplasticity window remains open for days after a single treatment.

Think of the relationship between ibogaine and noribogaine as a two-stage rocket. Ibogaine is the booster that initiates the launch: it drives the acute psychoactive experience, triggers the initial GDNF cascade, and engages the full spectrum of receptor targets. Noribogaine is the second stage that sustains the mission: it maintains serotonin transporter modulation, extends the neuroplasticity window, supports ongoing neurotrophic factor production, and provides continued kappa opioid receptor stimulation for remyelination. The combination of an acute, powerful initiation followed by a sustained, gentler continuation is what gives ibogaine its uniquely durable therapeutic profile.

Why Ibogaine May Be Uniquely Suited for Post-SSRI Recovery

No formal clinical trials have yet studied ibogaine specifically for PSSD or post-antidepressant anhedonia. It is important to be transparent about that. The case for ibogaine in this context is built on a convergence of preclinical research, mechanistic reasoning, anecdotal reports, and the documented failure of conventional approaches to address these conditions.

Here is why the mechanistic case is compelling. The core pathology of post-SSRI damage involves at least four overlapping problems: desensitized serotonin receptors, suppressed dopamine signaling, depleted neurotrophic factor production, and potentially compromised white matter integrity. There is no other known compound that addresses all four of these problems simultaneously. Ketamine addresses NMDA antagonism and neuroplasticity but does not upregulate GDNF or modulate opioid receptors. Classical psychedelics like psilocybin promote neuroplasticity through 5-HT2A agonism but do not engage the dopamine system directly or support remyelination. Bupropion modestly increases dopamine but does not promote structural repair. Vortioxetine has multimodal serotonin activity but lacks the neurotrophic factor upregulation and the opioid receptor modulation.

Ibogaine is the only compound that simultaneously recalibrates the serotonin transporter through a non-SSRI mechanism, supports dopamine neuron survival through GDNF upregulation in the VTA, promotes dendritic spine growth and synaptic plasticity, reduces excitotoxicity through NMDA antagonism, supports remyelination through kappa opioid receptor activation, modulates the mu opioid system to potentially resensitize the pleasure response, and reduces neuroinflammation through sigma receptor activity. This convergence of mechanisms maps directly onto the convergence of damage that SSRIs leave behind.

The analogy that best captures ibogaine's approach is this: imagine a city that has suffered years of infrastructure neglect. The roads are crumbling (myelin damage). The power grid is unreliable (dopamine suppression). The water system is contaminated (serotonin receptor dysfunction). And the construction workers have all gone home (neurotrophic factor depletion). A conventional treatment might fix one of these problems. Ibogaine sends in the construction workers (GDNF, BDNF, NGF), restores the power grid (dopamine recalibration), cleans the water supply (SERT recalibration), and repairs the roads (remyelination), all in one coordinated intervention.

Critical Safety Considerations

Ibogaine is not without serious risks, and anyone considering it must understand these risks fully before proceeding.

Cardiac Risk

The most significant risk of ibogaine is its effect on heart rhythm. Ibogaine and noribogaine can prolong the QTc interval, which in rare cases can lead to fatal cardiac arrhythmias. This risk is compounded by pre-existing cardiac conditions, electrolyte imbalances, and interactions with other medications. Any ibogaine treatment must be conducted under continuous cardiac monitoring by qualified medical professionals. A thorough cardiac workup including an EKG and blood panel should be completed before treatment. Individuals with pre-existing QTc prolongation, heart disease, or those taking medications that also prolong QTc should not take ibogaine.

Drug Interactions

Ibogaine has significant interactions with many medications. SSRIs and SNRIs should be fully discontinued and cleared from the system before ibogaine treatment, as the combination of ibogaine's serotonergic effects with residual SSRI activity could theoretically increase serotonin syndrome risk and compound cardiac effects. The appropriate washout period depends on the specific SSRI and its half-life. Fluoxetine (Prozac), with its exceptionally long half-life, may require a washout period of several weeks to months. Other medications that affect CYP2D6 metabolism, prolong QTc, or affect serotonin, dopamine, or opioid systems must also be carefully evaluated. Lithium, which some post-SSRI sufferers explore for neuroplasticity support, has potential cardiac interactions with ibogaine that must be carefully assessed by a qualified medical provider.

The Psychoactive Experience

Ibogaine produces a prolonged psychoactive experience lasting 24 to 36 hours that is qualitatively different from classical psychedelics. The experience is often described as oneirogenic (dream-like), involving vivid autobiographical visions, life review, and confrontation with emotionally significant memories. This can be psychologically intense and is not appropriate for individuals with active psychosis, schizophrenia spectrum disorders, or poorly controlled bipolar disorder. The experience, while often described as therapeutically valuable, requires appropriate psychological preparation and integration support.

Legal Status

Ibogaine is classified as a Schedule I controlled substance in the United States and is illegal in several other countries. It is legal or unregulated in many countries including Mexico, Canada, Brazil, New Zealand, South Africa, and several European nations. Treatment is most commonly sought at specialized medical clinics in Mexico, where cardiac monitoring, medical screening, and professional supervision are available. The legal landscape is evolving, and several jurisdictions are considering regulatory frameworks for ibogaine treatment.

Conclusion: The Case for Hope

If you are reading this document, you are likely someone who has been suffering from the aftereffects of antidepressant use and has been told, implicitly or explicitly, that there is nothing that can be done. That the emotional numbness, the anhedonia, the sexual dysfunction, the cognitive fog, are just something you have to live with. That message is not only unhelpful; based on the emerging science, it may not be true.

The research on ibogaine is still developing, and it would be irresponsible to present it as a guaranteed cure for post-SSRI damage. But the mechanistic evidence is substantial and specific. Ibogaine and noribogaine engage the exact receptor systems that SSRIs disrupt. They upregulate the exact neurotrophic factors that the damaged brain needs to repair itself. They promote the exact structural changes, dendritic spine growth, synaptic remodeling, and potential remyelination, that could reverse the neurological consequences of chronic serotonergic disruption. No other known compound combines all of these mechanisms in a single treatment.

The GDNF autoregulatory loop, in particular, represents something unprecedented in psychopharmacology: a mechanism by which a single treatment can restart the brain's own repair processes in a self-sustaining way. This is not masking symptoms. This is not compensating for damage with another drug. This is providing the brain with the biological tools it needs to heal itself.

Recovery is possible. The brain is not a static organ. It is a dynamic, adaptable system that retains the capacity for change throughout life. The dopamine neurons that have gone quiet are not necessarily dead. The serotonin receptors that have been downregulated can potentially be recalibrated. The neural connections that have been lost can potentially be rebuilt. What has been missing is the catalyst, the intervention powerful enough and broad enough to restart all of these processes simultaneously. Ibogaine may be that catalyst.

If you are considering ibogaine treatment, do your research thoroughly. Seek out reputable medical providers who offer comprehensive cardiac screening, continuous monitoring, and integration support. Consult with physicians who understand both the pharmacology of ibogaine and the specific neurochemical damage that antidepressants can cause. And approach the process with both hope and realism: ibogaine opens a door, but walking through it and doing the work of rebuilding your life on the other side is up to you.

Disclaimer: This document is for informational and educational purposes only. It is not medical advice and should not be used as a substitute for consultation with a qualified healthcare provider. Ibogaine carries serious medical risks including cardiac arrhythmia and death. Any use of ibogaine should be conducted under direct medical supervision with appropriate cardiac monitoring and screening. The author is not a physician.

Key Research References

Marton S, et al. (2019). Ibogaine Administration Modifies GDNF and BDNF Expression in Brain Regions Involved in Mesocorticolimbic and Nigral Dopaminergic Circuits. Frontiers in Pharmacology, 10:193. PMC6411846

He DY, Ron D. (2006). Autoregulation of Glial Cell Line-Derived Neurotrophic Factor Expression: Implications for the Long-Lasting Actions of the Anti-Addiction Drug Ibogaine. FASEB Journal, 20(13):2420-2422. PMID: 17023388

He DY, et al. (2005). Glial Cell Line-Derived Neurotrophic Factor Mediates the Desirable Actions of the Anti-Addiction Drug Ibogaine against Alcohol Consumption. Journal of Neuroscience, 25(3):619-628.

Ly C, et al. (2018). Psychedelics Promote Structural and Functional Neural Plasticity. Cell Reports, 23(11):3170-3182. PMC6082376

Govender D, et al. (2024). Ibogaine Administration Following Repeated Morphine Administration Upregulates Myelination Markers CNP and MBP mRNA and Protein Expression. Frontiers in Neuroscience, 18:1378841. PMC11303312

Calvey T, et al. (2026). Neurorestorative Properties of Ibogaine: Linking Multi-Receptor Affinities to Remyelination and Metabolic Restoration. Acta Neuropsychiatrica, 38:e26.

Chen L, et al. (2025). Case Report: Significant Lesion Reduction and Neural Structural Changes Following Ibogaine Treatments for Multiple Sclerosis. Frontiers in Immunology. PMC11839422

Carnicella S, Ron D. (2009). GDNF: A Potential Target to Treat Addiction. Pharmacology & Therapeutics, 122(1):9-18.

Glick SD, et al. (1997). Evidence for Roles of Kappa-Opioid and NMDA Receptors in the Mechanism of Action of Ibogaine. Brain Research, 749(2):340-343. PMID: 9138735

Peleg LC, et al. (2022). Post-SSRI Sexual Dysfunction (PSSD): Biological Plausibility, Symptoms, Diagnosis, and Presumed Risk Factors. Sexual Medicine Reviews, 10:91-98.

Frontiers in Pharmacology (2025). Ibogaine's Potential Role in Supporting Reward System Recovery Across Diagnostic Boundaries.