THRIVING MIND, PLLC

A PATIENT GUIDE

What I Wish Everyone Knew

before starting an antidepressant

The Informed Consent Conversation Most Patients Never Receive.

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—DISCLAIMER

This guide is for educational purposes only and does not constitute medical advice. All decisions regarding psychiatric medications should be made in consultation with a qualified healthcare professional. Do not stop or change any medication without speaking to your prescriber.

A NOTE BEFORE WE BEGIN

The informed consent conversation I give every new patient.

One of the most common questions I receive is: “How do I find someone who practices like you in my area?” The honest answer is that the kind of care I offer — genuinely informed, evidence-based, patient-centered psychiatric practice — remains rare. That is heartbreaking, and it is exactly why I created this guide.

The most important thing I can do as a psychiatric nurse practitioner is to teach my clients how to advocate for themselves. This guide attempts to constitute the informed consent conversation I give to each of my clients.

I often tell them: if you ever see anyone else, this is what you take into that appointment. I am sharing this guide, completely free, because every patient deserves this information — not just the clients who find me.

WHAT'S INSIDE

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CHAPTER / ONE

Your Brain Is Not Broken

The story you've been told about a "chemical imbalance" is not supported by the evidence.

The “Chemical Imbalance” Theory

If you are experiencing depression, anxiety, PTSD, OCD, or another mental health condition, there is something critical I want you to understand before you start any treatment.

The idea that your symptoms are caused by a “chemical imbalance” — specifically, too little serotonin in your brain — is not supported by the current scientific evidence. Despite being widely promoted for decades and remaining familiar to most patients, modern research has consistently failed to substantiate this theory. Concerningly, this theory is still taught to many patients when being placed on psychiatric medications. I have spent a considerable amount of my career re-educating patients, clinicians, and students on the lack of evidence for this theory.

—WHAT THE RESEARCH ACTUALLY SHOWS

In 2023, Moncrieff and colleagues published the most comprehensive review ever conducted on the serotonin hypothesis of depression in Molecular Psychiatry. Their conclusion:

“The main areas of serotonin research provide no consistent evidence of there being an association between serotonin and depression, and no support for the hypothesis that depression is caused by lowered serotonin activity or concentrations.”

— Moncrieff et al., Molecular Psychiatry, 2023

This does not mean that biology plays no role in mental health. It does. But the simplistic “broken brain” explanation has been used to justify over-reliance on medications while systematically underinvesting in the factors that actually shape mental health.

What Actually Influences Mental Health

Mental health symptoms arise from a complex interaction of factors, including:

PSYCHOLOGICAL & SOCIAL

- Trauma and adverse life experiences
- Relationship quality and attachment patterns
- Chronic stress and burnout
- Social connection and isolation
- Meaning, purpose, and identity
- Work and financial stressors

BIOLOGICAL & LIFESTYLE

- Sleep quality and quantity
- Nutrition and gut health
- Exercise and physical activity
- Genetics and family history
- Physical health conditions
- Nervous system dysregulation

> **IN PLAIN ENGLISH**
>
> *Your depression may be caused by unprocessed grief, a burnout-inducing job, or an emotionally abusive relationship. If we treat any of those as a chemical malfunction in your brain, we risk ignoring the very things that need to change for you to actually heal.*

Moncrieff, J., Cooper, R. E., Stockmann, T., Amendola, S., Hengartner, M. P., & Horowitz, M. A. (2023). The serotonin theory of depression: A systematic umbrella review of the evidence. Molecular Psychiatry, 28, 3243–3256. https://doi.org/10.1038/s41380-022-01661-0

CHAPTER / TWO

What Antidepressants Actually Do — And Don't Do

They are real tools with real effects. But reducing symptoms is not the same as resolving what caused them.

Antidepressants Don't Cure Anything. They Treat Symptoms.

Antidepressants — including SSRIs, SNRIs, NDRIs, and novel agents — do not cure depression, anxiety, OCD, or any other mental health condition. They treat symptoms. For many people, that distinction matters enormously.

Think of it this way: Tylenol can lower a fever and make you feel better. But the fever isn't what's actually wrong. A fever can be caused by an infection, cancer, an autoimmune condition, or other disease — knowing what is contributing to it is paramount to healing the underlying condition. If you never treat what's causing it, you are not getting better — you are just feeling better.

Antidepressants can work similarly. When someone feels more functional on medication, there is a real risk that the underlying stressors, traumas, or relationship problems that generated those symptoms go unaddressed indefinitely.

For some people, this tradeoff is worth it. Short-term symptom relief can create enough stability to engage in therapy, improve lifestyle habits, address underlying causes, and build a path toward genuine recovery. That is a legitimate and sometimes necessary use of medication.

Others, who believe there is something chemically wrong in their brain, may assume that their biology is fundamentally broken, and miss these signals.

For some, medication creates a method of tolerating an intolerable situation rather than changing it. This can have impacts on long term growth and meaningful change. Someone may never leave the job destroying their mental health because an SSRI makes it manageable. Someone may suppress anxiety that was trying to signal something important about a harmful relationship. This is not meant to be an argument against all use of medication. But rather an important discussion into how its use is applied.

> ***Symptom management is not the same as healing, and it should never replace it.***

Antidepressant Research Has Serious Problems

One of the most important things patients are rarely told: antidepressant research has well-documented limitations that significantly affect how we should interpret the evidence for these medications.

Problem 1: Publication Bias

In a landmark 2008 study published in the New England Journal of Medicine, Turner and colleagues compared all antidepressant trial data submitted to the FDA against what was actually published in the medical literature.

—WHAT THEY FOUND

Bottom line: the version of the evidence that doctors read was substantially more favorable than the complete data the FDA reviewed.

- Positive studies were almost universally published.
- Negative and mixed studies were frequently not published — or were reframed to appear positive.
- Effect sizes increased by 11–69% in the published literature compared to the complete FDA data.

Turner, E. H., Matthews, A. M., Linardatos, E., Tell, R. A., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. New England Journal of Medicine, 358(3), 252–260. https://doi.org/10.1056/NEJMsa065779

Problem 2: The Placebo Effect Is Enormous

When researchers look at the actual FDA trial data, they find something striking: patients receiving placebos improve

substantially too. The gap between medication and placebo is often far smaller than most patients have been led to believe.

—KEY FINDING

An influential analysis of FDA antidepressant trial data by Kirsch and colleagues (2008) found that the difference between drug and placebo fell **below the threshold for clinical significance** except in patients with very severe depression. Summarizing this dataset, Kirsch has estimated that roughly **82% of the drug response was duplicated by placebo**.

IN PLAIN ENGLISH

If much of the improvement seen in antidepressant trials also occurs with placebo, then the medication may not be doing as much of the work as patients are often led to believe. Hope, time, support, expectation, clinical attention, and the body's own healing capacity all matter.

And it raises the real informed-consent question: how much of the improvement is coming from the pill itself?

Kirsch, I., Deacon, B. J., Huedo-Medina, T. B., Scoboria, A., Moore, T. J., & Johnson, B. T. (2008). Initial severity and antidepressant benefits: A meta-analysis of data submitted to the Food and Drug Administration. PLOS Medicine, 5(2), e45. https://doi.org/10.1371/journal.pmed.0050045

Problem 3: Short-Term Trials, Long-Term Prescriptions

Most antidepressants were approved based on clinical trials lasting approximately 6–8 weeks. Yes, you read that correctly. The median trial length submitted to the FDA for antidepressant approval is around 8 weeks (Ward et al., 2025). Yet in real-world practice, patients routinely remain on these medications for years — sometimes decades.

Ward, W., Haslam, A., & Prasad, V. (2025). *Antidepressant trial duration versus duration of real-world use: A systematic analysis*. **The American Journal of Medicine, 138**(10), 1400–1407.e10. https://doi.org/10.1016/j.amjmed.2025.04.037

—THE EVIDENCE GAP

- The evidence base for antidepressant safety and effectiveness was largely built on 8-week trials.
- Long-term efficacy data is sparse or nonexistent for most antidepressants.
- The limited long-term data that does exist primarily examines relapse rates when medication is stopped — not whether continuous use leads to better outcomes.

IN PLAIN ENGLISH

We approved these medications based on 8 weeks of data. Patients are often prescribed them for 5 years or longer. That gap deserves to be part of every informed consent conversation.

CHAPTER / THREE

Side Effects: What You're Often Not Told

Every medication involves tradeoffs. Informed consent means knowing the ones that are usually left out of the conversation.

The Unspoken Side Effects

Every medication involves tradeoffs. Antidepressants are no exception — but some of the most debilitating and clinically significant side effects are consistently absent from informed consent conversations.

Most antidepressant side effects fall into a familiar, well-documented group — the ones your provider is likely to mention.

—THE COMMON, EXPECTED EFFECTS

Most are dose-dependent, tend to appear early, and often ease within the first few weeks. They are worth knowing — and worth reporting to your prescriber — but for most people they are manageable:

- Nausea or upset stomach
- Headache
- Sleep changes (insomnia or drowsiness)
- Dry mouth
- Appetite or weight changes
- Dizziness
- Fatigue or low energy
- Sweating, jitteriness, or restlessness

That list is not the problem. Those effects are real, but they are visible, expected, and usually discussed. **What concerns me more are the side effects that rarely make it into the conversation**. This is not an exhaustive review of every possible antidepressant side effect. It is a closer look at several of the most consequential risks — the ones patients deserve to understand before they begin treatment.

Emotional Blunting

Emotional blunting — the experience of feeling emotionally flat, muted, or disconnected — is one of the most commonly reported and least discussed side effects of antidepressants.

Patients describe it as:

- Feeling numb or emotionally “offline”

- Reduced ability to feel joy, excitement, or love
- Also reduced ability to feel sadness, grief, or fear
- "Watching life from a distance"
- Diminished creativity, motivation, or sense of aliveness

What makes this side effect especially problematic is how insidiously it can develop. Many of my clients only recognize the full weight of emotional blunting after months or years on medication — and many had assumed the flatness was just their depression. It often is not.

—WHAT THE RESEARCH SHOWS

Some degree of emotional blunting is commonly reported by people taking SSRIs or SNRIs, with surveys frequently finding rates in the range of 40–60%. In one multinational survey of 752 patients on antidepressants, 45% believed their medication was negatively affecting their emotional life, and just over one-third had considered stopping or had already stopped their medication as a result (Christensen et al., 2022).

Note: The Christensen et al. series was funded by H. Lundbeck A/S; the 40–60% prevalence estimate is consistent with independent clinical reviews.

Christensen, M. C., Ren, H., & Fagiolini, A. (2022). Emotional blunting in patients with depression. Part I: Clinical characteristics. Annals of General Psychiatry, 21, 10. https://doi.org/10.1186/s12991-022-00387-1

Sexual Dysfunction and PSSD

Sexual dysfunction is among the most common side effects of antidepressants, and among the least proactively discussed. It may include:

- Reduced libido
- Difficulty becoming or staying aroused
- Delayed or absent orgasm
- Erectile dysfunction

- Genital numbness or reduced sensation

—PREVALENCE

A widely cited meta-analysis by Serretti and Chiesa (2009) found rates of treatment-emergent sexual dysfunction ranging from 25.8% to 80.3% depending on the specific antidepressant. For SSRIs specifically, rates of 40–70% are commonly reported in the literature. In surveys asking patients directly, these numbers tend to be even higher.

Serretti, A., & Chiesa, A. (2009). Treatment-emergent sexual dysfunction related to antidepressants: A meta-analysis. Journal of Clinical Psychopharmacology, 29(3), 259–266. https://doi.org/10.1097/JCP.0b013e3181a5233f

Post-SSRI Sexual Dysfunction (PSSD)

—WHAT IS PSSD?

Post-SSRI Sexual Dysfunction (PSSD) is a condition in which sexual side effects persist after the antidepressant has been discontinued — sometimes for months or years, and in some cases indefinitely.

- Recognized by the European Medicines Agency (EMA) in 2019, leading to mandatory label updates across Europe.
- The FDA's current labeling for fluoxetine (Prozac) states explicitly: "Symptoms of sexual dysfunction occasionally persist after discontinuation of fluoxetine treatment." (DailyMed, 2023)
- PSSD was added to SNOMED CT international clinical terminology, enabling it to be formally documented in medical records.
- Reported symptoms include genital numbness, pleasureless or absent orgasm, loss of libido, and emotional blunting that persists after stopping the medication (with some documented cases lasting years or decades)

Exact prevalence is unknown due to underreporting and historical failures to track post-discontinuation effects. The condition is real, recognized by regulators, and should be part of every informed consent conversation before starting an antidepressant.

European Medicines Agency. (2019). Persistent sexual dysfunction after treatment with antidepressants and antipsychotics. EMA/513078/2019. · Healy, D., & Mangin, D. (2024). Post-SSRI sexual dysfunction: Barriers to quantifying incidence and prevalence. Epidemiology and Psychiatric Sciences, 33, e40. · U.S. Food and Drug Administration / DailyMed. (2023). Fluoxetine hydrochloride prescribing information.

Mood Elevation and the Induction of Mania

In some people, antidepressants do not just lift mood; they push it too far, producing hypomania or a full manic episode: racing thoughts, decreased need for sleep, elevated or irritable mood, impulsivity, grandiosity, rapid speech, and risky behavior.

This is well-documented. Treatment-emergent mania or hypomania has been reported with all major classes of antidepressants. It is more common with certain drugs, at higher doses— and it can be serious, both because of the impairment mania itself causes and because of what can happen next.

—IT'S IN THE OFFICIAL LABELING

This is not a fringe observation — mood elevation, hypomania, and mania are recognized risks in FDA-approved antidepressant labeling.

Both the **sertraline (Zoloft)** and **fluoxetine (Prozac)** prescribing information list *activation of mania/hypomania* as a labeled risk, report that mania or hypomania occurred in clinical trials, and direct clinicians to screen patients for bipolar disorder before starting treatment.

These are not opinions or fringe case reports. They are in the drugs' own official, FDA-approved labeling.

U.S. Food and Drug Administration / DailyMed. Sertraline (Zoloft) and fluoxetine (Prozac) prescribing information, Warnings and Precautions §5.4 — Activation of Mania/Hypomania; Screening Patients for Bipolar Disorder.

What can happen next. When someone becomes manic or hypomanic on an antidepressant, a common clinical response is to conclude that the person must have had bipolar disorder all along, and that the medication simply "unmasked" it. The diagnosis is changed. The treatment plan is often rewritten around a condition the person may never have shown a single sign of before that prescription.

There is real legitimacy to part of this concern, and it deserves to be stated: It is possible that antidepressants can reveal a bipolar vulnerability that had not yet declared itself, and for some people a manic switch could be evidence of bipolar disorder. That possibility deserves to be taken seriously, because the treatment implications are significant.

But treating *every* antidepressant-induced manic episode as proof of pre-existing bipolar illness is not supported by the evidence — and it is not even what the diagnostic manual says.

—WHAT THE DSM-5 ACTUALLY SAYS

The DSM-5 is explicit that timing and persistence matter. A full manic or hypomanic episode that continues beyond the physiological effect of antidepressant treatment may support a bipolar diagnosis. But symptoms that appear during treatment and resolve as the drug effect resolves should not automatically be treated as proof that someone was secretly bipolar all along.

American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders (5th ed.), criteria for manic and hypomanic episodes.

In other words, a manic reaction that appears on the medication and disappears when the medication is removed is, by the DSM's own definition, a drug effect — not a hidden lifelong illness. There are documented cases of people with no personal or family history of bipolar disorder who became manic on an SSRI, returned fully to baseline once it was stopped, and went years afterward with no further mood episodes. Labeling those individuals as permanently bipolar does not fit the evidence, and it carries real consequences: a diagnosis that may follow them for life, and medications — mood stabilizers, antipsychotics — that carry their own risks.

None of this means bipolar disorder isn't real, or that a manic switch should be ignored. It should always be taken seriously and evaluated carefully. The point is narrower, and it is the same point that runs through this entire guide:

A reaction to a drug is not the same as a lifelong diagnosis — and the difference between the two should be established carefully, not assumed.

IN PLAIN ENGLISH

If you became manic after starting an antidepressant and it settled once you stopped, that may have been the drug — not proof that you were secretly bipolar all along. It is worth a careful evaluation, not an automatic relabeling.

U.S. Food and Drug Administration / DailyMed. Sertraline (Zoloft) prescribing information. https://dailymed.nlm.nih.gov · U.S. Food and Drug Administration / DailyMed. Fluoxetine (Prozac) prescribing information. https://dailymed.nlm.nih.gov · Kimmel, R. J. (2013). Is antidepressant-associated mania always an evidence of a bipolar spectrum disorder? A case report and review of the literature. General Hospital Psychiatry, 35(5), 577.e1–577.e2. https://doi.org/10.1016/j.genhosppsych.2012.12.012 · Goldberg, J. F., & Truman, C. J. (2003). Antidepressant-induced mania: An overview of current controversies. Bipolar Disorders, 5(6), 407–420. https://doi.org/10.1046/j.1399-5618.2003.00067.x · Allain, N., et al. (2017). Manic switches induced by antidepressants: An umbrella review comparing randomized controlled trials and observational studies. Acta Psychiatrica Scandinavica, 135(2), 106–116. https://doi.org/10.1111/acps.12672 · American Psychiatric Association. (2013). Diagnostic and Statistical Manual of Mental Disorders (5th ed.).

Increased Risk of Suicidality

All antidepressants in the United States carry an FDA Black Box Warning — the most serious warning the FDA issues — regarding increased risk of suicidal thinking and behavior in children, adolescents, and young adults (under 25).

—FDA BLACK BOX WARNING

"Antidepressants increased the risk compared to placebo of suicidal thinking and behavior (suicidality) in children, adolescents, and young adults in short-term studies of major depressive disorder and other psychiatric disorders."

— U.S. Food and Drug Administration

In the FDA's pooled analysis of 24 pediatric trials, the risk of suicidality on antidepressants versus placebo was nearly double that of placebo (risk ratio 1.95; 95% CI 1.28–2.98), corresponding to

about 4% versus 2% of patients, a meaningful increase. This risk is most prominent during the early weeks of treatment, which is why close monitoring during initiation is critical.

To be clear: untreated severe depression carries its own serious suicide risk. Antidepressants may be beneficial or clinically indicated for some. But this risk is real, documented, and must be part of the informed consent conversation before a prescription is written.

U.S. Food and Drug Administration. (2007). Revisions to product labeling: Black box warning on antidepressants for suicidality in children, adolescents, and young adults. · Hammad, T. A., Laughren, T., & Racoosin, J. (2006). Suicidality in pediatric patients treated with antidepressant drugs. Archives of General Psychiatry, 63(3), 332–339.

CHAPTER / FOUR

Dependence and Withdrawal: What You Need to Know

Physical dependence is real and common. Withdrawal can be serious and debilitating.

Dependence and Withdrawal: Real & Common

Physical dependence on antidepressants is real, common, and increasingly well-documented in the scientific literature — yet it remains consistently understated in clinical practice and left out of many informed consent conversations.

—THE NUMBERS — A TALE OF TWO REVIEWS

Davies & Read (2019) conducted a systematic review of 14 studies and found that approximately 56% of patients experience withdrawal symptoms when stopping an antidepressant, with approximately 46% of those describing symptoms as severe.

Henssler et al. (2024) published a more methodologically rigorous meta-analysis of 79 studies in The Lancet Psychiatry. After accounting for symptoms also reported by people stopping placebo, they estimated that approximately **15% of patients** experience symptoms attributable to antidepressant withdrawal. Severe symptoms occurred in approximately **3%** of patients.

The true incidence likely sits somewhere between these estimates. Both reviews agree on one point: withdrawal is real, it can be severe, and patients deserve to know about it before they start.

Davies, J., & Read, J. (2019). A systematic review into the incidence, severity and duration of antidepressant withdrawal effects: Are guidelines evidence-based? Addictive Behaviors, 97, 111–121. · Henssler, J., Schmidt, Y., Schmidt, U., Schwarzer, G., Bschor, T., & Baethge, C. (2024). Incidence of antidepressant discontinuation symptoms. The Lancet Psychiatry, 11(7), 526–535.

What Withdrawal Looks Like

Withdrawal symptoms can be emotional, physical, cognitive, or neurological. They may include:

NEUROLOGICAL

- Brain zaps
- Dizziness / vertigo
- Perceptual disturbances
- Numbness or tingling

EMOTIONAL

- Intense anxiety
- Emotional instability
- Panic attacks
- Agitation / akathisia

PHYSICAL / COGNITIVE

- Insomnia
- Flu-like symptoms
- Nausea
- Cognitive difficulty

—A NOTE ON AKATHISIA

Akathisia — an intense, distressing inner restlessness and an unrelenting compulsion to move — deserves specific attention. It is one of the most severe and underrecognized symptoms associated with antidepressant use and discontinuation and is frequently mistaken for worsening anxiety or psychomotor agitation.

Akathisia can be profoundly distressing. In severe cases, it may contribute to extreme agitation, despair, or suicidal thinking, making prompt recognition and clinical response critical.

Withdrawal symptoms can range from uncomfortable to profoundly debilitating. Duration varies widely — from days to many months, sometimes longer. Davies & Read found that a substantial proportion of people experience withdrawal for longer than two weeks, and that prolonged withdrawal lasting months is not rare, and a substantial patient-reported literature describes withdrawal persisting for months or, in some cases, longer.

The Withdrawal-Relapse Error

> ***One of the most consequential and underrecognized errors in psychiatry is mistaking withdrawal for relapse.***

When someone feels worse after reducing or stopping an antidepressant, the worsening is often interpreted as proof that the medication was necessary — that the original depression or anxiety has returned, and that the person "needs" to stay on the drug long term.

Sometimes that may be true. Relapse can happen.

But in many cases, that interpretation is incomplete or wrong. **What looks like relapse may actually be withdrawal from the medication itself.** This distinction matters enormously.

Withdrawal can cause anxiety, panic, insomnia, low mood, crying spells, irritability, agitation, dizziness, brain zaps, nausea, flu-like symptoms, sensory changes, and emotional instability. Some of these symptoms look almost identical to depression or anxiety. If a clinician does not recognize withdrawal, the patient may be told, "See? This proves you still need the medication," when what it may actually prove is that the taper was too fast for that person's nervous system.

A few clues can help distinguish withdrawal from relapse. Withdrawal often begins soon after a dose reduction or missed doses, especially with shorter-acting medications. It may include physical or neurological symptoms that were not part of the original condition, such as brain zaps, dizziness, nausea, electric-shock sensations, vertigo, or sensory disturbances. It may also improve when the taper is paused, slowed, or carefully adjusted.

Relapse, by contrast, is usually a return of the person's original symptom pattern and often emerges more gradually, without the distinctive physical or neurological symptoms of withdrawal.

This does not mean every worsening during a taper is withdrawal. It means worsening during a taper should not automatically be labeled relapse. Patients deserve a careful assessment before being told they need to stay on a medication indefinitely.

> **IN PLAIN ENGLISH**
>
> *If you feel terrible after lowering or stopping an antidepressant, that does not automatically mean your depression or anxiety has returned. It may mean your body has adapted to the medication and the taper was too fast.*
>
> *The answer is not always "go back on forever."*
>
> *Sometimes the answer is: slow down, pause, stabilize, and taper more carefully.*

CHAPTER / FIVE

Medication Is Not the Only Option

Psychiatric medication is one tool. It is not the only tool — and the alternatives have real evidence behind them.

Psychiatric medication is one tool — sometimes a helpful one — but it should not be treated as the only path to healing. For some people, medication can create stability and reduce suffering. For others, especially when the deeper contributors to symptoms have not been addressed, the risks and limitations of medication deserve a more careful conversation.

When symptoms are shaped by trauma, chronic stress, sleep disruption, isolation, relationship dynamics, grief, burnout, or nervous system dysregulation, treatment should address those realities directly. The interventions below have meaningful evidence supporting their use for depression, anxiety, trauma-related symptoms, and related concerns — often with substantially better safety profiles than pharmacotherapy:

TREATMENT	EVIDENCE & NOTES
Psychotherapy (CBT, Interpersonal, Mindfulness Based, etc.)	Multiple psychotherapy approaches have strong evidence for depression and anxiety. Meta-analyses suggest psychotherapy can produce short-term effects comparable to antidepressants, and longer-term outcomes may favor psychotherapy or combined treatment over medication alone. Psychotherapy also does not create medication-induced physiological dependence. (Cuijpers et al., 2013; Karyotaki et al., 2016)
Trauma-Focused Therapy (EMDR, CPT)	For PTSD, trauma-focused therapies are among the most strongly supported treatments. These approaches directly target traumatic memories, avoidance, and trauma-related beliefs rather than relying only on symptom suppression.
Exercise	A 2024 network meta-analysis found walking, jogging, yoga, and strength training produced moderate reductions in depression comparable to antidepressants and psychotherapy. (Noetel et al., 2024)

TREATMENT	EVIDENCE & NOTES
Sleep Optimization	Bidirectional relationship with depression; improving sleep quality can significantly reduce depressive symptoms and may reduce the need for pharmacotherapy.
Nutrition & Gut Health	Emerging evidence links gut microbiome to mood regulation; anti-inflammatory dietary patterns associated with reduced depression risk. (Jacka et al., 2017).
Nervous System Regulation (Somatic therapy, breathwork, mindfulness, yoga, body-based practices)	Mental health symptoms are not only cognitive; they are physiological, too. Anxiety, trauma, panic, shutdown, insomnia, and hyperarousal often involve the body and nervous system. Body-based approaches aim to help patients regulate arousal, tolerate sensation, reconnect with the body, and rebuild a felt sense of safety. Evidence is still developing, but promising, especially for stress, anxiety, trauma-related symptoms, and emotional regulation.
Ketamine & Ketamine-Assisted Therapy	Ketamine and esketamine have strong evidence for rapid antidepressant effects, especially in treatment-resistant depression. Esketamine nasal spray is FDA-approved under a REMS monitoring program, while racemic ketamine remains off-label for depression. Ketamine-assisted psychotherapy is a promising emerging model that combines biological effects with therapeutic support and integration.
Social Connection & Community	Isolation is not just emotionally painful — it is physiologically stressful. Loneliness, disconnection, relational trauma, and lack of belonging can worsen depression, anxiety, sleep, inflammation, and nervous system dysregulation. Healing often requires more than insight or symptom reduction; it requires safe relationships, meaningful connection, community, and a life where the nervous system does not have to survive alone.

Cuijpers, P., et al. (2013). The efficacy of psychotherapy and pharmacotherapy in treating depressive and anxiety disorders. World Psychiatry, 12(2), 137–148. · Noetel, M., et al. (2024). Effect of exercise for depression. BMJ, 384, e075847. · U.S. Food and Drug Administration. (2019). FDA approves new nasal spray medication for treatment-resistant depression.

A More Complete Path

A more complete approach to mental health begins with a different question. Not simply: *What medication treats this symptom?* But rather: *What is this symptom connected to? What is it trying to signal? What has this person lived through? What is their body carrying? What relationships, environments, habits, losses, stressors, or nervous system patterns are shaping what they feel?*

Depression may be connected to grief, isolation, chronic stress, trauma, burnout, sleep disruption, inflammation, disconnection, or a life that no longer feels meaningful. Anxiety may be connected to unresolved threat, unsafe relationships, perfectionism, chronic over-responsibility, nervous system activation, or a body that has never fully learned what safety feels like. OCD, PTSD, insomnia, panic, and emotional dysregulation often have patterns beneath them that deserve careful attention — not just suppression.

Good treatment should take all of that seriously.

It should also include informed consent from the beginning. Patients deserve to know what a medication can do, what it cannot do, what risks come with it, what alternatives exist, how long the medication is intended to be used, and what stopping it may eventually require.

A prescription may reduce symptoms. But healing usually asks for more.

Healing may require grieving what was never grieved. Leaving what is no longer sustainable. Learning how to feel again. Repairing the body's sense of safety. Rebuilding connection. Changing patterns that once protected you but now keep you stuck. Creating a life your nervous system does not have to fight every day.

That is not quick work. But it is real work.

And for many people, it is the work that actually changes things.

> **IN PLAIN ENGLISH**
>
> *Medication can sometimes help you feel better. But feeling better is not always the same as healing.*
>
> *A more complete treatment plan should ask more than, "Which medication?" It should ask what happened, what hurts, what is missing, what needs to change, and what would help you actually get well.*

CHAPTER / SIX

Deprescribing: An Art and a Science

Many withdrawal problems are caused by stopping antidepressants too quickly.

If you are considering reducing or stopping an antidepressant, the most important thing to understand is this:

Many withdrawal problems are caused by stopping antidepressants too quickly, without proper support.

Coming off an antidepressant safely often takes far more time and care than most people — and many clinicians — expect. Some people taper with little difficulty. Others experience significant withdrawal symptoms, particularly after long-term use or higher doses. The difference often comes down to one thing: how the taper is done.

Why Standard Tapers Sometimes Fail

For many years, patients were routinely advised to reduce antidepressants over a few weeks using fixed dose reductions — for example, cutting from 20 mg to 10 mg to 5 mg and then stopping. While this works for some people, for others it is too fast. Not because they are weak or dependent in the conventional sense, but because of how antidepressants interact with the brain.

The relationship between dose and biological effect is not linear. At lower doses, even small reductions can produce disproportionately large changes in the brain's receptor activity. A reduction from 10 mg to 5 mg may create a much larger neurological shift than a reduction from 20 mg to 15 mg — even though both are a 5 mg cut on paper.

This helps explain one of the most common experiences reported by patients attempting to stop an antidepressant:

I felt fine through most of the taper.

The hardest part was the last few milligrams.

> **IN PLAIN ENGLISH**
>
> *Imagine dimming a light. Turning it from 100% brightness to 80% barely changes the room. But turning it from 5% brightness to complete darkness feels dramatic. For some people, antidepressants work similarly. The final stages of a taper can produce the largest adjustment the nervous system has to make, even though the dose appears small.*

Hyperbolic Tapering

To account for this, leading experts in deprescribing now recommend a strategy called hyperbolic tapering.

Rather than making equal milligram reductions at each step, hyperbolic tapering uses progressively smaller reductions as the dose gets lower. The goal is to create more consistent biological changes throughout the taper, giving the nervous system adequate time to adapt at each stage.

A common hyperbolic-style approach is:

Reduce by a percentage of the **current dose**, not the original dose.

THE PRINCIPLE IN PRACTICE

Traditional taper — equal milligram cuts:

20 mg → 10 mg → 5 mg → Stop

Hyperbolic taper — progressively smaller cuts:

20 → 18 → 16.2 → 14.6 → 13.1 → 11.8 → 10.6 → 9.5 → 8.6 → 7.7 → and so on

Note: This is an illustration of the concept, not a prescription. Individual tapers should be designed collaboratively with a knowledgeable clinician based on the specific medication, dose, duration of use, and the patient's symptom response.

This approach is described by Horowitz & Taylor in The Lancet Psychiatry (2019), reflected in the Maudsley Deprescribing Guidelines (2024), and now incorporated into guidance from the Royal College of Psychiatrists. Outcomes of hyperbolic tapering in real-world clinical practice have been documented in a 2023 prospective cohort study of nearly 4,000 patients in the Netherlands, which found that longer tapering trajectories were associated with lower withdrawal severity (van Os & Groot, 2023). It represents a meaningful shift from the "one-size-fits-all" schedules that have caused unnecessary suffering for many patients.

The Guiding Principle

Not everyone needs a long or highly gradual taper. Some people can reduce quickly and comfortably. Others require months or years. There is no universally correct speed.

—WHAT GUIDES A GOOD TAPER

- **The taper should be guided by the patient's response to the taper — not by an arbitrary schedule.**
- If withdrawal symptoms become significant, slowing down, pausing, or adjusting the dose is usually more effective than pushing through.
- Hyperbolic tapering often requires liquid formulations, compounded doses, or specialty tapering strips — all of which are available but require a clinician who knows how to use them.
- **A successful taper is not the fastest taper. It is the one the individual can tolerate.**

What makes someone more likely to need a slower taper?

Not everyone needs a long taper. Some people stop with few or minimal symptoms.

But slower tapering is often more important when someone has:

- long-term antidepressant use
- previous withdrawal symptoms
- high sensitivity to dose changes
- use of shorter half-life medications, especially paroxetine or venlafaxine
- high anxiety, insomnia, akathisia, or neurologic withdrawal symptoms
- multiple failed taper attempts
- major life stress during the taper

Horowitz, M. A., & Taylor, D. (2019). Tapering of SSRI treatment to mitigate withdrawal symptoms. The Lancet Psychiatry, 6(6), 538–546. · Horowitz, M. A., & Taylor, D. M. (2024). The Maudsley deprescribing guidelines. Wiley. · Royal College of Psychiatrists. (2021). Stopping antidepressants. · van Os, J., & Groot, P. C. (2023). Outcomes of hyperbolic tapering of antidepressants. Therapeutic Advances in Psychopharmacology, 13.

The Emotional Side of Deprescribing

Deprescribing is not only a medication process. It is often an emotional one, too.

For some people, antidepressants do more than reduce depression or anxiety. They may also dampen emotional intensity, mute grief, soften anger, blunt fear, or create distance from painful memories and body sensations. When the medication is gradually reduced, some of what has been muted may begin to come back online.

This can feel confusing and distressing if no one has prepared you for it.

A person may begin to feel more sadness, sensitivity, irritability, grief, anxiety, or emotional intensity during a taper and assume something has gone wrong. Sometimes those symptoms are withdrawal. Sometimes they are emotions, memories, stress responses, or nervous system patterns that were never fully processed.

This is why, in my view, deprescribing should not be reduced to dose changes alone.

A thoughtful taper pays attention to the whole person: sleep, stress, relationships, trauma history, body sensations, emotional capacity, nervous system regulation, and the pace at which the person can safely feel what is emerging.

I find that somatic processing can be especially useful here. It helps people notice what is happening in the body, build regulation skills, tolerate emotion without becoming overwhelmed, and create a greater sense of safety as sensations and feelings return.

In my experience, successful deprescribing requires both physiological support and emotional support.

The goal is not to force someone to "push through." The goal is to taper at a pace the nervous system can integrate.

CHAPTER / SEVEN

Questions I Wish Every Patient Asked

Not to challenge your providers — to create an informed conversation. Your care is better when you are an active participant in it.

The following questions are the ones I wish every patient would bring into their psychiatric appointments — not to challenge their providers, but to create an informed conversation. Your care is better when you are an active participant in it.

— BEFORE STARTING A MEDICATION

1. What do we know about the specific benefits of this medication for my condition?
2. What are the potential risks, including the long-term risks?
3. How large was the medication's benefit compared with placebo in the studies used to support it?
4. How long were the studies this medication was approved on?
5. What alternatives to medication should I consider for my situation?
6. What trauma, sleep, relationship, lifestyle, medical, environmental, or social factors might be contributing to my symptoms?
7. If the medication does help, how long are we planning for me to stay on it before reassessing?
8. If I decide to stop this medication later, what will that process look like?

— ABOUT SIDE EFFECTS

1. How common are sexual side effects with this medication, specifically?
2. What is Post-SSRI Sexual Dysfunction, and has it been reported with this medication or medication class?
3. How often do patients experience emotional blunting?
4. If I develop side effects, will the plan be to lower the dose, switch medications, add another medication, or stop?
5. How would I distinguish a side effect from a symptom of my condition?

— ABOUT STOPPING OR REDUCING

1. If I decide I want to stop this medication, what does that process look like?
2. How will we distinguish withdrawal from the return of my original depression or anxiety?
3. What should I do if withdrawal symptoms are severe?
4. Are liquid forms, compounded doses, tapering strips, or smaller-dose options available if needed?
5. What emotional or psychological support should be in place during the taper?

A Final Word

My goal is not to convince you to avoid medication.

My goal is to help you become an informed participant in your own mental health care — because informed patients get better outcomes, make more aligned choices, and are more likely to address the things that are keeping them stuck.

Patients deserve informed consent.

This guide represents much of the informed consent conversation I give every new patient I work with. If it has been useful, share it. If it has raised questions, bring them to your provider. If your provider is dismissive or unable to engage with these questions, it is reasonable to seek a provider who will engage with them.

You are allowed to ask hard questions.

You are allowed to want more than a prescription.

You are allowed to want to actually heal.

Nick Bischoff, APRN, PMHNP-BC

Psychiatric Nurse Practitioner | Thriving Mind PLLC

KETAMINE-ASSISTED PSYCHOTHERAPY · TRAUMA-INFORMED PSYCHIATRIC CARE · DEPRESCRIBING

—FINAL DISCLAIMER

This guide is for education and informed consent only. It is not medical advice, psychiatric treatment, diagnosis, psychotherapy, or a substitute for individualized care from a qualified healthcare professional.

Do not start, stop, taper, increase, reduce, or change any medication without working directly with a licensed clinician who understands your history, symptoms, medications, risks, and current situation.

Antidepressants can be helpful, appropriate, and sometimes necessary. They can also carry risks, side effects, withdrawal concerns, and unanswered long-term questions. This guide is not meant to tell you what decision to make. It is meant to help you ask better questions.

If you are experiencing suicidal thoughts, thoughts of harming yourself or others, mania, psychosis, severe agitation, confusion, or any mental health emergency, seek immediate help.

You deserve informed consent. You deserve careful care. And you deserve support that takes your whole story seriously.

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