Low-Dose Buprenorphine for BPD
A Patient Companion Guide

Understanding an Emerging Treatment for Interpersonal and Emotional Dysregulation

v0.2 Draft — August 2026

Contents

This document is a companion to the clinical guide *Low-Dose Buprenorphine in Severe Interpersonal and Affective Dysregulation: A Clinician Consideration Guide*, available at bpd.fyi/resources. It is written for patients and does not constitute medical advice. Discuss all treatment decisions with your prescribing clinician.

What This Guide Is For

You may have arrived here two different ways.

A clinician may have raised buprenorphine with you as a possible treatment for the emotional and interpersonal difficulties associated with borderline personality disorder (BPD) or similar presentations. If so, this guide explains what they are proposing and why.

More likely, you found this yourself, and the conversation with your clinician hasn't happened yet. That is the harder position, and most of this guide is written for it. Bringing an unfamiliar off-label use of a controlled medication to a prescriber is a specific kind of conversation, and it goes considerably better when you can explain *why* the idea makes biological sense rather than only that you read about it somewhere.

That is the purpose of the mechanism section that follows. It is not background. It is the part that makes the request a reasonable one to consider, and it is worth understanding well enough to say out loud in your own words.

This guide covers why a medication associated with addiction treatment is being considered for a completely different reason, what the evidence does and does not show, what to expect if you try it, and what to ask.

You are not being treated for addiction. The doses involved are far lower than those used in addiction medicine, and the purpose is different: not to manage cravings or withdrawal, but to address a possible deficit in the system that regulates social pain and attachment. It is the same receptor system either way. What differs is the problem being treated.

Why Buprenorphine? The Basic Idea

Your brain has a system for connection and social pain

Your brain produces its own opioid-like chemicals — called *endogenous opioids* — that play a central role in how you experience social connection, attachment, and the pain of rejection or loss. This isn't a metaphor: neuroscience research shows that the feeling of being socially rejected activates some of the same brain systems as physical pain, and your body's natural opioid system helps regulate both.

When you feel connected to someone you care about, your brain releases these chemicals. When that connection is threatened or lost, those chemical levels drop — and that drop is part of what makes separation, rejection, or abandonment feel so intensely painful.

In BPD, this system may be running too low

Researchers — including Barbara Stanley and Borwin Bandelow — have proposed that people with BPD may have lower-than-normal baseline levels of these natural opioid chemicals. If that's the case, it helps explain several things you may recognize:

- **Intense reactions to interpersonal stress.** When your baseline is already low, even small drops in opioid signaling (from a perceived slight, a missed text, a change in someone's tone) can feel catastrophic — because you're starting from a place of deficit.
- **The "overreaction" problem.** Other people may tell you that your reactions are disproportionate. From the outside, they may look that way. But from the inside, the neurobiological experience may actually be proportional to what your brain is registering, which is a larger relative drop from a lower baseline.
- **Chronic emptiness.** That persistent sense of hollowness or emotional numbness may reflect chronically low opioid tone rather than a character flaw or a failure of willpower.
- **Why certain behaviors provide temporary relief.** Self-injury, intense emotional states, substance use, and even high-intensity relationship dynamics can all trigger bursts of endogenous opioid release. If your baseline is low, these behaviors may be serving a biological function — your nervous system's attempt to get what it needs. This doesn't make them healthy or sustainable, but it may help explain why they're so hard to stop.

What buprenorphine may do

Buprenorphine has three properties that are relevant here:

1. **It partially activates the opioid system in a steady, controlled way** — potentially raising your baseline opioid tone closer to where it should be. "Partial" means there is a ceiling: past a certain point, more drug does not produce more effect, which is a large part of why buprenorphine is so much safer than a full opioid. The steadiness itself comes from how long it lasts. Buprenorphine stays in your system far longer than most opioids and releases from the receptor slowly, so levels stay relatively flat instead of peaking and dropping.

2. **It holds onto the receptor tightly.** This is why it blocks other opioids, including pain medication — see the note on that below. It has been proposed that the same property may blunt your body's *own* opioid surges, including the ones that follow self-injury or other things people do for relief. That has not been directly studied at these doses, and how much of it happens almost certainly depends on the dose, since receptor occupancy is much lower here than in addiction treatment. It is possible this is part of why some people need higher doses than others. But if you notice that something you used to do for relief no longer produces the same feeling, this may be why.
3. **It blocks a different part of the opioid system (called kappa receptors)** that is involved in the experience of stress-related misery, despair, and dysphoria. By blocking these receptors, buprenorphine may directly reduce the intensity of the worst emotional states — the ones that feel like existential dread or unbearable psychic pain.

The combined effect, in theory: your emotional baseline becomes more stable, extreme reactions to interpersonal stress become more proportional, and the worst lows become less severe. Not numb — more proportional.

> **The key point:** This is not about sedating you or suppressing your emotions. The goal is to correct a possible deficit in a specific brain system so that your emotional responses more closely match the actual size of what's happening in your life.

What Does the Evidence Actually Look Like?

The evidence is promising but limited:

What exists

A randomized controlled trial including people with BPD (Yovell et al., 2016). This study gave very low doses of buprenorphine to hospitalized people with severe suicidal thoughts, starting at 0.1–0.2 mg dissolving under the tongue and titrating up to a maximum of 0.8 mg per day. More than half the participants had BPD. Suicidal ideation dropped significantly compared to placebo.

People with BPD responded just as well to buprenorphine as people without it. What differed was the placebo response: people with BPD improved *less* on placebo than others did. That widens the gap between the medication and placebo in this group, which is a reason to think the medication is doing something real here rather than the improvement coming from expectation. That comparison was not the study's main question, so it should be read carefully. But it is the opposite of what people are often told about this diagnosis, and it is worth knowing. This remains the trial most directly relevant to BPD.

A second randomized controlled trial (Tucciarone et al., 2026). Conducted at Stanford and published in the *American Journal of Psychiatry*, this trial tested low-dose sublingual buprenorphine (0.2–0.8 mg/day) for four weeks in adults with major depressive disorder and significant suicidal ideation, started two days after a single ketamine infusion. Fifty people received ketamine and 45 completed at least a week of the follow-on treatment. By day 31, 78% of the buprenorphine group had at least a 50% reduction in suicidal ideation, compared with 48% on placebo. It is the first treatment shown to extend ketamine's antisuicidal effect.

What this trial offers is independent confirmation that low doses of buprenorphine act on suicidal ideation, from a large academic center, with a senior author who is a former president of the American Psychiatric Association. Its enrollment criteria were limited to major depressive disorder and did not include participants with BPD, so it is supporting evidence rather than direct evidence, and it is worth knowing that if you bring the paper to a clinician.

One further finding is worth noting: buprenorphine improved suicidal ideation but did not significantly improve depression scores. That suggests it is acting on something specific rather than lifting mood in general, which fits the

social pain model better than an antidepressant model.

One published case report (Hansen et al., 2022). A patient with BPD who was not using opioids was started on buprenorphine/naloxone and gradually increased to 6 mg. Before treatment, she had 41 crisis contacts in 15 months and spent an average of over 200 hours per hospital visit. After treatment, crisis contacts dropped to 12, then to zero after dose optimization. When she briefly stopped the medication, her symptoms returned — and resolved again when she restarted.

A body of neuroscience research supporting the idea that the opioid system is central to attachment and that BPD involves opioid system dysfunction.

What doesn't exist yet

No large clinical trial has been specifically designed to test buprenorphine for BPD as its primary question. There are no established treatment guidelines for this use. Your clinician is working from emerging evidence, clinical reasoning, and careful judgment, not from a textbook protocol.

It is worth naming that people with BPD are routinely excluded from trials like these, often listed under "severe psychiatric comorbidity." That exclusion is part of why the evidence you need does not exist yet, and it is not a statement about whether the treatment would work for you. If that exclusion stings to read, that reaction is reasonable.

This is an off-label use of the medication. "Off-label" means the FDA hasn't specifically approved it for this purpose, but it is legal for your doctor to prescribe it based on clinical judgment. Off-label prescribing is common across all of medicine.

What to Expect If You Start Treatment

How it's taken

Buprenorphine for this purpose is most often taken as a tablet or liquid held under the tongue (sublingual). You may be given a very low starting dose — much lower than the pills commonly available — which may involve dissolving a tablet in water and measuring small amounts with a syringe. Your clinician will explain the specific preparation if this applies to you.

This is a daily, scheduled medication — not something you take when you feel bad. It works by maintaining a steady level in your system over time, not by providing immediate relief in a crisis.

There is more than one formulation

Buprenorphine comes in several forms, and the differences matter more than most people are told.

Sublingual (under the tongue). Tablets or films. The most familiar route, and the most flexible for very low and precisely adjusted doses. Carries the dental risks described below.

Buccal film (e.g., Belbuca). Placed against the inside of the cheek, and manufactured in microgram strengths (75 to 900 mcg) rather than milligrams. This formulation is FDA-approved for chronic pain. It still dissolves in the mouth, so the dental considerations apply.

Transdermal patch (e.g., Butrans). A weekly skin patch delivering 5 to 20 micrograms per hour, also approved for chronic pain. Because nothing dissolves in your mouth, this route avoids the dental risk entirely, and it delivers a very steady level rather than a daily peak. The trade-off is that dose adjustments are coarser.

Why the numbers don't compare directly across formulations

This is worth understanding before you compare doses, because the same milligram figure can mean quite different amounts of drug depending on the route.

Only a fraction of a dose held in the mouth actually reaches your bloodstream. The rest is swallowed and broken down by the liver before it can do anything. How large that fraction is depends on the formulation:

Route	Roughly how much reaches the bloodstream
Sublingual (under the tongue)	About 30–50%
Buccal film (against the cheek)	About 45–65%
Transdermal patch	Dosed directly as the amount delivered

Two consequences follow.

A buccal film is stronger than the same number of milligrams sublingually. A 300 mcg buccal film and 0.3 mg of a sublingual tablet look identical on paper. The buccal film may deliver something closer to one and a half or two times as much drug into your system. If you switch between these routes, the dose usually needs to change, and switching at the same number is a common way to end up accidentally overdosed.

The patch numbers look tiny but aren't. Patches are dosed by what actually gets delivered, with no first-pass loss to account for. A 5 mcg/hour patch delivers about 120 mcg per day directly into your circulation. To get that much from a sublingual tablet you would need roughly 0.25 to 0.4 mg daily, depending on where you fall in that absorption range. So a 5 mcg/hour patch is in the same territory as a few tenths of a milligram sublingually, despite appearing to be a hundred times smaller.

These are population averages, and absorption varies quite a bit between individuals. Treat them as a way to understand why the numbers differ, not as a conversion to perform yourself. Your clinician should do the arithmetic, but it helps to know why the arithmetic is necessary.

Any of these can legally be prescribed for any indication a clinician judges appropriate; approval labels describe what was studied and submitted to the FDA, not the limits of what can be prescribed. In practice, though, clinicians differ in what they feel equipped to prescribe. A psychiatrist may decline a pain-labeled formulation on the grounds that they do not treat pain, in which case your primary care clinician may be the better person to ask.

If you have dental concerns, existing dental problems, or dry mouth from other medications, the patch is worth asking about specifically.

Starting doses are intentionally very low

Most people start at 0.1–0.2 mg per day. For comparison, the standard dose for addiction treatment is 8–24 mg per day. You are starting at a fraction of that.

Starting low matters because people who aren't used to opioid medications can be quite sensitive to them. Nausea is a common side effect at higher starting doses and can cause people to give up on a medication that might have worked at a lower dose. Starting low is a cautious choice.

The dose that works is highly individual

Where you start is not necessarily where you end up, and the range across reported cases is wide. Some people see meaningful improvement at 0.1 mg daily. In the published case report described earlier, crisis contacts did not stop

until the dose reached 6 mg. The trials used 0.1 to 0.8 mg. That is a sixtyfold spread, and there is currently no test that predicts where you will land.

This makes sense if the medication is correcting a deficit rather than producing a fixed drug effect, because the size of the deficit differs from person to person. It also means a dose that did nothing is not the same as a medication that doesn't work for you, and it is worth knowing that before you conclude anything from an early trial at a low dose.

The goal is the lowest dose at which you notice the change you are looking for. Finding it takes patience from both you and your clinician.

How long before you notice something

Sooner than you might expect. In both randomized trials, the buprenorphine and placebo groups had begun to separate within the first week or two of treatment, and both trials ran only four weeks in total. What people describe noticing first is usually subtle rather than dramatic: reactions slightly less intense, recovery from an emotional disturbance somewhat faster.

This means you should not have to wait months to learn something. If a week or two at a given dose produces no change at all, that is information worth acting on rather than sitting with. It does not necessarily mean the medication has failed — the far more common explanation is that the dose is still too low, and the next step is a conversation about increasing it rather than a decision to stop.

The distinction that matters is between *waiting* and *titrating*. Time spent adjusting the dose toward one that does something is time well spent. Time spent sitting at a dose that is doing nothing is not, and physical dependence accumulates with duration of exposure. So a long trial of a medication that never worked still leaves you with mild, temporary discomfort when you stop. That is a reason to be decisive rather than patient.

If you have reached a dose that is clearly doing nothing, or the side effects are not tolerable, stopping is a reasonable choice and an unsuccessful trial is useful information.

Side effects to know about

- **Nausea** — the most common side effect, and more likely if the dose is too high. Nausea may fade within one to two weeks at a steady dose.
- **Dizziness** — reported by roughly one in five participants in the 2026 Stanford trial, against none on placebo.
- **Oral burning or numbness** — reported by roughly one in six participants taking sublingual buprenorphine in that same trial, against none on placebo. Specific to formulations that dissolve in the mouth.
- **Sedation or drowsiness** — usually mild at these doses. Report if it affects your functioning.
- **Constipation** — a common opioid-class effect, typically mild at low doses.
- **Headache** — occasionally reported.

Important — dental risks with sublingual and buccal buprenorphine: In 2022 the FDA added a warning about dental problems associated with buprenorphine medicines that dissolve in the mouth, including cavities, tooth decay, gum injury, and tooth loss. These have been reported even in people with no prior history of dental problems, and even at low doses. The medication is mildly acidic, and it sits against the teeth while dissolving.

This risk is real and it is also manageable. Rinse your mouth with water after the medication has fully dissolved, swish and swallow, and *wait at least an hour before brushing* — brushing immediately can scrub acid into softened enamel. Keep up regular dental visits and tell your dentist you take this medication.

If dental problems are already a concern for you, ask about the transdermal patch. It bypasses the mouth completely. This is a common and legitimate reason to request a formulation change, and it is a much smaller request than the original prescribing decision was.

Important — pain medication interaction: Buprenorphine occupies the same receptors that pain medications (like morphine, oxycodone, etc.) use. While you're taking buprenorphine, standard opioid pain medications may not work as well or at all. This matters if you need emergency surgery, have a dental procedure, or go to the ER for an injury.

Carry written documentation of your buprenorphine use (dose, prescribing clinician, and the fact that it's for a psychiatric indication, not addiction). Show it to any emergency or surgical provider. Your clinician can provide a note for this purpose.

Monitoring and Follow-Up

Your clinician will monitor you regularly, especially in the first few months. This typically includes:

- **Monthly visits during dose adjustment** to check how you're responding, screen for side effects, and adjust the dose if needed.
- **A baseline drug screen and a prescription monitoring database check** before starting. These are standard for any controlled substance prescription. They are not a sign that your clinician suspects you of misusing the medication, and they are not something you should expect at every visit.
- **Tracking specific goals.** Before starting, you and your clinician should agree on what "improvement" would look like — for example, fewer crisis contacts, less frequent self-injury, better ability to attend therapy, or improved relationship stability. These concrete goals help both of you evaluate whether the medication is actually helping.
- **Screening for dissociation.** If you experience dissociative symptoms (feeling detached from yourself, feeling like things aren't real, memory gaps), let your clinician know. This may affect how the medication is dosed or delivered.

Once you're stable, visits may be spaced to every three months.

Things You Should Know

Physical dependence is expected — and it's not addiction

If you take buprenorphine regularly, your body will adapt to its presence. If you stop suddenly, you may experience withdrawal symptoms (discomfort, anxiety, sleep disruption, flu-like symptoms). This is called *physical dependence*, and it is a normal physiological response, the same way your body adapts to blood pressure medication or antidepressants.

Physical dependence is **not the same as addiction.** Addiction involves compulsive use, craving, loss of control, and continued use despite harm. At these low doses, buprenorphine's reinforcing properties are substantially reduced compared to other opioids.

If you decide to stop, do that with your clinician rather than on your own. In both trials, participants did not report significant withdrawal symptoms after the 4 weeks they took buprenorphine and discontinued without a taper. How best to come off buprenorphine at these doses is genuinely unsettled and it is one of the open questions the researchers behind the 2026 trial have called for study on. It is a decision to make together rather than one with a standard answer.

This is a trial, not a permanent commitment

You and your clinician are trying something based on emerging evidence. If it helps, continuing makes sense. If it doesn't help after an adequate trial, stopping is appropriate. You are not locked in.

One thing to expect if you do stop: in the 2026 Stanford trial, participants who came off buprenorphine showed a measurable return of suicidal ideation and depression scores within a week, while the placebo group did not. This is consistent with the case report described earlier, where symptoms returned on stopping and resolved again on restarting. If this medication works for you, it is likely working while you take it rather than producing a lasting change that persists after it stops. That is true of most psychiatric medications and is not a sign that something has gone wrong, but it is worth knowing before you start, and it is worth planning any discontinuation with your clinician rather than during a difficult week.

Hormonal fluctuations may affect how it works

If you experience cyclical estrogen changes — whether from a menstrual cycle or from hormone replacement therapy — you may notice that the medication seems less effective during certain phases. Estrogen affects opioid receptor sensitivity, so this is biologically expected. It does not necessarily mean you need a higher dose permanently. Mention any patterns you notice to your clinician.

Your observations matter

You know your internal experience better than anyone. If you notice that your reactions to interpersonal stress feel slightly less intense, that you recover faster from emotional disturbances, that the urge to self-injure is somewhat reduced, or that you're better able to use coping skills you've learned in therapy — those are meaningful signals. Similarly, if you notice worsening dissociation, increased emotional blunting, or any concerning changes, report them.

You are the primary observer in this process.

Common Questions

"Am I being treated for addiction?"

Not in the sense that matters clinically. You are not being treated for opioid use disorder, and the dose range used here (typically 0.1–1 mg daily, sometimes higher) is far below what is used in addiction treatment (8–24 mg daily). Someone who has been using full agonist opioids needs enough medication to occupy receptors that are already heavily adapted. That is a different situation from correcting a deficit.

But it is worth being honest that the two are not unrelated, and some people find the connection clarifying rather than shameful.

If the opioid system is dysregulated in BPD, then the behaviors that reliably trigger endorphin release — self-injury, intensely charged relationships, risk-taking, substance use — are doing something. They work. That is precisely why they are so hard to stop. Some people describe their attachments in frankly addictive terms: feeling unable to function when a particular person is unavailable, organizing their life around access to them, and experiencing something very like withdrawal when that access is lost. That description is not a misuse of the word.

What buprenorphine may do is raise the floor enough that those behaviors stop being the only thing that works.

And the rest of the work looks similar too. Recovery in both cases involves therapy, honest self-examination, understanding what a behavior was doing for you, and building patterns that meet the same need at a lower cost. Medication makes that work more possible. It does not do it for you.

This doesn't mean you should be routed into addiction treatment for BPD. It does mean the distance between these conditions may be smaller than either group is usually comfortable saying out loud.

"Will people think I'm on opioids?"

Buprenorphine at these doses should not cause the kind of impairment associated with opioid use. You should not feel "high," sedated to the point of impairment, or cognitively dulled. If you do, the dose may be too high, and you should tell your clinician.

You are not obligated to disclose your medications to anyone other than your healthcare providers. However, as noted above, emergency and surgical providers need to know.

"Why hasn't my psychiatrist mentioned this?"

This is an emerging area. Many psychiatrists are not yet familiar with the evidence base connecting the opioid system to BPD, and buprenorphine is still primarily associated with addiction medicine in most clinicians' training. This is changing, but slowly. Your clinician's willingness to consider this approach reflects engagement with current research.

"What if it doesn't work?"

Then you stop, with your clinician's guidance, and move on to other options. An unsuccessful trial is useful information, not a failure. It may narrow the search for what does help.

"Will this replace therapy?"

No. Buprenorphine is not a substitute for psychotherapy — particularly DBT or other structured approaches designed for BPD. What it may do is lower the emotional intensity enough that therapy becomes more accessible. Many people with BPD find it hardest to use their skills at exactly the moments they need them most, because that's when affect intensity is highest. If buprenorphine reduces peak intensity, the window in which skills are actually usable may widen.

Think of it as potentially making the work of therapy more possible, not replacing the work itself.

"What about naltrexone — I've heard that helps too?"

You should not take the two at the same time. If you are taking naltrexone in any form, including low-dose naltrexone (LDN), tell your clinician before starting buprenorphine. LDN is easy to forget to mention and does not reliably appear in prescription monitoring databases. There needs to be a washout period between stopping one and starting the other.

At 25 mg of naltrexone and above, it's well established: naltrexone displaces buprenorphine from the receptors, and losing that occupancy abruptly produces withdrawal — faster and sharper than stopping on your own, lasting a couple of days because naltrexone is long-acting. Whether the same holds in the LDN range has never been studied. Until it is, treat any milligram-dosed naltrexone as incompatible.

If the two do overlap by accident, tell your clinician and ask for supportive treatment. Opioid withdrawal is not typically life-threatening the way alcohol or benzodiazepine withdrawal can be, and trials that deliberately moved people from buprenorphine onto naltrexone found it milder than expected — though that was at addiction-treatment doses, far above these.

They are not simply opposites, and the difference explains the rest of this answer. Buprenorphine blocks too: it holds the receptor tightly enough that other opioids, including your own endorphins, cannot reach it. What sets it apart is that

it partially activates the receptor while sitting there, continuously, instead of clearing off after a few hours. LDN works the other way around — a brief block, then a rebound of your own endorphins once it lifts.

There is still a reason naltrexone comes up, and it may be worth raising with your clinician.

Because both act on the same receptor system, how sensitive you are to *one* may say something about how sensitive you will be to the other. Some people tolerate only microgram amounts of naltrexone, as low as 10 micrograms; others do fine at 0.5 to 4.5 mg. If you have taken LDN, that experience is information about your opioid system, and it may suggest where in the very wide buprenorphine range to begin. Starting too high is the most common way a trial fails for the wrong reason.

If LDN did little, or made things worse, that is not a reason to conclude the opioid system is the wrong target. LDN needs a strong endorphin rebound to recruit. Steady partial activation may be the better solution.

Dissociative symptoms are another reason. Dissociation is held in place partly by your own opioid signaling, and it is protective rather than broken — it developed because it worked. Opioid blockers do reduce dissociative symptoms; that is what the trials found. But they do it by lowering that barrier, and if the barrier is holding back grief, fear, or rage you have no other way to manage yet, that is not an improvement, whatever the symptom score says. Sensitivity here can be extreme — some people cannot tolerate an amount most prescribers would consider negligible.

This is the clearest argument for buprenorphine over an antagonist. It supplies opioid tone instead of removing it, so it may soften dissociative barriers without collapsing them. Some people who could tolerate no dose of naltrexone do well on buprenorphine below the smallest commercially available strength, which a clinician or compounding pharmacy has to prepare.

> **Worth discussing, not a protocol:** This dose-sensitivity relationship is a clinical observation, not a validated dosing rule, and there is no published conversion between the two medications. Raise it as a question, not as a plan. A fuller discussion is in the clinician guide at bpd.fyi/resources, which you are welcome to bring to your appointment. And if you do trial naltrexone first, the washout described above is mandatory before starting buprenorphine.

A Note on Stigma

You may feel uncomfortable about taking an opioid medication. You may worry about what others will think, or about what it means about you. These feelings are understandable, and they deserve to be taken seriously.

Here is what is true: your brain has a system that regulates how you experience connection, loss, and social pain. There is evidence that this system may not be functioning the way it should. A medication exists that may help correct that.

It is worth noticing what the discomfort is actually made of. Much of the stigma attached to buprenorphine is not really about the drug. It is inherited from how our culture treats people with addictions, who are widely assumed to have chosen their situation and to deserve what follows from it. If part of your unease is a wish not to be mistaken for one of those people, that is understandable, and it is also worth examining. They are not being treated fairly either, and a version of your argument that works by distancing yourself from them is weaker than one that does not need to.

You are not weak for considering this. You are not broken for needing it. You are making an informed decision about a biological system in your body, in collaboration with a clinician who is paying attention to the science.

For More Information

- **Full clinical guide** (written for your clinician): bpd.fyi/resources

- **If you dissociate**, or you're plural, there is a longer piece on why antagonists and buprenorphine differ here: bpd.fyi/posts/did-and-bpd
- **Contact**: contact@bpd.fyi

The studies named in this guide, if you want to hand a citation to a clinician:

Yovell Y, et al. *Am J Psychiatry* 2016;173:491–498. doi:10.1176/appi.ajp.2015.15040535

Tucciarone JM, et al. *Am J Psychiatry* 2026;183:409–419. doi:10.1176/appi.ajp.20250840

Hansen B, Inch KM, Kaschor BA. *Borderline Personal Disord Emot Dysregul* 2022;9:9. doi:10.1186/s40479-022-00181-1

This document was prepared for patient education purposes. It does not constitute medical advice. All treatment decisions should be made in collaboration with a qualified clinician.