Low-Dose Buprenorphine for BPD
A Patient Companion Guide

Understanding an Emerging Treatment for Interpersonal and Emotional Dysregulation

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This document is a companion to the clinical guide *Low-Dose Buprenorphine in Severe Interpersonal and Affective Dysregulation: A Primary Care 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

Your clinician may have mentioned buprenorphine as a possible treatment for emotional and interpersonal difficulties associated with borderline personality disorder (BPD) or similar presentations. This is probably not what you expected to hear — buprenorphine is best known as a treatment for opioid addiction.

This guide explains why a medication associated with addiction treatment is being considered for a completely different reason, what the evidence looks like, what to expect if you try it, and what questions to ask your provider.

You are not being treated for addiction. The doses involved are much lower than those used in addiction medicine, and the target is a different system in your brain.

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 Bernhard 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 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 two 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. This is different from a full opioid, which would produce a surge and then a crash. Buprenorphine's "partial" activation means it raises the floor without creating dramatic peaks.
2. **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?

You deserve an honest answer. The evidence is promising but limited.

What exists

One randomized controlled trial (Yovell et al., 2015). This study gave very low doses of buprenorphine (0.1–0.2 mg, dissolving under the tongue) to people with severe suicidal thoughts. It worked — suicidal ideation dropped significantly compared to placebo. A subgroup of participants who had BPD showed an especially strong response. However, this subgroup analysis wasn't the main focus of the study, so it needs to be interpreted carefully.

One published case report (Kaschor, 2022). A patient with BPD who was not using opioids was started on buprenorphine 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.

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 typically 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.

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 taking 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. Your clinician is trying to avoid that.

How long before you notice something

Some people notice subtle shifts within the first days or weeks — often not dramatic, but a sense that reactions are slightly less intense or that recovery from emotional disturbances is somewhat faster. Others may need dose adjustments before noticing meaningful change.

An adequate trial is generally considered to be at least 8–12 weeks at a dose that seems right. If you haven't noticed benefit by then, discontinuation may be appropriate.

Side effects to know about

- **Nausea** — most common, especially if the dose is too high too fast. Usually manageable and often resolves.
- **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 — 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.
- **Drug screening and prescription monitoring database checks.** These are standard safety measures for any controlled substance prescription. They are not a sign that your clinician suspects you of misusing the medication — they are part of responsible prescribing.
- **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, your clinician will help you taper gradually to minimize withdrawal. Do not stop abruptly on your own.

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.

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?"

No. The dose range used for this purpose (typically 0.1–1 mg daily, sometimes higher for more severe presentations) is far below what's used for addiction treatment (8–24 mg daily). The target is different: this aims to stabilize the brain system involved in social bonding and emotional regulation, not to manage opioid cravings or withdrawal.

"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 taper off gradually 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?"

Naltrexone is an opioid *blocker*, which is the opposite of what buprenorphine does. **You absolutely cannot take naltrexone and buprenorphine at the same time** — combining them will cause an acute withdrawal reaction that is intensely unpleasant and potentially dangerous. If you are currently taking naltrexone in any form (including low-dose naltrexone/LDN), tell your clinician before starting buprenorphine. There must be a washout period between stopping naltrexone and 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. The fact that the same medication is also used for addiction does not make your use of it an addiction any more than taking insulin for diabetes makes you a substance user.

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
- **Contact**: contact@bpd.fyi

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.