DELAMERE

Breaking the cycle

What the research shows about ketamine addiction

Read our findings

Systematic review of neurobiological mechanisms and psychotherapeutic innovations

- **70** studies reviewed
- **30** key studies tabulated
- Journal of Addictive Diseases (2026)

1. Who is most at risk?

Adolescents and young adults: disproportionately affected, with neurodevelopmental vulnerability in prefrontal control systems.

Polysubstance users: more complex impairment and a 40% higher relapse rate than ketamine-only users.

Women: higher burden where trauma, anxiety, depression and PTSD drive self-medication.

Trauma, emotional distress and socio-economic stress commonly intensify risk.

2. What ketamine addiction does to the brain and body

Prefrontal cortex impairment:
Poorer planning, impulse control and decision-making; studies reported a 35% reduction in decision-making efficiency.

Prefrontal-limbic stress sensitivity and dysconnectivity:
Relapse risk. reduced self-regulation and stronger compulsive use.

Reward systems:
Become less responsive to natural rewards and more dependent on ketamine-linked reinforcement.

Amygdala and emotional dysregulation:
Bbout 70% of chronic users show dysregulated amygdala activity.

HPA-axis stress dysregulation:
Elevated cortisol and reduced HRV increase stress sensitivity and relapse risk.

Maladaptive neuroplasticity:
Lower BDNF and disrupted default mode/salience network connectivity undermine recovery.

3. What helps in psychotherapy?

CBT: improves cognitive flexibility and planning; one study found cravings reduced by **50%** within 12 weeks.

Mindfuless-based interventions: normalize amygdala and HPA-axis activity; HPA-axis normalization was seen in **80%** of participants and linked to a **40%** decrease in relapse.

Neurofeedback and digital tools: promising adjuncts for emotional regulation, access and engagement.

Trauma-focused therapies (including EMDR): reduce amygdala over-reactivity and trauma-driven use; one study reported a 60% reduction in trauma symptoms and 70% abstinence at 1 year.

4. Clinical take-home

Stabilise arousal and stress

Restore executive control

Process trauma when ready

Embed relapse-prevention skills

Best care is staged, trauma-informed and neurobiologically informed

Biomarkers such as BDNF, HRV and cortisol may help personalise timing and intensity of treatment.

Peak neuroplastic opportunity may occur around 2-4 weeks post-cessation.

Culturally responsive and accessible models are still needed.

Based on Lomas, C. (2026). Breaking the cycle: a systematic review of neurobiological mechanisms and psychotherapeutic innovations in ketamine addiction.