



Envisioning a Path Forward: A Critical Narrative Review of the Brain Disease Model of Addiction (BDMA) after Three Decades of Debate

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Author's contribution

The sole author designed, analysed, interpreted and prepared the manuscript.

Article Information

DOI: <https://doi.org/10.9734/indj/2026/v23i5583>

Open Peer Review History:

This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: <https://pr.sdiarticle5.com/review-history/165525>

Review Article

Received: 02/07/2026

Accepted: 01/09/2026

Published: 05/09/2026

Abstract

For nearly three decades, the Brain Disease Model of Addiction (BDMA) has strongly influenced scientific, clinical, and public understandings of addiction while remaining the focus of sustained debate. This critical narrative review reassesses the BDMA by integrating evidence from addiction neuroscience, genetics, neuroimaging, behavioural economics, recovery research, stigma studies, and philosophical critique. The review examines evidence supporting a neurobiological basis for addiction alongside concerns regarding causation, agency, responsibility, social context, treatment implications, and stigma. The synthesis indicates that much of the continuing disagreement reflects differing definitions of “brain disease” rather than wholly incompatible evidence. Neurobiological findings demonstrate consistent involvement of reward, motivation, learning, and control systems, but they do not by themselves establish that addiction can be fully explained at a biological level. Conversely, psychosocial and choice-based accounts do not negate the relevance of neurobiological mechanisms. The reviewed literature therefore supports an integrative interpretation in which addiction emerges from interactions among biological vulnerability, learning history, socioeconomic conditions, environmental contingencies, and individual agency. This perspective has practical implications for prevention and treatment, favouring approaches that combine mechanism-specific and precision-oriented interventions with psychosocial support, recovery resources, and attention to stigma and social disadvantage. The review concludes that future progress depends less on defending a single explanatory label than on clarifying the meaning and limits of the brain-disease concept and applying multiple levels of explanation to research, clinical care, and public health.

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Keywords: Addiction; brain disease model; neurobiology; substance use disorder; integrative treatment.

1. Introduction

Few ideas in the field of substance use research have been as influential and as contested as the claim that addiction is fundamentally a brain disease. Leshner (1997), then director of the U.S. National Institute on Drug Abuse (NIDA), published a short but hugely influential paper declaring that "addiction is a brain disease and it matters". That single sentence has remarkably shaped how addiction is diagnosed, how clinicians are trained, how research funding is allocated, and how the public talks about drug use for nearly three decades. Yet the Brain Disease Model of Addiction (BDMA) has not achieved unanimous agreement among scientists, practitioners and the general public. Almost thirty years after Leshner's article, this debate has entered a new and more evidence-based phase. The publication of an edited volume, "Evaluating the Brain Disease Model of Addiction" (Heather et al., 2022), marked a turning point in this debate, as it brought together neuroscientists, philosophers, clinicians, and legal scholars in a single collection to examine the BDMA from every angle. This review revisits the BDMA debate in light of that volume and the research published since and poses three connected questions: What does the most recent evidence actually show? What does this evolving debate mean for how we prevent, treat and study substance use disorders? And what would a scientifically honest, clinically useful "cure" for addiction actually require? To answer these questions, this review draws on a wide range of evidence, including large-scale brain imaging studies, genetics research, behavioural economics, international surveys, and studies of stigma. By bringing evidence from these different fields together, it also highlights just how interdisciplinary this debate has become.

Recent scholarship continues to show that addiction research spans neurodevelopmental variation, neurobiological mechanisms, recovery processes, and environmental influences, reinforcing the value of evaluating brain-based and contextual explanations together (Miller et al., 2024; Engeln & Ahmed, 2025). Related reviews likewise emphasise that treatment and conceptual models can incorporate contextual contingencies and interacting biological, psychological, and social factors rather than relying on a single explanatory level (Harry et al., 2024; Agaga et al., 2026).

This review adopts a narrative, critical review methodology rather than a systematic one. Relevant literature was identified through targeted searches of PubMed, PsycINFO, and Google Scholar using terms including "brain disease model of addiction", "addiction neuroscience", and "addiction stigma", supplemented by manual searches of the reference lists of key sources, particularly the 2022 edited volume "Evaluating the Brain Disease Model of Addiction" (Heather et al., 2022) and subsequent commentary responding to it through 2026. Sources were prioritised for inclusion based on citation influence within the field, direct engagement with the central disease versus choice debate, and recency, rather than through a pre-registered or exhaustive systematic protocol.

Research gap: However, the literature considered in this manuscript remains distributed across neurobiological, psychosocial, philosophical, stigma-related, and treatment-focused lines of inquiry, and these strands are not always evaluated within a single updated account of what the BDMA does and does not explain. A focused synthesis is therefore needed to clarify how these strands can be interpreted together without treating either neurobiological evidence or contextual influences as sufficient on their own.

Objective: Accordingly, the objective of this review is to reassess the BDMA in light of the evidence and debate accumulated since 2022 and to examine its implications for prevention, treatment, research, and the meaning of a scientifically honest and clinically useful "cure" for addiction.

2. The Case for a Brain-Based Account

The neurobiological case for the Brain Disease Model of Addiction (BDMA) rests on a genuinely large body of scientific evidence obtained from decades of research in animals and humans. A substantial body of evidence has converged on a shared brain circuit involved in reward and motivation associated with addiction, consisting of the ventral tegmental area, nucleus accumbens, amygdala and prefrontal cortex of the brain. This neural circuit appears to be affected by virtually every class of addictive substances and is also implicated in other behavioural addictions (Koob & Volkow, 2016). Koob and Le Moal (1997) provided one of the earliest and most influential accounts of how this circuit changes with repeated drug use by proposing that addiction develops through a spiral of "hedonic homeostatic dysregulation". In simple terms, the brain's reward system becomes progressively less sensitive to normal pleasures and more focused on the drug itself, and this drives the vicious cycle of escalating substance use. Around the same time, Robinson and Berridge (1993) proposed a related but distinct account known as the incentive sensitisation theory, in which they distinguished between "wanting" a drug and "liking" it. They argued that, over time, the brain's wanting system can become highly sensitised to drug-related cues and produce intense cravings that persist even when the drug itself no longer feels particularly pleasurable. This also helps explain why relapse often occurs even after withdrawal symptoms have long faded.

Volkow and Koob (2015) later proposed a three-stage model of addiction consisting of a binge and intoxication stage, a withdrawal and negative affect stage, and a preoccupation and anticipation stage. They argued that repeated cycling through these stages produces a mix of heightened craving, a blunted ability to feel everyday pleasure, and weakened self-control. Volkow et al. (2016) expanded this case a year later in a widely cited review, in which they argued that decades of neuroscience research had convincingly linked addiction to reproducible and measurable changes in brain structure and function. Everitt and Robbins (2016) added another important piece to this account by highlighting how drug use often shifts over time from a voluntary action to an ingrained habit and gradually to compulsive use, as control shifts from goal-directed brain systems to more automatic ones. Supporters of the BDMA argue that this growing body of evidence from neuroscience has translated into real and practical benefits. It has led to the development of overdose-reversal agents such as naloxone, as well as maintenance and anti-relapse medications such as buprenorphine, methadone, naltrexone, and acamprosate, alongside newer brain stimulation techniques such as transcranial magnetic stimulation. The model also played a role in the passage of the U.S. Mental Health Parity and Addiction Equity Act

(2008), which required insurers to cover addiction treatment (Volkow & Koob, 2015). Framing addiction as a brain disease rather than a moral failing or lack of willpower is also credited with reducing stigma and encouraging more people to seek treatment (Koob & Volkow, 2016).

More recent scientific work has tried to refine this neurobiological account rather than abandon it. Bogdan et al. (2023) proposed a model called the Genetically Informed Neurobiology of Addiction (GINA), which combines genetic risk data with circuit-level brain science and suggests that a person's likelihood of developing addiction reflects an interaction between their genes, environment, and the timing of brain development. Ray et al. (2023) took a related but more clinical approach and argued for a shift towards precision medicine in addiction treatment. By using detailed behavioural testing frameworks, such as the NIAAA's Addictions Neuroclinical Assessment developed by Kwako et al. (2016), researchers can now identify distinct subtypes of substance use disorders (SUDs). It is hoped that profiling distinct subtypes based on biological evidence will eventually allow treatments to be matched to the specific mechanisms driving a given person's addiction, rather than relying on a one-size-fits-all approach. Both of these efforts represent more cautious and individualised versions of the brain disease claim than Leshner's original sweeping declaration.

Stubbs et al. (2023) furthered the biological account by analysing a large collection of prior neuroimaging studies that used lesion- and connectivity-based network mapping. They found that 91 percent of the reported brain abnormalities converged on a single, shared brain network spanning the prefrontal cortex, insula, and striatum, regardless of which drug was involved. A related lesion-mapping study similarly found that brain injuries capable of curing addiction tend to localise to this same circuit, offering convergent evidence from an entirely different method (Joutsa et al., 2022). This is among the strongest evidence yet that addiction may share a common underlying brain signature across very different substances, though convergence on a shared circuit does not by itself establish that this pattern reflects a disease process rather than the neural signature of any strongly reinforced behaviour more generally, a distinction examined more closely in the next section. Heilig et al. (2021) offered a related defence of the model, arguing for what they called "consilience", a coming-together of biological and social explanations. While they acknowledged that many criticisms of the BDMA have real merit, they maintained that its basic premise that addiction has a genuine neurobiological basis remains sound and warned that dismissing this basis outright could reduce access to care for people who need it.

3. Criticisms of the Brain Disease Model

Despite the prominence of the BDMA, critical objections in the literature have gradually become more precise and considerably more varied. Some of the earliest and most direct challenges came from within neuroscience itself. Hall et al. (2015) argued that group differences seen in brain-imaging studies are often modest in size, easily confounded by other factors, and vulnerable to publication bias, since studies that find dramatic differences are more likely to be published than studies that do not. They also pointed out that the BDMA has not clearly delivered more effective treatments than other approaches. Around the same time, Davies (2018) raised a more philosophical objection, arguing that much of the debate rests on a confused mixing of two separate questions: whether addiction has a neural cause and whether people struggling with it can still be held responsible for their actions. He stated that trying to answer both questions with a single model was philosophically incoherent from the start. Field et al. (2019) raised a closely related objection and argued that reducing addiction to a brain disorder oversimplifies a condition that is shaped by learning and context at every stage. Other critiques have focused on what gets left out of a purely brain-based account. Levy (2013) argued that merely showing that a person's brain functions differently is not, by itself, enough to prove that they have a disease. He further argued that whether a difference counts as an impairment depends heavily on a person's surrounding context and opportunities, not on brain scans alone. Satel and Lilienfeld (2014, 2017) made a related point from a clinical perspective, arguing that focusing so heavily on the brain distracts from the actual reasons people use drugs in the first place, such as self-medicating psychological pain, coping with socioeconomic stress, or managing difficult relationships. Hart (2017) sharpened this critique further, arguing that treating addiction primarily as a brain disease can actually promote social injustice by drawing attention away from the poverty, discrimination, and lack of opportunity that so often accompany problematic drug use.

Heyman (2022) examined a large body of epidemiological data to highlight that a substantial proportion of people who meet the clinical criteria for a substance use disorder eventually stop using without any professional treatment at all, though the exact proportion is highly dependent on definition and substance type. For alcohol use disorder, approximately 70 percent of affected individuals show improvement without formal intervention (Tucker et al., 2020). Population-level data similarly indicate that 9.1 percent of US adults reported having resolved a substance addiction problem at some point, roughly half without using any formal services (Kelly et al., 2017). Remission rates calculated from treatment-seeking clinical samples, however, are considerably lower and slower to emerge (Fleury et al., 2016), underscoring that natural-recovery estimates depend heavily on whether a population or a clinical sample is being described. This pattern nonetheless sits awkwardly with the idea of addiction as a uniformly "chronic and relapsing" disease, since most chronic diseases do not resolve on their own without intervention. Hogarth (2022) offered a related but distinct argument, suggesting that the persistence of addiction is often better explained by socioeconomic deprivation shaping people's choices than by any automatic or compulsive brain process. He argued that this points towards a very practical implication: research funding might produce better outcomes if redirected towards addressing poverty and social disadvantage rather than searching for ever more detailed brain mechanisms. Some scholars have gone further and questioned not just the science but the social and political forces that keep the BDMA so dominant. Alexander (2022) and Hammersley (2022) both argued that the model's popularity, particularly in the United States, owes as much to cultural and geopolitical history as to scientific necessity. Leighton (2022) raised a more technical concern, arguing that it is a category error or mereological fallacy to describe brain regions as "wanting" or "choosing" something, since only whole persons can be said to want or choose. Maier (2021) proposed a genuinely different framing: rather than asking whether addiction is a disease, he argued that it should be understood as a disability, involving real impairment and often real discrimination, regardless of how the disease question is eventually settled. This reframing

could shift the conversation away from a dispute over labels and towards more concrete questions about accommodation and support.

Historians of science have similarly argued that the BDMA's dominance is attributable to institutional and political history, tracing its rise to specific funding and policy decisions at the U.S. National Institute on Drug Abuse from the 1990s onwards (Courtwright, 2010; Vrecko, 2010). Not every critic has argued for abandoning the disease framing of the BDMA altogether. Snoek (2017) proposed a middle path, suggesting that addiction might involve a temporary "duress stage" in which a person's agency is significantly compromised but not eliminated, avoiding the extremes of treating people as either fully in control or entirely powerless. Pickard (2022) made a related call for agnosticism, arguing that addiction is too heterogeneous a category for a single verdict on the disease question, since some presentations may closely resemble a disease process while others remain substantially voluntary and responsive to incentives. Pedrero Pérez (2022) argued that the field is overdue for what philosopher Thomas Kuhn would call a paradigm shift, a point at which a dominant framework has accumulated so many unresolved problems that it needs replacing rather than patching. These accounts suggest that the BDMA's continued dominance owes more to funding structures and institutional inertia than to its explanatory success. A further strand of critique has questioned whether even the most impressive recent neuroscience findings settle the debate. Shared brain circuitry across different addictive substances, as found by Stubbs et al. (2023), is certainly consistent with a single, common pathway of reward dysregulation. However, critics point out that it is equally consistent with a simpler explanation: any strongly reinforced behaviour, whether or not it involves drugs, would recruit similar neural machinery because that is how learning works in general (Lewis, 2022). In other words, finding a consistent brain pattern behind addiction does not automatically prove that the pattern is pathological in a medical sense, a distinction already raised by Levy (2013). This same distinction sits at the heart of O'Mahony's (2025) more recent argument, which suggests that treating brain-based evidence as self-evident proof of disease reflects an assumption that researchers have rarely examined closely, rather than a conclusion that the evidence actually forces on us.

Finally, some of the more organised opposition to the BDMA has taken shape as a genuine scientific movement rather than a collection of individual papers. Heather et al. (2018) describe how, after a group of addiction researchers wrote a joint letter to the journal *Nature* challenging its uncritical endorsement of the brain disease framing, 94 addiction scholars from around the world signed on. This led to the founding of the Addiction Theory Network, a formal international group dedicated to developing and promoting alternatives to the BDMA. Its existence is itself a kind of evidence that, regardless of how influential the BDMA remains, it has never approached the unanimous consensus that it is often portrayed as representing. Sociological critiques have gone further, arguing that framing addiction primarily as a brain disease also serves institutional interests, reinforcing biomedical authority and, in some accounts, diverting attention from the political economy of substance use (Campbell, 2012; Reinman & Granfield, 2014).

4. Recent Evidence Since 2022

The 2022 publication of the book "Evaluating the Brain Disease Model of Addiction" marked a significant turning point in the debate on the BDMA. Rather than simply adding another voice to either side, the volume brought neuroscientists, philosophers, clinicians, and legal scholars together in one place to systematically test the model's assumptions, both empirical and conceptual. Several studies published since have continued this more careful reappraisal. Ochterbeck et al. (2024) took a more empirical approach to a question that is often debated only in the abstract: what do working addiction researchers actually believe? They surveyed 190 addiction researchers across 29 countries and found a striking but contradictory pattern. Most researchers valued brain-based explanations of addiction because they felt these explanations improved treatment access and reduced stigma. Yet an equally large majority still believed that people with addiction bear real personal responsibility for their condition and their actions. In other words, scientists working in the field do not necessarily see brain disease and personal responsibility as contradictory positions, even though much of the philosophical literature treats these two views as being in tension.

Liu et al. (2023) took a more direct empirical approach, using a meta-regression analysis to compare brain-activity measurements in people with addiction against those with recognised neurological diseases. Their results offered only mixed support, rather than a clean confirmation, for the idea that addiction closely resembles classic neurological conditions. More recently, Blithikioti et al. (2025) reframed the debate by identifying a basic, long-overlooked ambiguity: researchers on both sides have often meant different things by the phrase "brain disease" without realising it. Under a narrow definition, a condition only counts as a brain disease if it involves damage similar to well-established neurological diseases, such as visible structural injury. Under a broader definition, almost any psychological disorder would qualify, since virtually all mental processes have some basis in the brain. Much of the evidence discussed in this review, including the network-convergence findings from Stubbs et al. (2023), falls short of the narrow bar but looks more than sufficient under the broad one. Because proponents and critics have often been talking past each other using different implicit definitions, Blithikioti et al. (2025) argued that clarifying these terms is a necessary first step before the empirical debate can make further progress, arguably one of the most useful reframings that the thirty-year debate has received. This review takes the position that the broad definition is the more scientifically defensible starting point, since restricting "brain disease" only to conditions involving visible structural injury would exclude many uncontroversially biological psychiatric conditions altogether. The more productive question is therefore not whether addiction qualifies as a brain disease under either definition, but what follows from adopting the broad one: diagnostic frameworks, funding decisions, and public health messaging should stop treating the absence of visible structural damage as evidence against a genuine disorder, a standard rarely applied with the same rigour to other recognised psychiatric conditions. Complementing this, O'Mahony's (2025) analysis introduced the idea of epistemic injustice into the discussion, essentially asking whose knowledge and experience get taken seriously in this debate. He argued that treating the BDMA as a necessary tool for reducing stigma or expanding treatment access overstates its actual role, since many countries have successfully expanded access to addiction treatment without ever adopting a brain-disease framing at all. A parallel philosophical

critique has questioned whether the entire brain disease versus choice framing is itself the wrong starting point, arguing that this binary obscures more than it clarifies about how addiction actually develops (Goldberg, 2020).

5. Implications For Prevention and Treatment

This evolving debate is not merely an academic exercise. It has direct, practical consequences for how limited prevention and treatment resources are allocated. A strict version of the BDMA has historically favoured biomedical solutions, such as anti-craving medications, brain stimulation devices, and the search for biological markers of addiction. This emphasis has sometimes come at the expense of psychosocial and structural interventions that have strong evidence of population-level impact, such as taxation of addictive substances, restrictions on marketing, and regulations on access (Hall et al., 2015). Some newer models have attempted to bridge this gap directly. Acuff et al.'s (2023) contextualised reinforcer pathology model offers one promising approach. It shows that, across many different types of studies, increasing the availability and appeal of substance-free alternatives, such as stable employment, supportive relationships, and enjoyable recreational activities, reliably reduces substance use. This model is useful precisely because it works at both levels at once: it fits comfortably within a neurobiological account of reward while also taking behavioural and environmental factors seriously. Heilig et al. (2016) made a related point from a more purely neuroscientific angle, arguing that, for far too long, addiction neuroscience has ignored one of the most common features of real-world addiction: progressive social isolation and exclusion. They proposed that building social context directly into neuroscience research, rather than treating it as an afterthought, would make the resulting science far more clinically useful.

Gordon et al. (2024) recently highlighted a further, more hopeful development: newer, rapid-acting treatments such as psilocybin, ketamine, and ibogaine are showing early promise for reducing craving. However, they were careful to note that even if a medication can rapidly relieve craving, that relief still needs to be paired with behavioural interventions for the benefits to last. This underscores a point that runs through much of the reviewed literature: no purely biological “cure” is likely to succeed on its own when separated from a person's psychological and social context. For counselling and clinical practice specifically, the safest reading of the current evidence is an integrative one, rather than a decision to fully embrace or fully reject the brain disease framing. Clinicians can reasonably continue to use brain-based psychoeducation to help reduce a client's self-blame and shame while still avoiding the fatalistic message that recovery must wait for some future neurobiological breakthrough. Given that most people with a substance use disorder recover without formal treatment at all (Heyman, 2022), interventions that build a person's sense of agency, self-efficacy, and access to alternative sources of reward deserve at least as much attention as pharmacological approaches. Prevention efforts, meanwhile, may be better served by addressing the socioeconomic and developmental risk factors highlighted by both the GINA model (Bogdan et al., 2023) and the reinforcer pathology model (Acuff et al., 2023), rather than focusing narrowly on neurobiological vulnerability alone. This same logic underlies the growing recovery capital framework, which treats a person's social, physical, and psychological resources as central, measurable determinants of recovery on a par with any biological factor (Best & Hennessy, 2022).

The stigma-reduction argument for the BDMA also deserves more scrutiny than it typically receives in everyday clinical settings. Kelly et al.'s (2021) national randomised study found a more nuanced trade-off than a simple win or loss for the brain-disease framing. The term “chronically relapsing brain disease” produced the lowest attributions of personal blame of any label tested and increased perceived need for continuing care, but it also lowered prognostic optimism and increased perceived dangerousness relative to describing the same behaviour as a “problem”. No single label optimised every outcome simultaneously. This is consistent with Pennington et al.'s (2023) study, which similarly found that a brain-disease label reduced blame attributions while simultaneously raising attributions of genetic blame. Together, these findings suggest that whatever stigma-reduction benefits the brain-disease framing offers come with real trade-offs and depend heavily on which specific outcome is being measured, rather than indicating that the framing is straightforwardly effective or ineffective, as the broader BDMA literature sometimes assumes. For counsellors and prevention practitioners, the practical lesson is that deliberate anti-stigma programming, rather than brain-disease messaging, is likely the more reliable way to reduce stigma. Newer clinical research has extended this question directly into the therapy room, finding that whether brain-disease language helps or harms a client's confidence in their own capacity for recovery appears to depend heavily on how and when it is introduced in treatment (Casey et al., 2026).

6. Conclusion

Envisioning a genuine “cure” for addiction requires moving past the either/or framing that has shaped this debate since 1997. The most recent scholarship converges on a shared insight: addiction is neither purely a brain disease nor simply a matter of choice, but a condition shaped by multiple interacting factors. These include genetic vulnerability, changes in brain function, personal learning history, socioeconomic context, and individual agency, all interacting dynamically over the course of a person's life (Bogdan et al., 2023; Blithikioti et al., 2025). A useful way of thinking about this comes from Wiers and Verschure (2021), who argue that the field should move away from what they call the “broken brain” model, in which addiction is treated as damage to be fixed in isolation, and towards a systems-level model instead. In their framework, the addicted brain is understood as embedded within a person's physical and social environment, meaning that effective treatment needs to address a person's goals, self-image, and environment together with their brain chemistry, rather than targeting the brain alone. Chinchella and Hipólito (2025) arrive at a similar conclusion from a very different, more philosophical direction. Drawing on the philosopher Maurice Merleau-Ponty's distinction between the biological body and the lived, experienced body, they argue that a purely disease-focused model captures only part of what addiction actually involves. They propose shifting from a narrowly “cure-oriented” approach towards what they call a “care-oriented” one, which takes the individual's emotional and environmental dimensions seriously, not just what shows up on a brain scan. Legal and clinical ethics scholars have raised parallel points from their own disciplines: how a society settles the disease question directly shapes questions of criminal responsibility (Morse, 2022) and the ethical obligations owed to people in treatment (Rotgers, 2022).

An overall examination of the established and newly emerging literature suggests that a scientifically honest and clinically useful model of addiction should treat neurobiology as necessary but not sufficient on its own. Brain science can explain the mechanisms behind addiction, but mechanism is not the same as meaning, and understanding a mechanism alone has rarely been enough to produce lasting behaviour change by itself. The most promising path forward likely combines several elements: precision-medicine frameworks that group people by the specific mechanisms and context driving their addiction (Ray et al., 2023), interventions that work on both neurobiological and environmental levers at the same time, such as reinforcer-based approaches (Acuff et al., 2023), and far greater conceptual clarity about what the word 'disease' is actually doing whenever it appears in a given clinical or policy argument, building on the case made above for explicitly adopting the broader definition (Blithikioti et al., 2025). Ultimately, the lasting value of the BDMA controversy lies less in finally settling whether addiction "is" a brain disease and more in what the argument has forced researchers and clinicians to clarify along the way: effective prevention and treatment must operate on biological, psychological, and social terrains all at once. That integrated understanding, rather than any single neurobiological breakthrough, is the most defensible vision of a "cure" that the current evidence actually supports. For counselling practice in particular, this means resisting both extremes on offer in the literature: the belief that neurobiological explanation alone will eventually deliver a pharmacological cure and the opposing belief that biology is largely irrelevant and only psychological or social factors matter. The breadth of evidence examined in this review points towards a more pluralistic conclusion. Durable recovery seems to be best supported by interventions that work on multiple levels simultaneously. Counsellors, clinicians, and policymakers are likely to be better served by embracing this pluralistic reading of the evidence than by pledging allegiance to either side of a debate that has transformed and grown in the thirty years since its inception.

This review is subject to certain limitations. As a narrative and critical review rather than a systematic review, it did not follow a pre-registered search protocol or formal quality appraisal, and the methodological details and limitations of individual cited studies were not examined in depth. Several avenues for future research follow from this discussion: longitudinal designs are needed to clarify whether observed brain differences precede or result from addiction; empirical trials directly testing precision-medicine (Ray et al., 2023) and reinforcer-based (Acuff et al., 2023) approaches, ideally in combination, would help establish whether integrative interventions outperform single-lens approaches in practice; and further work is needed on the durability of rapid-acting pharmacological treatments when paired with psychosocial follow-up (Gordon et al., 2024), on recovery capital as an interventional target in its own right (Best & Hennessy, 2022), and on cross-cultural generalisability, given that much of this debate has so far been conducted primarily within North American and European contexts.

7. Limitation

This review is limited by its narrative and critical design. Consequently, study selection may reflect the review's thematic focus, and the methodological limitations of individual sources were not examined in equal depth. The literature considered also largely reflects debates conducted in North American and European contexts, which may limit broader generalisability. These limitations should be considered when interpreting the review's conclusions and proposed integrative direction.

Consent and Ethical Approval

It is not applicable.

Declaration of AI Use

This manuscript was prepared through the intellectual contributions of the author(s) to the study design, data analysis, interpretation of results, and scholarly content. Grammarly and ChatGPT were used solely to assist with grammatical refinement and reference formatting during manuscript revision. The author(s) accept full responsibility for the accuracy, integrity, and final content of the manuscript.

Competing Interests

Author has declared that they have no known competing financial interests OR non-financial interests OR personal relationships that could have appeared to influence the work reported in this paper.

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