Addiction as a Disorder of Connection: An Interdisciplinary Synthesis of Neuroscience, Trauma, and Social Capital

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Abstract

Background: The neurobiological disease model of addiction has produced important advances in understanding compulsive substance use, but it systematically fails to account for several empirical anomalies: high rates of natural recovery without formal treatment, the graded dose-response relationship between relational trauma and addiction risk, and the dramatic effect of social environment on substance self-administration demonstrated in animal models.

Objective: This review synthesises convergent evidence across five independent domains—neuroscience, trauma research, social epidemiology, environmental and naturalistic evidence, and treatment outcome science—to argue that addiction is fundamentally a disorder of connection, emerging as an adaptive response to chronic relational deprivation.

Methods: Narrative, non-systematic synthesis of peer-reviewed literature retrieved from PubMed, covering the neuroscience of social reward and allostatic dysregulation, adverse childhood experiences (ACE) research, social capital epidemiology, environmental models of addiction, and evidence-based recovery interventions.

Results: Five independent evidence streams converge on a shared conclusion: (1) drugs of abuse activate the same mesolimbic circuits evolved for social bonding; (2) childhood relational trauma predicts addiction severity in a dose-response relationship; (3) social capital deficits predict overdose mortality at the population level; (4) environmental and naturalistic evidence demonstrates that social context causally moderates compulsive use; and (5) the strongest predictors of recovery—therapeutic alliance, mutual-help group membership, peer support, and recovery capital—are all relational in nature.

Discussion: We propose a *Relational Deprivation Model* of addiction that extends rather than replaces the disease model. The policy implications are substantial: effective addiction treatment must address relational repair alongside pharmacology and behaviour change.

Keywords: addiction; social connection; adverse childhood experiences; social capital; attachment theory; relational deprivation; recovery capital; trauma-informed care

1. Introduction: The Limits of the Disease Model

The dominant biomedical model of addiction—consolidated across landmark publications over the past three decades (McLellan et al., 2000; Volkow and Blanco, 2023)—positions substance use disorder (SUD) as a chronic brain disease characterised by compulsive drug seeking, loss of control over use, and persistent neuroadaptation in reward, stress, and executive function circuits. This framework has been scientifically productive: it shifted the clinical response to addiction away from moral condemnation toward medical treatment, generated effective pharmacotherapies for opioid, alcohol, and nicotine use disorders, and produced detailed mechanistic accounts of how repeated substance exposure alters dopaminergic signalling, prefrontal cortical regulation, and amygdalar stress reactivity (Koob and Volkow, 2010; Volkow and Blanco, 2023).

Yet the disease model, in its standard form, faces three empirical anomalies that it cannot adequately explain.

The first is natural recovery. Natural recovery without formal treatment is far more common than the disease model predicts: large-scale epidemiological reviews document that substantial proportions of individuals meeting lifetime SUD criteria achieve sustained remission as their social circumstances stabilise, often in their late twenties and early thirties (Volkow and Blanco, 2023). If addiction were primarily a progressive neurobiological disease analogous to cancer or type 2 diabetes, spontaneous remission at this rate would be unexpected. What commonly changes in these individuals is not their neurochemistry but their social context: they form stable partnerships, secure employment, assume caregiving roles, and become embedded in communities of accountability. The transition is relational.

The second anomaly is the ACE dose-response. Felitti et al. (1998) documented in 9,508 adults that each additional category of adverse childhood experience (ACE)—abuse, neglect, household violence, parental loss—confers a graded, multiplicative increase in addiction risk. Individuals with four or more ACEs face a 4- to 12-fold increased risk of alcoholism and drug abuse. Dube

et al. (2003) extended this finding to illicit drug use, demonstrating that ACE score alone accounts for up to 67% of the attributable population risk for parenteral drug use. These are not random neurological injuries. They are specifically relational wounds: betrayals by caregivers, witnessed violence between attachment figures, abandonment by those responsible for protection. That they predict addiction so powerfully suggests that addiction is tracking relational rupture, not merely neurobiological vulnerability.

The third anomaly is the Rat Park finding. Alexander et al. (1978) demonstrated that rats provided with social housing and enriched environments self-administered morphine at dramatically lower rates than identically treated rats in isolated standard cages—even after the isolated rats had already established patterns of heavy use. The variable was not pharmacology, genetics, or dose: it was the social environment. The same substance, in the same species, produced compulsive use or casual use depending on whether the animal had access to social reward.

These three anomalies point toward a common missing variable: relational disconnection. This review argues that addiction is best understood as an adaptive response to chronic relational deprivation—the nervous system's attempt to manage unbearable pain in the absence of genuine connection. Recovery, on this account, is not the removal of the solution but the provision of genuine belonging. This argument does not require abandoning the disease model; it requires extending it. Neurobiological vulnerability is real, genetic heritability contributes substantially to risk, and pharmacological dependence is a genuine physical phenomenon. The claim here is complementary: that the most powerful risk factor for addiction, and the most powerful predictor of recovery, is relational.

The sections that follow develop this argument across five independent evidence domains: the neuroscience of social bonding and its overlap with addiction circuitry (Section 2), the trauma and attachment literature establishing the developmental pathway from relational rupture to substance use (Section 3), the social epidemiology of isolation and its relationship to overdose mortality (Section 4), animal and naturalistic human evidence demonstrating the causal role of social environment

(Section 5), and the treatment outcome literature showing that relational factors drive recovery (Section 6). Section 7 synthesises these into the Relational Deprivation Model and addresses major objections.

2. Neuroscience of Belonging: Shared Circuits and Allostatic Load

2.1. Drugs of Abuse Hijack an Ancient Social Bonding Circuit

The neural circuitry that mediates social attachment in mammals is the same circuit that drugs of abuse activate and dysregulate. Insel (2003) argued that social attachment is, at its neurobiological foundation, an "addictive" process: oxytocin and vasopressin activate the mesolimbic dopamine system in response to social cues, creating the same reinforcement architecture that underlies drug addiction. From this perspective, drugs are not foreign invaders of the brain—they are pharmacological shortcuts to a reward circuit that evolution built for social connection.

Burkett and Young (2012) extended this analysis across species, demonstrating that social attachment and drug addiction share mesolimbic dopamine, endogenous opioid, and corticotropin-releasing factor (CRF) circuitry. In species that form selective social bonds, the same opioid receptors that respond to attachment-related stimuli also respond to opioid drugs, and disruption of these receptors impairs both bonding and withdrawal equivalently. Borland (2025) provided a comprehensive mapping of nucleus accumbens (NAc) signalling, confirming that oxytocin, mu-opioid receptors, and endocannabinoids positively regulate rewarding social behaviours—affiliation, pair bonding, play—via the same NAc circuitry disrupted by chronic drug exposure. The kappa-opioid system, which negatively regulates social reward, is the same system recruited in drug withdrawal to produce dysphoria (Koob and Le Moal, 2008).

The clinical implication of this convergence was stated precisely by Alcaro and Panksepp (2011), who documented that the mesolimbic dopamine SEEKING system—phylogenetically ancient, evolved for approach toward food, safety, and social contact—is pathologically reorganised around

drug-specific memories in addiction, depleting the motivational resources needed for social reward. Zellner et al. (2011) framed this in terms that should restructure clinical assessment: what addicts "really want," at the level of affective neuroscience, is restoration of the social-emotional connection that SEEKING circuits were designed to pursue. Substances are not chosen over connection; they are chosen in the absence of it.

2.2. Allostatic Load: From Social Deprivation to Neurobiological Vulnerability

If the first argument is that drugs substitute for absent social reward, the second is that chronic social deprivation creates the neurobiological terrain in which this substitution becomes necessary. Koob and Le Moal (2001) developed the allostatic model of addiction in its initial form, and Koob and Le Moal (2008) elaborated it: repeated drug exposure progressively dysregulates the brain's reward set point, recruiting anti-reward systems—CRF, dynorphin, norepinephrine—that produce a persistent state of anhedonia, dysphoria, and stress sensitivity between drug exposures. Withdrawal is not merely physical discomfort; it is a state of emotional pain closely analogous to social separation distress.

The crucial extension is that chronic relational deprivation activates the same anti-reward systems through a different entry point. George et al. (2012) provided evidence consistent with the view that within-system (dopamine downregulation) and between-system (CRF upregulation) neuroadaptations accumulate during repeated stress exposure, creating a neurobiological vulnerability state that closely resembles that produced by repeated drug withdrawal. Koob and Schulkin (2019) confirmed that stress-driven CRF dysregulation operates across all three stages of the addiction cycle and parallels the neurobiological cost of chronic social isolation.

This means that individuals who experience chronic disconnection—through childhood trauma, poverty-driven isolation, or community disintegration—arrive at first drug exposure with a reward system already in deficit. Substances do not create their vulnerability; they exploit a vulnerability that social deprivation created first.

2.3. Social Reward Sensitivity as a Developmental Vulnerability Window

Rademacher et al. (2017) confirmed that the dopaminergic reward system processes social stimuli analogously to nonsocial rewards, with oxytocin activating dopaminergic reward pathways specifically in response to social cues. Disruption of this system is a shared vulnerability across addiction and multiple developmental and psychiatric conditions. Beard et al. (2022) synthesised fMRI evidence across 28 studies showing that substance use positively correlates with ventral striatal social reward activity and negatively with amygdalar social-stress responses during adolescence—the critical window during which social reward circuitry is being calibrated. This bidirectional neural link between social reward sensitivity and addiction vulnerability in adolescence suggests that an adolescent who uses substances heavily is often one whose social reward circuits are poorly supported: overstimulated by social pain and understimulated by genuine social belonging.

The therapeutic implication is direct: interventions that restore the experience of genuine social reward—through therapeutic relationship, peer community, and meaningful belonging—are not merely supportive adjuncts to pharmacological treatment. They are targeting the specific neurobiological deficit that substances are compensating for.

3. Trauma as the Missing Variable: ACEs, Complex PTSD, and the Developmental Arc

Relational trauma occupies the central mechanistic role linking adverse childhood experience to adult addiction vulnerability. Post-traumatic stress disorder (PTSD)—and especially its complex form (C-PTSD)—represents the clinical expression of this linkage, mediating the pathway from childhood relational rupture to neurobiological dysregulation and compulsive substance use. The subsections below trace this developmental pathway from dose to disorder.

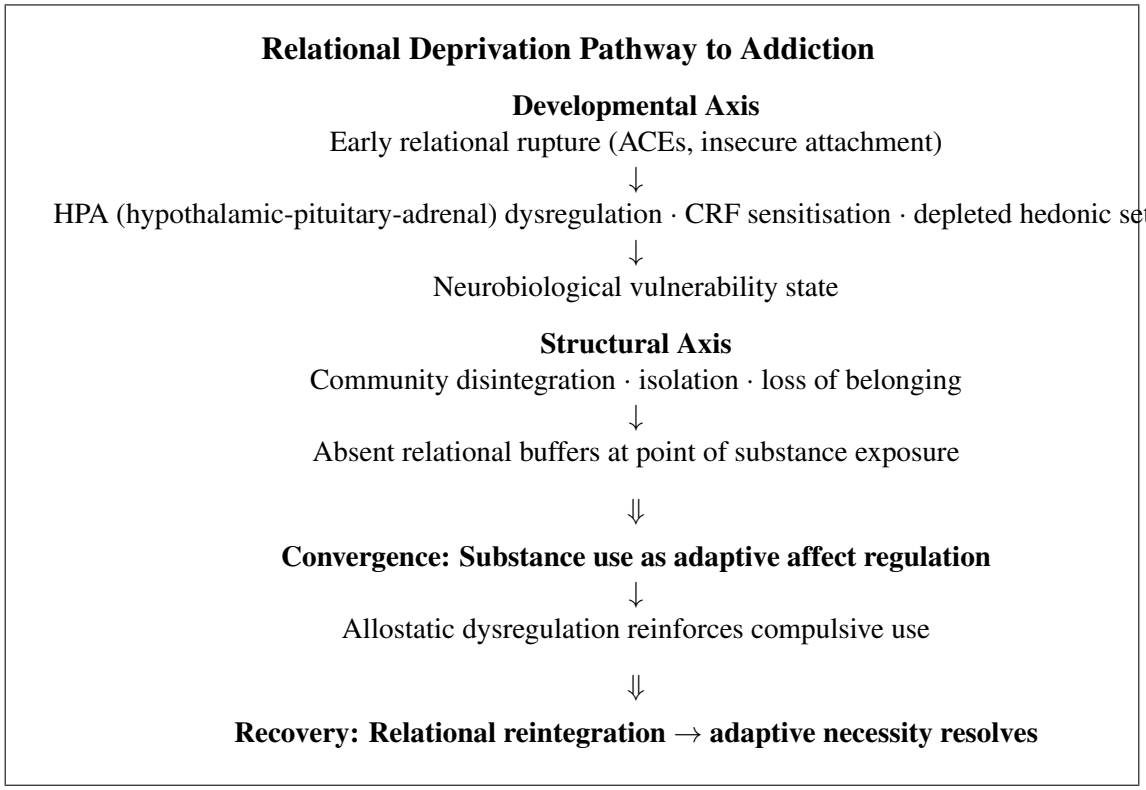

Figure 1: Conceptual model of the Relational Deprivation pathway. The developmental axis (early relational rupture producing neurobiological vulnerability) and the structural axis (community disintegration removing relational buffers) converge to create conditions under which substances function as adaptive affect regulators. Recovery is represented as the provision of genuine relational belonging that removes the adaptive necessity of substance use.

3.1. The ACE Study: Relational Betrayal as the Dose

The Adverse Childhood Experiences (ACE) Study, initiated by Felitti et al. (1998) in 9,508 adults, established one of the most replicated dose-response relationships in public health: each additional category of ACE confers a graded, multiplicative increase in risk for addiction, depression, cardiovascular disease, and early mortality. For alcoholism, those with four or more ACE categories faced a 4- to 12-fold increased risk compared to those with no ACEs. Dube et al. (2003) extended this to illicit drug use across 8,613 adults, finding that ACE score accounts for up to 67% of the attributable population risk for parenteral drug use. Anda et al. (2006) synthesised findings across 17,337 adults, documenting a graded relationship between ACE score and 18 health outcomes, linking cumulative childhood stress to broad neurodevelopmental impairment.

What is critical to recognise is that ACEs are not random misfortunes. They are specifically relational betrayals: sexual, physical, or emotional abuse by caregivers; neglect by those responsible for protection; exposure to domestic violence between attachment figures; parental substance abuse or mental illness that renders attachment figures emotionally unavailable. The dose-response between ACEs and addiction is therefore a proxy measure of accumulated relational rupture.

Recent meta-analyses confirm these relationships in contemporary samples. Among people with opioid use disorder, Santo et al. (2021) found pooled childhood maltreatment prevalence of 38–43% across 62 studies ($N = 21{,}871$)—roughly three to four times the population baseline. Rakovski et al. (2024) synthesised 58 studies ($N = 170{,}749$) finding that unspecified childhood maltreatment increased alcohol use risk approximately four-fold in youth, with 9 of 10 specific maltreatment-substance combinations showing significant associations.

3.2. Complex PTSD: The Relational Wound as Clinical Entity

Judith Herman's foundational theoretical contribution was to identify disconnection—from self, from others, and from meaning—as the central injury of trauma, not the traumatic event itself (Herman, 1992). This framing anticipates the ICD-11 categorisation of Complex PTSD (C-PTSD),

which codifies three additional disturbance clusters beyond standard PTSD: affect dysregulation, negative self-concept, and disturbances in relationships. All three are explicitly relational.

Harvey et al. (2025) confirmed in a substance use treatment sample ($N = 72$) that C-PTSD was substantially more prevalent than PTSD alone (34.8% vs. 5.8%), and that the severity of each C-PTSD cluster was significantly and positively associated with psychological dependence—a pattern not found for standard PTSD. Patel et al. (2025) found in a meta-analysis that individuals with comorbid PTSD and SUD showed 18–24 percentage points higher prevalence of childhood complex trauma than those with either condition alone. Kirsch et al. (2025) synthesised evidence showing that early life stress drives lasting HPA-axis dysregulation, altered stress-reward-executive circuitry, and comorbid mood disorders—neurobiological pathways that produce earlier SUD onset, greater relapse risk, and treatment resistance.

These findings establish that the trauma-addiction connection is not merely correlational. It operates through specific mechanisms—affect dysregulation, impaired stress response, negative self-concept, and relational disturbance—each compounding the others and creating conditions under which substances function as the most immediately available means of self-regulation.

3.3. Attachment Theory: The Developmental Architecture of Connection Failure

John Bowlby's attachment theory (Bowlby, 1982) provides the developmental architecture that links early relational experience to adult vulnerability. Children with insecure or disorganised attachment develop internal working models of unavailability, unpredictability, or danger—models that generalise to shape the individual's expectations of all subsequent relationships, including therapeutic ones. Schindler (2019) reviewed 34 cross-sectional and 3 longitudinal studies along with 1 meta-analysis, finding consistent confirmation that insecure attachment is associated with substance use disorder across all study designs, and that the relationship is bidirectional: insecure attachment predicts substance use, while ongoing substance use further impairs attachment capacity, creating an escalating cycle. Unterrainer et al. (2018) synthesised 12 studies finding significant

relationships between SUD and insecure attachment in 10 of 12 studies, specifically confirming fearful-avoidant attachment patterns in heroin-dependent individuals.

Maunder and Hunter (2001) synthesised the MEDLINE literature to propose a three-mechanism model of how insecure attachment increases disease risk: through heightened stress susceptibility, through reliance on external affect-regulators including substances, and through altered help-seeking behaviour that reduces access to care. All three mechanisms are expressions of the same underlying deficit: the absence of a reliable internal model of connection.

4. Social Capital, Isolation, and Structural Disconnection

4.1. Social Capital as a Population-Level Protective Factor

Robert Putnam's social capital framework (Putnam, 2000) identified civic participation, social trust, and community network density as key determinants of public health outcomes. At the population level, the prediction of the connection-disorder thesis is direct: communities with higher social capital should show lower addiction and overdose rates, independent of income, and the data support this prediction.

Zoorob and Salemi (2017) conducted an ecological study of 49,664 county-years across the United States (1999–2014) and found that counties in the highest quintile of social capital were 83% less likely to fall in the high-overdose mortality category and 75% less likely to fall in the moderate-overdose category compared to counties in the lowest quintile ($p < 0.01$). This effect was not explained by poverty, healthcare access, or urbanicity. Kawachi et al. (1997) established an earlier structural correlate: across 39 U.S. states, lack of social trust correlated $r = 0.76$ with income inequality and independently predicted age-standardised mortality across multiple causes. Kawachi and Berkman (2001) subsequently documented that social network deficits predict psychological distress through both main-effect and stress-buffering pathways—the same affective states that substances are used to manage.

4.2. Loneliness as a Physiological State

Loneliness is not simply a subjective feeling; it is a measurable physiological state with distinct neuroendocrine, inflammatory, and sleep-architecture signatures. Hawkley and Cacioppo (2010) documented that loneliness triggers implicit hypervigilance for social threat, disrupts HPA axis regulation and immune function, and independently predicts increased morbidity and mortality. A "loneliness regulatory loop" explains how cognitive biases toward threat interpretation perpetuate social disconnection and drive health-damaging coping behaviours including substance use.

Holt-Lunstad et al. (2015) provided the definitive meta-analytic evidence, demonstrating across 148 prospective studies that social isolation increases all-cause mortality odds by 29% (odds ratio [OR] = 1.29), loneliness by 26% (OR = 1.26), and living alone by 32% (OR = 1.32). These effect sizes are comparable to smoking as a mortality risk factor. Masi et al. (2011) found in an intervention meta-analysis that addressing maladaptive social cognition—the internal working model of social threat—produced the largest effect sizes in loneliness-reduction trials, confirming that perceived rather than merely objective isolation drives the health risk.

Among older adults, Zhang et al. (2025) found in a multilevel analysis ($N = 2{,}774$) that individual-level loneliness independently predicted substance misuse, tobacco use, and binge drinking, while neighbourhood belonging was negatively associated with all three behaviours. Shield et al. (2022) exploited the COVID-19 pandemic as a natural experiment, documenting in a national Canadian survey ($N = 5{,}892$) that loneliness co-mediated the relationship between pandemic stress and increased alcohol consumption—directly linking acute social disruption to increased substance use in a general population.

4.3. Deaths of Despair: Structural Disconnection at Scale

The geography of the opioid and addiction crisis is not random. Case and Deaton (2015) documented that all-cause mortality for middle-aged white non-Hispanic Americans reversed decades of decline between 1999 and 2013, driven primarily by drug and alcohol poisonings, suicide, and chronic liver

disease—a pattern unique among wealthy nations. This reversal was concentrated in people without college education: precisely the population that had experienced the most severe deindustrialisation and erosion of the social institutions—stable employment, union membership, civic organisations, religious communities—that had historically provided belonging and purpose.

Yeung et al. (2026) confirmed that deaths of despair rose from 28.60 per 100,000 in 1999 to 71.99 per 100,000 in 2023—a 151% increase—with drug-related fatalities contributing approximately 75% of the increase after 2014. The pattern is a population-level expression of the same dynamic documented at the individual level: when the relational structures that confer belonging, purpose, and identity are removed, addictive and suicidal behaviour rises as adaptation to unbearable disconnection.

5. The Rat Park Experiments and the Causal Role of Social Environment

5.1. Alexander et al. (1978): Social Context as the Primary Determinant of Compulsive Use

Alexander et al. (1978) conducted a series of experiments—the Rat Park studies—that remain among the most influential proof-of-concept demonstrations that social environment causally determines patterns of substance use. Rats given social housing in a large, enriched environment (Rat Park) and rats held in isolated standard laboratory cages were both provided with access to morphine solution and plain water, following an initial forced-exposure period, then observed in free-choice conditions. The isolated rats consumed significantly more morphine throughout the choice period and increased their consumption during experimentally induced escalation cycles. The socially housed animals, by contrast, did not show the same pattern of escalating consumption during those cycles. Critically, isolated rats already dependent on morphine who were transferred to Rat Park conditions voluntarily reduced their consumption—demonstrating that the effect was environmentally modulated and not irreversible.

This finding directly challenges the pharmacological determinism implicit in early addiction models,

which held that addictive substances were intrinsically compulsion-inducing regardless of context. Rat Park showed that social environment—specifically the presence or absence of social reward—was the dominant variable. The implication is not that pharmacology is irrelevant but that it operates in a relational context that determines whether exposure transitions to compulsive use.

Several methodological limitations of the Rat Park paradigm warrant explicit acknowledgement. First, as an animal model, it does not map directly onto human experience: rat social behaviour, reward circuitry, and the subjective meaning of belonging differ in important ways from human sociality. The forced-exposure method (morphine provided as the sole drinking fluid during the initiation phase) also created abnormally high baseline exposure that may not reflect typical human initiation patterns. Second, replication of the original findings has been inconsistent; some subsequent rodent studies have failed to reproduce the magnitude of the environmental effect, particularly with drugs other than opioids or under different housing protocols. Third, rodent models cannot capture distinctively human dimensions of addiction—narrative meaning, identity, stigma, or the role of language and culture in shaping substance use. The Rat Park findings are therefore best understood as providing *proof-of-concept evidence* that social environment can causally modulate compulsive drug use at a neurobiological level, not as a complete model of human addiction. The argument for the relational hypothesis does not rest on Rat Park alone; it is triangulated against the human literatures reviewed in Sections 2, 3, 4, and 6.

5.2. Human Analogs: Vietnam, Natural Recovery, and Policy Evidence

The Vietnam veteran data provide the most powerful human analog. Robins (1993) reported on the outcomes of army enlisted men evaluated between 1972 and 1974 following service in Vietnam, during which cheap, high-purity heroin was abundantly available and approximately 20% met criteria for heroin addiction in-country. Upon return to the United States—to families, communities, and social roles—approximately 88–95% of those who had been addicted ceased use without formal treatment. Robins characterised this as "rapid recovery from heroin addiction" and identified the contextual change—from a socially disconnected, threat-saturated war environment to reconnected

civilian life—as the primary explanatory variable.

This finding cannot be explained through pharmacological models, which would predict severe, treatment-requiring withdrawal and high relapse rates following intense prior use. It is directly predicted by the connection model: when the social conditions that made substance use adaptive were removed and replaced by genuine belonging, the compulsion attenuated.

Portugal's 2001 decriminalisation reform, combined with substantial investment in social reintegration services—housing, employment, community support—produced dramatic reductions in HIV transmission, drug-related incarceration, and overdose mortality over the following decade (Hughes and Stevens, 2010). International heroin-assisted treatment trials in Switzerland, the Netherlands, and other countries similarly demonstrated that providing stable relational and social context for severe, long-term heroin-dependent individuals reduced crime, improved social functioning, and enabled gradual reduction in use (Strang et al., 2015). In both cases, the active ingredient was relational: the provision of dignity, structure, and community belonging.

6. Recovery as Relational Reintegration

6.1. Recovery Capital: Relational Resources as the Primary Predictor

The recovery capital framework—the aggregate of personal, social, and community resources available to support recovery (White, 2009; Laudet and White, 2008)—was first tested prospectively by Laudet and White (2008) in a longitudinal study of inner-city poly-substance users ($N = 312$). Higher baseline recovery capital—particularly social supports, 12-step affiliation, and community belonging—significantly predicted sustained recovery, higher quality of life, and lower stress at one-year follow-up, above and beyond treatment modality or medication status.

This finding has an underappreciated inequality implication. The well-documented disparity in recovery rates between high- and low-income individuals is partly explained by differential access to treatment but substantially explained by differential access to relational capital. Higher-income

individuals recover at higher rates in large part because they retain or can reconstruct the family support, social networks, and community structures that constitute the scaffolding of sustained recovery. Expanding access to treatment without expanding relational capital leaves the structural driver of inequality in recovery rates unaddressed.

6.2. Alcoholics Anonymous and Mutual Help: The Relational Mechanism

The most widely studied recovery intervention is Alcoholics Anonymous, and the largest meta-analytic evidence base now clearly supports its efficacy through a specifically relational mechanism. Kelly et al. (2020) conducted a Cochrane systematic review of 27 studies ($N = 10{,}565$) and found that manualised AA and 12-step facilitation increased continuous abstinence at 12 months compared to cognitive-behavioural therapy (relative risk [RR] = 1.21, 95% CI 1.03–1.42, high-certainty evidence), with substantial healthcare cost savings. Kelly et al. (2012), analysing mechanisms across the Project MATCH dataset ($N = 1{,}726$), found that social network changes and increased abstinence self-efficacy were the primary mediators of AA's effect on drinking outcomes, explaining 43–67% of the AA-outcome relationship. AA works not primarily through its spiritual content or its cognitive restructuring steps—it works because it embeds the individual in a community of shared experience, accountability, and witnessed recovery. Kelly et al. (2018) confirmed these social network mechanisms operate comparably in young adults, with peer similarity providing additional benefit in this age group.

6.3. Trauma-Informed Care: Treating the Relational Wound

Seeking Safety (Najavits, 2002) is the most studied behavioural intervention for co-occurring PTSD and substance use disorder. Najavits et al. (2006) found in a randomised controlled trial (RCT) with adolescent females ($N = 33$) that Seeking Safety produced significantly better outcomes than treatment-as-usual on substance use and trauma-related symptoms. In a related RCT of a trauma-informed cognitive-behavioural approach in incarcerated women with PTSD/SUD ($N = 49$), Zlotnick et al. (2009) found that the CBT arm showed significantly greater PTSD symptom reduction

compared to standard care, with 53% of the intervention group achieving PTSD remission by six months post-release—demonstrating that trauma-focused approaches produce meaningful between-group gains even in justice-involved populations with severe comorbidity. Najavits et al. (2020) argued in a narrative review of treatment options for PTSD/SUD comorbidity that present-focused, relational models show superior public health scalability compared to purely trauma-processing or substance-focused approaches.

This is consistent with the broader literature showing that therapeutic alliance—the quality of the relational bond between therapist and client—is the single most consistent predictor of outcome across all psychotherapy modalities, including addiction treatment (Meier et al., 2005). Myers et al. (2025), in a scoping review of 135 articles, confirmed that therapeutic alliance was the most frequently studied dimension of patient-centred care in SUD (35.6% of studies) and showed consistently positive associations with outcomes; trauma-informed care was the third most studied dimension and also associated with better results.

6.4. Peer Support: Relational Credibility as Active Ingredient

Peer support specialists—individuals with lived experience of addiction and recovery—provide a form of relational credibility that clinical professionals cannot offer. Wu et al. (2025) found that peer support workers employ distinctively relational engagement strategies—disclosing lived experience and "humanising the individual"—that differ in both function and form from clinician strategies. These strategies enable recovery capital building through connective, experiential methods unavailable to non-peer providers. Belenko et al. (2021) found in a pilot RCT in drug court ($N = 76$) that peer support reduced rearrests and improved treatment engagement, and Watson et al. (2024) demonstrated in a pragmatic emergency department RCT that peer support specialists increased opioid use disorder treatment linkage and reduced recurrent overdose—both with particular promise in justice-involved and high-acuity populations whose histories of relational betrayal make trust in professional helpers acutely difficult. The relational mechanism in peer support is not simply empathy—it is specifically the demonstration that relational reintegration is

achievable, embodied by the peer specialist as living evidence.

7. Theoretical Integration: The Relational Deprivation Model

7.1. A Unified Framework

The five evidence streams reviewed above converge on what we term the **Relational Deprivation Model of Addiction**. The model has two axes (see Figure 1 and Table 1).

The *developmental axis* proceeds from disrupted early attachment through allostatic dysregulation to adult vulnerability: early relational rupture (ACEs, attachment insecurity) produces lasting changes in stress-response systems—HPA dysregulation, CRF sensitisation, amygdalar hyperreactivity—that deplete the social reward circuit's hedonic set point and create a neurobiological state in which substances provide the fastest available path to affect regulation. This pathway operates on a continuum; each additional unit of relational deprivation incrementally increases vulnerability.

The *structural axis* proceeds from community-level social capital erosion through isolation to triggering context: deindustrialisation, institutional disintegration, and community collapse remove the social structures—stable employment, civic participation, religious community, neighbourhood cohesion—that provide adult belonging and purpose. The individual exposed to substances in this context lacks the relational buffers that moderate use into non-compulsive patterns.

These axes converge at the same point: the use of substances as an adaptive solution to unmet belonging needs in a dysregulated nervous system. Recovery, in this model, is the re-creation of the relational conditions under which the solution is no longer necessary.

7.2. Addressing the Major Objections

Genetic heritability. Addiction has substantial genetic heritability (approximately 40–60%), which might appear to support a primarily biological aetiology independent of relational factors. However, McLellan et al. (2000) noted that genetic heritability for drug dependence is comparable to asthma

and type 2 diabetes—conditions universally understood as requiring environmental triggers for expression. Many of the genetic variants associated with addiction risk heighten stress reactivity and sensitivity to environmental context (Volkow and Blanco, 2023), meaning the same genes may amplify both vulnerability to relational deprivation and responsiveness to relational repair. Genetic risk and relational environment are interacting, not competing, determinants.

Physical dependence. Pharmacological dependence—tolerance, withdrawal—is a real physiological phenomenon. The critical distinction, as McLellan et al. (2000) documented, is between physical dependence and addiction: many individuals take opioids chronically for pain management and become physically dependent without developing compulsive, loss-of-control use. Physical dependence is a condition of the body; addiction is a condition of the self in relation. The key determinant of whether dependence transitions to addiction is not the pharmacology but the individual's access to alternative sources of meaning, connection, and felt safety.

Individual variation. Not everyone who experiences poverty, childhood adversity, or community collapse becomes addicted. This is correct and important, but it does not refute the relational model—it requires the model to be probabilistic and multifactorial, which it is. Variation in relational capital, co-occurring protective factors (secure adult attachments, meaningful work, spiritual community), and access to care all moderate the risk conferred by relational deprivation. The model does not predict deterministic causation; it identifies relational deprivation as the primary population-attributable risk factor.

7.3. Policy and Treatment Implications

If addiction is fundamentally a relational disorder, several policy implications follow that extend well beyond the conventional addiction treatment system. First, housing stability must be treated as a component of addiction treatment rather than a prerequisite for it; the Housing First evidence base demonstrates that providing unconditional housing to chronically homeless individuals with severe SUD improves health outcomes without increasing substance use (Tsemberis et al., 2004). Second,

social prescribing—referral to community-based, non-clinical activities including mutual aid, arts programmes, volunteering, and employment support—must be integrated into addiction treatment pathways. Third, peer support must be resourced as a clinical intervention, not a supplementary service.

Portugal's 2001 reform provides the existence proof: when a society treats addiction as a public health and social reintegration problem—investing in housing, employment, community services, and decriminalisation—outcomes improved across multiple key indicators, including HIV transmission rates, drug-related incarceration, and overdose mortality (Hughes and Stevens, 2010).

7.4. Limitations of this Review

Several limitations of this synthesis warrant explicit acknowledgement. First, as a narrative, non-systematic review, it is susceptible to selection bias: evidence consistent with the relational deprivation framework may have been weighted more heavily than inconsistent findings. Systematic reviews and meta-analyses were prioritised where available, but the overall evidence synthesis has not been subjected to formal PRISMA procedures. Second, the literature base is predominantly North American and Northern European; it is unclear whether the relational mechanisms described generalise to collectivist cultural contexts in which social embeddedness is structurally higher. Third, the human evidence for the causal role of social environment in addiction is largely observational; the experimental evidence (Rat Park) is from animal models with acknowledged translational limitations. Fourth, the review does not systematically address sex and gender differences in relational pathways to addiction, which represent an important future research priority.

8. Conclusion

This review has synthesised convergent evidence across five independent domains to argue that addiction is best understood as an adaptive response to chronic relational deprivation, and that recovery requires relational reintegration rather than abstinence alone. Neuroscience demonstrates

Table 1: Evidence Integration Summary: The Relational Deprivation Model across Five Domains

Domain	**Evidence Type**	**Key Finding**	**Relational Mechanism**
Neuroscience	Circuit overlap studies; allostatic load models	Drugs activate social bonding circuitry; disconnection depletes hedonic set point	Substances substitute for absent social reward (Insel, 2003; Burkett and Young, 2012)
Trauma / ACEs	Cohort studies; meta-analyses	4–12$\times$ addiction risk with $\geq$4 ACEs; 67% population-attributable risk for IV drug use	ACEs are relational betrayals; trauma disrupts affect regulation (Felitti et al., 1998; Dube et al., 2003)
Social Capital	Ecological studies; longitudinal cohorts	83% lower overdose in high social capital counties; loneliness increases all-cause mortality OR = 1.29 (not overdose-specific)	Community belonging buffers exposure; isolation removes protective context (Zoorob and Salemi, 2017; Holt-Lunstad et al., 2015)
Environmental / Naturalistic	Animal models; veteran cohorts; policy natural experiments	Social housing dramatically reduced compulsive use in Rat Park; 88–95% of Vietnam heroin users recovered on repatriation; Portugal decriminalisation reduced HIV, incarceration, and overdose	Social context causally moderates whether exposure transitions to compulsive use (Alexander et al., 1978; Robins, 1993; Hughes and Stevens, 2010)
Treatment Evidence	RCTs; Cochrane reviews; scoping reviews	AA RR = 1.21 vs. CBT; therapeutic alliance strongest predictor across modalities	Recovery requires relational embedding alongside pharmacology (Kelly et al., 2020; Meier et al., 2005)

that drugs of abuse activate circuits evolved for social bonding and that chronic disconnection creates the allostatic vulnerability that makes substances pharmacologically rewarding. Trauma research establishes a dose-response relationship between relational ruptures in childhood and adult addiction risk. Social epidemiology shows that community-level social capital deficits and individual loneliness predict overdose mortality at the population level. Environmental and naturalistic evidence—from the Rat Park animal model, the Vietnam veteran cohort, and the Portuguese decriminalisation reform—demonstrates that social context causally moderates whether substance exposure transitions to compulsive use. And the treatment outcome literature converges on relational factors—therapeutic alliance, mutual-help community, peer support, and recovery capital—as the primary drivers of sustained recovery.

Several critical questions remain unanswered. The temporal and causal directionality of the loneliness-addiction relationship requires prospective experimental resolution. The neurobiological mechanisms of social reward substitution remain incompletely characterised. The scalability of relational interventions in under-resourced clinical settings is an unproven challenge. And the degree to which digital tools can supplement human connection for isolated individuals in low-resource contexts is a legitimate future avenue—one that requires rigorous clinical evaluation before deployment.

What the evidence already supports, however, is sufficient to reorient a treatment system. Clinicians who ask only "what substance is the patient using and how frequently" are missing the more fundamental diagnostic question: "who does this person belong to, and what is preventing them from belonging?" The answer to the second question is, in most cases, the answer to the first.

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Conceptualisation, literature synthesis, and writing were conducted by the authors. All authors approved the final manuscript.

Conflict of Interest

The authors declare no conflict of interest.

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