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Mini Review

Why Addiction, Compulsion, and Craving Share the Same Brain Circuit: Is it Called "Reward Deficiency Syndrome (RDS)?"

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Abstract

Neuroscience suggests we've been treating symptoms, not the system [1]. Addiction is usually described as many different problems including alcohol, opioids, stimulants, gambling, compulsive eating, and many behavioral addictions [2]. What if addiction isn't about drugs or behavior at all—but about a brain that cannot experience reward normally? Medicine treats these conditions separately. Policy regulates them separately. Research often studies them separately [3].

But the brain does not.

Across these conditions, relapse rates remain stubbornly high [4]. Prevention struggles to gain traction. Patients often move from one diagnosis to another with little lasting relief. The pattern is hard to ignore. What if these behaviors are not separate diseases at all—but different expressions of the same underlying brain dysfunction?

Neuroscience increasingly suggests exactly that.

Keywords: Reward Deficiency Syndrome; Addiction; Dopamine; Reward Circuitry; Compulsive Behavior

Introduction

Again and again, researchers return to the same circuitry: the brain's reward system [5]. Centered on dopamine signaling between the ventral tegmental area, the nucleus accumbens, and the prefrontal cortex, this network governs motivation, reinforcement, and the capacity to experience satisfaction from everyday life as affected by stress [6]. When it functions well, effort feels worthwhile. Goals feel meaningful. Emotional balance is possible.

When it does not, life feels flat

From a neurobiological perspective, this framework is distinct from current diagnostic systems such as the DSM, which categorize addiction based on observable behaviors rather than underlying reward-circuit mechanisms.

Neuroscientists have a name for this shared vulnerability: Reward Deficiency Syndrome, first coined by Blum., *et al*. in 1996 [7]. The dopaminergic system, and in particular the dopamine D2 receptor, has been profoundly implicated in reward mechanisms in the brain. Dysfunction of the D2 dopamine receptors leads to aberrant substance-seeking behavior (alcohol, drugs, tobacco, and food) and other related behaviors (pathological gambling, Tourette's syndrome, and attention-deficit hyperactivity disorder). Variants of the D2 dopamine receptor gene are proposed as important common genetic determinants of Reward Deficiency Syndrome.

The term describes a hypodopaminergic state in which the brain's reward circuitry fails to respond adequately to ordinary experiences. Pleasure is muted. Motivation fades. Satisfaction becomes elusive. In this state, the brain seeks compensation—anything capable of producing a rapid dopamine increase (see Figure 1).

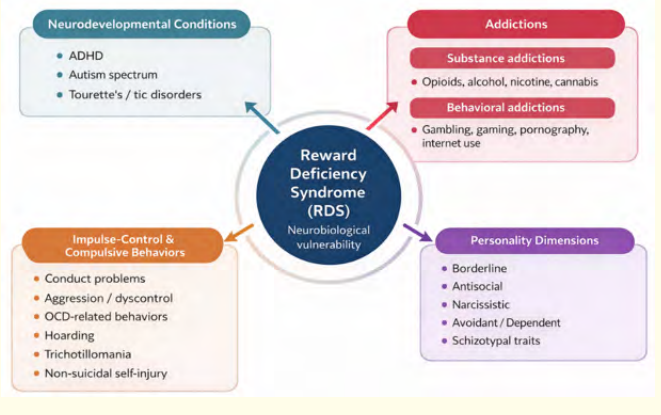

Figure 1

Figure 1 visually summarizes the core premise of Reward Deficiency Syndrome by illustrating how hypodopaminergic reward circuitry can give rise to diverse substance-related and behavioral addictions through a shared neurobiological pathway.

Literature selection approach

This narrative mini-review draws on peer-reviewed literature identified primarily through PubMed and related biomedical databases. Emphasis was placed on foundational and contemporary studies published approximately between 1990 and 2025 that address reward circuitry, dopaminergic signaling, genetic vulnerability, and addictive or compulsive behaviors. Sources were selected to provide neurobiological, clinical, and translational perspectives rather than to serve as a systematic review.

Shared clinical expression of reward deficiency

Together, this body of work supports a shared reward-circuit vulnerability across addictive behaviors.

Alcohol. Opioids. Stimulants. Gambling. Binge eating. Compulsive shopping. Gaming. Endless scrolling.

Each offers temporary relief, followed by a deeper deficit.

Seen through the lens of Reward Deficiency Syndrome, the traditional boundary between substance use disorders and so-called "behavioral addictions" dissolves. These are no longer separate categories. They are different strategies for solving the same biological problem: how to raise dopamine signaling in a reward system that cannot sustain it on its own.

This framework helps explain why people often migrate from one addiction to another [8]. A patient who stops drinking may begin gambling. Someone who quits opioids may develop compulsive eating or shopping [9,10]. The behavior changes. The circuitry does not.

Over time, what begins as choice becomes craving. Craving becomes compulsion. Control erodes.

This shared mechanism helps explain one of addiction medicine's most persistent paradoxes: why treating the drug or behavior alone so often fails [11]. Detoxification may stop use [12]. Abstinence may reduce harm. Even medication can blunt symptoms. But when the underlying reward circuitry remains impaired, craving persists. Relapse is not a mystery. It is a predictable neurobiological outcome.

A growing body of research points to common vulnerabilities beneath these conditions. Genetic variations affecting dopamine

receptors, transporters, and metabolic pathways recur across substance use disorders and compulsive behaviors [13]. These risks are further shaped by experience—early stress, trauma, chronic pain, and repeated exposure to high-dopamine stimuli[14]. Biology and environment converge on the same system via epigenetic [15].

Estimates suggest that a substantial portion of the population carries some degree of reward vulnerability. In a modern environment saturated with fast, artificial rewards, that vulnerability can become a tipping point. Yet our diagnostic frameworks lag behind this science. Manuals such as the DSM were designed to reliably classify behavior, not to identify biological risks. They describe what addiction looks like after it has fully emerged. They offer little insight into why it begins—or why it so often returns. As a result, intervention usually starts late, after years of neuroadaptation have already occurred.

The implications are profound. If addiction, compulsion, and craving reflect a shared reward-circuit dysfunction, prevention must begin earlier [16,17]. Risk identification—routine in cardiology and oncology—becomes plausible in behavioral health. This is not about predicting destiny. It is about recognizing vulnerability before damage accumulates.

Applied practically, this framework supports earlier identification of reward vulnerability through educational awareness, clinical screening, and population-level risk stratification. In clinical settings, it may inform more personalized prevention strategies that emphasize resilience, stress regulation, physical activity, and social connection before compulsive behaviors become entrenched. At a public-health level, recognizing shared reward-circuit vulnerability reframes addiction prevention as a systems challenge rather than a series of isolated disorders.

Treatment, too, looks different through this lens. The goal shifts from suppressing behavior to restoring healthy reward signaling. That means integrated approaches aimed at stabilizing—not spiking—the reward system: behavioral therapy, social connection, physical activity, stress regulation, and emerging biologically informed strategies designed to normalize dopamine tone [18]. Recovery, in this model, is no longer defined solely by abstinence. It is defined by the return of motivation, meaning, and the ability to experience pleasure from ordinary life.

There is also a moral shift embedded in this science. When craving is understood as impaired circuitry rather than weak will, stigma loosens its grip. Patients are no longer failing treatment. Treatment is failing to address the system beneath the symptoms [19].

Conclusion

While Reward Deficiency Syndrome provides a unifying neurobiological framework, it is not the sole explanatory model of addiction. Alternative perspectives—including habit learning, allostatic load, and psychosocial or environmental models—offer valuable insights into specific aspects of addictive behavior. These frameworks are not mutually exclusive. Rather, Reward Deficiency Syndrome is presented here as an integrative lens that helps explain shared vulnerability across diverse addictive and compulsive phenotypes, complementing existing diagnostic and behavioral models.

In summary, addiction is not simply about substances. Nor is it a collection of unrelated disorders. At its core, it is a disorder of reward—one we have been treating in pieces, long after it takes hold. Recognizing the shared circuit does not solve the problem overnight. But it finally gives us a coherent place to begin.

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MPL and KB developed the first draft. MPL thereafter revised and all co-authors edited and approved the final manuscript.

Conflict of Interest

KB holds patents for KB220 and the GARS test.

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