

SERGEI KALUTSKIY



ANATOMY OF RELAPSE

WHY WE KEEP GOING
WHEN WE WANT TO STOP

18+

Sergei Kalutskiy

**Anatomy of Relapse. Why We Keep
Going When We Want to Stop**

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Relapse isn't a failure of character. Its the predictable outcome of an internal conflict - one that gets worse, the harder you fight it. This book draws on seventeen years of lived experience and a synthesis of modern addiction science to explain why conventional approaches work when they work and fail when they fail. What you'll find here isnt another method. Its the map that shows how methods work internally - spanning major approaches from Internal Family Systems to Motivational Interviewing and Gestalt Therapy. This book doesnt blame addiction on weakness and doesnt deny agency. It offers a framework that is neither brain disease nor moral failing - but a predictable solution to an unsolvable problem, built into the architecture of the mind itself.

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Anatomy of Relapse

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Introduction

This book is divided into two parts: Theory and Practice.

Part I presents a new perspective on the mechanism behind all forms of addictive behavior. Part II examines existing treatment approaches — why they help some people and fail others, and how they can be reinterpreted to become genuinely effective in light of this theory.

The book is built around thought experiments. Each one is simple, intuitive, and requires no specialized knowledge.

Modern psychology often feels inaccessible. Behind complex terminology and professional concepts, the living human meaning is easily lost. My aim is to look behind the curtain of scientific language, set aside the heavy jargon, and reveal the problem in its simplest form. You will find that many seemingly complicated theoretical constructions conceal an elementary truth — one that becomes visible through the framework this book proposes.

You will also find that despite the apparent diversity of treatment methods — from spiritual practices to clinical psychotherapy — they ultimately come down to a small number of fundamental ideas.

There are two distinct questions about addiction that are frequently conflated.

First: why does a person begin using, and why do they continue? The answer involves needs, genetic predisposition, environment, personal history, trauma, and many other factors. This book does not attempt to answer that question comprehensively.

Second: why can a person who sincerely wants to stop fail to do so? That is the central question of this book.

These two questions require different explanations. A person may use substances for years without experiencing any inner conflict — and there is nothing mysterious about that. The mystery emerges the moment a genuine desire to stop appears, only to reveal that desire alone is not enough.

Part I Theory

Chapter 1. A Unified Perspective

This book does not attempt to examine individual forms of addiction separately — drug dependence, overeating, smoking, compulsive shopping, or internet addiction. Its aim is different: to uncover the universal internal mechanism behind all forms of addictive behavior and, on that basis, to propose a general theory — one that explains why some treatment methods prove effective while others do not.

These disorders undoubtedly differ in the harm they cause to the body, in the substances involved, and in their impact on personality. Yet a closer look reveals that all addictions share a striking number of common features. They tend to develop according to the same cyclical pattern: relapse, guilt, and a renewed firm intention to "quit forever."

Five universal characteristics can be identified across any addiction.

1. Loss of Control

Loss of control over one's own behavior is the defining feature of addiction. It is precisely what distinguishes addiction from a merely harmful habit.

Nora Volkow, Director of the U.S. National Institute on Drug Abuse (NIDA), describes addiction as a condition associated with dysfunction in the neural systems responsible for self-control and behavioral regulation (Volkow & Li, 2005).

An addicted person typically recognizes their condition and genuinely wishes to stop. Whether the issue is drugs, smoking, or overeating, the individual wants to stop but cannot follow through on their own decision.

Addiction, then, should be understood not primarily as substance misuse but as an impairment of self-regulatory capacity. If a behavior remains fully subject to voluntary control, it is difficult to call it an addiction.

2. From Reward to Relief

A widespread misconception is that addicted individuals continue using simply because they enjoy it: that people with obesity cannot resist tasty food, drug users seek euphoria, and alcoholics drink for pleasure.

A closer look shows that continued use is not always associated with pleasure. The experience may be neutral or even unpleasant. Philosophical analyses of addiction emphasize that compulsive behavior can persist long after it ceases to provide genuine enjoyment, functioning instead as a way of avoiding discomfort rather than producing positive experience (Kennett, Matthews, & Snoek, 2013).

Empirical data support this shift. The affective processing model shows that once addiction is established, the primary motivation for using becomes the alleviation of negative feelings rather than the pursuit of reward (Baker, Piper, McCarthy, Majeskie, & Fiore, 2004). At the neurobiological level, this corresponds to an allostatic shift: the baseline of the reward system drops, and the substance is required not for pleasure but to restore a neutral state (Koob & Le Moal, 2001).

The urge to use is therefore driven more often by an attempt to relieve intense internal tension than by any desire for pleasure. The symptoms may vary — from irritability and anxiety to nausea and tremor — but they are almost always present. Continued use becomes not a source of pleasure but a means of escaping suffering.

This distinction between seeking pleasure and avoiding suffering is fundamental to everything that follows.

3. Withdrawal Syndrome

Withdrawal is often assumed to occur only in drug addiction or chronic alcoholism. In reality, withdrawal-like states are also observed in behavioral addictions such as gambling, shopping, and overeating.

Stanton Peele, analyzing the phenomenon of romantic addiction, noted that the end of addictive relationships is accompanied by pronounced distress, anxiety, and experiences functionally similar to withdrawal (Peele & Brodsky, 1975).

Contemporary research confirms similar patterns in other behavioral addictions. Pathological gambling, for example, is associated with withdrawal-like symptoms — irritability, restlessness, and sleep disturbances — comparable in structure to withdrawal from substance dependence (Yau & Potenza, 2015).

Withdrawal syndrome, then, is not exclusive to chemical addiction. It is a universal response of the self-regulatory system to the loss of a familiar means of stabilization.

4. Propensity for Relapse

One of the most consistent features of addiction is the high likelihood of returning to previous behavior. As early as the 1970s, researchers showed that relapse curves for alcoholism, smoking, and heroin dependence follow strikingly similar patterns: a sharp decline during the first three months of abstinence, followed by a plateau (Hunt, Barnett, & Branch, 1971).

Clinical data indicate that the risk of relapse remains elevated long after treatment ends. In this respect, addiction resembles other chronic illnesses — diabetes, hypertension, asthma: relapse rates in addiction do not exceed rates of treatment non-adherence in these conditions (McLellan, Lewis, O'Brien, & Kleber, 2000).

5. A Shared Neurobiological Basis

According to the American Society of Addiction Medicine (ASAM), addiction is a chronic brain disease affecting systems of motivation and memory (ASAM, 2019).

Despite differences in pharmacological action, psychoactive substances engage similar neural systems related to reward, motivation, and learning — including the mesolimbic dopamine pathway (Koob & Volkow, 2010). Neuroimaging studies show that comparable circuits are activated in behavioral addictions such as gambling (Potenza, 2006).

Understanding addiction as a brain disorder has important implications. It tells us that addiction is neither a moral failing nor a defect of character, but a disruption of regulatory mechanisms grounded in neurobiology. In this sense, the term *brain disease* is justified: all forms of addiction rely on similar systems of motivation, reward, and memory.

But this does not imply helplessness. The brain is not an external machine that has malfunctioned independently of the person. Psychological processes and neurobiology are simply two levels of description of the same system.

To say that addiction is a brain disease is about as accurate as saying that a computer virus is a disease of a computer. Yet a virus cannot be eliminated by replacing hardware — it requires changes at the level of software. Likewise, in addiction, neural circuits reshape through experience, decisions, and new modes of self-regulation.

The neurobiological model explains where the disruption occurs, but not how it persists. The task of this book is to describe that mechanism at the psychological level.

All forms of addiction share common features: cyclical progression, loss of behavioral control, the presence of withdrawal, a high probability of relapse, and involvement of the same neural systems.

Taken together, these observations suggest that addiction is a universal disorder of will and motivation — not a specific case of substance misuse.

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Chapter 2. The Drug-as-Disease model

In popular understanding, addiction is seen as a disease caused by the substance itself — one that follows a predictable course.

According to this model, regular drug use gradually produces tolerance: the previous dose no longer generates the desired effect, and the individual must consume increasing amounts to achieve the same level of pleasure.

When use stops, withdrawal emerges — a painful condition explained as the body having "adapted" to the substance and now demanding it again. This discomfort, together with an irresistible craving caused by bodily intoxication and brain adaptation, is believed to trap the person in a lifelong cycle of addiction.

This view, although widespread, does not hold up under careful examination. Contemporary research shows that addiction cannot be reduced to the formula "substance → habituation → disease." Its nature is far more complex.

Consider five major misconceptions underlying the traditional model.

1. The Myth of Tolerance

Tolerance refers to physiological adaptation that requires increased doses to achieve the same effect. It is often assumed that a drug user is inevitably doomed to escalate dosage without limit.

In reality, limitless dose escalation is a myth. For most individuals, this process eventually stabilizes. Moreover, tolerance is not a unique marker of addiction — it also develops with many ordinary medications, such as antidepressants, which do not produce compulsive drug-seeking.

Norman Zinberg's studies of opiate users showed that among participants tracked over time, nearly half maintained stable doses for years, 27% reduced their intake, and only 13% increased it (Zinberg, 1984). Zinberg demonstrated that controlled opiate use is possible when social rituals and rules regulate consumption — in other words, context, not the substance alone, plays the decisive role.

Large epidemiological datasets further indicate that most addicted individuals stop using without formal treatment, typically between the ages of 30 and 40. Addiction appears, then, not as a lifelong sentence but as a disorder with a characteristic natural history — one in which recovery is the norm rather than the exception (Heyman, 2009). From a neurobiological perspective, Marc Lewis makes a similar argument: addiction is not a brain disease but a form of deep learning. The brain reorganizes through repeated experience, yet the same plasticity allows it to reorganize again (Lewis, 2015).

2. The Myth of "Hooked After the First Time"

Popular warnings claim that a single dose is enough to enslave a person. Neither clinical experience nor historical evidence supports this.

Alcohol and nicotine clearly do not produce addiction after first exposure; repeated use is required. The same applies even to so-called "hard" drugs. Before the modern war on drugs, opiates were widely included in common medicines and tonics (Brecher, 1972; Courtwright, 1982). Despite broad availability, addiction rates remained relatively low.

In the United Kingdom, heroin was long prescribed for cough and migraine without triggering a widespread epidemic of addiction (Berridge, 1996).

Systematic analyses of medical opioid prescribing show that dependence develops in roughly 8–12% of patients receiving long-term treatment — significantly lower than commonly assumed, though far from zero (Vowles et al., 2015). These findings challenge both extremes: the myth of instant addiction and the opposite belief that prolonged use is entirely safe.

George Vaillant's decades-long longitudinal research on alcoholism showed that even individuals who later described their addiction as sudden had in fact developed dependence gradually,

over many years. The experience of "instant addiction" is often a retrospective distortion of memory (Vaillant, 1983).

3. The Myth of Inevitability with Prolonged Use

Another widespread belief holds that prolonged use inevitably leads to addiction. At first glance this seems intuitive: the longer a substance is used, the stronger physiological and psychological adaptation should become. Reality is more complex.

Consider patients receiving medically supervised postoperative analgesia. They are often allowed to regulate their own opioid dosage — conditions that would appear ideal for addiction to take hold. Yet studies consistently show that once pain subsides, patients discontinue use without difficulty. Addiction does not develop (Schug, Merry, & Acland, 1991).

Perhaps the largest natural experiment in history occurred during the Vietnam War. In the late 1960s and early 1970s, a substantial proportion of American soldiers in combat regularly used heroin, marijuana, and amphetamines. Estimates suggest that more than 35% used opioids at least once and about 20% used them regularly. When the U.S. Department of Defense learned of these figures, widespread panic followed. Experts predicted an epidemic of addiction among returning veterans.

The research led by Lee Robins revealed a dramatically different outcome. Among soldiers who tested positive for drugs before demobilization, only 5% continued using during the first year after returning home. Most stopped on their own, without treatment, detoxification, or rehabilitation programs (Robins, 1993).

Long-term follow-up showed that only about 12% resumed use, and even those cases were typically episodic. Roughly nine out of ten stopped almost immediately upon returning to civilian life.

Why? Researchers emphasized the decisive role of context. Drug use in combat served specific functions — coping with stress, fear, pain, and insomnia. Returning home radically changed circumstances: the sources of tension disappeared, social bonds and values were restored, and the need for the drug lost its meaning. The same individuals, the same physiology, the same substances — but a different environment, and the habit dissolved.

4. The Problem with Animal Experiments

Since the mid-twentieth century, the concept of self-perpetuating addiction has relied heavily on animal experiments. In "Skinner boxes," rats or monkeys could press a lever to self-administer drugs. The results appeared straightforward: animals rapidly escalated intake and sometimes consumed drugs to the point of death.

These findings were long interpreted as direct proof that drugs themselves inevitably produce addiction. Canadian psychologist Bruce Alexander questioned the experimental conditions themselves, proposing that the critical factor was social isolation, not the substance. To test this, he created "Rat Park" — an enriched environment with space, toys, food, and social companions (Alexander, Beyerstein, Hadaway, & Coombs, 1981).

The results were striking. Rats in enriched environments showed little interest in morphine solution, preferring ordinary water. Even previously drug-exposed animals often abandoned drug consumption.

Alexander's findings sparked debate. Replication attempts produced mixed results — some confirming the effect, others finding weaker outcomes (Petrie, 1996). Nevertheless, the central insight — that environment plays a decisive role — received substantial support from later research. Enriched environments significantly reduce cocaine self-administration and drug-seeking behavior even after prolonged exposure (Chauvet, Lardeux, Goldberg, Jaber, & Solinas, 2009).

The issue, then, was not whether Alexander was correct in every detail, but that standard experiments with isolated animals systematically overestimated the addictive power of substances by neglecting context.

5. The Myth of Withdrawal as the Primary Driver

It is often assumed that physiological withdrawal symptoms are what force people to resume using. This belief rests on an unexamined conflation: the entire experience of cessation — restlessness, craving, obsessive thinking, psychological pain, a sense of unbearable pressure — is attributed to the body's chemical reaction to the absence of a substance.

Yet only a narrow subset of withdrawal symptoms is genuinely physiological in origin. Tremor, nausea, sweating, tachycardia, and in severe cases seizures and delirium — these reflect the body's acute response to the removal of a substance to which it has adapted. They are medically serious and, in the case of alcohol and benzodiazepines, potentially life-threatening. Discontinuation should occur under medical supervision.

But the broader phenomenology of withdrawal — the restlessness, the obsessive preoccupation with use, the sense of mounting internal pressure, the psychological pain that people describe as unbearable — cannot be accounted for by chemical deficit alone. These symptoms appear with comparable intensity in behavioral addictions where no substance is involved at all. Pathological gambling produces irritability, restlessness, and sleep disturbance structurally similar to substance withdrawal (Yau & Potenza, 2015). Internet gaming disorder is accompanied by craving and distress upon cessation that activate the same neural systems as drug withdrawal (Dong, Wang, Du, & Potenza, 2017; Grant, Potenza, Weinstein, & Gorelick, 2010). The end of addictive relationships triggers a state neurobiologically comparable to withdrawal syndrome (Fisher, Brown, Aron, Strong, & Mashek, 2010).

Similar symptoms — anxiety, irritability, sleep disturbance — also occur during caffeine cessation and antidepressant discontinuation, neither of which involves substances classified as narcotics (Fava, Gatti, Belaise, Guidi, & Offidani, 2015).

If the full range of withdrawal could be explained by the body's chemical readjustment, these phenomena would not exist. The fact that they do suggests that most of what people experience as "withdrawal" is not physiological in the strict sense — it is psychological, and requires a different explanation.

Moreover, even the genuinely physiological component does not explain addiction. Modern medicine can alleviate physical withdrawal relatively effectively. If withdrawal alone explained addiction, treatment would not be so difficult. Yet relapse often occurs weeks or months after the body is already physiologically free of the substance. The primary triggers are not physical craving but emotional states, interpersonal conflicts, and situational pressures (Marlatt & Gordon, 1985).

Research on craving supports this conclusion from another angle. Craving is not even a necessary condition for use — people often engage in substance use automatically, without conscious desire. Craving intensifies primarily when habitual behavior is blocked (Tiffany, 1990). Experimental studies further demonstrate that craving depends less on physiological state than on perceived availability: it increases when individuals believe use is possible and decreases when they consider it impossible (Wertz & Sayette, 2001). Craving is not a mechanical bodily signal but a response mediated by expectations and context.

Contemporary reviews confirm this: the relationship between subjective craving and actual relapse is considerably weaker than commonly assumed. Craving is an important but insufficient predictor of use; it reflects internal conflict rather than directly causing behavior (Sayette, 2016).

The fact that detoxification alone rarely resolves addiction points to the central conclusion: physiological symptoms, despite their severity, are not the primary driving force. They account for only a fraction of the withdrawal experience, and even that fraction does not explain why people return to use. The full phenomenology of withdrawal — the pain, the obsession, the unbearable pressure — demands an explanation that goes beyond chemistry. That explanation is the subject of the next chapter.

The evidence is clear: a substance by itself does not create addiction without the involvement of psychological and social factors.

Addiction is not a chemical trap into which the brain falls. It is a specific form of relationship between a person and a substance — one that arises only within a particular context and serves a particular function.

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Chapter 3. Withdrawal Syndrome

Withdrawal syndrome is the set of physical and psychological symptoms that arise when a behavior is discontinued or when the dose of a substance (or activity) to which tolerance and dependence have developed is sharply reduced. It reflects the body's adaptation to a stable source of regulation — and the imbalance that follows when that source is removed.

Withdrawal is characteristic of all forms of addiction. The symptoms differ, but the underlying mechanism is the same.

Neuroimaging studies show that the same neural networks activated in opioid or cocaine addiction are also involved in gambling and internet addiction — particularly systems related to reward, expectation, and impulse control (Dong, Wang, Du, & Potenza, 2017; Grant, Potenza, Weinstein, & Gorelick, 2010).

The destructive effects of psychoactive substances on personality are well known: increased antisocial behavior, criminalization, deterioration of social relationships. But such consequences are not unique to drugs. In the classic report *Licit and Illicit Drugs*, Edward Brecher showed that heroin and nicotine dependence share similar structural patterns. He described postwar Germany, where deprivation of tobacco led to theft, begging, prostitution, and the sale of personal belongings — behaviors scarcely distinguishable from those associated with heroin addiction (Brecher, 1972).

This universality is further emphasized in *Love and Addiction* by Stanton Peele and Archie Brodsky. The authors describe addiction as a general psychological mechanism rather than the effect of a particular substance. Addictive relationships, they argue, can erode personality as deeply as chemical dependencies (Peele & Brodsky, 1975).

"People can become addicted to other people in the same way they become addicted to drugs. We use the term 'addiction' not metaphorically, but literally."

In romantic addiction, the sustaining force is not the pursuit of intimacy but fear of loss — above all, fear of loneliness. The individual does not consciously choose destructive relationships; they are already embedded in them and act under an irrational terror of losing a psychological source of stability.

Modern neurobiological findings support this parallel. Social rejection and the breakup of close relationships activate neural systems involved in physical pain and substance withdrawal. The loss of a significant partner can produce a state neurobiologically comparable to withdrawal syndrome (Fisher, Brown, Aron, Strong, & Mashek, 2010).

Fear of Loss as the Driving Force

From this follows a key assumption: addiction is governed not by the pursuit of acquisition but by the fear of deprivation. Habituation must come first — without it, withdrawal cannot exist. Through prolonged use or long-term attachment, the substance or partner stops being experienced as something acquired and begins to feel like something that was always there. The threat of loss triggers anxiety and compels the person to restore equilibrium at any cost.

In neurobiology, this shift from seeking pleasure to avoiding suffering is described as an allostatic process. With chronic use, the baseline of the reward system shifts downward: the new "normal" falls below the pre-addiction state. The substance is no longer needed to produce pleasure but to return the person to a minimally tolerable condition. Addiction is maintained not by reward but by attempts to prevent an escalating deficit (Koob, 2013).

There is always a reason for using. It may seem trivial, but it is never absent.

Alcohol for relaxation and anxiety reduction. Food for alleviating boredom or unpleasant emotions. Caffeine for an energy boost. Nicotine for short-term stress regulation. Over time, the substance or behavior becomes integrated into the system of self-regulation. Its loss understandably provokes panic.

Importantly, fear of loss does not arise immediately. It emerges only when the object begins functioning as a regulator of difficult internal states rather than as episodic pleasure. This is why moderate users do not experience withdrawal: you cannot fear losing what has not yet become psychologically necessary.

Addiction as Preservation of a Familiar Order

Addictive behavior, in this sense, is not a pursuit of pleasure but an attempt to preserve an established psychological order. This is why withdrawal occurs not only when drugs are discontinued. Almost any stable behavior integrated into psychological homeostasis produces a similar reaction when disrupted.

Phones, social media, music, exercise, news consumption — abandoning any of them often leads to irritability, anxiety, and a persistent urge to "restore things as they were." This is not weakness of will. It is a regulatory system deprived of a key stabilizing element.

A particularly illustrative example is the cessation of long-term anabolic steroid use. What is lost is not merely a chemical effect but an identity. The person confronts the experience: "I am losing part of who I was." Research shows that abandoning behaviors tied to self-identity evokes not only discomfort but strong resistance, because it threatens the basic sense of self (Oyserman, 2009).

Discontinuation of meaningful behavior is experienced not simply as stopping an action but as losing a part of one's identity.

Two Modes of Withdrawal

Withdrawal arises both when homeostasis is disrupted involuntarily and when it is disrupted voluntarily.

A revealing contrast illuminates the nature of withdrawal. Imagine forced abstinence: the substance is confiscated, and the person makes no effort to stop wanting it. There is no ambivalence — only deprivation. What does this person actually experience? Not the classic withdrawal syndrome. They experience mobilization: anger, agitation, a focused drive to get what was taken. They scheme, demand, negotiate, beg. The energy moves outward — toward action, toward the object. This is frustrated desire, and it looks nothing like withdrawal.

Now consider the opposite: voluntary abstinence. The substance is still available — no one has taken it away. The person wants to use and is simultaneously preventing themselves from using. What do they experience? Something entirely different. The classic withdrawal pattern: muscular tension, restlessness that leads nowhere, diffuse suffering without a clear target. The energy is not directed outward — it is turned against itself. The person is not mobilized; they are immobilized. They are not fighting to obtain something; they are fighting themselves.

This contrast is diagnostic. If withdrawal were simply caused by the absence of a substance — a chemical deficit — then forced and voluntary abstinence should produce identical symptoms. The chemical state, after all, is the same. But the experiences are nothing alike. Forced abstinence produces mobilization. Voluntary abstinence produces suffering.

This mechanism was described by Fritz Perls, who argued that so-called psychogenic pain arises from inhibited impulses. Suppressed tears become muscular tension and headache — not because of physiological defect, but because of internal conflict.

In *Gestalt Therapy*, written with Paul Goodman and Ralph Hefferline, Perls explained the mechanism as follows:

"If you slightly open a faucet and try to hold the water back with your finger, you will feel how increasingly difficult this becomes. This is a direct analogy to what happens in internal conflict... If you clench your fists tightly, you will soon develop a cramp. A 'psychogenic' or 'functional' headache is a phenomenon of the same kind. You are about to cry, but you restrain the impulse, as if squeezing your head so as not to appear weak or to avoid giving others the satisfaction of seeing you cry." (Perls, Hefferline, & Goodman, 1951)

A firm decision to "quit" works the same way. It simultaneously generates an impulse and attempts to oppose it. The result is not energized action but painful self-restraint. Withdrawal suffering cannot be explained solely by chemical deficit; it emerges from the conflict between the need to restore equilibrium and the prohibition against acting.

Pain as a Consequence of Suppression

One might assume that pain itself activates the person. Experience suggests the opposite: physical and emotional pain tend to paralyze rather than mobilize. During withdrawal, people often do not act — they lie immobilized by suffering, unable even to move. This is not a fight response but a state of arrested action.

Contemporary research supports this. Models of emotion regulation show that chronic suppression of emotional experience increases physiological arousal and subjective distress rather than reducing them. Attempts to control feelings produce a paradoxical effect: the stronger the suppression, the greater the distress (Gross, 2015).

Consider the pre-relapse state. A person in severe withdrawal — immobilized, trembling, unable to think clearly — makes the decision to use. That decision alone produces relief before the substance is consumed. The person mobilizes, plans, acts with purpose and efficiency. No pharmacological event has occurred; no molecule has been ingested; no half-life has shifted. The only variable that changed is the decision to stop suppressing. If withdrawal were a chemical deficit, a decision could not resolve it — just as a decision to see a doctor does not relieve nerve pain, and a decision to eat does not halt food poisoning. Real detoxification proceeds on the body's timeline regardless of the individual's intentions. Withdrawal, by contrast, resolves — at least in its psychological core — the instant the decision changes.

The problem is not insufficient desire or weak willpower. The decision to stop does not break the cycle. Suffering is not chosen deliberately; it emerges as a consequence of suppression. From within this state, only two options appear to exist: relapse or endure the fight. And as long as the fight continues, it brings pain with it.

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Chapter 4. Internal Conflict

Two central phenomena of addiction still require explanation. First: why does the compulsive, almost manic drive to restore "things as they were" disappear after a person has endured withdrawal? And second: why does this drive return after relapse — even following years of abstinence?

To approach these questions, consider a thought experiment.

Imagine yourself standing in an empty room. On the opposite side lies something of immense value — money, a car, any object you deeply desire. It is only a few steps away, yet an invisible wall stands between you and the goal.

You begin searching for a way past the obstacle. At first cautiously, then more insistently — you push, strike, try to go around. But the wall does not yield. The harder you try, the stronger your frustration grows: the desired object remains just out of reach. Gradually, active effort gives way to despair, then helplessness. Eventually you give up — not because you are exhausted, but because you stop believing the wall can be overcome.

This experience is a metaphor for withdrawal syndrome.

Perceived Availability

The drive to restore equilibrium persists for as long as the person believes the object remains attainable. While that belief holds, anxiety and panic remain active. When it collapses — when the path back comes to feel genuinely closed — the drive loses its purpose, and calm emerges.

The belief that pleasure remains within reach — its perceived availability — is the key factor distinguishing active addiction from abstinence. As long as a person believes things can be restored, tension, fear, and anxiety persist. When this belief collapses — when it becomes clear that the path back is closed — the person stops expending energy on futile attempts, and the pain subsides.

Experimental data support this mechanism. Reviews of studies on perceived opportunity to use show that subjective craving increases when individuals believe use is possible and decreases when they believe it is impossible (Wertz & Sayette, 2001). Craving is not a mechanical signal from the body but a response mediated by expectations.

To endure withdrawal, then, is to persist until the mind accepts that return is impossible. At that point, the drive to act loses its purpose, and the system settles into a new equilibrium.

Abstinence and Its Fragility

The period of abstinence is marked by a paradoxical shift: uncontrolled desire disappears, replaced by indifference. This does not mean the substance becomes aversive — rather, belief in its availability vanishes.

But abstinence is inherently fragile. A single breach of internal separation — a seemingly minor step backward — signals to the mind that the object is available again. Hope returns, and with it fear, anxiety, and pain. A new cycle begins.

Even a single episode of use after a long period of abstinence cannot meaningfully poison the body, yet it instantly restores confidence in the attainability of pleasure. That alone is enough to return a person to active addiction. The problem is not that the body remembers the substance, but that the mind remembers the possibility.

Marlatt and Gordon described this as the abstinence violation effect: a lapse produces not merely a physiological response but a cognitive shift — loss of perceived control, guilt, and the feeling that "everything is ruined," dramatically increasing the likelihood of continued use (Marlatt & Gordon, 1985). Within the model proposed here, this effect has a more precise explanation: relapse restores perceived availability, and with it the entire internal conflict.

To re-enter abstinence, the person must pass through psychological withdrawal again — until the mind once more accepts that access to the addictive object is closed.

The Drama of Addiction

It is relapse that makes addiction so destructive. Every addicted person eventually reaches a "bottom" — a state of fear, pain, and despair that motivates the decision to endure withdrawal. Yet even after achieving abstinence, vulnerability remains. A single misstep can undo years of sobriety, and the guilt and helplessness that follow can plunge a person into depression and despair.

If recovery depends on belief in the availability of the substance, an inevitable question arises: who persuades — and who is being persuaded? To enter abstinence, a person must convince themselves that the object is no longer accessible. During relapse, the same person convinces themselves that use is again possible.

Before every relapse, an internal dialogue unfolds: one part of the self persuades, promises, and rationalizes; another resists, doubts, and fears. This raises a deeper question: who makes the promise "it will never happen again," and who is the one to whom that promise is given?

To understand how this belief collapses and is restored, we must abandon the notion of the mind as a single, unified agent. Viewing addiction solely as a person suppressing panic — or solely as a person overwhelmed by panic before an obstacle — remains incomplete.

These two figures are parts of a single whole, interacting within one psyche.

It becomes reasonable, then, to assume that addiction is not merely a disorder of will or emotion but a stable conflict between two parts of personality: one striving for liberation, the other fearing permanent loss of the possibility of use. Between them unfolds the drama of addiction — dialogue, promises, self-deception, and relapse. Only by recognizing this internal division can we understand why addiction persists for years, even when craving appears long gone.

But if two incompatible positions truly coexist within a single psyche, how is this possible at all? How can one person simultaneously know that use is destroying their life and yet act as though it does not matter?

Answering this question requires turning to the mechanism that makes such coexistence possible — dissociation.

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Chapter 5. Dissociation

How can two contradictory positions coexist within a single mind? How can a person simultaneously understand that a fear is unfounded and yet continue acting as though the danger were real?

To answer this, we must abandon the intuitive notion of the mind as a completely unified and continuous subject. The conflict observed in addiction is not merely metaphorical. It rests on a real psychological mechanism — dissociation, in which control, responsibility, and access to information become functionally divided among different parts of the self.

The Hidden Observer

Among the first systematic investigations of such division were the hypnosis experiments of American psychologist Ernest Hilgard. These studies showed that in trance states, individuals could cease consciously perceiving certain stimuli — sound, pain, or touch — while still responding to them indirectly (Hilgard, 1977).

Participants were given the suggestion that they could no longer hear. Following this instruction, they indeed stopped responding to speech and external sounds. Yet when Hilgard addressed a possible "hidden part" of the mind and asked for a prearranged signal, participants performed the action without conscious awareness of why. After emerging from trance, they reported no intentional effort and could not explain their behavior.

Hilgard called this the "hidden observer" — suggesting that part of the mind continues to perceive and process information while remaining inaccessible to conscious control.

Importantly, the hidden observer is not a separate personality or structural entity. In Hilgard's terminology, it is a descriptive label for a functional position that arises during dissociation — a state in which awareness and control temporarily diverge.

Later research confirmed the phenomenon and refined its interpretation. The hidden observer is not merely a passive recorder; it can selectively process information and generate autonomous responses, though within a restricted contextual framework (Woody & Sadler, 2008).

Informational Isolation

Hilgard's experiments and subsequent studies demonstrated that dissociated parts of the mind may possess unequal access to information. The following experiment illustrates the principle.

Participants were told that a "hidden part" of their mind would control processes associated with either the right or the left cerebral hemisphere. They were asked to memorize lists containing abstract and concrete words. Half were informed that abstract words were processed only by the right hemisphere and concrete words by the left; the other half received the opposite instruction. After being given the suggestion to forget the words, none could consciously recall any of them. However, when researchers made contact with the corresponding part of the mind, participants recalled only the words supposedly accessible to that part. The information existed but was functionally isolated.

The hidden observer retains the essential cognitive capacities of the primary self: it perceives events, understands speech directed toward it, evaluates situations, and selects responses. The difference lies not in diminished intelligence but in contextual limitation. It lacks the autobiographical perspective, intentions, and value system of the integrated personality — and exists only temporarily, for as long as necessary to compensate for lost control.

Dissociative Fugue

If hypnosis reveals dissociation under laboratory conditions, clinical phenomena demonstrate it in natural form. One of the clearest examples is dissociative fugue.

During a fugue state, a person abruptly leaves their familiar environment and loses access to autobiographical memory — while retaining intelligence, skills, and socially appropriate behavior.

They may find employment, interact normally with others, make decisions, and live an apparently ordinary life without awareness of their prior identity (American Psychiatric Association, 2022).

A well-known case described by William James involved the minister Ansel Bourne, who suddenly left home and lived for several weeks in another city under a different name, leading an orderly and unremarkable life. He later "awoke," fully recovering memory of his former identity but recalling nothing of the fugue period — and experiencing profound emotional shock upon learning what had occurred (James, 1890).

Classical descriptions of fugue show that the phenomenon does not represent psychological disintegration but a compensatory response. The person does not flee life itself but an experience that has become psychologically intolerable. Rather than collapsing, the mind redistributes its functions to preserve continued existence.

Modern research supports this interpretation. Dissociation is increasingly viewed not as dysfunction but as a regulatory mechanism that emerges when experiences cannot be integrated into the current self-model. Dissociative phenomena exist along a continuum — from everyday attentional disengagement to clinical conditions — reflecting the mind's general capacity for functional division (Holmes et al., 2005).

Fugue illustrates a crucial principle: relinquishing responsibility at one psychological level does not eliminate functioning. Instead, those functions are transferred to another, temporarily autonomous process.

Why Splitting Occurs

What triggers this mechanism of division? Why can the mind not simply contain contradiction within a single self?

An answer emerges from Daniel Wegner's theory of ironic processes, which describes how deliberate attempts to suppress a thought make its recurrence more likely. The classic example: the instruction not to think about a white bear. To avoid the thought, one must constantly monitor whether it has appeared — thereby continually activating it (Wegner, 1994).

When a person tries not to think about something, two incompatible positions arise: one process generates the forbidden thought, while another monitors and suppresses it. It is impossible to simultaneously be the one producing the thought and the one controlling it. A cognitive contradiction emerges that cannot be sustained within a single unified agent.

Experimental findings show that this effect intensifies under cognitive load, stress, and emotional strain — the very conditions characteristic of addiction (Wegner & Erber, 1992). The greater the need for control, the less effective control becomes.

At this point the mind functionally divides: one line of processing continues generating the prohibited content, while another enforces control. Dissociation appears not as pathology but as the logical consequence of an irreconcilable contradiction within one subject.

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