



Relapse as a Failure of Inference: A Precision-Collapse Model of Return-to-Use in Substance Use Disorders

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Abstract

Background: Relapse is the central unsolved problem of addiction medicine, with return-to-use rates commonly estimated at 40 to 60 percent within a year of treatment. The dominant neurobiological accounts, including the brain-disease model, incentive-sensitization theory, the allostasis (“dark-side”) model, the actions-to-habits-to-compulsions framework, and the impaired-response-inhibition-and-salience-attribution (iRISA) model, powerfully explain how vulnerability develops and endures, yet each treats relapse largely as the re-expression of a stable trait. They under-specify the acute question that most concerns clinicians and patients: why does a particular person return to use at a particular moment?

Methods: This is a conceptual, integrative synthesis. Drawing on computational neuroscience (active inference and predictive processing), interoception research, addiction neurobiology, and the clinical relapse-prevention literature, we integrate seminal and recent work into a single mechanistic account and derive testable predictions.

Results: We advance the Precision-Collapse Model (PCM), which reframes relapse not as reactivation of disease but as a transient, state-dependent failure of precision control: a momentary collapse in the precision-weighting of prediction errors that permits entrenched maladaptive priors (“using will relieve this”) to dominate policy selection. Canonical relapse triggers (stress, negative affect, cue exposure, sleep loss, and interoceptive perturbation) are reinterpreted as sharing one computational signature: each transiently biases the precision balance toward aversive interoceptive prediction errors and away from exteroceptive and goal-directed counter-evidence. Incentive-sensitization, allostasis, habit and compulsion, and iRISA are shown to be special cases of this dysregulation.

Conclusion: PCM shifts the unit of analysis from disease to inference and from trait to event, reframes mindfulness-based and interoceptive therapies as “precision retraining,” and specifies a roadmap for a computational relapse signature combining interoceptive assessment with ambulatory sensing. It reduces stigma by casting relapse as a predictable dynamical event rather than moral or biological inevitability.

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Introduction

Addiction is routinely described as a chronic, relapsing condition, and it is the second word in that phrase that has proven most resistant to intervention [1]. Even among individuals who complete evidence-based treatment, return-to-use within the first year is the modal outcome rather than the exception, and the neurobiological adaptations that sustain vulnerability can persist for years after the last exposure [2]. The human and economic costs of this durability are enormous, and they fall disproportionately on communities already burdened by structural disadvantage. If addiction science has a defining problem, it is not why people begin to use, for social, genetic, and pharmacological answers to that question are comparatively mature, but why people who have decided, often at great cost, to stop find themselves using again.

The field is not short of explanatory frameworks. The brain-disease model locates the disorder in drug-induced neuroadaptation of reward, stress, and executive circuits [2]. Incentive-sensitization theory holds that repeated exposure sensitizes mesolimbic “wanting” systems so that drug cues acquire pathological incentive salience, generating cue-triggered urges that persist long after “liking” has faded [3-5]. The allostasis or “dark-side” model describes a shift from positive to negative reinforcement, in which anti-reward and brain-stress systems create a chronic dysphoric set point that drug use transiently relieves [6-8]. The actions-to-habits-to-compulsions account maps a devolution of behavioral control from prefrontal, goal-directed systems toward dorsal-striatal, stimulus-response habit, and ultimately compulsion [9,10].

The impaired-response-inhibition-and-salience-attribution (iRISA) model integrates these strands

around prefrontal dysfunction, in which drug cues assume heightened value while inhibitory control weakens [11,12].

Each of these frameworks is illuminating, and none is wrong. But they share a structural feature: they are, in the main, trait theories. They describe the durable substrate that makes a person relapse-prone. What they under-specify is the state event itself: the transition, sometimes over minutes, from stable abstinence to use. A person carries the same sensitized cue systems, the same allostatic set point, the same habit architecture on the Tuesday they remain abstinent and the Wednesday they relapse. Something changed in the interval, and that something is not captured by a theory pitched at the level of stable pathology. Relapse is dynamical; most of our models are static.

A complementary tradition offers a natural language for dynamics. Computational psychiatry recasts brain function as probabilistic inference, and the active-inference or predictive-processing framework in particular treats perception, learning, and action as the minimization of prediction error under a hierarchical generative model [13-16]. Within this framework, a single quantity, precision (the confidence or inverse variance assigned to a prediction error), governs how strongly evidence updates belief and, crucially, how strongly competing internal models compete for the control of action. Precision is not a trait; it is a state variable that fluctuates from moment to moment with arousal, affect, sleep, and context.

The objective of this paper is to exploit that fact. We argue that relapse is best understood not as the reactivation of a disease but as a failure of inference: specifically, a transient collapse of precision control that momentarily hands behavioral control to a maladaptive prior. We call this the Precision-Collapse Model (PCM).

The remainder of the paper situates PCM within the computational and interoceptive literatures, states the model formally enough to be falsifiable, shows that the major existing theories fall out of it as special cases, reinterprets clinical interventions in its terms, and specifies the empirical program that would confirm or refute it.

Materials and Methods

This is a theoretical contribution, and intellectual honesty requires that its methodology be described as such: no primary data were collected, and no formal meta-analytic pooling was undertaken. The paper is an integrative synthesis whose method is conceptual rather than statistical. Its validity rests not on a sampling frame but on the coherence, parsimony, and falsifiability of the framework it produces.

The synthesis drew on four literatures. First, the computational-neuroscience literature on predictive processing, the free-energy principle, and active inference, including the specific extension of these ideas to addictive choice. Second, the neurobiological literature on interoception and the insular cortex in substance use disorders. Third, the classical mechanistic theories of addiction (incentive-sensitization, allostasis and opponent-process, the habit and compulsion account, and iRISA), which any new model must either subsume or contradict.

Fourth, the clinical literature on relapse prevention and mindfulness- and interoception-based interventions, which supplies both a test bed and a translational target.

Sources were identified through iterative searching of biomedical databases and citation tracing from seminal papers, prioritizing (a) foundational statements of each theory in their authors' own words and (b) the most recent authoritative reviews, so that the synthesis reflects the current state of each field rather than a single historical snapshot. Where a claim is contested or where evidence is preliminary, this is flagged in the text. The deliberate limitation of a narrative integrative synthesis, its susceptibility to selective emphasis, is mitigated here by the requirement, imposed in Section 3.7, that the resulting model generate specific predictions capable of being wrong.

Results

The Precision-Collapse Model

A Minimal Predictive-Processing Primer

Under active inference, the brain is a prediction machine that maintains a hierarchical generative model of the causes of its sensory input and acts to make its predictions come true [13,14]. Perception is the updating of beliefs to reduce the mismatch (the prediction error) between expected and actual input, and action is the selection of policies expected to minimize prediction error over time. What determines how much any given prediction error influences belief and behavior is its precision: a high-precision error commands attention and drives learning, whereas a low-precision error is discounted as noise. Precision is therefore the brain's gain-control or gating parameter, and its regulation is itself an inferential problem the brain must solve continuously.

This framework makes a striking neuromodulatory proposal. In Friston and colleagues' formulation, dopamine does not simply signal reward or reward prediction error; it encodes the precision of beliefs about which actions are worth taking, the confidence assigned to policies and to the cues that afford them [17]. This reframes, without discarding, the influential temporal-difference account in which phasic dopamine encodes a reward prediction error [18,19]. Precision-weighting is the hinge on which the present argument turns, because a disorder of precision is a disorder of confidence, and confidence, unlike reward value, is intrinsically state-dependent and fast-changing.

Addiction as Maladaptive High-Precision Priors

Applied to addiction, active inference yields a distinctive claim: the core pathology is not simply an over-valued reward but an over-confident belief. Schwartenbeck and colleagues showed that maladaptive choice can be modeled as optimal inference under a suboptimal generative model. That is, the behavior is not irrational given the agent's priors; the priors themselves are the problem [20]. Extending this, embodied and predictive-processing accounts describe addiction as the entrenchment of high-precision priors, for example "using will relieve this distress," that come to dominate the predictive hierarchy and resist revision even in the face of contradictory evidence [21]. Chronic drug exposure, on this view, does not merely strengthen a habit; it inflates the precision of a belief, rendering the

agent pathologically certain that a specific action will resolve a specific bodily state.

This is a subtle but consequential shift. A merely strong preference can be outweighed by new information; a high-precision prior actively suppresses the very prediction errors that would otherwise correct it. The addicted generative model becomes self-sealing: contradictory outcomes are down-weighted before they can teach. This explains a clinical commonplace that reward-based accounts strain to capture, namely that people relapse while fully, cognitively “knowing better,” and often while no longer expecting much pleasure from use [4].

The Interoceptive Core

If the maladaptive prior is about a bodily state, then the prediction errors that matter most are interoceptive, the signals about the internal condition of the body. A substantial literature places interoception and its principal cortical hub, the insula, at the center of addiction. Paulus and Stewart proposed that addiction reflects a discrepancy between predicted and actual internal states, a bodily prediction error, that biases approach and avoidance and, ultimately, the decision to use [22]. Craving itself has been construed as an interoceptive inference [23,24]. The causal weight of this circuitry is difficult to overstate: focal insular damage can abolish cigarette addiction outright, and lesions across the brain that disrupt addiction converge on a common insula and anterior cingulate network rather than a single site [25-27]. Prospectively, lower insular and striatal reactivity during reward processing predicts subsequent relapse at the level of the individual [28].

Interoception thus supplies the model’s content: the priors that go awry in addiction are, characteristically, priors about how the body will feel and what will fix it. It also supplies a measurable channel. If relapse is a precision failure, the interoceptive stream is where we should expect to detect it.

The Model Stated

The Precision-Collapse Model makes one central claim: **a relapse event is a transient, state-dependent collapse of precision control in which the balance of precision shifts so that entrenched maladaptive priors capture policy selection.** Three coupled shifts constitute the collapse:

an up-weighting of aversive interoceptive prediction errors, so that a spike of distress, withdrawal, or craving is treated as urgent, high-confidence evidence demanding action; (2) a down-weighting of exteroceptive and contextual counter-evidence and of the goal-directed “recovery” generative model (the reasons, plans, and identities that normally compete with the using prior); and (3) a loss of top-down prefrontal precision supply, so that the executive system fails to lend confidence to the recovery model, and control defaults to the faster, habitual controller [9,11]. These three shifts are summarized in Figure 1

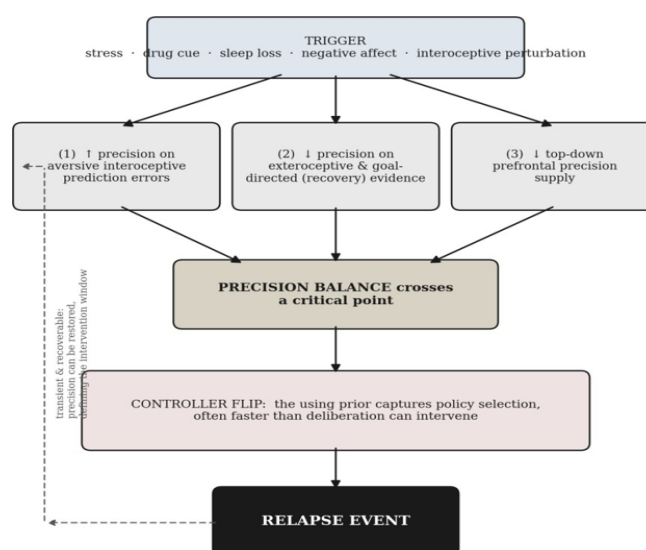


Figure 1: The Precision-Collapse Model. A State-Level trigger drives Three Coupled Shifts in Precision that

Converge to Move the Precision Balance Past a Critical Point, Flipping Behavioral Control from the Goal-Directed Recovery Model to the Entrenched using Prior. Because the Collapse is Transient and Recoverable, it Defines a Window in which Intervention is Possible.

The essential move is to treat relapse as a bifurcation in a dynamical system rather than a threshold crossing on a single dimension of craving. Precision balance is a control parameter; when a trigger perturbs it past a critical point, the system reorganizes abruptly: control flips from the recovery model to the using prior, often faster than deliberation can intervene. This accounts for the phenomenology patients report: relapse that arrives suddenly, feels ego-dystonic, and is poorly predicted by the person's stable intentions. The trait models describe the shape of the landscape; PCM describes the ball rolling off the ridge. Figure 2 renders this as a deforming potential landscape.

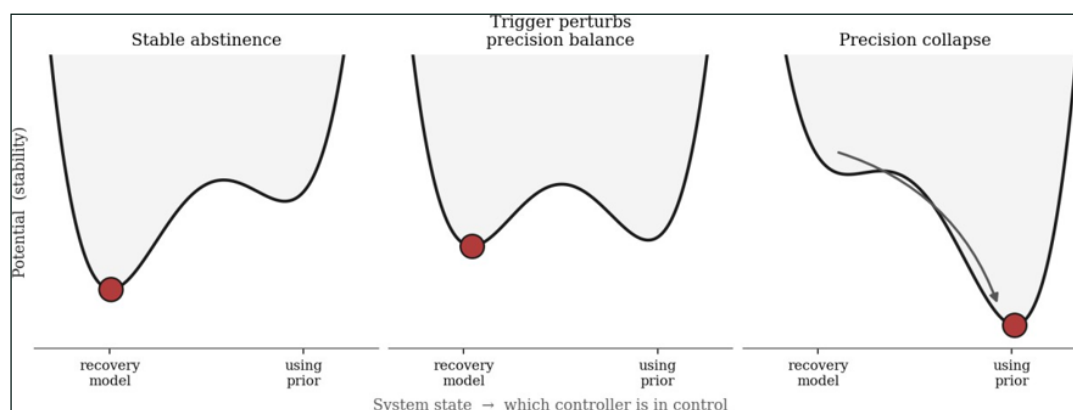


Figure 2: Relapse as a Bifurcation in a Potential Landscape. In Stable Abstinence (left) the Recovery Model Occupies a deep, Protected Basin. A Trigger Perturbs the Precision Balance and Lowers the Barrier (center). When Precision Collapses (right), the Landscape tilts until the Recovery Basin no Longer holds and the State Rolls into the Using Prior. The Transition is Abrupt and State-Dependent Rather than a Gradual slide Along a Single Craving Dimension.

Precision collapse is, by construction, transient and recoverable. This is a feature, not a bug: it explains why the same individual, with the same underlying pathology, is relapse-vulnerable at some moments and robust at others, and it identifies the window in which intervention is possible.

Existing Theories as Special Cases

PCM is intended to subsume rather than replace the major accounts, each of which describes a chronic distortion of precision that lowers the threshold for acute collapse:

Incentive-sensitization becomes chronically elevated precision on drug cues: sensitized “wanting” is, in inferential terms, pathological confidence that a cue signals an available, need-satisfying outcome, imbuing it with attention-commanding salience independent of hedonic “liking” [3,5]. Dopamine’s role as a precision signal makes this translation almost literal [17].

Allostasis and opponent-process becomes a shift in the priors about baseline bodily state together with amplification of aversive interoceptive prediction errors: the anti-reward set point raises the resting level of “something is wrong” signals, so that ordinary perturbations more readily exceed the precision threshold for the relief-seeking prior [6-8].

Actions-to-habits-to-compulsions becomes a chronic reallocation of precision from the goal-directed to the habitual controller: as prefrontal, model-based inference loses precision relative to dorsal-striatal, model-free control, the system is pre-disposed to the controller flip that PCM places at the heart of the relapse event [9,10].

IRISA becomes a deficit in the prefrontal supply of top-down precision to the recovery model, the third of the three shifts above, providing the neuroanatomical substrate for collapse [11,12].

The parsimony of the account is its principal theoretical claim: four influential frameworks, developed largely in parallel, describe distortions of a single quantity, and the relapse event is what happens when a state-level perturbation exploits those distortions.

Relapse Triggers Share a Computational Signature

PCM predicts that the heterogeneous, clinically familiar precipitants of relapse should share one underlying effect: each biases the precision balance in the direction of collapse. Table 1 maps common triggers onto this signature.

Relapse trigger	Proximal effect on the system	Precision consequence (predicted)
Acute stress / HPA activation	Elevated arousal and threat signaling	Up-weights aversive interoceptive prediction errors; narrows to the relief-seeking prior

Relapse trigger	Proximal effect on the system	Precision consequence (predicted)
Negative affect / dysphoria	Amplifies the allostatic “wrongness” signal	Raises the precision of the “using will relieve this” prior above threshold
Drug-cue exposure	Sensitized cue reactivity	Injects high-precision evidence that a satisfying outcome is available
Sleep deprivation / fatigue	Degrades prefrontal function	Reduces top-down precision supplied to the goal-directed recovery model
Interoceptive perturbation (pain, hunger, withdrawal)	Bodily signal misread through biased priors	Miscategorizes a benign or non-drug state as a using-relevant one
Abstinence-violation, or a “slip”	Prior-confirming outcome after a lapse	Sharpens the using prior and collapses the recovery model (see 4.1)

Table 1: Common Relapse Triggers Reinterpreted as Convergent Perturbations of Precision Balance. The Claim is not that these Triggers are Identical but that they share a Final Common Computational Pathway.

Falsifiable Predictions

A conceptual model earns its place only if it can be wrong. PCM generates several predictions that distinguish it from trait accounts:

First, measures of interoceptive precision (for example, calibrated interoceptive accuracy and metacognitive confidence tasks) should predict the timing of relapse over and above trait severity, and should degrade in the hours preceding a lapse, not merely differ between relapsers and non-relapsers as stable groups.

Second, within-person state manipulations that restore precision balance (slow-paced breathing, interoceptive exposure, mindful attention to bodily signals) should reduce lapse probability specifically by increasing tolerance of aversive interoceptive prediction errors, a mediation that is measurable.

Third, computational modeling of sequential choice tasks administered longitudinally should reveal state-dependent precision parameters (for example, inflated policy precision on drug-relevant options, or reduced precision on goal-directed options) that shift in the peri-relapse window, rather than static parameters that differ

only between groups.

Fourth, neuromodulatory or pharmacological manipulations that alter the insula, anterior cingulate, and prefrontal precision circuitry should move relapse risk in the direction PCM specifies, and lesion evidence should continue to converge on that circuit [25,27].

Should interoceptive-precision dynamics fail to track relapse timing within individuals, or should the triggers in Table 1 prove to have no common precision signature, the model is disconfirmed.

Discussion

The contribution of the Precision-Collapse Model is threefold. It shifts the unit of analysis from disease to inference and from trait to event, giving the field a principled language for the acute transition that clinicians actually manage. It is parsimonious, deriving four major frameworks from distortions of a single, well-characterized quantity. And it is translational, because precision is something the nervous system already regulates and that behavioral and neuromodulatory interventions can plausibly target.

Clinical Implications

PCM reinterprets several established interventions as, in effect, precision-retraining.

Mindfulness-based relapse prevention teaches patients to attend to craving and bodily discomfort with acceptance and non-judgment, letting urges rise and fall without acting. This is precisely the operation of declining to treat an aversive interoceptive prediction error as high-precision evidence demanding action [29,30]. Studies of these programs find that awareness and acceptance mediate reductions in craving, consistent with the proposed mechanism [30]. Mindfulness-Oriented Recovery Enhancement targets the interface of attention, appraisal, and emotion in a manner readily restated as the re-weighting of interoceptive and reward-relevant precision [31].

The classic abstinence-violation effect, the disproportionate spiral that can follow a single “slip”, receives a clean reformulation. Under PCM, a lapse supplies exactly the prior-confirming, high-precision evidence (“I have used; I am using again”) that sharpens the using prior and collapses the recovery

model, producing a self-amplifying cascade [32]. This reframing is not merely descriptive: it implies that the therapeutic target after a slip is the precision of the interpretation, not the slip itself.

Most consequentially, PCM motivates a computational relapse signature for just-in-time intervention. Because precision collapse is transient and state-dependent, ambulatory sensing of its correlates (autonomic arousal, sleep, affect, and brief interoceptive probes delivered on a smartphone) could in principle detect the peri-collapse window and trigger a targeted micro-intervention before the controller flips. This reframes digital mental health for addiction away from generic check-ins toward mechanism-guided, closed-loop support.

Dissolving the Disease-Versus-Choice Dichotomy

Debates about addiction have long been polarized between a brain-disease framing and a choice or learning framing [2,21]. PCM is neither, and in being neither it dissolves the opposition. Relapse is disturbed inference in a coupled agent-environment system: the machinery is intact and doing exactly what inference machinery does, but it is doing so under distorted priors and mis-regulated precision, in an environment richly structured around use. This is not a moral failure, and it is not a fixed lesion; it is a predictable dynamical event. That framing has an ethical payoff. It reduces the stigma that attaches both to “weak will” and to “broken brain,” and it restores a defensible sense of agency: precision is trainable, the window is real, and the person is a participant in stabilizing their own inference.

Limitations and Future Directions

The model is conceptual and awaits formal implementation. Precision is not yet cleanly and non-invasively measurable in humans, and interoceptive-accuracy measures remain contested; without careful operationalization, a precision account risks the unfalsifiability that has dogged some applications of the free-energy principle. The predictions in Section 3.7 are offered as a hedge against that risk, and the immediate priorities are a formal computational instantiation (for example, a Markov-decision-process model whose precision parameters are fit to longitudinal choice data) and prospective, within-person studies that test whether interoceptive-precision dynamics track relapse timing. The framework should also be examined transdiagnostically: if PCM is correct,

behavioral addictions and, more speculatively, other disorders of compulsion should show analogous precision dynamics, and the interoceptive channel should generalize beyond substances to any relief-seeking prior anchored in a bodily state.

Conclusion

Relapse has resisted a century of theory in part because we have kept asking a trait question about a state event. The Precision-Collapse Model proposes that a relapse is a failure of inference: a transient collapse of precision control that lets an over-confident, body-anchored prior capture the wheel. The account unifies the field's major frameworks as chronic distortions of a single quantity, reinterprets our best psychological treatments as ways of retraining that quantity, and specifies the measurement science (interoceptive precision assessed longitudinally and ambulatorily) that could turn relapse from an unpredictable catastrophe into a detectable, interruptible event. The task now is to build that measurement science, and to find out whether the model is wrong.

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