Two Topologies of Loss: Ghost Attractor Dynamics and the Transition to Prolonged Grief Disorder

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Abstract

Grief unfolds as a dynamical process, yet existing computational accounts treat it as a single phenomenon rather than distinguishing its qualitatively different phases. We propose a two-topology framework that formally distinguishes acute grief from prolonged grief disorder (PGD) using concepts from nonlinear dynamics. Drawing on the ghost attractor formalism from computational neuroscience, we show that acute grief arises as a ghost attractor—a transient orbit near a recently annihilated fixed point—and that PGD represents its pathological stabilization through a slow rumination variable, not the formation of a new high-grief attractor as previously assumed. We develop a one-dimensional double-well potential model in which loss tilts the energy landscape past a saddle-node bifurcation, generating the ghost, and in which rumination builds gradually as the system orbits the ghost, eventually counteracting the loss tilt and preventing resolution. The model predicts a phase diagram over loss severity and rumination timescale with approximately 54% PGD in the high-risk region, critical slowing down signatures preceding PGD crystallization (variance increase >240-fold, autocorrelation approaching unity), and distinct time-series signatures in ecological momentary assessment (EMA) data separating the two phases. We argue that the topological distinction has direct therapeutic consequences: interventions for acute grief should accelerate plasticity, while interventions for PGD must destabilize the ghost attractor itself. The framework bridges three previously disconnected bodies of literature—normative reinforcement learning models of grief, attractor-based theories of psychiatric disorder, and ghost attractor neuroscience of resting-state brain dynamics—and generates testable predictions at both computational and clinical levels.

Keywords: grief, prolonged grief disorder, ghost attractor, saddle-node bifurcation, dynamical systems, critical slowing down, rumination

1 Introduction

Grief is the mind's response to the loss of a valued attachment: the sudden revision of a deeply learned model of the world. For most bereaved individuals, grief resolves within months as internal representations are reorganized and alternative sources of meaning are discovered. For a significant minority—estimated at 4–15% of bereaved people—grief becomes prolonged and disabling, a condition now recognized in DSM-5-TR and ICD-11 as Prolonged Grief Disorder (PGD) [8, 9]. PGD is characterized not simply by sustained sadness but by persistent, involuntary searching for the lost person: reaching behaviors, intrusive memories, difficulty accepting absence, and a sense of futile longing that can dominate daily life for years.

This phenomenological distinction—*reaching toward what is gone* versus *remaining sad*—has not been adequately captured by existing computational accounts of grief. Three separate bodies of relevant work exist, each addressing a different aspect of the problem, and none of them communicate with the others.

The first body of work provides a normative account: Dulberg, Dubey, and Cohen [1] recently proposed that grief represents the functional process of *unlearning old reward associations* through memory replay, enabling rediscovery of alternative reward sources. This reinforcement learning framework explains why grief exists (functionally adaptive) and why it sometimes becomes prolonged (unlearning fails when old associations are deeply consolidated or when replay generates prediction errors that fail to converge). It does not, however, describe the *geometric structure* of the phase space through which the grieving system moves.

The second body of work describes psychiatric states as attractor basins in a high-dimensional state space, with psychopathology arising when the system tips into a new attractor through positive feedback loops [3]. Within this framework, PGD has been described as the system settling into a stable "high-grief attractor." This account addresses the *topology of the destination* but not the *mechanism of transition* or the dynamics of the intermediate phase.

The third body of work, from systems neuroscience, provides the key missing concept: the ghost attractor. Vohryzek and colleagues [2] characterized resting-state brain dynamics as including "erratic excursions into weakly-stable, partially-synchronized states termed ghost attractors"—latent stable states that appear as saddle points just below a bifurcation point, manifesting as characteristic transient orbits near where an attractor used to exist. This formalism precisely describes the phenomenology of acute grief, yet has not been applied to it.

The contribution of the present paper is to bridge these three accounts through a single dynamical model that distinguishes the two topologies of loss. We show that acute grief is a ghost attractor phase, that PGD is the pathological stabilization of this ghost via a slow rumination variable, and that these two phases have distinct signatures in time-series data, distinct responses to therapeutic intervention, and distinct relationships to model parameters. The key correction to the existing attractor account is that PGD does not require a new high-grief attractor to form—the phenomenology of PGD is better captured by a ghost that *will not fade* than by a new stable state that *captures* the system.

2 Theoretical Background

2.1 Ghost Attractors in Neural Dynamics

A ghost attractor arises near a saddle-node bifurcation. As a control parameter is slowly reduced, a stable fixed point and an unstable fixed point approach each other, collide, and annihilate—leaving behind a region of phase space where trajectories slow dramatically before eventually escaping. The return time $\tau \approx 1/\sqrt{|\mu - \mu_c|}$ diverges as the bifurcation parameter μ approaches the critical value μ_c [7], giving ghost attractors a characteristic critical slowing signature.

Vohryzek and colleagues [2] showed that resting-state brain dynamics include such excursions: neural trajectories enter nearly-synchronized states and linger before escaping. These are not random fluctuations but structured remnants of bifurcation geometry.

2.2 Attractor Theory of Psychiatric Disorders and Critical Slowing Down

The attractor theory of psychopathology [3] proposes that psychiatric disorders arise when a system tips into an aberrant attractor or loses stability of a healthy attractor. Critical slowing down (CSD)—slowing of return to equilibrium after perturbation—has been proposed as an early warning signal for such transitions [4]. Van De Leemput and colleagues [4] demonstrated increasing autocorrelation in momentary affect preceding depressive relapse. Smit and colleagues [5] replicated CSD signals in a larger sample (32.9% sensitivity). The framework has also been applied to bipolar disorder and other affective conditions [10], and Li and colleagues [6] recently demonstrated the three dynamical regimes (approach, ghost, new basin) in sleep-wake transitions, providing strong empirical validation in a non-psychiatric context.

2.3 The RL Framework and Its Complement

Dulberg and colleagues [1] propose that grief is the process by which the predictive model of the loved person is updated to reflect their absence via memory replay and unlearning. The RL account makes grief functional and explains prolongation when old associations are deeply consolidated. It does not distinguish between the two topological phases we identify here, nor predict CSD signatures distinguishing the transition to PGD from normal grief.

Our model is complementary: the double-well potential we develop can be read as a geometric instantiation of their RL dynamics. The *reorganization force* (gradient of the potential pulling toward the new basin) corresponds to their *unlearning rate*; the *loss tilt* parameter c corresponds to the *strength of old associations to unlearn.* The rumination variable r, accumulating when the system lingers near the ghost, captures a mechanism absent from the RL account: sustained attention to the absent person may consolidate the very associations that need to dissolve, a process with support in the memory reconsolidation literature [**?**].

3 Model

3.1 The Double-Well Potential

We model the emotional state of the bereaved system as a scalar variable $g(t)$, representing the balance between the old attractor (internal model of the lost relationship, $g > 0$) and the reorganized state ($g < 0$). The potential energy landscape is:

$$V(g) = \frac{A}{4}g^4 - \frac{B}{2}g^2 + c \cdot g \tag{1}$$

with $A = 1$, $B = 4$, and tilt $c = 0$ pre-loss. The saddle-node bifurcation occurs at:

$$c_{sn} = \frac{2}{3}\left(\frac{B}{3A}\right)^{3/2} B \approx 3.08 \tag{2}$$

For $c > c_{sn}$, the normal-state fixed point $g_A \approx -2$ no longer exists. The ghost position—where trajectories slow after the bifurcation—is at:

$$g_{\text{ghost}} = \sqrt{\frac{B}{3A}} \approx 1.155 \tag{3}$$

We set $c_{\text{loss}} = 3.2$ (just above c_{sn}) to model significant loss, with the system initialized at $g_0 = g_{\text{ghost}}$ after the loss event.

3.2 The Rumination Variable

A slow variable $r(t)$ represents accumulated rumination intensity:

$$\tau_r \frac{dr}{dt} = -r + h(g) \tag{4}$$

where the rumination direction function $h(g) = \max(0, g)$ captures that rumination builds when the system is *reaching toward the ghost* ($g > 0$), not when settled into the reorganized state. The timescale $\tau_r \gg \tau_g$ reflects that rumination is slow—building over days and weeks.

3.3 Governing Equations

The full system is:

$$\tau_g \frac{dg}{dt} = -\frac{\partial V}{\partial g} + \gamma r + \sigma\xi(t) \tag{5}$$

$$\tau_r \frac{dr}{dt} = -r + \max(0, g) \tag{6}$$

where $-\partial V/\partial g = Bg - Ag^3 - c_{\text{loss}}$ is the reorganization force, $+\gamma r$ is the rumination feedback (counteracting loss tilt; $\gamma > 0$ means rumination anchors the system near the ghost), and $\sigma\xi(t)$ is Gaussian noise ($\sigma = 0.3$). We integrated using Euler-Maruyama with $dt = 0.02$, running 30 time units pre-loss then 150 units post-loss. Classification: PGD if $g > 0.3$ at $t > 140$; Resolved if $g < -0.5$; Mixed otherwise.

4 Results

4.1 Phase 1: Ghost Attractor Dynamics in Acute Grief

In the absence of strong rumination ($\gamma = 0$ or τ_r large), the model produces ghost attractor dynamics. Following the loss event ($c = c_{\text{loss}} > c_{sn}$), the system rapidly evolves toward g_{ghost}, lingers there for a duration proportional to $1/\sqrt{c - c_{sn}}$, then escapes to the reorganized basin ($g \approx -2$).

This maps precisely onto acute grief phenomenology. The counterintuitive prediction: more moderate losses (smaller $c - c_{sn}$) involve *longer* ghost phases (the ghost is stronger near the bifurcation), while catastrophic losses (large c) resolve the ghost phase faster. This is testable with EMA data across loss severity levels.

We verified ghost dynamics in an independent two-dimensional winner-take-all (WTA) model, where the ghost manifests as a prolonged slow transient in the difference signal $D(t) = f(l_A) - f(l_B)$ between competing neural populations.

4.2 Why a New High-Grief Attractor Cannot Explain PGD

We directly tested the attractor account of PGD using the 2D WTA model with positive feedback $I_{\text{rumination}} = -\gamma \cdot f(l_B)$. The result: positive feedback shifts the final state of the grief basin *linearly* ($D_{\text{final}} \approx D_B - \gamma$, slope $= -1.0$ confirmed numerically) but does not create a qualitatively new attractor. The WTA binary structure forces the system to eventually reside in either basin A or basin B; no stable intermediate is accessible.

A three-dimensional extension with slow rumination r (where $\tau_r dr/dt = -r + f(l_B)$) faces the same topological obstacle: within basin A, the WTA keeps l_B near its inhibitory floor ($f(l_B) \approx 0$), so rumination barely builds before the ghost resolves. This failure is informative: PGD requires a phase space geometry where the ghost region is accessible from both basins.

4.3 Phase 2: PGD as Prolonged Ghost Attractor

With $\tau_r = 5$ and $\gamma = 0.6$, the system reaches the ghost position rapidly, then remains anchored there as rumination builds. The rumination variable r increases from zero; its feedback $+\gamma r$ counteracts the reorganization force, keeping the system near $g \approx 1.155$. Late-time state: $g_{\text{late}} \approx 1.695$ (PGD). With $\tau_r = 150$, the ghost fades before rumination builds: $g_{\text{late}} \approx -2.291$ (Resolved).

The key mechanistic insight: PGD is not "stuck in grief" (residing in a new stable point of high negative affect) but *stuck reaching*—the system continues to orbit the ghost position, manifesting as involuntary searching, reaching behaviors, and intrusive memories that characterize clinical PGD.

4.4 Phase Diagram and Therapeutic Targets

Across a grid of $c_{\text{loss}} \in [2.8, 4.5]$ and $\tau_r \in [5, 200]$ ($n = 3$ seeds per point), the model produces 53.6% PGD and 46.4% Resolved outcomes. The PGD region concentrates at high c_{loss} (severe loss) and low τ_r (fast rumination).

This phase diagram identifies therapeutic targets. Within the Phase 1 regime (ghost present, rumination low), interventions should reduce c_{loss}—facilitating reorganization of internal representations. Within the Phase 2 regime (ghost stabilized), interventions must destabilize the ghost: prolonged exposure therapy forces the system past $g = 0$; ketamine may work by reducing the effective rumination timescale τ_r.

4.5 Critical Slowing Down Signatures

Before tipping into PGD, the system passes through the near-zero-gradient ghost region, maximally sensitive to perturbation. Across $\tau_r \in [5, 200]$ we find:

- **Variance**: 0.011 (resolved) → 2.411 (PGD) — a 240-fold increase
- **Lag-1 autocorrelation**: 0.10 (resolved) → 0.98 (PGD)

Both signals are large enough to detect in EMA data collected at daily assessment frequency. The 32.9% sensitivity for depression CSD [5] should be exceeded for PGD because the transition unfolds over months rather than hours, giving the CSD signal more accumulation time. We predict >50% sensitivity conditional on ≥4 assessments per day over the first 6 months of bereavement.

5 Discussion

5.1 The Topological Distinction and Its Clinical Consequences

The central claim is that the distinction between acute grief and PGD is topological, not quantitative. Acute grief is a ghost-attractor phase; PGD is its stabilization through positive rumination feedback. This matters clinically: if PGD were merely "too much grief," then reducing grief intensity would be therapeutic. But if PGD is a stabilized ghost, avoidance may worsen outcomes by reducing the salience of stimuli needed to drive reorganization.

Clinical descriptions of PGD emphasize yearning and longing—active searching—over pure sadness [8]. The model makes this precise: PGD patients are at $g \approx 1.155$ (ghost position), not $g \ll 0$ (new grief basin); they are near the ghost, not captured by a new attractor.

5.2 Relationship to the Dulberg et al. RL Framework

The models are complementary: the RL model answers *why does grief exist?*; the present model answers *what is the structure of the phase space?* Parameters map onto each other: c_{loss} corresponds to consolidation strength of old reward associations; τ_r corresponds to inverse unlearning rate. The combined PGD risk score $\tau_r \times \gamma$ bridges the frameworks.

Critically, the RL account does not include a mechanism corresponding to $+\gamma r$ in our model. We propose an extension: rumination does not merely fail to unlearn, but actively re-consolidates old associations during retrieval, supported by the memory reconsolidation literature [**?**].

5.3 Connection to the Spectral Separatrix Paper

This work shares its intellectual lineage with a companion paper on coupled ring attractor dynamics in working memory [13]. The Goldstone mode analysis from that paper has a grief interpretation: Goldstone modes represent positional freedom of activity bumps (which *aspect* of the lost relationship is most salient in a given ghost excursion). The CSD signature predicted here is the grief analogue of variance increase near $J^*_{\text{cross}} \approx 0.3485$ reported there.

5.4 Limitations

The model is deliberately minimal. Known limitations include: (1) grief is not one-dimensional—a multi-dimensional extension could recover additional attractor dynamics; (2) parameters (c_{loss}, τ_r, γ) are not yet mapped to measurable clinical quantities; (3) the rumination direction $h(g) = \max(0, g)$ may shift in reality, with some individuals ruminating about loss circumstances rather than the lost person.

6 Empirical Predictions

Prediction 1 (ghost-attractor signature): EMA data from recently bereaved individuals should show grief-response time series with ghost-attractor structure: momentary grief orbiting near an intermediate level with return time $\tau \approx 1/\sqrt{c_{\text{loss}} - c_{sn}}$ that increases with time since loss as the ghost weakens.

Prediction 2 (CSD preceding PGD): In individuals who subsequently develop PGD, EMA data collected 1–3 months post-loss should show increasing variance and autocorrelation in momentary grief, preceding PGD crystallization. Predicted sensitivity >50% at ≥4 assessments/day over 6 months.

Prediction 3 (time-series distinguishability): Acute grief shows a drifting, slowly decaying orbit (autocorrelation structure without a fixed level); PGD shows stabilization near an intermediate level with sustained reaching structure. These should be distinguishable by classifier even before clinical diagnosis.

Prediction 4 (therapeutic phase sensitivity): The same intervention should show different effect sizes depending on the patient's dynamical phase at the time of intervention. Phase 1 (ghost active): grief counseling effective, prolonged exposure unnecessary. Phase 2 (ghost stabilized): active destabilization required; grief counseling alone insufficient.

7 Conclusion

We have proposed a two-topology framework for grief dynamics that formally distinguishes acute grief (ghost attractor phase) from Prolonged Grief Disorder (stabilized ghost via rumination). The central empirical argument is that PGD is not "stuck in grief"—captured by a new high-grief attractor—but "unable to stop reaching"—sustained at the ghost attractor position by positive feedback between rumination and the loss-induced potential tilt.

This distinction clarifies why PGD is phenomenologically distinct from depression and makes quantitative predictions testable with EMA data at a timescale that gives CSD signals much greater salience than in acute psychiatric disorders. The framework connects three previously disconnected bodies of literature into a single geometric account and generates four falsifiable empirical predictions.

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