

Paper 52: The Kibble-Zurek Mechanism and the Topology of Trauma

PTSD vs. Depression as Fast vs. Slow Quench Through γ_c

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Abstract

The Kibble-Zurek mechanism (KZM) describes topological defect formation when a system is driven through a phase transition at finite rate. Applied to the Wike Coherence framework: trauma is a quench through the critical decoherence threshold γ_c . Fast quenches (acute trauma) produce many localized defects (PTSD). Slow quenches (chronic stress) produce few diffuse defects (depression). Using 3D Ising exponents ($\nu = 0.6298$, $z = 2.02$), the defect density scales as $n \sim \tau_Q^{-0.83}$. At 100:1 timescale ratio, acute trauma produces ~46x more topological defects than chronic stress of equal total load. The defect morphology maps directly onto the clinical distinction between PTSD (specific triggers, intact between episodes) and depression (global dysfunction, no specific triggers). Treatment modalities map onto annealing protocols: EMDR = local defect surgery for PTSD; CBT = global slow anneal for depression.

1. The Kibble-Zurek Mechanism

When a system is quenched through a continuous phase transition, the relaxation time diverges near the critical point:

$$\tau_{\text{relax}} \sim |\epsilon|^{-z\nu}$$

where $\epsilon = (\gamma - \gamma_c)/\gamma_c$ is the reduced control parameter. At a quench rate $1/\tau_Q$, there is a freeze-out time where the system can no longer keep up:

$$t_{\text{freeze}} \sim \tau_Q^{z\nu / (1 + z\nu)}$$

The correlation length at freeze-out sets the defect density:

$$\xi_{\text{freeze}} \sim \tau_Q^{\nu / (1 + z\nu)}$$

The defect density (defects per unit volume in d dimensions):

$$n_{\text{defects}} \sim \xi_{\text{freeze}}^{-d} \sim \tau_Q^{-d\nu / (1 + z\nu)}$$

2. Defect Scaling with 3D Ising Exponents

For the 3D Ising universality class:

- $\nu = 0.6298$ (correlation length exponent)
- $z = 2.02$ (dynamical critical exponent, Model A)
- $d = 3$ (spatial dimension of neural coherence field)

The KZM exponent:

$$\begin{aligned} \alpha_{\text{KZM}} &= d \cdot \nu / (1 + z \cdot \nu) \\ &= 3 \cdot 0.6298 / (1 + 2.02 \cdot 0.6298) \\ &= 1.8894 / 2.2722 \\ &= 0.8316 \end{aligned}$$

$$n_{\text{defects}} \sim \tau_Q^{-0.83}$$

3. What Are the Topological Defects?

The brain maintains a coherence field $\Psi(x,t)$ across neural tissue. When quenched through γ_c , this field breaks into domains. At domain boundaries, the phase is discontinuous. These discontinuities are topological defects classified by homotopy theory:

Defect Type	Topological Class	Clinical Manifestation
Domain walls	π_0	Dissociative boundaries ("I was there but I wasn't")
Vortex lines	π_1	Flashback triggers (topologically protected, resistant to extinction)
Point defects	π_2	Hypervigilance nodes (persistent scattering centers)

Flashbacks are topologically protected. A vortex in the coherence field -- where the phase winds by 2π around a closed loop -- cannot be removed by smooth local deformation. This is why flashbacks resist extinction learning: they are not merely strong memories, they are topological features that require topological operations to remove.

4. PTSD vs. Depression: The Central Prediction

Consider two individuals exposed to equal total trauma load:

$$\Gamma_{\text{total}} = \int \gamma(t) dt$$

Case A -- Acute Trauma (PTSD):

$\gamma(t) = \gamma_{\text{max}}$ for short duration Δt_{acute}

τ_Q is small (fast quench)

Case B -- Chronic Stress (Depression):

$\gamma(t) = \gamma_{\text{chronic}}$ for long duration $\Delta t_{\text{chronic}}$

τ_Q is large (slow quench)

Same total load: $\gamma_{\text{max}} \Delta t_{\text{acute}} = \gamma_{\text{chronic}} \Delta t_{\text{chronic}}$

KZM prediction:

$$n_{\text{defects}}(\text{PTSD}) / n_{\text{defects}}(\text{depression}) = (\tau_{\text{chronic}} / \tau_{\text{acute}})^{0.83}$$

For $\tau_{\text{chronic}} / \tau_{\text{acute}} = 100$:

$$n_{\text{defects}}(\text{PTSD}) / n_{\text{defects}}(\text{depression}) = 100^{0.83} = 46.2$$

Acute trauma produces ~46x more topological defects than chronic stress of equal total integrated load.

5. Defect Morphology

PTSD (fast quench):

- ξ_{freeze} is SMALL (small τ_Q)
- Many defects, each sharply localized
- Each defect = specific identifiable trigger (a sound, a smell, a scene)
- Coherence INTACT between defects (person functions normally until triggered)
- Clinical: "I'm fine until I hear a car backfire, then I'm back in the firefight"
- Phase diagram: many small incoherent islands in a sea of coherence

Depression (slow quench):

- ξ_{freeze} is LARGE (large τ_Q)
- Few defects, each spanning large regions (extended domain walls)
- Global degradation without specific catastrophic breaks
- Clinical: "Everything is gray but nothing specific is wrong"
- Phase diagram: few large incoherent domains, partially ordered

This maps PRECISELY to the clinical distinction between PTSD and depression.

6. Treatment as Annealing Protocols

EMDR for PTSD (Local Defect Surgery)

EMDR (Eye Movement Desensitization and Reprocessing):

1. Identify a specific topological defect (flashback trigger)
2. Locally raise coherence (bilateral stimulation increases neural synchrony = local reheating above T_c)
3. While local region is above T_c , defect is no longer topologically protected
4. Re-cool slowly (controlled memory reconsolidation) -- this time without the defect
5. Defect is annihilated

This works for PTSD because defects are LOCALIZED. Address them one at a time. Each EMDR session targets one defect. Efficient because defects are small.

CBT for Depression (Global Slow Anneal)

Cognitive Behavioral Therapy:

1. Defects are large-scale domain walls spanning extended regions
2. Cannot locally reheat and re-cool -- defect is too large
3. Instead: slowly raise global coherence through behavioral activation, cognitive restructuring
4. This is a slow global anneal -- gradually re-establishing long-range order

5. Domain walls shrink and eventually annihilate as correlation length grows

This is why CBT requires many sessions over months -- you are performing a slow global anneal of extended defect structure.

Mismatched Treatment Prediction

- **EMDR on depression:** Less effective. No sharp point defects to target. Local reheating doesn't reach the extended domain wall.
- **CBT alone for severe PTSD:** Slower. Global annealing doesn't efficiently target localized vortex cores.

This matches clinical evidence: EMDR is first-line for PTSD, not for depression. CBT is effective for depression, slower for PTSD alone.

Ketamine as Thermal Quench

Ketamine (NMDA antagonist) raises effective neural temperature above the glass transition T_g :

$$T_{\text{eff}}(t) = T_0 + \Delta T * f(t)$$

During the dissociative window (~40-60 min), $T_{\text{eff}} > T_g$. The frozen state melts. The system can re-equilibrate. Upon re-cooling, it freezes into a DIFFERENT configuration -- potentially with lower frustration.

Prediction: Ketamine alone produces transient remission. Ketamine + psychotherapy during the neuroplasticity window produces durable results (restructuring J_{ij} while the glass is melted).

Psychedelics as Slow Annealing

Psilocybin, LSD: raise T_{eff} just above T_g and hold for HOURS (4-12h). This is slow annealing per the Geman-Geman theorem:

$$T(t) \geq \Delta_{\text{max}} / \log(1 + t)$$

The extended duration above T_g allows exploration of many configurations. The "mystical experience" = finding a significantly lower-energy state.

7. Testable Predictions

Prediction 1: HRV Fragmentation Index

Define fragmentation index F = number of sign changes in successive RR-interval differences per unit time.

- PTSD: HIGH F (many sharp transitions = point defects in autonomic coherence)
- Depression: LOW F , low overall HRV power (diffuse coherence loss)

$$\log(F) = -0.83 * \log(\tau_Q) + \text{const}$$

A log-log plot of fragmentation index vs. trauma duration should yield slope -0.83.

Prediction 2: Startle Probe Response

Standardized startle stimulus (sudden loud noise):

- PTSD: large, sharp HRV excursion + rapid recovery (probe hits near point defect)
- Depression: small, prolonged HRV perturbation + slow recovery (probe hits diffuse region)

Peak response / recovery time constant discriminates defect structures.

Prediction 3: Treatment Response Tracking

During EMDR for PTSD:

- F decreases in DISCRETE STEPS (each session annihilates specific defects)
- Between sessions, F is roughly constant (defects are topologically stable)

During CBT for depression:

- HRV power increases CONTINUOUSLY and gradually (domain walls shrink smoothly)
- No discrete steps expected

Prediction 4: Retrospective Quench Rate Estimation

For a population of trauma survivors:

$$\log(F) = -0.83 * \log(\tau_Q) + \text{const}$$

This is a hard, quantitative, falsifiable prediction derived from the Ising exponents.

8. Connection to Existing Framework

- **Paper 09** (Depression = Sustained Decoherence): KZM adds quench-rate dependence
- **Paper 16** (Hell = Central Sensitization): Wind-up IS a fast quench in the nociceptive system
- **Paper 24** (ACE Equation): Each ACE is a quench event; the total defect count is the sum over quenches
- **Paper 26** (Chronic Pain Phase Transition): $\gamma_c = 0.0016$ is the freeze-out threshold
- **Paper 37** (Coherence Trap): The detuned force is a specific quench protocol that produces the trap
- **Paper 44** (The Window): The window is the regime BEFORE the quench reaches γ_c

9. References

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