FLOW HIJACKED · LECTURE 50

AFTER DANGER

PTSD, Trauma, and the Altered Rules of Return

THE ORGANISING QUESTION

How can a response built to protect life become a regime that life can no longer easily leave?

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Educational scientific lecture · Not diagnosis, individual treatment, or emergency guidance

CONTENTS

PART I

AFTER DANGER: THE PUZZLE OF PERSISTENCE

A protective response can outlive the conditions that made it necessary.

1

Abstract

FLOW HIJACKED SYNTHESIS

This lecture begins with a problem that a diagnostic checklist can name but cannot, by itself, explain. A person survives an event, or a sequence of events, that required vigilance, rapid action, concealment, endurance, or emotional narrowing. Later, some of those responses remain available with extraordinary speed. A sound enters the body before it has become a thought. A neutral face acquires the force of a warning. Sleep becomes shallow. Attention is recruited toward what might go wrong. Avoidance brings immediate relief and quietly removes the experiences through which safety might become believable. The calendar says "after." The organism is not always able to return on command.

Post-traumatic stress disorder is real, serious, and often functionally costly. It is also heterogeneous. Exposure is not diagnosis; acute distress is not inevitably chronic illness; a questionnaire threshold is not a clinician-administered assessment; and PTSD is not interchangeable with grief, depression, anxiety, dissociation, moral injury, pain, sleep disruption, or the ordinary suffering that can follow an extraordinary event. Population research shows that potentially traumatic exposure is common, while PTSD risk varies substantially with the event, prior history, social conditions, measurement system, and time window [Benjet et al., 2016] [Liu et al., 2017] [Koenen et al., 2017]. Longitudinal research does not yield one canonical course. Recovery, persistence, delayed expression, fluctuation, and recurrence all occur, while estimates depend on sampling, attrition, definitions, and follow-up design [Steinert et al., 2015] [Brooks & Greenberg, 2024].

The argument therefore moves beyond the familiar cartoon of an "overactive amygdala" opposed by an "underactive prefrontal cortex." PTSD implicates learning and memory, context processing, threat discrimination, bodily regulation, sleep, attention, action, meaning, relationships, and interacting neural systems. These are not separate chapters pasted onto a disorder. They form a coupled brain–body–person–environment system. The same outward symptom may emerge through different pathways, and different states may be hidden beneath a similar total score. Functional impairment matters in its own right, across domestic, interpersonal, occupational, civic, and other domains, and cannot be inferred perfectly from symptom count [Jellestad et al., 2021].

Flow Hijacked develops one central proposal: **Threat-State Return Asymmetry**, or TSRA. The hypothesis is that trauma can alter conditional transition rules. Entry into a defensive state may become faster, more probable, or less dependent on discriminating contextual evidence, while return to safe engagement becomes slower, less reliable, or more dependent on unusually strong conditions. This is not claimed as an established mechanism, a hidden diagnosis, or a property of every person with PTSD. Related evidence comes from intensive longitudinal, learning, physiological, and dynamic-network research, but it is nonuniform and the joint asymmetry has not yet been demonstrated as a unitary PTSD process [Hertz-Palmor et al., 2026] [Littleton et al., 2023] [Lewis et al., 2023] [Danböck et al., 2024] [Wu et al., 2026]. A large multisite analysis that found no robust PTSD-group differences in several resting-state dynamic-connectivity measures is therefore part of the theory, not an inconvenience to be hidden [Tomas et al., 2025].

Later sections formalize TSRA through entry thresholds, cue-specific transient gain, generalisation width, dwell and escape times, recovery half-time, contextual gating, and alternative-state accessibility. Attractor geometry, metastability, hysteresis, network controllability, and non-normal transient amplification are used as mathematical possibilities with explicit evidential labels. No direct evidence located by the cutoff establishes that PTSD *is* a non-normal operator, that it occupies one deeper neural attractor, or that treatment changes basin depth. A useful model must risk being wrong.

The lecture also audits influential language. Bessel van der Kolk and *The Body Keeps the Score* receive serious treatment because the book helped many readers understand that trauma has embodied consequences. Yet cultural importance is not mechanism evidence. Autonomic, interoceptive, sensory, and somatic dysregulation can be investigated without claiming that an autobiographical episode is literally stored in muscle or fascia. The same discipline governs treatment: established trauma-focused psychotherapies outrank novelty, while body-oriented care, neuromodulation, medicines, digital interventions, and psychedelic approaches are judged separately by design, comparator, durability, adverse events, replication, access, and regulatory status.

October 7 and its prolonged aftermath appear as a substantial naturalistic case study, not as the organising centre of PTSD. The science is especially valuable where pre-event baselines or repeated measures exist; it is especially vulnerable where continuing threat is described as though it had ended. Throughout, the central human correction remains simple. Persistence is not proof of weakness, and adaptation is not proof that suffering is harmless. Recovery need not mean forgetting. It may mean that safety becomes discriminable, other actions become reachable, and the future is no longer governed entirely by the rules that once protected life.

CORE SENTENCE

PTSD is approached here not as a broken person remembering badly, but as a heterogeneous protective system whose learned rules of entry, persistence and return may have changed.

2

Author’s Note — Writing About a System That Tried to Protect

FLOW HIJACKED SYNTHESIS

I want to begin with a restraint. To write about trauma is to enter a field in which explanation can help, but can also take too much possession of another person’s life. A concept may illuminate one pattern and obscure another. A brain image can make suffering appear objective while quietly replacing the person who lives it. A diagnosis can open care, recognition, and language; it can also be heard as a verdict about identity. Even the word *recovery* can become cruel when it is used as a timetable imposed from outside.

Flow Hijacked approaches PTSD because the central puzzle of the project is persistence: how a response that made sense under one set of conditions can continue to organize perception, bodily readiness, action, relationship, and the future after those conditions have changed. That question is intellectually demanding. It is also morally demanding. If we begin by calling the response irrational, we miss what it learned. If we call every persistent response adaptive, we miss the suffering and constriction it can now produce. If we describe the brain as damaged, we can turn a history into a destiny. If we romanticise resilience, we can make people feel that their pain is a failure of character.

My role in this lecture is not to speak on behalf of everyone who has lived through trauma. There is no single interior voice of PTSD, no universal sequence from event to symptom, and no honest way to convert one story into a prevalence estimate or a mechanism. I will therefore use first person only to state an authorial commitment: I want the model to remain accountable to the person, and I want the person to remain larger than the model. I will not supply an invented vignette to make the science feel intimate. The intimacy is already present in what is at stake: sleep, trust, concentration, touch, work, parenting, solitude, movement through public space, and the ability to imagine a next hour that is not organized by defence.

The scientific language will sometimes become formal. We will speak about hidden states, transition kernels, stochastic processes, effective connectivity, controllability, and finite-time amplification. Mathematics can protect clarity when it tells us exactly what a claim would require us to measure. It becomes harmful when it is used as costume—when a basin is drawn and the drawing is allowed to stand in for a human experiment. Every formal object in this lecture must therefore return to lived meaning. An entry rate is about how often an ordinary cue recruits defence. An escape time is about how long it takes before speech, movement, sleep, or connection becomes available again. State accessibility is about whether a person has more than one possible next move.

The same restraint applies to authority. Foundational clinicians and theorists matter because they changed the questions the field was able to ask. They do not become correct in every detail because their language travelled widely. New neuroscience matters because it offers finer measurement. It does not become clinically decisive because its images are vivid. The largest study may miss an important subgroup; the smallest study may discover a signal and greatly overestimate it. The honest lecture has to hold all of this at once.

There is compassion in refusing false certainty. It leaves room for continuing danger, for cultural and developmental difference, for the coexistence of strength and impairment, and for recovery that does not look cinematic. It also leaves room for responsibility. Understanding why a defensive policy persists does not mean that every action taken under its influence is harmless. It means that responsibility can be pursued with a model rich enough to support change rather than shame.

CORE SENTENCE

I will use theory to make experience more intelligible, never to make a person smaller than the theory.

3

The Question That Organises the Lecture

FLOW HIJACKED SYNTHESIS

The question is not merely why a traumatic memory returns. It is this: **How can a response built to protect life become a regime that life can no longer easily leave?** The wording matters. "Response" acknowledges function. "Regime" suggests coordinated patterns rather than a single symptom. "Leave" directs attention toward transitions, not only toward what is present once the state has already arrived.

Suppose the condition of a person at time t is represented by a multiscale state vector

$$\mathbf{x}_t = (\mathbf{n}_t, \mathbf{a}_t, \mathbf{m}_t, \mathbf{b}_t, \mathbf{s}_t, \mathbf{e}_t),$$

where $\mathbf{n}_t$ denotes neural configuration, $\mathbf{a}_t$ autonomic and interoceptive condition, $\mathbf{m}_t$ memory and belief variables, $\mathbf{b}_t$ available actions, $\mathbf{s}_t$ social-relational conditions, and $\mathbf{e}_t$ the external environment. None of these components is directly observed in full. We encounter partial measurements: a symptom report, heart rate, sleep, behaviour in a task, words in an interview, a brain signal, or the testimony of someone who has watched the person change.

A general transition model can be written as

$$\mathbf{x}_{t+\Delta t} \sim K_{\boldsymbol{\theta}}(\cdot \mid \mathbf{x}_t, \mathbf{u}_t, \mathbf{c}_t, \mathcal{H}_t),$$

where $K_{\boldsymbol{\theta}}$ is a conditional transition kernel, $\mathbf{u}_t$ contains current inputs, $\mathbf{c}_t$ represents context, $\mathcal{H}_t$ is the relevant history, and $\boldsymbol{\theta}$ contains learned or slowly changing parameters. The empirical question is not whether this notation is elegant. It is whether trauma is followed, in some people, by reproducible changes in $\boldsymbol{\theta}$: changes that make particular defensive transitions more available and return transitions less available even when current evidence would support a different response.

That question reorganizes the lecture. Intrusions matter not only because they are memories but because they can trigger state entry. Avoidance matters not only because it is a symptom but because immediate relief can alter the future data available to the system. Sleep matters because transition thresholds tomorrow may depend on regulation tonight. Social support matters because another person can change both the actual environment and the interpretation of it. Shame and moral injury matter because threat can concern identity, belonging, or moral standing rather than bodily injury alone. Treatment matters not only because a symptom score falls but because a wider repertoire of states and actions may become reachable.

Longitudinal work already shows that averages conceal sharply different courses and that early symptoms, appraisals, regulation, and context can evolve together [Beaudoin et al., 2023] [Littleton et al., 2023]. Person-specific intensive modelling has begun to estimate relations among triggers, current threat, trauma memory, appraisals, and coping strategies, while also revealing that different individual networks can share only part of their organisation [Hertz-Palmor et al., 2026]. These studies justify asking transition questions. They do not establish one master kernel for PTSD.

The model must also distinguish three sources of persistence. The environment may still be dangerous. A learned policy may remain calibrated to earlier danger. Or ongoing danger and prior learning may be coupled so tightly that neither can be understood alone. Clinical language becomes inaccurate if it attributes the first entirely to pathology; romantic language becomes inaccurate if it treats the second as cost-free wisdom. The central task is to estimate the relation between current hazard, remembered hazard, predicted hazard, and the action policy they jointly recruit.

This is why the lecture will move across scales without reducing one to another. A synapse cannot by itself explain a household. A household cannot replace physiology. A network state is not an identity. Yet each level can constrain the transitions available at the others. The organising question is therefore not "Where is PTSD?" It is "Under what conditions does the system move, remain, and return—and how could those conditional rules become changeable again?"

CORE SENTENCE

The scientific object of this lecture is not a frozen symptom state but the conditional rule that determines what the whole system can become next.

4

Protection Is Not Pathology

BROADER SCIENTIFIC INFERENCE

Before discussing persistence, we have to respect the intelligence of defence. In danger, the organism should not deliberate as though every interpretation were equally likely. Attention narrows. Heart rate and respiration may change. Muscles prepare. Pain can recede from awareness. Ambiguous cues are given a less generous reading. Memory privileges what may help survival. Social behaviour can shift toward seeking protection, shielding others, hiding, obeying, confronting, freezing, or escaping. No single sequence describes everyone, and no simple "fight, flight, freeze" ladder captures the range. The governing point is that speed, bias, and bodily mobilisation can be appropriate when the cost of a missed threat is very high.

Recognising that protective role does not require us to say that every response was optimal, or that everything learned during danger should be preserved. It requires only that we begin with the conditions under which the system had to choose, not with the comfort of hindsight.

This is also why exposure cannot be treated as pathology. Cross-national surveys find potentially traumatic events across much of the population, while only a subset of exposures are followed by PTSD, and risk differs sharply by event type, prior exposure, social position, and other conditions [Benjet et al., 2016] [Liu et al., 2017] [Koenen et al., 2017]. The distinction protects two truths. Trauma exposure can matter profoundly even when no disorder develops. And a person can meet criteria for PTSD without their response being evidence of defective character.

In decision-theoretic terms, a defensive policy π_D can be understood relative to unequal errors. Let $H_t \in \{0, 1\}$ denote the presence of a consequential threat, and let $a_t \in \{\text{defend}, \text{engage}\}$. The locally optimal action minimizes conditional expected loss:

$$a_t^* = \arg\min_a \mathbb{E}[L(a, H_t) \mid \mathcal{I}_t],$$

where $\mathcal{I}_t$ is the information available at that moment. If $L(\text{engage}, 1)$—the loss from failing to defend during real danger—is enormous, then a lower threshold for defensive action can be rational. This equation is not a claim that the nervous system solves an explicit optimization problem, and it does not justify every defensive act. It makes visible why "false alarm" and "overreaction" are retrospective terms. From inside uncertain danger, the asymmetry of possible losses matters.

Pathology is therefore not located in the existence of fear, vigilance, startle, withdrawal, or distress. It concerns pattern, duration, context, impairment, and loss of flexibility. A response can have been protective when learned and become costly when it generalises too widely, activates too readily, fails to update, or excludes too many other actions. It can also remain partly protective and partly costly at the same time. Someone living under recurring violence may need vigilance and still suffer from the impossibility of rest. Someone who avoids one setting may reduce immediate distress while progressively surrendering work, connection, movement, or care.

This framing refuses two moral errors. The first is to shame the person for a system that learned under severe conditions. The second is to glorify suffering as though adaptation removes the need for treatment or social change. A protective origin is not a benign outcome. It is the beginning of an explanation that preserves dignity while asking exactly where the present costs arise.

CORE SENTENCE

A response should be judged in relation to the danger, information and costs under which it was learned—not mistaken for a defect merely because it was powerful.

5

When the Event Ends but the Regime Continues

BROADER SCIENTIFIC INFERENCE

An event can end in minutes. A learned regime has no such obligation. It may persist through recurrent intrusions, avoidance, altered sleep, scanning, bodily alarm, restricted movement, and changes in trust or interpretation. This persistence is often described as though the person simply fails to recognise that the past is over. That can be misleading. The person may know the date, place, and facts perfectly well. What has not updated may be the conditional readiness of the system: which cue is treated as sufficient, how rapidly defence is recruited, how long it remains active, and how much evidence is required before ordinary engagement feels possible.

Prospective studies show that early post-trauma distress can decline substantially on average while a subset of people follow persistent, fluctuating, or later-worsening courses [Shalev et al., 1998] [Santiago et al., 2013]. Longer naturalistic studies reveal wide variation in remission and chronicity, shaped partly by social conditions and co-occurring mental and physical problems [Steinert et al., 2015]. Recurrence after improvement is also real, but the literature uses inconsistent definitions, preventing a stable pooled estimate [Brooks & Greenberg, 2024]. These findings support persistence and heterogeneity. They do not, by themselves, reveal the mechanism that generates either.

A minimal way to express history dependence is to let the fast state $\mathbf{x}_t$ evolve under parameters $\boldsymbol{\theta}_t$ that themselves change slowly:

$$d\mathbf{x}_t = \mathbf{f}(\mathbf{x}_t, \mathbf{c}_t; \boldsymbol{\theta}_t)\, dt + \mathbf{G}(\mathbf{x}_t)\, d\mathbf{W}_t,$$
$$d\boldsymbol{\theta}_t = \varepsilon\, \mathbf{g}(\mathbf{x}_t, \mathbf{c}_t, \mathcal{H}_t)\, dt, \qquad 0 < \varepsilon \ll 1.$$

Here $\mathbf{W}_t$ represents unresolved fluctuation, $\mathbf{c}_t$ current context, and ε the separation between faster moment-to-moment dynamics and slower learning or adaptation. During danger, repeated defensive states can alter $\boldsymbol{\theta}_t$. When danger recedes, $\mathbf{x}_t$ may change quickly—one can leave the scene—while $\boldsymbol{\theta}_t$ changes more slowly. The equation does not prove that PTSD follows this system. It specifies one reason an ended event and a persistent response are not contradictory.

Persistence can be maintained across levels. Avoidance prevents disconfirming experience. Insomnia increases next-day reactivity. Bodily sensations become evidence for threat. A shame-based interpretation changes the meaning of social ambiguity. Repeated isolation removes co-regulation and practical support. Pain, alcohol use, and other coping strategies may form reciprocal loops rather than simple one-way consequences. None is necessary in every person; several may interact in ways that help maintain the pattern.

The word *regime* should therefore be used carefully. It does not mean a permanent essence, and it does not require a literal neural attractor. It means that multiple variables can become coordinated so that the present state helps reproduce the conditions of its own return. A sleepless night changes attention; attention changes cue sampling; cue sampling changes arousal; arousal changes sleep. The loop can continue even when no one element is uniquely causal.

The humane implication is not that the person should "just update." Updating requires evidence the system can receive, actions it can perform, sleep in which learning can consolidate, relationships that are safe enough to trust, and sometimes treatment that makes contact with avoided material tolerable. Knowledge that the danger is over may be genuine while access to safety remains unreliable. Recovery is then not a correction of ignorance. It is a change in the rules through which evidence becomes action.

CORE SENTENCE

The past can remain active without being confused with the present, because learned transition rules may change more slowly than the world that trained them.

6

When Danger Has Not Fully Ended

BROADER SCIENTIFIC INFERENCE

"Post-traumatic" is a chronological term. It is not always an ecological description. People may live after an attack while rockets continue, after displacement while return remains impossible, after captivity while a loved one remains captive, after abuse while contact with the perpetrator or institution continues, or after combat while redeployment is expected. In such conditions, the scientific problem changes. The environment cannot be treated as a cleared laboratory in which every alarm is necessarily a false alarm.

Let $h_t = P(H_t = 1 \mid \mathcal{I}_t)$ represent the estimated current hazard, based on available information $\mathcal{I}_t$. Let τ_t denote the system's threshold for recruiting defence. A stylised decision rule might be

$$a_t = \text{defend} \quad \Longleftrightarrow \quad h_t\, C_{\text{miss}} > (1 - h_t)\, C_{\text{alarm}} + \tau_t,$$

where C_{miss} is the expected cost of missing real danger and C_{alarm} the cost of unnecessary defence. When real hazard remains high or poorly observable, increased vigilance may be partly calibrated. When prior learning lowers τ_t, the same environment may also evoke more defence than current evidence alone would predict. Both can be true. The equation is a conceptual decomposition, not a clinical test.

Research conducted during prolonged war and recurrent escalation makes this boundary unavoidable. Studies of continuous traumatic stress find that displacement, exposure, dissociation, social support, trust, income loss, and renewed escalation can accompany changing distress while the threatening context remains active [Harwood-Gross et al., 2026] [Amsalem et al., 2026]. An October 7 cohort with an August 2023 pre-event baseline found higher later proportions of screen-defined probable PTSD and depression among directly exposed participants while the war remained ongoing; these were self-report screening outcomes, not clinician-confirmed diagnoses [Levi-Belz et al., 2025]. These studies tell us about people under pressure. They do not permit us to subtract the pressure and label the remainder "the disorder."

The distinction has practical and ethical consequences. Safety learning cannot be reduced to persuading someone that the world is safe when it is not. Avoidance of a genuinely dangerous route is not evidence of pathological generalisation. Sleep disruption in a setting of alarms, caregiving demands, or unstable shelter cannot be read solely as an internal maintenance mechanism. Treatment may still reduce unnecessary suffering, restore discrimination, or widen choice, but it cannot ethically substitute for physical safety, housing, social support, institutional trust, or the end of violence.

Continuing danger also disrupts simple outcome language. If symptoms fall while threat remains, that may reflect adaptation, exhaustion, effective coping, improved protection, altered reporting, or several processes together. If symptoms rise after renewed escalation, that is not necessarily delayed pathology emerging from nowhere. If a person remains highly vigilant, the question is not "Why have you failed to recover?" but "Which parts of this response are tracking current conditions, which have generalised beyond them, and what would a safer range of response look like here?"

Flow Hijacked therefore keeps external hazard inside the model. The person is not a closed nervous system reacting to memories in a vacuum. News, sirens, legal proceedings, community tensions, family uncertainty, economic loss, and the availability of trustworthy others alter both what danger is and what safety can mean. A theory of return that ignores the world may call social reality a symptom. A theory that attributes everything to the world may miss learned persistence that treatment could change. The scientific discipline is to estimate both without forcing either to disappear.

CORE SENTENCE

No model of traumatic persistence is humane or valid if it declares the danger over before the person's world has actually become safer.

7

A First View of the Altered Rules of Return

NEW HYPOTHESIS

We can now state the central Flow Hijacked proposal precisely. **Threat-State Return Asymmetry (TSRA)** is the hypothesis that trauma may alter conditional transition rules so that defensive states are entered more readily, rapidly, or with less context-specific evidence, while return to safe engagement becomes slower, less reliable, or more dependent on favourable conditions. The claim concerns an asymmetry between entry and return. It is not another name for high symptom severity, and it does not say that PTSD is a single state.

Let $S_t \in \{E, D\}$ denote, for the moment, two coarse latent regimes: engaged or defensively organised. For a context c and history $\mathcal{H}_t$, define the transition intensities

$$\lambda_{E\to D}(t \mid c, \mathcal{H}_t) = \lim_{\Delta t \downarrow 0} \frac{P(S_{t+\Delta t} = D \mid S_t = E, c, \mathcal{H}_t)}{\Delta t},$$

$$\lambda_{D\to E}(t \mid c, \mathcal{H}_t) = \lim_{\Delta t \downarrow 0} \frac{P(S_{t+\Delta t} = E \mid S_t = D, c, \mathcal{H}_t)}{\Delta t}.$$

A dimensionless asymmetry contrast can then be written as

$$\mathcal{A}(c) = \log \frac{\lambda^{\text{post}}_{E\to D}(c)}{\lambda^{\text{base}}_{E\to D}(c)} - \log \frac{\lambda^{\text{post}}_{D\to E}(c)}{\lambda^{\text{base}}_{D\to E}(c)}.$$

Positive $\mathcal{A}(c)$ means that, relative to an appropriate baseline, entry has accelerated, return has slowed, or both. The baseline might be a pre-trauma within-person period, a stable low-symptom period, or a carefully matched counterfactual; these are not equivalent. Neither a symptom total nor one resting-state scan can identify both intensities.

TSRA proposes six measurable coordinates: **entry threshold**, the evidence needed to recruit defence; **cue-specific transient gain**, the amplification of a small input; **generalisation width**, the spread of the learned danger category; **dwell or escape time**, persistence after entry; **recovery half-time**, return toward an individual reference after controlled perturbation; and **alternative-state accessibility**, the reachable non-defensive actions and their cost. The coordinates can dissociate: rapid entry may accompany rapid recovery, while a higher entry threshold may coexist with hours-long persistence. Identical weekly symptom totals can conceal different dynamics.

Adjacent evidence makes the hypothesis plausible without proving it. Person-specific data reveal differing networks among triggers, current threat, appraisals, memories, and strategies [Hertz-Palmor et al., 2026], while nonlinear analyses identify heterogeneous post-disaster adjustment dynamics [Littleton et al., 2023]. A small neuroimaging study reported less frequent but more stable anomalous states in PTSD; estimated stability is not an attractor [Wu et al., 2026]. In 71 participants, dissociation related nonlinearly to autonomic response, but predicted immobility, staring, and emotional-numbing markers were not confirmed [Danböck et al., 2024]. Conversely, a 30-site resting-state analysis of 1,035 trauma-exposed participants found no robust PTSD-group differences in dwell, transitions, or static and dynamic connectivity [Tomas et al., 2025]. That null constrains any universal TSRA.

The hypothesis makes risky predictions. Repeated, context-controlled within-person measurements should yield partly separable, reliable entry and return parameters; add prospective information beyond exposure and symptom totals; vary with context as preregistered; sometimes mediate treatment benefit; and generalise from one perturbation to another or to daily life. If these predictions fail in adequately powered, preregistered, independently replicated studies—or a simpler severity or measurement model predicts equally well—TSRA should be weakened or abandoned.

The concept is useful only if it changes what we measure. Instead of asking merely how distressed someone became, we ask: how did the state begin, how fast did it rise, what kept it active, what permitted it to release, and what could the person do afterward? That shift does not make pain mathematical. It makes the mathematics answerable to the temporal structure of pain.

CORE SENTENCE

TSRA proposes that trauma may change not only how strongly defence is felt, but the unequal rules by which defence is entered and safe engagement is regained.

PART II

WHAT PTSD IS, AND WHAT IT IS NOT

Diagnosis matters because suffering, symptoms and neighbouring conditions are not interchangeable.

8

Trauma Exposure Is Not a Diagnosis

BROADER SCIENTIFIC INFERENCE

The word *trauma* carries several meanings at once. In ordinary language, it can name an event, an injury, a lasting psychological consequence, or the felt magnitude of an experience. In diagnostic research, those meanings have to be separated. A potentially traumatic event is an exposure. Post-traumatic stress disorder is one possible pattern of response, defined not by exposure alone but by a particular configuration of symptoms, time, impairment, and exclusions. The experience can transform a life without producing PTSD; PTSD can be severe without representing the only consequence of what happened.

This distinction is not pedantic. Cross-national surveys find that exposure to potentially traumatic events is widespread, while PTSD occurs in a much smaller and highly conditional subset [Benjet et al., 2016] [Koenen et al., 2017]. Risk varies with exposure type and history: assaultive violence, repeated exposure, prior adversity, and the surrounding social environment do not carry interchangeable probabilities [Liu et al., 2017]. Even those broad findings are population estimates, shaped by the interview, diagnostic system, countries sampled, and retrospective recall. They cannot be transferred wholesale to one person or one event.

Formally, let E_i denote qualifying exposure for person i, $D_i(t)$ a PTSD diagnosis at time t, $\mathbf{z}_i$ person-level and historical conditions, $\mathbf{c}_i(t)$ the current context, and $\mathcal{M}$ the measurement procedure. Then the relevant object is not E_i by itself but

$$p_i(t) = P[D_i(t) = 1 \mid E_i, \mathbf{z}_i, \mathbf{c}_i(t), \mathcal{M}] .$$

Qualifying exposure is required by the diagnostic definition, yet it is nowhere near sufficient. The conditional probability changes when the measurement changes, when the follow-up window changes, and when the denominator changes from a general population to directly exposed survivors. This is why the phrase “a traumatised population” can be scientifically dangerous: it may refer to people exposed, people distressed, people above a symptom threshold, or people with a diagnosed disorder.

The distinction also protects trauma-exposed people who do not develop PTSD. They are not untouched, and they have not necessarily “processed” the event in some superior way. They may grieve, sleep badly, become angry, revise their identity, experience bodily symptoms, or require practical and psychological support. Conversely, a stable-low symptom trajectory does not prove that the event was minor. Human consequence is broader than one diagnostic outcome.

This allows us to acknowledge the magnitude of an event without assigning every person the same outcome, and to acknowledge the magnitude of a response without reducing the event’s full meaning to one diagnosis.

For research, a trauma-exposed comparison group is often more informative than an unexposed group alone. If both trauma-exposed groups show a neural, bodily, or cognitive difference from people who were not exposed, that difference may track exposure rather than PTSD. If the PTSD group differs from trauma-exposed controls, the result is more specific, though still not necessarily causal or diagnostic. Without this separation, the biology of surviving danger can be mislabeled as the biology of disorder.

For public language, the rule is equally direct. Do not convert the magnitude of an event into an assumed diagnosis. Do not demand a diagnosis before acknowledging harm. Ask what was experienced, what has changed, how long it has lasted, what function has been lost, which other conditions may be present, and how the conclusion was reached. The event matters. The response matters. Their relationship must be studied rather than declared.

CORE SENTENCE

Exposure tells us what a person encountered; diagnosis asks what pattern followed, for how long, with what impairment, and by what method it was established.

9

The Diagnostic Architecture of PTSD

EMPIRICAL RESULT

A PTSD diagnosis is not a vote taken among unpleasant symptoms. It has an architecture. In the DSM-5 family of definitions, a qualifying exposure is followed by requirements across intrusion, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity, together with duration, clinically significant distress or impairment, and exclusion conditions. The ICD-11 architecture is more compact and organised differently. These systems overlap substantially, but they do not carve the clinical space in identical ways. A prevalence estimate, trial sample, or biological comparison inherits the architecture used to create it.

The logical form matters. Let Q represent qualifying exposure; let B, C, M, A represent the required symptom-cluster conditions; let T represent the duration requirement; F clinically significant distress or functional impairment; and X satisfaction of the relevant exclusion rules. A stylised DSM-like diagnostic rule is

$$D = \mathbf{1}\{Q \wedge B \wedge C \wedge M \wedge A \wedge T \wedge F \wedge X\}.$$

The indicator $\mathbf{1}\{\cdot\}$ equals one only when the stated conjunction is satisfied. This representation is deliberately schematic: it omits item-level counts and clinical judgement. Its purpose is to show why a high total on a general distress scale, or even several intense post-traumatic symptoms, is not automatically the same object as a diagnosis. The pattern and its boundaries matter.

Structured measurement was strengthened historically by the Clinician-Administered PTSD Scale, which anchored symptom frequency and intensity to behavioural descriptions [Blake et al., 1995]. The CAPS-5 adaptation demonstrated strong initial reliability and validity in military veterans, while leaving the ordinary cautions about transport across populations, languages, and settings [Weathers et al., 2018]. A recent systematic review found multiple DSM-5- and ICD-11-based instruments with useful psychometric evidence, but also substantial variation in cutoffs and underrepresentation of some populations [Coeur et al., 2026]. There is no measurement device whose label alone abolishes context or judgement.

Architecture changes interpretation. Intrusive recollection without avoidance may be agonising but may not satisfy the full diagnostic rule. Sleep disruption and irritability can occur in PTSD, depression, pain, substance withdrawal, caregiving strain, or ongoing danger. Emotional numbing may reflect several processes. A person can experience serious impairment with a subthreshold configuration, and another can cross a symptom threshold while retaining more function in a particular domain. The categorical decision is clinically useful; it does not make the underlying dimensions disappear.

Even within the same diagnosis, there are several ways to satisfy the criteria. The same total score therefore does not guarantee the same symptom configuration, severity within each cluster, or treatment need.

Nor does diagnosis locate the disorder in one place. The criteria describe a pattern at the level of experience and function. They do not assert that every symptom shares one neural mechanism. Two people can satisfy the same diagnostic architecture through different item combinations, histories, and maintaining processes. The diagnosis can therefore serve as a shared clinical language while an individual formulation asks a different question: what is producing and sustaining this person's current pattern?

The lecture will respect both functions. It will not dissolve PTSD into generic suffering, because diagnostic specificity affects evidence and care. It will not reify the checklist into a natural essence, because a classification system is a disciplined rule for organising observations, not a complete causal theory of a person.

CORE SENTENCE

A PTSD diagnosis is a structured clinical inference about a patterned syndrome—not a synonym for exposure, distress, or one elevated score.

10

Diagnosed PTSD, Probable PTSD and PTSS

EMPIRICAL RESULT

Three expressions recur throughout the literature: **diagnosed PTSD**, **probable PTSD**, and **post-traumatic stress symptoms**, or PTSS. They are related; they are not interchangeable. Much confusion in public reporting begins when the instrument disappears from the sentence.

In this lecture, *diagnosed PTSD* is reserved for a clinician-administered or structured diagnostic assessment, or for clearly documented administrative clinical ascertainment whose limitations are stated. *Probable PTSD* means that a person crossed a threshold on a validated self-report screening instrument. *PTSS* refers to symptom severity, a cluster score, or a dimensional measure without claiming that a diagnostic threshold was established. Acute stress, nonspecific distress, grief, anxiety, depression, dissociation, and moral injury retain their own names.

Screeners are valuable. They make large cohorts, repeated measurements, and rapid service planning possible. They can sensitively identify people who may need fuller assessment. But a cutoff is not a microscopic boundary in nature. A recent review of DSM-5 and ICD-11 instruments found that cutoffs for the widely used PCL-5 varied across studies and populations, with veteran and medical samples disproportionately represented [Coeur et al., 2026]. Work on subthreshold PTSD likewise shows that alternative definitions classify different groups, even while subthreshold symptom configurations can carry genuine clinical significance [Klein et al., 2024].

The mathematics of screening makes the language rule unavoidable. If sensitivity is $Se = P(+ \mid D)$, specificity is $Sp = P(- \mid \neg D)$, and the prevalence of the target diagnosis in the assessed population is $\pi = P(D)$, then the positive predictive value is

$$PPV = P(D \mid +) = \frac{Se\,\pi}{Se\,\pi + (1 - Sp)(1 - \pi)}.$$

Even if sensitivity and specificity were fixed—which they are not perfectly—the proportion of screen-positive people who meet a full diagnostic standard changes with π. In a high-risk clinical sample and a broad community survey, the same score can therefore have different implications. Translation, literacy, cultural expression, time since exposure, and the purpose of assessment add further variation.

Clinician-administered interviews are not infallible either. They require training, judgement, reliable administration, and a population for which the instrument is valid. The original and DSM-5 versions of CAPS improved standardisation and showed strong psychometric properties in their validation settings [Blake et al., 1995] [Weathers et al., 2018]. Yet interview quality, recall, rapport, language, dissociation, comorbidity, and administrative context can still shape ascertainment. “Diagnosed” names a stronger process; it does not mean metaphysical certainty.

These distinctions become especially important after disasters, attacks, and wars. A rapid survey may responsibly report elevated PTSS or the proportion crossing a probable-PTSD threshold. It cannot simply announce the prevalence of clinical PTSD unless its ascertainment supports that noun. Correct wording does not minimise suffering. It prevents one kind of evidence from borrowing the authority of another.

The rule also protects longitudinal interpretation. A movement from above to below a screening threshold is not necessarily diagnostic remission; a small reduction that remains above threshold may still accompany major functional improvement; and stable symptom severity can hide changes in which symptoms are active. Every consequential number should travel with its instrument, threshold, population, and assessment window.

CORE SENTENCE

The noun must match the measurement: diagnosis, screen-positive classification and symptom severity are different scientific objects even when they concern the same suffering.

11

Symptoms, Function and the Shape of Impairment

BROADER SCIENTIFIC INFERENCE

Symptoms tell us how a disorder is expressed. Function tells us what the expression does to a life. The two are connected, but neither is a perfect substitute for the other. Someone may report fewer intrusions yet remain unable to work in a crowded room. Another may continue to experience nightmares while rebuilding intimacy, study, parenting, or public movement. A symptom total can improve before the world reopens; functional improvement can also precede later symptom change without, by itself, proving what caused it.

A systematic review and meta-analysis found substantial average impairment associated with PTSD across general tasks, mobility, self-care, domestic life, relationships, major life areas, and community or civic participation [Jellestad et al., 2021]. Comparisons with other mental disorders were smaller than comparisons with healthy groups, which is exactly what should be expected if impairment is serious but not specific to PTSD. The studies were observational and vulnerable to comorbidity and selection. They establish burden at group level, not the functional future of an individual.

The measurement problem can be written as a joint outcome rather than a single axis. For person i at time t, let

$$\mathbf{y}_{it} = \begin{bmatrix} \mathbf{s}_{it} \\ \mathbf{f}_{it} \end{bmatrix}, \qquad \mathbf{s}_{it} \in \mathbb{R}^p, \quad \mathbf{f}_{it} \in \mathbb{R}^q,$$

where $\mathbf{s}_{it}$ contains symptom dimensions and $\mathbf{f}_{it}$ functioning across q domains. A complete outcome model must estimate not only change in $\mathbf{s}$ but the cross-lag structure

$$\begin{aligned} \mathbf{s}_{i,t+1} &= A\mathbf{s}_{it} + B\mathbf{f}_{it} + \boldsymbol{\varepsilon}^{(s)}_{it}, \\ \mathbf{f}_{i,t+1} &= C\mathbf{s}_{it} + D\mathbf{f}_{it} + \boldsymbol{\varepsilon}^{(f)}_{it}. \end{aligned}$$

The matrices B and C allow lagged dependence in both directions; they do not, without stronger assumptions, establish causal influence. Session-level analysis in a psychotherapy trial found complex reciprocal relations, with symptom-to-function and function-to-symptom associations differing by phase and symptom cluster [Benfer et al., 2024]. Because cross-lag estimates depend on model specification and the parent trial, the study does not prove one universal causal sequence. It does show why function cannot remain an afterthought.

The shape of impairment is personal and ecological. Avoidance may restrict commuting but leave solitary work intact. Hypervigilance may be almost invisible in structured settings and exhausting at home. Emotional detachment can protect performance during crisis while damaging intimacy. Poverty, discrimination, unsafe housing, pain, caregiving, and inaccessible treatment can convert moderate symptoms into severe disability; stable resources can buffer some effects without erasing the disorder. Function is produced by an interaction between a person's capacities and the demands or supports of an environment.

There is no single "level of functioning" that summarises a life. An action may remain possible only at the cost of severe exhaustion, and someone may appear to function because other people or structures quietly carry part of the load. Good measurement must also ask what it takes to sustain the performance.

This matters for treatment goals. A fall in a scale score is meaningful, but it is not the whole recovery. We must also ask whether sleep is restorative, concentration usable, relationships tolerable, movement less constrained, work or study more reachable, and ordinary uncertainty less likely to trigger total defence. Conversely, continued symptoms do not cancel regained life. The aim is not to force a person to perform wellness. It is to understand which possibilities have reopened and which remain costly.

CORE SENTENCE

The clinical significance of PTSD is revealed not only by what a person feels, but by which parts of life remain reachable while those feelings are present.

12

Acute Stress, Delayed Expression and Time

BROADER SCIENTIFIC INFERENCE

Time is not merely a line beneath the diagnosis. It changes what the observation means. Intense reactions in the immediate aftermath of danger may include intrusion, disorientation, sleep disturbance, autonomic arousal, avoidance, numbing, grief, anger, or dissociation. Some people meet criteria for acute stress disorder during the early diagnostic window; many distressed people do not. Neither acute distress nor acute stress disorder is a declaration of a chronic future.

Early symptoms carry information, but prediction remains imperfect. Prospective work comparing questionnaires with clinician-administered assessment found that early measures predicted later PTSD better than chance, while recovery was easier to predict and the structured interview performed better than questionnaires [Shalev et al., 1997]. A recent individual-participant-data meta-analysis across acute-care cohorts found that early PTSD and depressive symptom dimensions contributed information about later persistence, while still leaving substantial unexplained variation [Ratanatharathorn et al., 2026]. The result is not that early suffering should be ignored. It is that risk and destiny are different nouns.

The temporal distinctions are clearer in a multistate process. Let S_t take values in $\{A, L, P, R\}$: acute stress A, low or subthreshold symptoms L, PTSD P, and remission R. Conditional transition intensities are

$$q_{jk}(t \mid \mathcal{H}_t) = \lim_{\Delta t \downarrow 0} \frac{P(S_{t+\Delta t} = k \mid S_t = j, \mathcal{H}_t)}{\Delta t}, \qquad j \neq k.$$

An early transition $A \to P$, a later transition $L \to P$, improvement $P \to R$, and recurrence $R \to P$ are different paths. They may have different predictors and mechanisms. A single assessment cannot tell them apart, and a study with a late first measurement may call a case “delayed” that had substantial unmeasured symptoms earlier.

Likewise, two widely spaced assessments can miss fluctuation, intervals of remission, or new exposures occurring between them.

Systematic longitudinal evidence shows a strong average decline in PTSD burden during the first year after many forms of trauma, alongside persistent and later-onset courses whose frequency differs by intentionality, sample, and definition [Santiago et al., 2013]. Long-term reviews show even wider variability in recovery, chronicity, and recurrence [Steinert et al., 2015]. A dedicated review of recurrence could not produce a stable pooled estimate because studies defined and measured recurrence too differently [Brooks & Greenberg, 2024]. Methodological disagreement is therefore part of the temporal finding.

Time also changes intervention claims. Acute stress may be self-limiting, and treating every early response as incipient disorder risks pathologising natural recovery. Waiting passively for severe impairment to resolve can also be costly. Evidence for early pharmacological prevention remains limited and intervention-specific [Bertolini et al., 2024]. The ethical response is proportionate: provide safety, practical support, monitoring, and access to assessment; distinguish broad humane care from unproven universal prevention; and intensify treatment according to need rather than an imagined clock shared by everyone.

Delayed expression should never be interpreted as fabrication merely because the person functioned earlier. Symptoms may have been subthreshold, hidden, differently named, buffered by structure, or reactivated by new stress. Nor should every later difficulty be attributed automatically to the earlier event. Longitudinal assessment has to consider new exposures, depression, grief, pain, substance use, sleep, and changing social context.

CORE SENTENCE

Time does not simply reveal whether PTSD is present; it distinguishes emergence, persistence, remission and recurrence—different transitions that one snapshot cannot see.

13

Grief, Depression, Anxiety, Dissociation and Moral Injury

BROADER SCIENTIFIC INFERENCE

Trauma rarely respects the borders of one diagnosis. After loss, violence, captivity, betrayal, disaster, or war, a person may experience PTSD alongside grief, depression, anxiety, dissociation, moral injury, pain, sleep disturbance, or substance-related difficulty. Overlap is common. Equivalence is false.

PTSD is organised around a qualifying exposure and a characteristic pattern of re-experiencing, avoidance, negative cognition or mood, and altered arousal or reactivity. Grief is organised around loss and the relationship to the person or world that is gone; yearning and sorrow are not simply fear symptoms. Depression can involve pervasive low mood, diminished interest or pleasure, hopelessness, and altered energy beyond trauma-linked cues. Anxiety may centre on future uncertainty, panic, social evaluation, or generalized worry. Dissociation concerns alterations in presence, continuity, self-experience, or the felt reality of the world. Moral injury refers to consequences of perpetrating, failing to prevent, witnessing, or being betrayed around events that violate deeply held moral expectations. It is not itself a synonym for PTSD.

The data support both overlap and separability. Prospective trauma research found early co-occurrence of PTSD and major depression, with comorbidity associated with greater severity and poorer functioning [Shalev et al., 1998]. Four-year youth data after an earthquake identified both shared and distinct post-traumatic-stress and depressive symptom trajectories [Liang et al., 2021]. Among bereaved family surrogates, prolonged-grief, post-traumatic-stress, and depressive symptoms formed co-occurring but nonidentical courses [Wen et al., 2023]. Moral-injury reviews report associations with PTSD, depression, anxiety, suicidality, and behavioural outcomes, while also exposing inconsistent constructs and largely observational evidence [Hall et al., 2022] [Williamson et al., 2018]. Shame is moderately associated with post-traumatic symptoms at the meta-analytic level, but that association does not establish one direction of causation [López-Castro et al., 2019].

A useful measurement model permits shared and specific variance:

$$\mathbf{y}_t = \mathbf{\Lambda}_g g_t + \mathbf{\Lambda}_P p_t + \mathbf{\Lambda}_A a_t + \mathbf{\Lambda}_G \gamma_t + \mathbf{\Lambda}_\Delta \delta_t + \mathbf{\Lambda}_M \mu_t + \mathbf{\Lambda}_\Xi \xi_t + \boldsymbol{\epsilon}_t,$$

where g_t is general distress; p_t, a_t, γ_t, δ_t, μ_t, and ξ_t denote PTSD-specific, anxiety, grief, depressive, moral-injury, and dissociative processes respectively; and each $\mathbf{\Lambda}$ is a corresponding loading vector or matrix. This is a conceptual factorisation, not a validated universal model. Its discipline is that a shared item—sleep disturbance, concentration difficulty, withdrawal, guilt—cannot be assumed to reveal which latent process generated it.

The distinction changes care and meaning. Exposure work aimed at trauma memories is not automatically grief therapy. Reassurance does not resolve moral betrayal. Treating depression may improve sleep and energy without addressing cue-linked reliving. Grounding may help a dissociative episode without settling guilt or repairing trust. At the same time, insisting on perfectly separate boxes can fragment the person and force one dominant diagnosis to carry the whole story.

The Flow Hijacked lens asks how these processes couple. Nightmares can intensify next-day vigilance; depression can reduce engagement with corrective experience; shame can drive concealment; moral injury can alter trust in institutions or in oneself; grief can keep the lost relationship continuously present; alcohol or other short-latency relief can narrow the available response repertoire. A coupled system still requires precise nouns. We can model interaction without erasing difference.

CORE SENTENCE

Trauma-related suffering may be coupled across diagnoses and meanings, but compassion becomes more precise—not less—when grief, depression, anxiety, dissociation and moral injury keep their own names.

14

PTSD and Complex PTSD

BROADER SCIENTIFIC INFERENCE

Complex PTSD emerged from the clinical recognition that prolonged, repeated, and interpersonal trauma can be followed by difficulties not fully described by fear, intrusion, and avoidance alone. Judith Herman's formulation enlarged the field's attention to affect regulation, self-concept, relationships, dissociation, and development [Herman, 1992]. Historical importance, however, does not settle diagnostic ontology.

ICD-11 treats PTSD and complex PTSD, or CPTSD, as related sibling diagnoses. CPTSD includes the core PTSD pattern together with disturbances in self-organisation: affect dysregulation, a persistently negative self-concept, and relational disturbance. It is not simply the label for "very severe PTSD," and it is not automatically assigned because trauma was prolonged. DSM-5-based practice organises much of the same clinical territory differently and does not use an identical CPTSD diagnosis. Studies must therefore name the system and instrument rather than speaking as though all definitions were interchangeable.

A latent-profile study supported distinguishable PTSD-like and CPTSD-like configurations in a treatment-seeking sample [Cloitre et al., 2013]. The International Trauma Questionnaire was developed to measure the ICD-11 structure and has supporting psychometric evidence, but it remains a self-report instrument rather than a clinician-administered diagnosis [Cloitre et al., 2018]. A 2026 meta-analytic confirmatory factor analysis across 57 studies found support for clinically meaningful structure while also reporting that a correlated seven-factor model fit better than the commonly proposed higher-order solution and that some subscales had limited reliability [Kindred et al., 2026]. The evidence therefore supports useful distinction without establishing two immutable natural kinds.

Formally, let the latent symptom vector be

$$\boldsymbol{\eta}_i = \begin{bmatrix} \boldsymbol{\eta}_i^{\mathrm{PTSD}} \\ \boldsymbol{\eta}_i^{\mathrm{DSO}} \end{bmatrix}, \qquad \mathbf{y}_i = \boldsymbol{\Lambda}\boldsymbol{\eta}_i + \boldsymbol{\epsilon}_i,$$

where $\boldsymbol{\eta}^{\mathrm{PTSD}}$ represents core PTSD dimensions and $\boldsymbol{\eta}^{\mathrm{DSO}}$ disturbances in self-organisation. Evidence that a particular loading matrix $\boldsymbol{\Lambda}$ fits observed covariance does not, by itself, prove that the corresponding categories are biological attractors, distinct causes, or permanent identities. Competing dimensional, factor, network, and clinical-formulation models can reproduce parts of the same data.

The boundaries with dissociation and borderline personality disorder also require care. Dissociative experience is particularly relevant in some CPTSD samples, but it does not define every case [Hyland et al., 2020]. Reviews have challenged premature certainty about complex PTSD and shown how conclusions change with criteria and comparison groups [Resick et al., 2012] [Brewin et al., 2017]. Longitudinal captivity research suggests that CPTSD-like classifications can be associated with enduring functional and health burdens, while relying on self-report algorithms rather than establishing clinician diagnoses [Zerach et al., 2019].

The person-facing meaning should remain humane. "Complex" does not mean incomprehensible, untreatable, or damaged beyond return. It signals that the consequences may extend into regulation, identity, and relationship, and that treatment planning may need to address safety, skills, trauma memories, dissociation, social context, and preference in a thoughtful sequence. It does not prove that everyone requires the same phase-based path, nor that direct trauma-focused work is inherently unsafe.

Duration of trauma alone does not determine the diagnosis. Exposure type, developmental timing, available relationships, meaning, and the present pattern all matter, but none can replace assessment of symptoms and functional impairment.

CORE SENTENCE

Complex PTSD names a broader, evidence-supported but still debated clinical configuration—not a deeper kind of broken person and not a claim that complexity has only one treatment sequence.

15

Many Trajectories, No Single Fate

BROADER SCIENTIFIC INFERENCE

If we average every post-trauma course into one line, the line often falls. That average can be true and still describe almost no one. Beneath it may be people with stable-low symptoms, rapid improvement, gradual recovery, persistent high symptoms, delayed worsening, fluctuation, remission, or recurrence. The names change across studies. So do the proportions. Heterogeneity is the robust finding; the exact taxonomy is not.

Reviews of post-trauma trajectories consistently find multiple courses rather than one inevitable progression [Galatzer-Levy et al., 2018] [Steinert et al., 2015]. Ten-year veteran data likewise show persistent, improving, and changing symptom patterns over long follow-up [Moye et al., 2023]. Related major-life-stressor trajectory research shows that the apparent prevalence of "resilience" is sensitive to model assumptions, baseline placement, measurement error, missing data, and how much change a class is allowed to contain [Infurna & Luthar, 2016]. A trajectory labelled stable-low in one analysis must not become "the resilient type" in a person's identity.

The standard latent-mixture formulation makes both the value and the risk visible. If $\mathbf{y}_i = (y_{i1}, \dots, y_{iT})$ is person i's measured symptom course, then

$$p(\mathbf{y}_i \mid \mathcal{M}_K) = \sum_{k=1}^{K} \pi_k \, p(\mathbf{y}_i \mid z_i = k, \boldsymbol{\theta}_k), \qquad \pi_k \geq 0, \quad \sum_{k=1}^{K} \pi_k = 1.$$

The model $\mathcal{M}_K$ assumes K latent classes with weights π_k and class-specific parameters $\boldsymbol{\theta}_k$. The fitted class z_i is not observed; it is inferred. Change K, the curve shape, covariance structure, missing-data assumption, or assessment schedule, and the classification may change. External-validation work has reproduced broad trajectory families while failing to distinguish some clinically important remitting and nonremitting courses reliably [Tomas et al., 2022]. The mathematics gives a compressed description of data, not a passport stamped with a future.

Delayed expression and recurrence make that warning even more important. A low early score does not confer immunity. Improvement does not erase vulnerability. A recurrence review found such inconsistent definitions and estimates that no pooled prevalence could be justified [Brooks & Greenberg, 2024]. At the other end, persistent symptoms in a study window do not prove lifelong chronicity. Observation ends before a person does.

Trajectories also differ by outcome. PTSD symptoms and depression can move together or diverge. Function can recover on a different clock. Grief may remain intense while fear-related symptoms fall. Perceived growth can coexist with substantial distress and, without prospective change measures, does not establish objective improvement [Frazier et al., 2009] [Benight et al., 2026]. A serious longitudinal picture therefore needs several axes: symptoms, diagnosis, function, sleep, social connection, meaning, and current exposure. It should also show uncertainty around the line rather than polishing it into certainty.

There is an ethical reason to preserve this complexity. Calling resilience the dominant trajectory can counter fatalism, but it can also become a social demand: most people recover, therefore you should have recovered. Calling PTSD chronic can validate the need for sustained care, but it can also be heard as a sentence. Neither message is justified at the level of an individual. Population trajectories describe conditional histories under particular measurements and circumstances.

Flow Hijacked will use trajectories as evidence compatible with a transition system that is plastic, path-dependent, and plural. Some pathways narrow; some reopen; some are altered by new danger or support. The next Parts ask how learning, memory, bodily state, and context may help produce those paths. They begin from a premise already earned here: no group average, latent class, diagnosis, or difficult month has the authority to become a person's fate.

CORE SENTENCE

A trajectory is a pattern inferred from observations, not an identity—and no statistical class has the right to close an individual future.

PART III

LEARNING DANGER, LEARNING SAFETY

The system learns danger quickly; safety often has to become credible by experience.

16

Fear Acquisition Is Only the Beginning

BROADER SCIENTIFIC INFERENCE

Fear conditioning gives us a clean experimental grammar for one part of traumatic learning. A previously neutral cue, conventionally written CS^+, is paired with an aversive outcome, US. A comparison cue, CS^-, is not. Over trials, the CS^+ can begin to evoke expectancy, autonomic change, startle, action preparation, or conscious fear before the aversive outcome arrives. This matters because experience can teach a nervous system to anticipate danger rather than merely react after harm.

One compact learning rule is

$$\hat{r}_t = \mathbf{w}_t^\top \mathbf{x}_t, \qquad \delta_t = r_t - \hat{r}_t, \qquad \mathbf{w}_{t+1} = \mathbf{w}_t + \alpha_t \, \delta_t \mathbf{x}_t.$$

Here $\mathbf{x}_t$ is the vector of cues present on trial t; $\mathbf{w}_t$ contains their learned predictive weights; r_t is the observed outcome; $\hat{r}_t$ is the expected outcome; δ_t is prediction error; and α_t is a learning rate. The equation is a useful abstraction, not a literal description of one PTSD circuit. Human threat learning recruits multiple response systems, and their measures can disagree. A person can know verbally that a cue is safe while their startle, skin conductance, attention, or urge to leave tells a different story.

Some PTSD experiments report altered threat or safety responding, with effects differing by learning phase and outcome measure [Wicking et al., 2016] [Lewis et al., 2023]. A harmonised fear-conditioning study spanning 2,199 participants likewise shows that task variables are major sources of neural variability [Radua et al., 2025]. These results support taking threat learning seriously. They do not justify saying that everyone with PTSD "learns fear faster," or that a laboratory CS^+ reproduces betrayal, combat, captivity, sexual violence, disaster, or continuing danger.

The counterevidence is central, not ornamental. In a multiverse analysis, conclusions about extinction retention changed across defensible analytic choices, and most PTSD–trauma-exposed-control comparisons were not statistically significant [Lewis et al., 2023]. A large preregistered 2026 analysis found no group difference in conditioning or extinction between trauma-exposed people with and without diagnosed PTSD on its primary physiological outcomes [Davidson et al., 2026]. Those null findings do not erase every positive study. They tell us that the phenotype is not a single robust laboratory abnormality waiting to be read from one curve.

Even within one experiment, acquisition has several possible shapes. The danger cue can evoke a higher response, the safety cue can fail to settle, discrimination can emerge slowly, or expectancy can change while physiology does not. Averaging these into one "fear score" hides the difference between response magnitude, learning rate, uncertainty, and decision threshold. Each makes a different prediction about persistence.

Most importantly, acquisition cannot by itself explain persistence. The original learning may have been swift and exquisitely appropriate. The harder questions begin afterward: Does the system distinguish danger from resemblance? Can it learn safety? Can that learning be retrieved tomorrow and elsewhere? Does behavior permit disconfirming evidence to arrive? Fear acquisition supplies the opening move, not the whole game.

CORE SENTENCE

PTSD cannot be read from how fear begins, because persistence lives in what the system distinguishes, retrieves, and does next.

17

Extinction Is New Learning, Not Erasure

BROADER SCIENTIFIC INFERENCE

When a conditioned danger cue is repeatedly presented without the aversive outcome, defensive responding often declines. This is called extinction. The name is unfortunate if it suggests that the old learning has been extinguished like a flame. A better account is that the organism learns something additional: "In these conditions, this cue no longer predicts the same outcome." The original danger association can remain available while a newer safety or non-threat memory competes with it.

That competition can be represented schematically as

$$P(R_t = 1 \mid x_t, c_t) = \sigma[\beta_D(c_t)V_D(x_t) - \beta_S(c_t)V_S(x_t)], \qquad \sigma(u) = \frac{1}{1+e^{-u}}.$$

R_t denotes a defensive response, x_t the current cue, and c_t the context. V_D and V_S are learned danger and safety values; $\beta_D(c_t)$ and $\beta_S(c_t)$ are context-dependent retrieval weights. The expression is conceptual. It does not assert that the nervous system stores two scalar memories, or that a logistic function is a clinical mechanism. It clarifies one point: declining fear can reflect stronger access to competing safety learning without deletion of the danger history.

Return-of-fear phenomena make this distinction visible. Responding can reappear after time has passed, after an aversive event occurs again, or when the cue is encountered outside the extinction context. These are commonly described as spontaneous recovery, reinstatement, and renewal. They are not evidence that extinction "failed" in every sense. They show that what was learned and what is retrieved are different questions. Contemporary reviews of fear-learning circuitry and reconsolidation both treat memory updating as conditional, distributed, and more complex than trace deletion [Marek & Sah, 2018] [Lee, 2009].

In PTSD, this distinction protects us from two equal mistakes. The first is pessimistic: if the danger memory remains, recovery must be counterfeit. The second is triumphalist: if distress falls during a safe session, the trauma memory has been erased. Neither follows. A three-day conditioning study found stronger return-of-fear measures in a small PTSD group when extinction memory was tested in a new context, but it did not show an incapacity to learn anything during extinction [Wicking et al., 2016]. Across studies, acquisition, within-session extinction, retention, and next-day recall must therefore be named separately [Lewis et al., 2023].

A falling response within one session is especially ambiguous. It can reflect safety learning, fatigue, habituation, strategic regulation, or a change in what the participant thinks the experimenter will do. Evidence for durable extinction requires later testing, preferably across time and context, and it should preserve expectancy, behavior, and physiology as distinct outcomes. A smooth curve today is not yet proof of accessible safety tomorrow.

This is also why successful treatment need not make an autobiographical event inaccessible. The relevant change may be that recollection no longer predicts present catastrophe, that bodily activation becomes tolerable, that action is no longer governed by escape, or that safety learning retrieves across a wider range of contexts. Remembering and being returned are not the same operation.

CORE SENTENCE

Extinction does not delete danger; it builds a rival future whose retrieval must become credible in the present context.

18

Extinction Recall Depends on Context

EMPIRICAL RESULT

Learning safety in one place is not identical to retrieving safety in another. Laboratories reveal this with an A–B–C design: danger is learned in context A, extinction occurs in context B, and recall is tested in B, A, or a new context C. The physical cue may be unchanged, yet defensive responding can return because the surrounding context changes which memory is treated as relevant.

A latent-context formulation makes the problem explicit:

$$P(z_t = k \mid x_{1:t}, c_t) \propto p(x_t \mid z_t = k, c_t) \sum_j P(z_t = k \mid z_{t-1} = j) P(z_{t-1} = j \mid x_{1:t-1}, c_{t-1}).$$

Here z_t is an unobserved—or latent—environmental state, $x_{1:t}$ is the sequence of observations, and c_t denotes contextual information. The transition term expresses how likely one inferred state is to follow another. This is a model of inference, not a measured "PTSD state." Its value is to show why the same doorway, tone of voice, bodily sensation, or time of day can be interpreted differently depending on where it appears and what history accompanies it.

Direct PTSD evidence is suggestive but not uniform. In one small fMRI experiment, adults with PTSD showed greater differential skin conductance and amygdala–hippocampal response than two control groups when extinction memory was tested in a novel context; arousal to the nominal safety cue was also elevated [Wicking et al., 2016]. In a larger two-day cross-diagnostic study, healthy participants showed a gradual increase in dynamic connectivity during extinction that was not seen in PTSD and anxiety groups; only in controls did those changes relate to next-day recall [Wen et al., 2022]. Neither study measures a latent context directly, and neither establishes a universal neural signature.

Prospective findings sharpen the uncertainty. Reduced extinction learning before deployment predicted later post-traumatic symptoms in one military cohort [Lommen et al., 2013]. A conceptual replication in 529 firefighters did not reproduce that prospective prediction: baseline symptoms and later stressor severity predicted follow-up symptoms, whereas pre-trauma extinction learning did not [Lommen & Boddez, 2022]. The responsible conclusion is not that context-dependent safety recall is irrelevant. It is that one laboratory task cannot yet serve as a general vulnerability test.

Context is also nested. The room is embedded in a relationship; the relationship in an institution; the institution in a period of peace, uncertainty, or ongoing threat. Internal context matters too: sleep deprivation, pain, intoxication, heart rate, and dissociation can alter what is retrieved. A credible account of recall therefore needs more than a label on a background image. It needs a model of which contextual features actually gate memory.

In lived terms, a person may feel steadier in a clinician's office and still become overwhelmed in a car, a bedroom, a crowd, or an official building. That does not mean the first learning was fraudulent. It means safety has not yet become portable enough. The scientific question is therefore not only whether responding falls, but where, when, with whom, and under what bodily conditions the new learning can be retrieved.

CORE SENTENCE

Safety that cannot be recalled where life happens is real learning, but not yet usable safety.

19

Generalisation: When the Category of Danger Widens

BROADER SCIENTIFIC INFERENCE

Learning would be useless if it applied only to an exact repetition. A nervous system must generalise: after one dangerous encounter, it should recognise sufficiently similar situations without waiting to be harmed again. The problem is not generalisation itself. The problem is resolution. If the category becomes too wide, resemblance begins to count as recurrence.

For a cue represented by a feature vector $\mathbf{x}$, one simple generalisation kernel is

$$G(\mathbf{x}, \mathbf{x}_0) = \exp\left[-\frac{1}{2}(\mathbf{x} - \mathbf{x}_0)^\top \boldsymbol{\Sigma}^{-1}(\mathbf{x} - \mathbf{x}_0)\right].$$

$\mathbf{x}_0$ is the learned danger cue, and the covariance matrix $\boldsymbol{\Sigma}$ determines how similarity is measured and how broad the gradient becomes across different feature directions. A wider gradient is one formal way to represent more stimuli inheriting threat value. Human generalisation is not literally Gaussian, however, and meaning cannot be reduced to visual distance. A uniform, a smell, a facial expression, a silence, and a loss of control can be linked at a conceptual level even when they share few sensory features.

PTSD studies have reported broader responding across graded perceptual cues. In one veteran sample, generalisation involved visual, insular, locus-coeruleus-region and thalamic responses alongside weaker vmPFC-related safety connectivity [Morey et al., 2015]. In women with abuse-related PTSD, the pattern was not simply "more startle": slower danger judgments around the safety cue and variation linked to self-reported spreading of triggers across life were also observed [Lis et al., 2020]. Another study reported altered generalisation at a conceptual rather than merely perceptual level [Morey et al., 2020]. These are small or moderate cross-sectional studies, so they cannot establish a universal mechanism.

Prospective evidence is particularly valuable. Greater fear generalisation in Dutch firefighters predicted post-traumatic symptom severity two years later [Lommen et al., 2024]. Temporal order strengthens the case that generalisation is more than a consequence measured after symptoms are established, but observational prediction still does not prove causation, and it does not turn a group association into an individual diagnostic instrument.

Gradient height and width must also be separated. A person may respond strongly but narrowly around the learned cue, or modestly across a very broad range. They may discriminate perceptually while generalising by meaning, relationship, or bodily state. Future studies should estimate these dimensions rather than treating every elevated response to a near cue as the same process. That distinction is crucial for the later TSRA proposal: easier entry and broader generalisation are related possibilities, not synonyms.

The humane implication is subtle. A broadened danger category is not stupidity, weakness, or a refusal to "move on." Under uncertainty, false negatives can be catastrophically costly. The system may rationally choose sensitivity over specificity when it has learned that apparently safe situations can turn dangerous. Yet a once-protective widening can later make safe places, people, sensations, and futures inaccessible. Recovery would then require not amnesia but finer discrimination: the capacity to register similarity without treating similarity as identity.

CORE SENTENCE

Overgeneralisation is not "too much fear"; it is a loss of resolution in deciding what truly belongs to danger.

20

Threat Discrimination and the Work of Safety Signals

BROADER SCIENTIFIC INFERENCE

A safety signal is not merely the absence of an alarm. It is information that helps the organism distinguish conditions under which harm is unlikely from conditions under which vigilance is warranted. In differential conditioning, the central quantity is often the separation between responses to CS^+ and CS^-. A person can respond strongly to danger and still discriminate well; or respond moderately to both cues and discriminate poorly.

Signal-detection theory provides one useful description:

$$d' = \Phi^{-1}(P(\text{alarm} \mid D)) - \Phi^{-1}(P(\text{alarm} \mid S)),$$

where D and S denote danger and safety trials and Φ^{-1} is the inverse standard-normal distribution. Larger d' means greater separation under the model's assumptions. The response criterion—how much evidence is required before sounding the alarm—is a different parameter. This matters because high alarm rates may reflect reduced discriminability, a deliberately cautious criterion, or both. The formula is not interchangeable with skin conductance, startle, expectancy ratings, or clinical judgment; each samples a different channel.

Some PTSD experiments point particularly to safety processing. In a three-context study, the PTSD group showed elevated self-reported arousal not only to the danger cue but also to the safety cue [Wicking et al., 2016]. Work on abuse-related PTSD similarly found slower risk assessment around a safety cue and heterogeneity related to real-world generalisation [Lis et al., 2020]. Such findings fit the idea that persistence can involve difficulty using safety evidence, not simply amplified response to danger.

Intervention experiments offer mechanistic clues, but they require disciplined language. A placebo-controlled study reported that dexamethasone altered extinction and safety discrimination in a modest PTSD sample [Michopoulos et al., 2017]. In healthy participants, prazosin administered during threat learning later reduced responding to the safe stimulus during extinction and re-extinction [Homan et al., 2017]. The latter is bridge evidence, not evidence that prazosin restores safety learning in people with PTSD. Neither result licenses routine clinical use from a laboratory endpoint.

Discrimination also depends on the costs of error. When missing a true threat could be catastrophic, a conservative system will accept more false alarms. The same observable response can therefore arise from noisy evidence or from a rational shift in criterion. Experiments that do not estimate both sensitivity and criterion risk calling prudence a perceptual deficit. Clinical interpretation must ask what the person believes a missed alarm would cost.

Safety signals are also relational and ecological. A locked door, a predictable schedule, a trusted person, control over distance, or the ability to stop an interaction may change what a cue means. Their value depends on credibility. Reassurance that contradicts present conditions is not safety learning; it can become another source of mistrust. Under continuing threat, a low threshold for alarm may remain adaptive. The scientific and clinical task is not to demand calm, but to determine whether evidence is being discriminated accurately enough for the world the person actually inhabits.

CORE SENTENCE

Recovery needs more than weaker alarms; it needs trustworthy evidence about what is safe now.

21

Avoidance as an Intelligent Short-Term Policy

BROADER SCIENTIFIC INFERENCE

Avoidance is often described as if it were simply irrational. That is poor science and poor listening. If a street, person, smell, memory, conversation, or internal sensation predicts overwhelming danger, moving away can be the most intelligent action available. Avoidance reduces exposure, buys time, preserves functioning, and sometimes prevents real harm. Under continuing danger, it may remain necessary.

A minimal decision model writes the selected action as follows. Let the current hidden environmental condition be $z_t \in \mathcal{Z}$, with belief distribution $z_t \sim b_t$:

$$a_t^* = \arg\min_{a \in \mathcal{A}} \mathbb{E}_{z_t \sim b_t} \left[C_{\text{harm}}(a, z_t) + C_{\text{arousal}}(a, z_t) + C_{\text{opportunity}}(a, z_t) \right].$$

$\mathcal{A}$ is the available action set; b_t is the person's current belief distribution over the hidden condition z_t; and the three state-dependent costs represent possible harm, immediate physiological or emotional burden, and what must be surrendered by avoiding. When danger is judged likely and the immediate cost of activation is high, escape can be optimal. The long-term problem emerges when $C_{\text{opportunity}}$—lost mobility, intimacy, work, sleep, care, or corrective learning—accumulates outside the horizon of the decision.

The influential cognitive model developed by Ehlers and Clark places maladaptive behavioral and cognitive strategies within a continuing sense of current threat [Ehlers & Clark, 2000]. This is a theoretical architecture, not a direct causal assay. Meta-analytic work nevertheless finds robust associations between post-traumatic symptoms and broader emotion-regulation difficulties, including experiential avoidance and thought suppression [Seligowski et al., 2015]. These constructs must not be collapsed. Avoiding a place, suppressing a thought, numbing bodily sensation, scanning for exits, and declining an unsafe relationship may perform different functions.

Prospective laboratory evidence is sobering. In 502 firefighters, task-derived avoidance learning was associated with neither baseline nor later PTSD symptoms [de Haart et al., 2021]. That null result challenges any claim that one avoidance task reveals a general predisposition to PTSD. Intensive longitudinal studies also show that temporal relations vary: in one wartime sample, arousal forecast later intrusions and negative cognition or mood, while avoidance forecast little [Gelkopf et al., 2019]. Elsewhere, thought avoidance participated in temporal symptom networks, but those exploratory associations did not identify a causal master variable [Greene et al., 2018].

Time horizon changes the apparent rationality of the action. An exit that prevents collapse tonight may make work, treatment, or relationship repair harder next month; yet a future cost cannot simply be imposed on someone whose present capacity is exhausted. The humane alternative is not forced confrontation. It is to widen the action set—adding pacing, choice, accompaniment, preparation, and the right to stop—so that approach is no longer equivalent to helpless exposure.

So the useful question is not "Why does this person avoid?" asked with accusation already built in. It is: What danger does the action prevent now, what information does it prevent from arriving, what life does it protect, and what life does it gradually make impossible? Function comes before judgment.

CORE SENTENCE

Avoidance can be a sensible rescue in the next minute and a costly teacher across the next year.

22

Relief as Reinforcement

BROADER SCIENTIFIC INFERENCE

Avoidance does more than remove a person from a cue. It often produces relief. That immediate fall in anticipated danger or bodily activation can reinforce the action, making it more likely to be selected again. In learning theory this is negative reinforcement: "negative" means that an aversive state is removed, not that the person has behaved badly.

Let $A(s_t)$ represent aversive load in state s_t. The relief generated by an action can be written

$$\rho_t = A(s_t) - A(s_{t+1}), \qquad \delta_t^Q = \rho_t - c(a_t) + \gamma V(s_{t+1}) - Q(s_t, a_t),$$

where $\rho_t > 0$ is immediate relief, $c(a_t)$ is the action's immediate cost, γ discounts future value, $V(s_{t+1})$ is expected value after the transition, and $Q(s_t, a_t)$ is the prior value assigned to the action. If leaving rapidly reduces aversive load, the temporal-difference error δ_t^Q can strengthen the escape policy even when no external catastrophe was actually prevented. This is a formal illustration, not a claim that subjective relief or PTSD can be read from a single reinforcement-learning parameter.

The asymmetry of information is crucial. After leaving, the person directly experiences relief. They do not observe the counterfactual—what would have happened had they remained, with support and genuine control, long enough for new evidence to emerge. The action therefore receives a vivid teaching signal while the safety of the unchosen path remains unknown. Emotional-processing theory framed exposure as activation of a fear structure in the presence of incompatible information [Foa & Kozak, 1986]. Avoidance can interrupt precisely that informational encounter, although this influential account is not itself proof of one universal treatment mechanism.

Clinical association supports a maintenance role for avoidance, but the temporal picture is not simple. In a 20-year study of Israeli veterans, avoidance was particularly stable, while earlier hyperarousal predicted later avoidance and intrusion [Solomon et al., 2009]. Yet laboratory avoidance learning failed to predict subsequent symptoms in a large firefighter cohort [de Haart et al., 2021]. These findings can coexist: the function of lived avoidance may be consequential even when a simplified task does not capture it, and avoidance may sometimes follow arousal rather than initiate it.

Small "safety behaviors" can participate in the same learning loop, but their effects are not predetermined. Sitting near an exit, checking a route, or bringing a trusted person may prevent full disconfirmation; it may also provide enough control for the person to remain and learn. Their function has to be observed. Removing every support in the name of pure learning can turn a manageable experiment into another experience of powerlessness.

Relief is not the enemy. It is often desperately needed. The question is whether relief becomes the only reliable route out of activation. If every escape is rewarded and every tolerable encounter is prevented, the policy can grow more certain while the world grows smaller. That loop is plausible and clinically useful, but its strength and direction must be tested within people and contexts rather than presumed from diagnosis.

CORE SENTENCE

Relief can train a prison without anyone choosing imprisonment.

23

Prediction Error and the Possibility of Updating

EMPIRICAL RESULT

Learning changes when what happens differs from what was expected. That discrepancy is prediction error. A signed error distinguishes better-than-expected from worse-than-expected outcomes; an unsigned error, $|\delta_t|$, marks surprise regardless of direction. Neither is automatically corrective. A surprise can strengthen danger, weaken it, shift attention, or be assigned to a different hidden cause.

A flexible update can be written

$$\delta_t = y_t - \hat{y}_t, \qquad \theta_{t+1} = \theta_t + \alpha_t \, g_t \, \delta_t, \qquad g_t = P(z_t = z_{\text{old}} \mid y_{1:t}, c_t).$$

y_t is the observed outcome, $\hat{y}_t$ the prediction, θ_t a learned parameter, and α_t a learning rate. The gate g_t represents the inferred probability that the current observation belongs to the old situation rather than a new latent cause z_t. If a safe outcome is classified as an exception—"only because the therapist was here"—then g_t may be small and the old threat model changes little. This is a computational proposal, not an observed neural switch.

There is direct, still-limited PTSD evidence for altered model-based updating. In 85 participants, computational estimates of value and latent-state updating during fear conditioning, together with related neural activity, covaried with PTSD symptom severity [Letkiewicz et al., 2022]. Another study found increased associability-modulated learning from losses in PTSD, consistent with altered unsigned prediction-error updating rather than a generic excess of fear [Brown et al., 2018]. In a sample of 56 trauma-exposed adults, greater re-experiencing severity was associated with a lower modeled probability of inferring multiple latent causes during extinction [Norbury et al., 2022]. These parameters are inferred from behavior under a chosen model; they are not directly observed states and have not become diagnostic markers.

Signed and unsigned errors ask different questions. The sign says whether the outcome was safer or more dangerous than predicted. Magnitude says how surprising it was and may alter attention or associability. A system can register surprise accurately yet update little if the evidence is assigned low reliability, if the context is treated as exceptional, or if a competing model explains the event better. "Prediction error occurred" is therefore the start of an analysis, not its conclusion.

Prediction error also helps explain why mere contact with a reminder is insufficient. If an encounter unfolds exactly as expected, little revision is required. If surprise is overwhelming, it may be encoded as fresh danger. If safety is attributed to a narrow circumstance, it may not generalise. Updating is most plausible when new evidence is noticeable, tolerable, credible, and assigned to the model that needs revision.

That formulation is compatible with exposure and reconsolidation accounts, but it does not prove that one error signal mediates all effective therapy. It gives us sharper questions: What did the person predict? What actually occurred? Was the discrepancy registered? Which context or cause received it? Did behavior change outside the session? The possibility of recovery lies not in manufacturing surprise, but in enabling evidence to reach the governing model.

CORE SENTENCE

Change becomes possible when surprise is not merely endured but assigned to the right cause and allowed to revise the model.

PART IV

MEMORY, CONTEXT AND PREDICTION

Memory returns through context, prediction and action, not through a single storage metaphor.

24

Trauma Memory Is Not a Recording

BROADER SCIENTIFIC INFERENCE

A recording preserves an input and plays it back. Human memory does neither. Remembering is a present act of reconstruction, constrained by what was encoded, what has been learned since, the cue that initiates retrieval, the current context, and the purposes of the remembering system. This does not mean memory is arbitrary or that an account of trauma is untrustworthy. It means that fidelity, vividness, confidence, sensory intensity, temporal order, and voluntary control are different properties.

A probabilistic description is

$$p(m \mid q, c, \mathcal{K}) \propto p(q \mid m, c)\, p(m \mid \mathcal{K}),$$

where m is a candidate remembered event representation, q is the retrieval cue, c is the present context, and $\mathcal{K}$ is accumulated knowledge. The likelihood term expresses how well a candidate memory explains the cue in this context; the prior term expresses its accessibility given the person's learned model. This equation is a formal metaphor for reconstructive retrieval, not a claim that testimony can be assigned a simple Bayesian probability or that one can infer historical accuracy from confidence.

PTSD research shows that memory difficulties are not confined to one traumatic episode. A 2026 multilevel meta-analysis of trauma-exposed adults compared diagnosed PTSD with trauma-exposed controls across 90 studies and found average impairments, most consistently in verbal episodic and working memory; substantial heterogeneity and possible publication bias remained [Sulejmani & Pop-Jordanova, 2026]. This is evidence for group-level cognitive differences, not a memory test for PTSD and not proof of permanent damage.

Those average differences can have several contributors: sleep disruption, depression, medication, pain, attention captured by threat, and chronic stress may all affect performance. A neuropsychological score cannot by itself reveal whether the original trauma was encoded differently, whether retrieval is currently obstructed, or whether limited cognitive resources are being diverted elsewhere. Mechanism requires designs capable of separating those alternatives.

When trauma-related autobiographical memories are evoked, neuroimaging meta-analyses implicate distributed systems involved in self-reference, autobiographical retrieval, salience, and sensory processing rather than a single "trauma-memory centre" [Thome et al., 2020] [Sartory et al., 2013]. These studies average small, methodologically varied samples. They cannot tell us whether a particular recollection is accurate, nor whether its neural pattern caused symptoms.

The distinction between content and mode of return is decisive. Someone may retain a coherent factual account yet be involuntarily flooded by images or bodily states. Another person may recall facts but struggle with temporal order. Someone else may have ordinary autobiographical gaps without PTSD. The memory can remain accessible while its prediction of present danger changes; conversely, detailed recall can coexist with severe impairment.

This is why the metaphor of a recording is so damaging. It tempts us to imagine one frozen object that must be replayed, recovered, or erased. The scientific problem is more dynamic: how different memory systems, cues, bodily states, contexts, appraisals, and actions make some aspects of the past available now—and with what consequences. The event happened once. Its reconstruction is a living process.

CORE SENTENCE

A trauma memory is not a recording played back; it is a present act of reconstruction constrained by what was learned.

25

Fragmentation, Sensory Memory and What the Evidence Permits

EMPIRICAL RESULT

Many people describe traumatic remembering as sensory, discontinuous, temporally confused, or difficult to put into words. Those experiences deserve to be heard without forcing them into a universal theory. “Fragmentation” has been used to mean several things: a person’s felt sense that memory is disorganized; a narrative judged by another person to lack chronology or coherence; fewer causal connectives; gaps in recall; or the predominance of images, sensations, and perceptual detail. These measures are not interchangeable.

The evidence is correspondingly mixed. A review of 19 empirical studies found more consistent links between PTSD-related pathology and sensory or perceptual references and disturbed temporal qualities than between PTSD and narrative fragmentation itself. The fragmentation evidence was inconclusive, and measurement validity was a major problem [O’Kearney & Perrott, 2006]. A later review of 16 studies found that associations between dissociation and fragmentation were strongest when both were reported by the same person. The relationship largely disappeared for trait dissociation or independently and computationally coded narratives [Bedard-Gilligan & Zoellner, 2012]. That pattern raises the possibility of shared method variance without making subjective disorganization unreal.

Treatment data further constrain a strong fragmentation theory. In 77 adults with chronic PTSD receiving prolonged exposure or sertraline, objective, rater-based, and self-report features of trauma narratives did not show consistent fragmentation changes that tracked treatment modality or response [Bedard-Gilligan et al., 2017]. In another small trauma-exposed sample, independently rated and language-derived measures of integration did not predict symptom severity after covariate adjustment, although self-rated disorganization remained associated with symptoms [O’Kearney et al., 2011]. These studies do not prove that organization never matters. They show that changing narrative fragmentation is not established as a necessary mechanism of recovery.

Sensory intensity also should not be confused with a memory existing outside all language. Speech can become difficult under high arousal; a recollection can arrive first as an image, smell, pressure, or bodily alarm; and words can feel inadequate to what occurred. None of this establishes that traumatic memories are universally nonverbal, stored intact in the body, or inaccessible to ordinary reconstructive processes. Neuroimaging symptom-provocation studies implicate distributed autobiographical and self-referential systems and do not support one obligatory language-region shutdown [Sartory et al., 2013].

Better work would preregister what fragmentation means, compare trauma narratives with equally emotional non-trauma memories, separate subjective experience from independent coding, and test whether change in the proposed feature precedes functional improvement. Without that temporal and measurement discipline, a compelling description can quietly become an untested mechanism.

The careful position is neither dismissal nor myth. Some trauma memories are experienced as fragmented and unusually sensory. The prevalence, mechanism, and clinical significance depend on how those terms are measured, on dissociation and arousal, on the kind of trauma, and on the moment of retrieval. A person does not have to produce a perfectly ordered narrative to deserve belief or care. Nor must recovery be judged by whether the story becomes chronologically elegant.

CORE SENTENCE

Trauma memory can feel sensory, disordered, or wordless without science licensing one universal architecture of fragmentation.

26

Hippocampal Contextualisation: Pattern Separation and Completion

FLOW HIJACKED SYNTHESIS

The present often resembles the past without being the past. To act well, a nervous system must preserve both similarity and difference. Two complementary computational ideas help: pattern separation makes overlapping experiences more distinguishable; pattern completion allows a partial cue to retrieve a fuller stored representation. Both are adaptive. Separation without completion would make recognition fragile; completion without enough separation would let a fragment summon the wrong whole.

If $\mathbf{r}_i$ and $\mathbf{r}_j$ are neural or representational patterns for two events, their normalized similarity can be written

$$s_{ij} = \frac{\mathbf{r}_i^\top \mathbf{r}_j}{\|\mathbf{r}_i\|_2 \|\mathbf{r}_j\|_2}.$$

Pattern separation can be represented as a transformation that reduces s_{ij} for confusable inputs; pattern completion as retrieval

$$m^* = \arg\max_{m \in \mathcal{M}} p(m \mid q, c),$$

where a partial cue q and context c select the most probable memory m^* from a set $\mathcal{M}$. These equations clarify a computational tension. They do not demonstrate that PTSD is a failure of either operation, and a similarity metric is not an individual biomarker.

The hippocampus is central to contextual and relational memory, but simple localization is misleading. In a small three-context PTSD experiment, altered return of fear involved hippocampal, amygdala, prefrontal, and autonomic measures together [Wicking et al., 2016]. Threat can also affect behavioral pattern separation while recruiting prefrontal control; one experimental study found a right-dlPFC effect rather than the expected hippocampal result [Balderston et al., 2017]. Conceptual fear generalisation in PTSD further suggests that what counts as "similar" can be semantic and relational, not merely perceptual [Morey et al., 2020].

Bridge evidence is provocative. Experimental work on context-impoverished memories showed that restricted context encoding produced broader and more persistent fear, while later contextual updating could improve or misdirect responding depending on where updating occurred [Zinn et al., 2020]. That study offers a computational mechanism relevant to PTSD; it does not establish the mechanism in diagnosed PTSD. Likewise, smaller average hippocampal volumes are repeatedly reported at group level, but twin evidence suggests that at least some difference may precede trauma, so anatomy cannot tell a single causal story [Logue et al., 2018] [Gilbertson et al., 2002].

The balance between completion and separation may also be state-dependent rather than fixed. High arousal, poor sleep, a matching bodily sensation, or a strong contextual cue could temporarily favor completion; another moment may permit fine discrimination. Repeated within-person tasks across contexts would test that possibility more directly than a single scanner average.

The Flow Hijacked use of these concepts is therefore functional. A cue may complete into a whole threat situation too readily, while contextual details that distinguish "then" from "now" fail to constrain retrieval strongly enough. That is a testable synthesis, not an established ontology. It predicts measurable confusion among highly similar contexts, excessive retrieval from partial cues, and improvement when contextual discrimination becomes more reliable. It would weaken if those operations do not differ after symptoms, exposure, sleep, medication, and comorbidity are controlled.

CORE SENTENCE

Context lets a nervous system register resemblance without mistaking resemblance for recurrence.

27

Intrusions and the Involuntary Return of the Past

EMPIRICAL RESULT

An intrusion is not simply a strong memory. It is a memory-related event that enters awareness without being deliberately summoned and is difficult to disengage from. It may arrive as an image, phrase, bodily sensation, sound, emotion, or compressed scene. Intrusions are central to PTSD, but they also occur after trauma without PTSD and in other conditions. Their presence alone does not establish a diagnosis.

A conceptual observation model is

$$P(I_t = 1) = \sigma\big(\eta_0 + \eta_q Q_t + \eta_b B_t + \eta_a A_t - \eta_u U_t\big)\,,$$

where I_t indicates an intrusion at time t, Q_t is cue–memory match, B_t bodily-state congruence, A_t current arousal, and U_t effective top-down control. The coefficients η express candidate contributions, not known universal effects. The equation is meant to prevent a single-cause story: an intrusion can become more probable through several routes, and the relevant configuration can change within the same person.

A systematic review connecting cognitive science and clinical research treats intrusive trauma memories as a distinct, measurable target while documenting substantial variation in paradigms and mechanisms [Iyadurai et al., 2019]. Neuroimaging meta-analysis of trauma-related autobiographical memory implicates broad self-referential, salience, and memory systems rather than one replay device [Thome et al., 2020]. Another symptom-provocation meta-analysis highlighted retrosplenial and precuneus involvement alongside amygdala and anterior-cingulate effects, with important inconsistency among small studies [Sartory et al., 2013]. Distributed activation is not evidence that an episode is re-enacted exactly as it occurred.

Control research adds a more specific clue. In an experimental memory-suppression paradigm, PTSD was associated with altered predictive control; when proactive control failed, prediction-error responses linked to intrusions were less effectively downregulated by reactive control [Leone et al., 2022]. This is direct mechanistic evidence within one task, not proof that all clinical intrusions arise from failed suppression. Indeed, forceful thought suppression can itself be counterproductive, and the relation between control, acceptance, attention, and intrusion depends on timing and strategy.

Intrusion, flashback, and ordinary unwanted recollection also require separation. They can differ in sensory vividness, loss of present orientation, duration, conviction, and bodily recruitment. Counting events alone misses whether a person can recognize "this is a memory," remain anchored to the room, and recover afterward. Frequency, state-entry intensity, and return time are distinct dynamical outcomes; improvement in one need not imply improvement in all.

The language matters. An intrusion is not evidence that a person is choosing the past, refusing the present, or failing to exercise willpower. Nor is it necessarily a complete and accurate replay. It is a present event generated when memory accessibility, cue similarity, bodily state, context, and control align in a particular way. Treatment can reduce its frequency, its intensity, its credibility as evidence of current danger, or the time needed to recover afterward. Those are different outcomes and should be measured separately.

CORE SENTENCE

An intrusion is not the past returning unchanged; it is the present losing control over how the past becomes active.

28

Reconsolidation: Updating Without Myth

BROADER SCIENTIFIC INFERENCE

After initial consolidation, a memory is not necessarily sealed forever. Under some conditions, retrieval can make aspects of it labile before they are restabilized—a process called reconsolidation. The phrase "under some conditions" carries most of the science. Reactivation is not identical to destabilization; destabilization cannot usually be observed directly in clinical work; and a retrieved memory may be strengthened, left unchanged, linked to a new context, or updated.

A boundary-sensitive model is

$$M_{t+1} = \begin{cases} \mathcal{R}(M_t, \varepsilon_t, c_t), & D_t = 1, \\ M_t, & D_t = 0, \end{cases} \qquad D_t \in \{0, 1\}.$$

M_t is the current memory representation, ε_t is new prediction-error information, c_t is context, and $\mathcal{R}$ is a restabilization operator. D_t indicates whether destabilization actually occurred. In most human clinical studies D_t is latent; it is inferred after the fact. This makes failed updating scientifically ambiguous: the intervention may be ineffective, or the memory may never have entered the proposed window.

Reconsolidation is a well-developed memory framework [Lee, 2009], and systematic synthesis finds clinically promising signals across consolidation- and reconsolidation-oriented procedures [Astill Wright et al., 2021]. A propranolol meta-analysis reported modest average reductions across heterogeneous healthy and clinical samples, while emphasizing methodological differences [Pigeon et al., 2022]. But the PTSD trial record does not tell one triumphant story. A randomized study of 60 adults reported greater symptom reduction when propranolol preceded traumatic-memory reactivation [Brunet et al., 2018]. A later double-blind trial in 66 adults found substantial improvement in both propranolol and placebo-reactivation groups, without an overall post-treatment advantage for propranolol [Roullet et al., 2021]. Three small psychophysiological studies also found no group differences in reactivity or symptoms [Wood et al., 2015].

Even a positive clinical comparison would not identify the molecular route by itself. To support a reconsolidation mechanism, studies need a credible reactivation manipulation, a priori boundary conditions, an appropriate control, outcomes tied to the reactivated memory, and later tests of reinstatement, renewal, or spontaneous recovery. Improvement immediately after repeated retrieval can be valuable while still being mechanistically nonspecific. The mechanism claim and the clinical benefit claim must travel on separate evidential tracks.

These results prohibit the claim that every act of remembering rewrites the memory, that propranolol erases trauma, or that symptom improvement proves reconsolidation blockade. Exposure, expectancy, repeated contact, therapeutic attention, spontaneous change, and measurement can all contribute. Return-of-fear and durable functional outcomes matter more than an attractive mechanism label.

The useful promise is quieter. Memory can sometimes be updated rather than merely suppressed, and treatment may create conditions in which old predictions lose control over present action. The research task is to identify the boundary conditions prospectively: mismatch strength, retrieval duration, memory age and strength, arousal, context, timing, and individual variation. "Reconsolidation" should name a testable process, never serve as a magic word.

CORE SENTENCE

Retrieval can open a memory to change, but "opened" is an empirical condition, not a therapeutic incantation.

29

Threat Priors in Predictive Processing

FLOW HIJACKED SYNTHESIS

Perception is not passive receipt. A predictive system uses prior learning to infer the hidden causes of incomplete and noisy sensory input. In Bayesian notation,

$$p(z_t \mid y_t, c_t) = \frac{p(y_t \mid z_t, c_t)\, p(z_t \mid c_t)}{\sum_{z' \in \mathcal{Z}} p(y_t \mid z', c_t)\, p(z' \mid c_t)}.$$

$z_t \in \mathcal{Z}$ is one of a discrete set of possible hidden states—such as danger or safety—y_t is current sensory evidence, and c_t is context. The prior $p(z_t \mid c_t)$ reflects what was plausible before the new evidence arrived; the likelihood $p(y_t \mid z_t, c_t)$ describes how expected that evidence would be under each state. The posterior is the revised belief. This formalism does not require a person to hold a conscious belief, and it does not mean the brain literally evaluates this sum.

Trauma can rationally alter priors. After a severe or repeated event, danger is no longer a remote theoretical possibility. Ambiguous footsteps, a closed door, a bodily surge, or a delay in communication may carry a different posterior probability because the learned base rate has changed. If threat was concealed, unpredictable, or delivered by a trusted person, evidence once treated as reassuring may also lose reliability. A "strong threat prior" is therefore not synonymous with irrationality.

Predictive-processing accounts of stress and trauma offer a coherent vocabulary for this shift, but they remain theoretical integrations rather than a confirmed unitary account of PTSD [Krupnik, 2020]. Direct computational evidence is narrower. Model-derived value and latent-state updating during conditioning have covaried with PTSD severity [Letkiewicz et al., 2022]. Greater re-experiencing has been associated with a lower modeled probability of inferring multiple latent causes during extinction [Norbury et al., 2022]. People with PTSD have also shown difficulty predicting ongoing events in two experimental tasks [Eisenberg et al., 2023]. Each result depends on a particular sample, task, and model; none measures a single "trauma prior."

Priors are plural and hierarchical. Expectations about a sound, a person, a place, one's own body, and the stability of the world can change differently. A person may expect a room to be safe but expect their own panic to become uncontrollable. Calling both "the threat prior" is convenient, but a serious model must say which level is altered and what observation could revise it.

The framework becomes useful when it generates discriminating predictions. If priors dominate ambiguous evidence, group differences should be greatest when cues are uncertain and should shrink when safety evidence becomes reliable. If the problem instead lies mainly in sensory noise, manipulations of signal quality should matter more. If altered action policy drives the effect, differences should depend on whether participants can escape or sample further information. These alternatives must be compared rather than renamed in Bayesian language.

Within Flow Hijacked, threat priors are one component of a larger system. Bodily states supply evidence, contexts gate retrieval, actions determine what will be sampled next, and social environments determine whether reassurance is credible. The framework earns its place only if it predicts more than ordinary descriptions such as hypervigilance or negative appraisal.

CORE SENTENCE

A strong threat prior can make the world appear to keep proving the prior right.

30

Precision, Ambiguity and Uncertainty

FLOW HIJACKED SYNTHESIS

Bayesian updating depends not only on what prior and sensory evidence say, but on how reliable each is estimated to be. Precision is inverse uncertainty. In a simple Gaussian case,

$$\mu_{\text{post}} = \frac{\pi_p \mu_p + \pi_y y}{\pi_p + \pi_y}, \qquad \pi_p = \sigma_p^{-2}, \quad \pi_y = \sigma_y^{-2}.$$

μ_p is the prior mean, y the current observation, and π_p and π_y their respective precisions. When prior precision is high and sensory precision is low, ambiguous evidence moves the posterior little. When trustworthy new evidence is precise, it can produce substantial revision. This is a didactic special case; lived threat inference is hierarchical, non-Gaussian, action-dependent, and distributed across time.

Precision is not confidence in the everyday sense, and high precision is not always pathological. In a stable dangerous environment, a reliable threat expectation should govern behavior. In a volatile environment, yesterday's safety may genuinely say little about today. The difficult case is miscalibration: evidence is treated as more or less reliable than conditions warrant, or uncertainty itself becomes so costly that the system repeatedly chooses vigilance, escape, or premature closure.

The PTSD evidence here is indirect and heterogeneous. In a treatment-seeking sample of 116 veterans, intolerance of uncertainty was associated with PTSD severity beyond measured covariates [Raines et al., 2019]. Experimental work has found difficulty predicting unfolding events in PTSD [Eisenberg et al., 2023], and computational conditioning studies report symptom-related differences in value or latent-state updating [Letkiewicz et al., 2022]. These findings make precision-based hypotheses plausible. They do not show that researchers have directly measured a global "precision-weighting deficit," and intolerance of uncertainty is not identical to computational precision.

Ambiguity can enter at several levels. The cue may be noisy: Was that sound a threat? The context may be uncertain: Is this place governed by the old rules? Volatility may be uncertain: How quickly do rules change? Social evidence may be unreliable: Can this person's reassurance be trusted? Collapsing these into one score would lose the mechanism. A rigorous study should manipulate or estimate them separately and test out-of-sample predictions.

Precision may also be allocated dynamically. Attention can increase the gain on external threat cues; interoceptive focus can increase the influence of heartbeat, breath, or tension; fatigue can reduce the reliability of contextual detail. None of these is necessarily a stable trait. The same person may weight evidence differently at noon and at three in the morning, alone and with a trusted other, rested and sleep-deprived. That within-person variation is a central empirical target.

The humane implication is that uncertainty is not a failure of courage. For many people, uncertainty was where danger lived. Asking the system to assign high precision to verbal reassurance before repeated experience supports it is asking for belief without evidence. Recovery may involve making safety evidence clearer, more controllable, and more portable—not simply instructing the person to lower a prior.

CORE SENTENCE

Precision decides which evidence is allowed to change the mind.

31

Active Inference, Avoidance and Missing Corrective Evidence

FLOW HIJACKED SYNTHESIS

Predictive processing concerns how observations revise beliefs. Active inference adds a consequential step: action changes the observations that will become available. We do not only infer the world from data; we move, look, withdraw, seek help, scan, freeze, approach, or avoid, thereby selecting part of the next data stream.

One formal expression assigns each policy π an expected free energy,

$$G(\pi) = \mathbb{E}_{q(o,s|\pi)}\left[\ln q(s \mid \pi) - \ln p(o,s)\right], \qquad q(\pi) \propto \exp[-\gamma G(\pi)].$$

s denotes hidden states, o future observations, q the agent's predictive distribution under policy π, $p(o,s)$ a preferred generative model, and γ policy precision. Depending on assumptions, $G(\pi)$ can be decomposed into expected cost or risk and an epistemic term reflecting information gain. The equations describe a model family. They do not establish that people with PTSD minimize this exact quantity or possess one abnormal value of γ.

Avoidance becomes especially important because it can be both protective and information-selecting. Leaving a genuinely dangerous place prevents harm. Leaving an ambiguous but safe situation may also prevent the observation that catastrophe did not occur. The action produces immediate relief, while the unchosen counterfactual remains invisible. Over time, a policy can therefore preserve the model that keeps selecting it. This resembles the continuing-current-threat architecture of cognitive PTSD theory [Ehlers & Clark, 2000] and active-inference accounts of interoceptive psychopathology [Paulus et al., 2019], but resemblance is not causal proof.

Direct evidence is modest. PTSD studies of latent-cause inference and value updating show that computational quantities can relate to re-experiencing or symptom severity [Norbury et al., 2022] [Letkiewicz et al., 2022]. Yet a prospective task study in 502 firefighters found that avoidance learning did not predict later symptoms [de Haart et al., 2021]. That null result matters: "avoidance maintains PTSD" at a clinical level does not imply that every formal avoidance parameter is a vulnerability marker. Models must predict which avoidance, under which uncertainty, in which environment, over what timescale.

The strongest test would manipulate information availability while preserving autonomy. If missing corrective evidence is causal, then supported approach that permits observation should update beliefs more than equal-duration contact in which the person remains certain they cannot act. If agency, not information, is decisive, the pattern should differ. Active-inference language is useful only when it forces such competing predictions.

The framework also guards against blame. If action has been shaped by credible danger, relief, loss of control, and unreliable safety signals, the policy is learned rather than chosen from nowhere. Change cannot be demanded by withholding safety. It requires conditions in which exploration is tolerable, control is genuine, and new evidence can be encountered without violating the person's boundaries.

This section prepares the central Flow Hijacked hypothesis but does not prove it. Threat-State Return Asymmetry will ask whether trauma changes not merely beliefs or symptoms, but conditional rules for entering and leaving defensive states. Active inference supplies one possible maintenance loop: actions selected under threat prevent the observations that might reopen other policies. The loop must still be measured, compared with alternatives, and allowed to fail.

CORE SENTENCE

When action repeatedly blocks disconfirming experience, a protective policy can preserve the world-model that keeps selecting it.

PART V

BEYOND THE AMYGDALA

PTSD is distributed across interacting systems, not located in one frightened region.

32

Why the Two-Region Cartoon Fails

BROADER SCIENTIFIC INFERENCE

The familiar picture is easy to remember: an overactive amygdala sounds the alarm while an underactive prefrontal cortex fails to restrain it. It helped move PTSD away from moral judgment and toward biology. But as a scientific account, it is now too small. It turns many forms of learning, remembering, sensing, acting and regulating into a contest between two anatomical labels. It also implies a fixed direction of abnormality—more activity here, less activity there—when the observed direction often changes with the task, cue, comparison group and moment of measurement.

Meta-analytic evidence already required a broader view. A synthesis of emotional-processing studies found amygdala and insula hyperactivation across anxiety-related diagnoses, not uniquely in PTSD, while reported prefrontal and cingulate differences varied across tasks and regions [Etkin & Wager, 2007]. A PTSD-focused meta-analysis subsequently supported interacting salience, affective, memory and regulatory systems rather than an isolated amygdala–prefrontal imbalance [Patel et al., 2012]. Even symptom-provocation studies implicated retrosplenial cortex, precuneus, anterior cingulate, sensory-association cortex and autobiographical-memory processes alongside the amygdala [Sartory et al., 2013]. The pattern is distributed before it is simple.

A more disciplined description treats a measured regional response as conditional:

$$\Delta y_r = f_r(q, \tau, \kappa, \chi, \mathbf{s}, \mathbf{m}, \boldsymbol{\theta}) + \varepsilon_r.$$

Here, Δy_r is the observed change in signal from region r; q is the cue; τ is its timing within learning or recall; κ is task demand; χ is context; $\mathbf{s}$ represents the person's current physiological and psychological state; $\mathbf{m}$ includes medication, sleep and comorbidity; $\boldsymbol{\theta}$ denotes relatively stable individual characteristics; and ε_r contains measurement error and unmodeled variation. The response, fitted function and error share the study-specific imaging-contrast units; other inputs must be encoded and scaled explicitly. This is not offered as a fitted law of PTSD. It is a formal reminder that "activity in a region" has no context-free meaning.

The empirical contradictions are therefore informative rather than embarrassing. One conditioning study found reduced, not elevated, amygdala responsivity to trauma-related stimuli in PTSD [Diener et al., 2016]. In an imagery study, a significant left-amygdala increase appeared only in male combat veterans, despite inverse amygdala–medial-prefrontal relationships more broadly [Shin et al., 2004]. A much larger 32-site analysis detected group-level differences in resting connectivity among amygdala, hippocampal and related regions, yet those findings remained associations among signals at rest—not a diagnostic circuit and not a causal account of a person's symptoms [Hinojosa et al., 2026].

Imaging measurements add another boundary. Blood-oxygen-level-dependent fMRI is an indirect, temporally blurred correlate of local metabolic and vascular activity. Functional connectivity commonly means statistical dependence, not anatomical wiring or directional control. The apparent network depends partly on preprocessing, parcellation, motion correction, time scale and statistical threshold. A group contrast cannot tell us that every member of one group has the reported pattern, or whether the pattern preceded trauma, followed it, reflected treatment, or arose from a correlated condition.

Leaving the cartoon behind does not mean that the amygdala or prefrontal cortex is irrelevant. It means asking a better question: how do memory, valuation, bodily regulation, sensory routing, attention, action selection and social context become coordinated at particular moments? PTSD is not situated in a frightened spot. It is expressed through changing relations among systems—and through what those relations allow the whole person to perceive, predict and do.

CORE SENTENCE

PTSD is not an alarm region overpowering a control region; it is a heterogeneous disturbance in how distributed systems coordinate under particular conditions.

33

The Amygdala Is a Participant, Not a Verdict

BROADER SCIENTIFIC INFERENCE

The amygdala matters. The error lies not in including it, but in asking it to explain too much. It is a collection of nuclei with different cellular compositions and connections, participating in learned significance, uncertainty, attention, autonomic preparation and the selection of responses. A change in one amygdala measure is therefore neither a verdict that danger is present nor a neural certificate that PTSD exists.

Even the phrase "amygdala activation" compresses several decisions into one number. If $a_k(q, \tau)$ denotes the measured response of amygdala subdivision k to cue q at task time τ, the reported contrast is typically

$$\Delta a_k = a_k(q_{\text{target}}, \tau) - a_k(q_{\text{control}}, \tau).$$

Both terms and their difference use the experiment's imaging-signal units. The sign depends on the control condition, habituation, novelty, anticipation, learning phase, cue ambiguity and analysis model. A negative contrast need not mean that the amygdala is globally inactive; a positive contrast need not mean that it is continuously overactive. The contrast describes a relation constructed by an experiment.

Across studies, that relation is not uniform. Meta-analytic work found amygdala hyperactivation during emotional processing in PTSD, but also across other anxiety conditions, limiting diagnostic specificity [Etkin & Wager, 2007]. Trauma-imagery research reported sex-dependent results, with a significant left-amygdala increase confined to male combat veterans in one study [Shin et al., 2004]. Conversely, a conditioning experiment found reduced amygdala responsivity to trauma-related stimuli in the PTSD group [Diener et al., 2016]. Such findings do not cancel one another. They show that cue meaning and task phase matter, and that "more amygdala" is not a stable phenotype.

Nor is the amygdala a single unit. Recent work has examined connectivity profiles of its subregions rather than treating the structure as homogeneous [Haris et al., 2025]. A large consortium analysis found associations between particular amygdala-nucleus volumes, symptom dimensions and childhood adversity, but the results were nucleus-specific and correlational rather than evidence for one volumetric PTSD signature [Mufford et al., 2026]. A 32-site mega-analysis likewise detected resting-connectivity differences involving amygdala and hippocampal seeds, yet these remained overlapping group distributions rather than individual-level verdicts [Hinojosa et al., 2026].

The question of origin is also unresolved. A 2026 monozygotic-twin study attempted to distinguish familial from unique environmental contributions to amygdala hyperresponsivity [Beucke et al., 2026]. Its design is unusually informative because genetically identical twins provide leverage that an ordinary case-control comparison cannot. But only 26 twin pairs were studied. The result deserves attention, not conversion into a universal causal story.

Within the Flow Hijacked lens, the amygdala is best understood as one participant in a conditional transition. Its response may help bias perception, bodily readiness and action toward defense, but whether the whole system enters, sustains or leaves a defensive state depends on hippocampal context, cortical valuation, thalamic and sensory routing, brainstem and autonomic processes, learned avoidance, present danger and social conditions. The same measured amygdala response can have different consequences in different system configurations.

This distinction is humane as well as technical. A person is not "ruled by an overactive amygdala." Nor is recovery the victory of a rational cortex over an irrational animal brain. Both metaphors erase the adaptive history of the response and the distributed work required for return.

CORE SENTENCE

The amygdala can bias a transition toward defense, but it neither contains PTSD nor determines the state of the person by itself.

34

Hippocampus, vmPFC and dACC: Context, Value and Action

BROADER SCIENTIFIC INFERENCE

If the amygdala helps register learned significance, it still cannot determine what a cue means here, now, for this person. That requires context, remembered relations, current value and possible action. The hippocampus, ventromedial prefrontal cortex (vmPFC) and dorsal anterior cingulate cortex (dACC) participate in those functions, but none maps cleanly onto a single psychological verb. "Context center," "safety center" and "fear center" are labels of convenience, not anatomical truths.

One useful formal description begins with a latent context z_t that cannot be observed directly. From present and past observations $\mathbf{y}_{1:t}$, the system forms a belief distribution

$$b_t(z) = P(z_t = z \mid \mathbf{y}_{1:t}, \mathbf{c}_{1:t}) ,$$

where $\mathbf{c}_{1:t}$ contains spatial, temporal, bodily and social context. Action can then depend on both this belief and anticipated value:

$$\pi(a_t \mid b_t) \propto \exp[\beta\, Q(a_t, b_t)] .$$

Here, π and b_t are dimensionless probability distributions, Q is expected value in the task's chosen units, and the inverse-temperature parameter β has reciprocal-value units and controls how selectively the higher-valued action is chosen. These equations describe a computational problem, not a one-to-one assignment of b_t to hippocampus, Q to vmPFC or action to dACC. Neural implementation is distributed and reciprocal.

Structural evidence illustrates both the reality and modest scale of hippocampal findings. In an ENIGMA-PGC study of 1,868 participants, PTSD was associated with a small average hippocampal-volume difference, $d = -0.17$; distributions substantially overlapped, and the amygdala-volume difference did not survive region-wise correction [Logue et al., 2018]. A discordant-twin study suggested that smaller hippocampal volume may partly precede trauma-related PTSD vulnerability [Gilbertson et al., 2002]. Yet prospective work in soldiers also associated post-service hippocampal-volume reduction and weaker hippocampus–vmPFC connectivity with greater subsequent post-traumatic symptoms [Admon et al., 2013]. Together, these findings permit both antecedent vulnerability and experience-related change. They do not establish one universal sequence.

The vmPFC is often presented as a brake on the amygdala. That metaphor is too mechanical. Depending on task and context, vmPFC-related signals can participate in valuation, inferred safety, extinction recall and the integration of present information with prior learning. Reduced coupling does not prove that a cortical brake has failed, and increased coupling does not necessarily mean recovery. Direction, timing and the nodes being coupled all matter.

The dACC is similarly irreducible to "fear expression." It participates in monitoring conflict, action relevance, effort and autonomic coordination across many conditions. A resting-fMRI study of drug-naïve participants with PTSD found altered amygdala–dACC interactions and coupling with prefrontal, parietal, visual and fusiform regions [Li et al., 2021]. Because that study was cross-sectional and its interaction measure did not establish directed causal influence, it supports dysconnectivity in one sample—not a universal top-down or bottom-up mechanism.

The more defensible synthesis is relational. The hippocampus may help discriminate which situation is being revisited; vmPFC-associated computations may contribute to what the current situation is worth and whether safety evidence is credible; dACC-associated computations may help organize attention, bodily demand and action when competing responses matter. PTSD can involve disturbance in any of these relations without requiring uniform damage to any node. What persists may be not a broken module, but a context-sensitive coordination policy that too often selects protection and too slowly releases it—a possible component of Threat-State Return Asymmetry.

CORE SENTENCE

Context, value and action emerge from coordinated relations among hippocampal, prefrontal, cingulate and wider systems—not from three regions performing three fixed jobs.

35

Insula and Salience: What Demands the Organism Now

BROADER SCIENTIFIC INFERENCE

A sound in the street, a tightening chest, an ambiguous face and a sudden memory are physically different events. Yet each can become the most important fact in the world for a few seconds. The insula and the broader salience network help us study this prioritization: how signals from outside and inside the body come to demand attention, reorganize processing and prepare action. They do not constitute a "trauma center," and salience is not another word for fear.

A conceptual expression makes the distinction clear:

$$\mathcal{S}_t = \Phi\left(\mathbf{e}_t, \mathbf{i}_t, \hat{\mathbf{e}}_t, \hat{\mathbf{i}}_t, \mathbf{g}_t, \mathbf{c}_t\right).$$

Here, $\mathcal{S}_t$ is a dimensionless modeled priority score; $\mathbf{e}_t$ and $\mathbf{i}_t$ are scaled exteroceptive and interoceptive signals; their hats denote predicted signals; $\mathbf{g}_t$ represents current goals; and $\mathbf{c}_t$ is context. The function Φ is deliberately unspecified. It does not claim that the brain computes one scalar called salience, or that the insula alone implements it. It says that urgency depends on relations among sensation, expectation, bodily state, goal and setting.

PTSD research supports involvement of this interface, but not one direction of abnormality. An integrative review connected interoceptive processing, fear learning and PTSD while emphasizing that the proposed links span several partially tested mechanisms [Joshi et al., 2023]. In firefighters with PTSD, trauma-related interference was associated with stronger left-insula activation and weaker patterns of insular functional connectivity [Lee et al., 2022]. These are meaningful group-level findings in a particular occupational and experimental context, not evidence that all people with PTSD possess the same insular state.

Specificity is an even stronger limitation. A meta-analysis found altered dorsal mid-insula activation during interoception across psychiatric disorders [Nord et al., 2021]. That transdiagnostic result matters: it suggests that some insular differences may index disturbed bodily attention, arousal or regulation shared by several conditions rather than PTSD itself. Depression, panic, dissociation, medication, sleep loss, pain and cardiorespiratory differences can all affect the processes being measured.

Prospective evidence adds a temporal dimension while also demonstrating why caution is necessary. In police recruits studied before and after repeated occupational trauma exposure, acute-stress-induced changes in salience-network coupling predicted later perceived stress, but not overall post-traumatic stress symptoms. Increased salience-network–cerebellar coupling was related to higher clinician-rated PTSD symptoms, particularly intrusions [Zhang et al., 2022]. The same study therefore contains both a promising network signal and a boundary: a predictor of one later outcome did not generalize to the diagnosis as a whole.

The salience network is often described as a switch between default-mode and executive systems. That metaphor is useful only if it remains a metaphor. Network switching is distributed, the relevant nodes belong to multiple configurations, and inferred connectivity does not reveal a command center. In lived terms, the important possibility is subtler: some systems may assign urgent priority to weak, ambiguous or bodily cues because those cues once carried crucial information. That is not irrationality. It is protection operating under changed probabilities. Whether this priority becomes a persistent defensive state depends on what happens next across memory, action, autonomic regulation and context.

Part VI will bring the body explicitly into this loop. For now, the crucial point is that salience is relational. A heartbeat is not inherently a threat signal, and an insular response is not inherently pathological. Meaning emerges from the model in which the signal is received.

CORE SENTENCE

The insula and salience network help determine what demands attention now, but urgency arises from a distributed relation among body, cue, prediction, goal and context.

36

Default-Mode and Frontoparietal Systems

BROADER SCIENTIFIC INFERENCE

PTSD affects more than the detection of threat. It can alter how experience is related to the self, how attention is allocated, how a goal is held in mind and how the present is distinguished from an autobiographical past. These functions bring default-mode and frontoparietal systems into the account. Their names can mislead: the default-mode network is not simply "off task," and the frontoparietal network is not a single executive. Both are statistical organizations of coordinated activity, with boundaries that depend on method and time scale.

Default-mode regions—including medial prefrontal, posterior cingulate, precuneus and related temporal structures—are frequently engaged when cognition concerns autobiographical memory, self-relevance, social inference or internally generated scenes. A meta-analysis of trauma-provocation studies found PTSD-related activity in retrosplenial cortex and precuneus, alongside anterior cingulate, amygdala and sensory-association differences [Sartory et al., 2013]. This distributed pattern is more consistent with re-experiencing as a reconstruction involving self, memory and sensory attention than with a stored image released by one region.

Frontoparietal systems contribute to maintaining goals, selecting information and flexibly configuring task demands. But "more control" is not always the relevant solution. A system can exert strong control in the service of scanning, suppressing, avoiding or maintaining distance from experience. What matters is not the quantity of control in the abstract, but which goal it serves, what information it admits and how readily the configuration can change.

That changing organization can be represented by time-dependent community membership. Let $w_{ik}(t) \in [0, 1]$ denote the degree to which node i participates in network k at time t, with

$$\sum_{k=1}^{K} w_{ik}(t) = 1.$$

A node need not belong permanently to one network. Its functional allegiance can shift with memory, task, arousal and context. The equation is a modeling convention, not a claim that the brain literally carries labeled network memberships.

The empirical literature is much thinner than the popularity of these network labels suggests. A systematic review of neuroimaging during self-appraisal located only five eligible PTSD studies. It linked altered self-appraisal with default-mode regions and with systems involved in control, salience and valuation, but methodological and trauma-history differences prevented quantitative meta-analysis [Agathos et al., 2024]. The result makes self-related processing visible while showing how little evidence warrants a universal "disrupted self network."

A community study of 569 adults approached the problem across several biopsychosocial domains. Poor sleep, temperament and impulsivity were consistently related to symptom severity, while connectivity among default-mode, central-executive and salience regions explained additional variance [Bainter et al., 2024]. Additional variance is not destiny, diagnosis or mechanism. The design did not establish whether network differences generated symptoms, followed them or reflected another shared influence.

The distinction between network content and network dynamics should remain clear. This section concerns the kinds of work distributed systems can support: autobiographical location, self-appraisal, goal maintenance and flexible attention. The next temporal question is whether their relations assemble differently from moment to moment. A static group association cannot answer that question, and a network name cannot substitute for the mental operation actually measured.

The honest conclusion is therefore neither "networks explain everything" nor "networks tell us nothing." Default-mode and frontoparietal descriptions are useful coordinate systems for testing how self-related and goal-directed processes interact. They become misleading when drawn as fixed boxes, when "connectivity" is made synonymous with communication, or when a group difference is converted into a permanent architecture of the traumatized person.

CORE SENTENCE

Default-mode and frontoparietal systems matter because trauma can alter the coordination of self, memory and goal—not because either network has one fixed PTSD state.

37

Thalamus, PAG, Locus Coeruleus and Striatum

BROADER SCIENTIFIC INFERENCE

A cortex-only account leaves out systems that route sensory information, coordinate defensive action, regulate arousal and shape which actions become worth repeating. The thalamus, periaqueductal gray (PAG), locus coeruleus and striatum belong in a distributed PTSD model. But they should not be collapsed into a "primitive survival brain." Each is internally heterogeneous, densely connected and involved in ordinary adaptive life as well as threat.

The thalamus is not a passive relay. Its nuclei participate differently in sensory selection, cortical coordination, memory and motor preparation. A 2026 structural-covariance analysis segmented 25 nuclei per hemisphere in 2,784 participants, including 1,306 with PTSD. It found stronger intrathalamic and thalamocortical covariance on average, with different patterns related to avoidance, hyperarousal and comorbid depression [Steele et al., 2026]. Structural covariance, however, is correlated anatomical variation across people. It is not an axonal tract, a moment-to-moment interaction or evidence of causal routing.

High-resolution resting-state work in 397 participants likewise found nucleus-specific rather than globally "high" or "low" thalamic connectivity. Pulvinar connectivity with sensorimotor and salience regions was weaker, medial geniculate connectivity with sensorimotor cortex was stronger, and canonical networks coupled differently to distinct thalamic nuclei [Steele et al., 2026]. These results make the network account more precise while remaining cross-sectional group associations.

The PAG contributes to organizing defensive physiology and action, but human imaging cannot simply read "freeze," "fight" or "shutdown" from a PAG signal. In a small task-fMRI dynamic-causal-modeling analysis, PTSD was associated with stronger estimated excitatory influence from PAG toward precuneus and medial-prefrontal regions during subliminal trauma-related cues [Terpou et al., 2020]. The model favored bidirectional interactions, which is important. Yet effective connectivity is an estimate conditional on a restricted model space; it is not direct observation of neural causation, and the comparison used healthy rather than trauma-exposed controls.

The locus coeruleus is a small brainstem noradrenergic nucleus implicated in arousal, vigilance and adaptive gain. Direct human PTSD measurement remains technically sparse. In one fear-generalization study, veterans with PTSD generalized learned fear toward higher-intensity cues while engaging a distributed set that included visual, insular, thalamic and locus-coeruleus regions, alongside weaker vmPFC safety-related connectivity [Morey et al., 2015]. The finding supports a coordinated generalization process. It does not establish that tonic "excess norepinephrine" explains PTSD, nor that a locus-coeruleus signal determines subjective danger.

The striatum extends the model from threat detection to action selection, reinforcement, habit and reward. A cross-sectional multimodal study reported hippocampus–striatum hyperconnectivity and reduced temporal variability across trauma-related groups, while reduced tract integrity was confined to participants with comorbid postconcussive symptoms and PTSD [Rangaprakash et al., 2017]. Because mild traumatic brain injury and PTSD were entangled and the classifier lacked independent validation, the phrase "imaging biomarker" in the title should not be mistaken for clinical readiness.

Together, these structures show why persistence cannot be reduced to fear intensity. Sensory routing can favor particular inputs; arousal can alter gain; defensive patterning can prepare the body; action systems can make avoidance immediately valuable. Flow Hijacked treats their coordination as a possible pathway by which a small cue acquires large consequences. The empirical literature supports the components. It does not yet establish one closed-loop mechanism, one direction of influence or one shared configuration across PTSD.

CORE SENTENCE

Subcortical and brainstem systems help route, amplify and act on significance, but their contribution to PTSD is distributed, conditional and not reducible to a primitive alarm circuit.

38

Network Reconfiguration and Dynamic Connectivity

BROADER SCIENTIFIC INFERENCE

A conventional connectivity map averages an entire scan. It can tell us that two signals covary, but not whether their relation is stable, intermittent or confined to brief episodes. For a condition defined partly by transitions—into intrusion, vigilance, avoidance or dissociation—that loss of time may be decisive. Dynamic connectivity asks not only which systems are related, but when, for how long and in what sequence.

For signals $x_i(s)$ and $x_j(s)$, a sliding-window estimate can be written

$$C_{ij}^{(w)}(t) = \frac{\sum_{s \in w_t} \left[x_i(s) - \bar{x}_{i,w_t}\right] \left[x_j(s) - \bar{x}_{j,w_t}\right]}{\sqrt{\sum_{s \in w_t} \left[x_i(s) - \bar{x}_{i,w_t}\right]^2 \sum_{s \in w_t} \left[x_j(s) - \bar{x}_{j,w_t}\right]^2}},$$

where x_i and x_j are signals in native or standardized units, $\bar{x}_{i,w_t}$ is the mean within temporal window w_t centered on t, and $C_{ij}^{(w)}(t)$ is dimensionless. Recurring matrices $C^{(w)}(t)$ may then be clustered into estimated states $z_t \in \{1, \dots, K\}$. Their transition structure is summarized by

$$P_{ab} = P(z_{t+1} = b \mid z_t = a), \qquad D_a = \mathbb{E}[L_a],$$

where P_{ab} is the estimated probability of moving from state a to state b, and D_a is expected dwell time, based on a run length L_a. Occupancy, switching rate and recurrence can also be calculated. None of these quantities is observed without assumptions: window length, number of clusters, parcellation, motion correction and sampling rate help determine the apparent states.

Several PTSD studies nevertheless show why temporal analysis is worth pursuing. A 2016 MEG study associated combat-related PTSD with high-frequency hypersynchrony and reduced local signal variability in a left temporal network involving hippocampus and amygdala [Mišić et al., 2016]. A 2017 resting-fMRI study found that dynamic measures classified PTSD better than static connectivity in its single-site sample [Jin et al., 2017]. During extinction learning, patient groups failed to show the gradual connectivity increase observed in healthy controls across default-mode, frontoparietal and somatomotor systems [Wen et al., 2022]. A multilayer analysis later found lower switching in particular networks among treatment-naïve adults with PTSD [Suo et al., 2023].

These signals are not yet a universal phenotype. The largest direct test in the corpus—a harmonized ENIGMA-PGC analysis—found no robust PTSD-group differences in dynamic connectivity, dwell time or transition count [Tomas et al., 2025]. A 2026 resting-state study found increased variability in some connections but no adjusted group difference in state transitions [Hu et al., 2026]. Conversely, a small 2026 energy-landscape study reported less frequent but more stable anomalous states in PTSD [Wu et al., 2026]. Different sample composition and analytic choices may expose different phenomena, but that explanation itself must be tested rather than used to rescue every positive result.

The resulting boundary is sharp. Some tasks, regions and cohorts show altered temporal organization. The evidence does not show that every person with PTSD has globally reduced flexibility, nor that an estimated connectivity state is a biological attractor. Dwell time is not basin depth. A state transition extracted from fMRI is not yet the lived transition into terror or shutdown.

Dynamic methods become scientifically powerful when they make risky predictions: which cue will precede which transition, whether the transition replicates within a person, whether it forecasts function beyond symptom history, and whether successful treatment changes it before symptoms improve. Until then, network dynamics are a promising measurement program, not a completed ontology.

CORE SENTENCE

PTSD may alter when networks assemble and release, but estimated connectivity states are method-dependent signals—not yet proven attractors or universal transition laws.

39

Why No Brain Biomarker Diagnoses PTSD

BROADER SCIENTIFIC INFERENCE

A statistically significant brain difference is not a diagnostic test. A classifier that separates two research groups is not automatically a clinically useful biomarker. To diagnose an individual, a signal must survive new scanners, new sites, different trauma histories, medication, comorbidity, demographic variation and the full range of people who arrive in real clinical settings. No neural measure in the present corpus has crossed that threshold for standalone PTSD diagnosis or treatment selection [Patel et al., 2012] [Zilcha-Mano et al., 2020].

The base-rate problem alone prevents accuracy from being a portable property. If D denotes PTSD, T^+ a positive test, Se sensitivity, Sp specificity and $\pi = P(D)$ the prevalence in the population being tested, then

$$P(D \mid T^+) = \frac{\mathrm{Se}\,\pi}{\mathrm{Se}\,\pi + (1 - \mathrm{Sp})(1 - \pi)}.$$

The probability that a positive result is correct changes when π changes, even if sensitivity and specificity remain fixed. A model developed in a treatment-seeking veteran sample cannot therefore inherit the same positive predictive value in an emergency department, community survey or adolescent clinic. It must also be calibrated, so that

$$P(D = 1 \mid \hat{p} = p) \approx p,$$

where $\hat{p}$ is the predicted probability. Discrimination without calibration can rank people while systematically misrepresenting their risk.

Existing findings illustrate the distance between discovery and clinical use. The largest structural consortium study found only a small average hippocampal-volume association, $d = -0.17$, with substantial overlap between individuals [Logue et al., 2018]. A resting-connectivity machine-learning study reported 70.6% accuracy for distinguishing PTSD from trauma-exposed controls and 76.7% for distinguishing PTSD alone from PTSD with major depression [Zilcha-Mano et al., 2020]. Those results are scientifically interesting, but a modest research sample and internal out-of-sample procedure do not establish transportability to independent health systems.

Prospective prediction is possible, yet still limited. In 162 recent trauma survivors, connectome-based modeling predicted clinician-rated symptom severity at one month and fourteen months with correlations of $\rho = .18$ and $\rho = .24$, but not at six months, where $\rho = .03$ [Ben-Zion et al., 2025]. These are associations with a continuous outcome, not a validated diagnostic instrument. Meanwhile, a large multisite analysis found no robust group differences in several static and dynamic resting-connectivity measures [Tomas et al., 2025]. A 32-site mega-analysis did identify amygdala- and hippocampus-centered connectivity differences [Hinojosa et al., 2026], showing that replicable group signals and failed individual diagnosis can coexist.

Five hurdles recur: feature selection can overfit; scanner and preprocessing differences can shift the signal; PTSD is heterogeneous and commonly comorbid; diagnostic labels themselves contain measurement error; and a biomarker must add useful information beyond exposure history, symptoms, functioning and clinical assessment. A final hurdle is clinical impact: even a reproducible predictor matters only if acting on it improves outcomes without creating inequity or harm.

Brain research remains valuable without a diagnostic scan. It can test mechanisms, reveal heterogeneity, identify measures worth following longitudinally and generate treatment hypotheses. But clinical diagnosis still concerns exposure, symptoms, duration, distress, impairment, neighbouring conditions and the person's context. A scan cannot certify that suffering is real, and the absence of a proposed signature cannot invalidate it.

The humane implication is important. People do not need neuroimaging proof to deserve care. Nor should a colorful brain map be allowed to convert a probabilistic group association into a permanent identity.

CORE SENTENCE

Brain measures can illuminate PTSD research, but no current neural signal is validated to diagnose the individual or determine the legitimacy, course or treatment of their suffering.

PART VI

THE BODY IN THE LOOP

The body does not merely carry symptoms; it participates in the loop that estimates danger and safety.

40

Interoception: The Body as Evidence

BROADER SCIENTIFIC INFERENCE

Interoception is the nervous system's continuing estimation of conditions inside the body: heartbeat, breathing, temperature, visceral tension, pain, fatigue, nausea, pressure and the urge to move or become still. These signals are not a private report delivered to a brain that has already decided what the world means. They help constitute the evidence from which danger, safety and action-readiness are inferred.

That matters in PTSD because a bodily change can be both a consequence of threat estimation and a cue for further threat estimation. A faster heart, a restricted breath or a sudden visceral drop may follow an external reminder. Once learned, the sensation itself may acquire predictive meaning. A review of interoception in fear learning proposes that internal responses elicited during trauma can later function as conditioned cues, but this is a mechanistic framework, not proof of one universal PTSD process [Joshi et al., 2023]. The preceding discussion of the insula located one set of neural participants; interoception is the larger process of sensing, estimating and acting on the internal milieu.

Interoception also contains distinctions that everyday language collapses. Accuracy in detecting a heartbeat is not the same as how much attention a person gives the body, confidence in the judgment, distress about a sensation or willingness to remain with it. A person can detect a change accurately and interpret its implications catastrophically; another can report little sensation while their physiology changes strongly. "More awareness" is therefore not automatically therapeutic.

A randomized trial illustrates that boundary. One hundred active-duty service members and veterans with probable PTSD received four brief modules of interoceptive acceptance training or an active healthy-habits control. Training improved acceptance of uncomfortable sensations, and that change statistically mediated later symptom reduction, but the intervention had no direct effect on PTSD symptoms [Kearns et al., 2025]. The mediation is hypothesis-generating: a short, self-report study cannot establish that acceptance caused symptom change, and a null primary contrast must not be converted into efficacy by an indirect path.

A predictive formulation can make the reciprocal loop precise. Let o_t^{int} denote interoceptive observations at time t, s_t a latent bodily state and c_t context. An approximate posterior is obtained by minimizing variational free energy,

$$q^\star(s_t) = \arg\min_q F_t[q], \qquad F_t[q] = \mathbb{E}_q\big[\log q(s_t) - \log p(o_t^{\text{int}}, s_t \mid c_t)\big] .$$

Here q is a normalized approximate density, p the generative joint density, and F_t a dimensionless log-probability functional, conventionally expressed in nats. The influence of a mismatch is not determined by its size alone. In predictive-processing notation, a precision-weighted error can be written $\varepsilon_t = \Pi_t(o_t^{\text{int}} - \hat{o}_t^{\text{int}})$, where observations and predictions share declared physiological or standardized units and the precision matrix Π_t has inverse-squared units. If bodily threat signals are assigned high precision while contextual safety signals are assigned little, a modest palpitation can dominate inference. This is a formal bridge and a Flow Hijacked interpretation, not a parameter already estimated as a general mechanism in diagnosed PTSD.

Nor does the model say that bodily sensations are false. Tachycardia can reflect illness, exertion, medication, sleep loss, pain or current danger. A constricted breath can be both physiologically real and contextually misread. Compassionate formulation therefore asks two questions together: what is happening in the body, and what has the system learned that this sensation predicts? The point is not to argue a person out of experience. It is to discover whether new, tolerable experiences can let the body become evidence for more than one possible future.

CORE SENTENCE

In PTSD, the body can become part of the evidence for danger—not because it literally stores an event, but because sensation, prediction, context and action continually teach one another.

41

Allostasis and Autonomic Regulation

FLOW HIJACKED SYNTHESIS

Homeostasis is often imagined as a thermostat correcting deviation from a fixed set point. Allostasis is more anticipatory: the organism changes regulation in advance of expected demand. Heart rate rises before effort. Attention narrows before impact. Blood flow, breathing, endocrine output and muscle readiness are coordinated around what the system believes the next moment will require. Under actual threat, this is not malfunction. It is protection arriving early.

The difficult question is what happens when prediction and regulation keep preparing for conditions that are no longer reliably present—or when danger really does continue, but only intermittently. PTSD research reports average differences in psychophysiological reactivity, yet the effects vary across resting conditions, startling sounds, trauma reminders and other challenges [Pole, 2007]. The evidence does not reveal one autonomic switch stuck permanently in "on." Some people show elevated mobilization; some show blunted responses; some alternate; some differ little from trauma-exposed controls on the measure selected.

Brain and body also need not move in lockstep. A modest cross-sectional neuroimaging study found weaker synchronization between autonomic responses and connectivity within a central autonomic network in PTSD [Thome et al., 2017]. In the prospective AURORA cohort, physiological variables such as heart rate, blood pressure and high-frequency HRV interacted with hypothalamic connectivity differently according to later PTSD status, but these associations remained preliminary and group-level [Seligowski et al., 2024]. "Desynchronization" in one analysis is not a universal law, and correlation among signals does not establish which component drove the others.

Flow Hijacked represents allostatic regulation as coupled fast and slow dynamics. Let $x_t \in \mathbb{R}^n$ describe a fast neural–behavioral state, $a_t \in \mathbb{R}^m$ autonomic and endocrine action, and θ_t slowly changing regulatory parameters. A conceptual stochastic system is

$$\begin{aligned} dx_t &= f(x_t, a_t, c_t; \theta_t)\, dt + B\, u_t\, dt + \Sigma_x\, dW_t^{(x)}, \\ \tau_a\, da_t &= [\pi(x_t, c_t) - a_t] dt + \Sigma_a\, dW_t^{(a)}, \\ d\theta_t &= \epsilon\, h(a_t, \ell_t, r_t)\, dt, \qquad 0 < \epsilon \ll 1. \end{aligned}$$

Here c_t is context, u_t an external or therapeutic input, ℓ_t accumulated load, r_t restorative conditions, B maps inputs into the state, Σ_x and Σ_a are noise-amplitude matrices, and $W_t^{(x)}$ and $W_t^{(a)}$ are Wiener processes. Time t and the autonomic time constant τ_a use the same chosen unit; the drift functions carry state-per-time units. The separation $\epsilon \ll 1$ expresses a crucial possibility: rapid defensive responses can settle long before slowly learned expectations and regulatory settings change. These equations are a Flow Hijacked synthesis. PTSD studies have not jointly identified these state variables or shown that one parameter drift explains the disorder.

Allostatic load should therefore not become a moralized "wear-and-tear score," and autonomic regulation should not be equated with calmness. Healthy flexibility includes vigorous mobilization when action is needed and sufficient braking when it is not. The clinically meaningful question is whether responses are context-sensitive, proportionate and capable of recovery. A person whose body prepares quickly is not failing at relaxation. Their system may be making an expensive prediction in the service of protection.

This framing changes the aim from suppressing every arousal signal to widening regulatory range. Sleep, breathing, movement, medication, psychotherapy, relational safety and material security can each alter part of the loop, but none is a universal reset. Recovery is not a permanently low pulse. It is the regained capacity to mobilize, settle, connect and choose in accordance with the present. In Threat-State Return Asymmetry, not only the intensity of entry matters; so do the cost and speed of return.

CORE SENTENCE

The autonomic problem in PTSD is not simply "too much arousal"; it may be a loss of context-sensitive range and an expensive delay in returning from protection to participation.

42

Heart-Rate Variability Without a Magic Number

BROADER SCIENTIFIC INFERENCE

Heart-rate variability, or HRV, describes variation in the intervals between successive heartbeats. It is not the same as heart rate, and it is not a direct window onto courage, safety or mental health. Time-domain indices, frequency-domain indices and nonlinear measures extract different properties from an interval series; they cannot be substituted casually for one another.

For normal-to-normal intervals $RR_1, \ldots, RR_N$, ordinarily measured in milliseconds, a common short-term measure is

$$\mathrm{RMSSD} = \sqrt{\frac{1}{N-1}\sum_{i=1}^{N-1}\left(RR_{i+1} - RR_i\right)^2}.$$

RMSSD has the same time unit as the RR_i intervals. Respiratory sinus arrhythmia and high-frequency HRV are often used as indices influenced by cardiac parasympathetic regulation. "Influenced by" is the necessary phrase. Respiration, posture, physical fitness, age, medication, substance use, time of day, recording length, artifact correction and sleep can all change the result. The frequently used low-frequency/high-frequency ratio is not a clean meter of sympathetic–parasympathetic balance.

Across studies, there is a reproducible average signal, but it is smaller and less specific than popular accounts imply. A meta-analysis of 55 studies and 6,689 participants found a small association between PTSD and lower resting respiratory sinus arrhythmia, $g = -0.26$, with moderate heterogeneity and some indication of publication bias [Campbell et al., 2019]. Other meta-analyses reported lower RMSSD and high-frequency HRV on average and modest differences during stress [Schneider & Schwerdtfeger, 2020]. One synthesis reported much larger effects, but it was based on fewer participants, showed considerable heterogeneity and often compared PTSD groups with healthy rather than trauma-exposed controls [Ge et al., 2020]. The comparator matters: a contrast with never-traumatized healthy adults mixes effects of trauma exposure, health, medication and diagnosis.

The transdiagnostic boundary is equally important. An umbrella review covering 442 primary studies across 19 mental disorders judged reduced HRV evidence suggestive for PTSD and several other conditions, not uniquely diagnostic of PTSD [Wang et al., 2025]. A low value cannot identify the disorder, disclose the cause of distress or prescribe the right treatment.

Dense measurement begins to ask a better question than a single resting average. In four days of ambulatory ECG from 169 World Trade Center responders, daily PTSD symptoms predicted slightly lower HRV about five hours later, whereas HRV did not predict subsequent symptoms in the fitted model [Slavish et al., 2024]. The estimated symptom-to-HRV path was small, and the design did not prove causality. A different cohort, timescale or preprocessing choice could produce another direction. Still, the study illustrates why temporal order and within-person change are more informative than declaring that someone "has low vagal tone."

For Flow Hijacked, HRV is best treated as one noisy observation channel in a multivariate system. A potentially useful future measure would ask whether an individual's response and recovery change from their own validated baseline across context—not whether they cross a universal "resilience" threshold. That requires high-quality ECG reference, respiration control, preregistered signal processing, external validation and proof that using the measure improves care without intensifying body surveillance. None of those requirements is satisfied by a consumer score alone.

CORE SENTENCE

HRV carries information about regulation at the group and possibly within-person level, but no single value can diagnose PTSD, measure safety or summarize a human nervous system.

43

HPA Dynamics and the Cortisol Myth

BROADER SCIENTIFIC INFERENCE

Cortisol stories are irresistible because they turn a complex disorder into a direction: too high, too low, exhausted or permanently dysregulated. The hypothalamic–pituitary–adrenal axis does not behave like a fuel gauge. It is a delayed, pulsatile, circadian feedback system whose output depends on when, where and why it is measured.

A minimal delayed model makes the problem visible. Let $H(t)$ denote hypothalamic drive, $A(t)$ pituitary ACTH and $C(t)$ circulating cortisol. Then a conceptual regulatory system can be written

$$\begin{aligned}\dot{H}(t) &= I(t) - \alpha_H H(t) - \phi(C(t-\tau)),\\ \dot{A}(t) &= k_{HA} H(t) - \alpha_A A(t),\\ \dot{C}(t) &= k_{AC} A(t) - \alpha_C C(t) + \rho\cos(\omega t + \varphi) + \sigma\,\eta(t).\end{aligned}$$

Here H, A and C use declared assay or normalized concentration scales; $I(t)$ collects inputs per unit time; $\alpha_{\bullet}$ are clearance-rate constants; k_{HA} and k_{AC} are coupling rates; ϕ is glucocorticoid negative feedback after delay τ; $\rho\cos(\omega t + \varphi)$ represents circadian modulation; and $\sigma\eta(t)$ denotes unresolved fluctuations in concentration per unit time. A morning saliva sample, 24-hour urine, hair concentration, awakening response and laboratory stress reaction are therefore different observations of this system, not interchangeable readings of one latent quantity.

An influential early study reported lower 24-hour urinary cortisol in people with PTSD [Yehuda et al., 1990]. That finding generated important hypotheses, but later synthesis refused a universal rule. Across 37 studies, 828 adults with PTSD and 800 controls showed no overall basal cortisol difference: pooled SMD $= -0.12$, with a confidence interval crossing zero. Lower values appeared only under particular matrices, comparison groups, trauma histories, sex compositions and sampling times [Meewisse et al., 2007]. A later meta-analysis of 108 studies and 6,484 participants found small reductions in morning and 24-hour cortisol, alongside a cortisol awakening increase and no consistent moderator pattern across the full set [Schumacher et al., 2019]. A salivary-cortisol synthesis also found a modest lower morning signal, but could not turn it into a diagnostic tool [Pan et al., 2018].

Contradiction is informative here. Hair cortisol in one military study was higher in both PTSD and deployed groups relative to non-deployed personnel, suggesting an exposure-related rather than PTSD-specific difference [Schumacher et al., 2022]. In a psychotherapy meta-analysis, symptoms improved while changes in basal cortisol and the cortisol awakening response were essentially null [Schumacher et al., 2018]. At the study-aggregate level, symptom improvement therefore did not require a detectable mean change in those selected hormone measures; conversely, a hormone difference alone does not explain symptoms.

The same laboratory result can also carry different meanings. A momentary concentration may change because pulse amplitude changed, because the sample fell at a different phase, because clearance differed, or because feedback responded differently to challenge. One cross-sectional assay struggles to distinguish these possibilities. Doing so requires repeated samples at known times, challenge-linked measurements, hierarchical models and longitudinal designs that separate, as far as possible, antecedent risk, exposure consequence and adaptation to the present.

The humane consequence is not that biology is irrelevant. It is that no person should be told their trauma has produced a single endocrine defect on the basis of a slogan or isolated assay. Medication, sleep, depression, pain, menstrual and reproductive context, metabolic health, current danger and time since trauma all enter the interpretation. In continuing threat, anticipatory HPA activity may also remain contextually meaningful rather than evidence that the system has failed to realize the event is over.

CORE SENTENCE

PTSD has no universal cortisol direction; the HPA axis is a timed feedback process, and every claim must specify the matrix, moment, comparison group and context.

44

Inflammation and Neuroimmune Constraint

BROADER SCIENTIFIC INFERENCE

Immune signalling is one route by which infection, injury, stress, sleep, metabolism and behavior can influence one another. It is scientifically reasonable to ask whether inflammatory processes contribute to PTSD persistence or its physical-health burden. It is not reasonable to replace an "overactive amygdala" story with an "inflamed brain" story.

A 2015 meta-analysis found higher average concentrations of several peripheral inflammatory markers in PTSD, including interleukin-6, interleukin-1β and interferon-γ. Yet most marker analyses had very high heterogeneity, and medication, comorbid depression, assay and collection time explained substantial variation; possible publication bias appeared for interleukin-1β [Passos et al., 2015]. A later systematic review likewise found immune differences but showed that medication-stratified results could change the apparent pattern [Yang & Jiang, 2020]. These are population-level associations in peripheral blood, not direct measurements of a person's brain or a disease-specific immune state.

Newer work strengthens the signal while sharpening its modest scale. A 2026 meta-analysis of 32 studies included 2,667 people with PTSD and 8,166 controls. C-reactive protein was higher on average, but the pooled effect was small, $g = 0.188$, varied by serum versus plasma and sex, and was moderated by body-mass index [Yang et al., 2026]. A large multicohort analysis linked PTSD with soluble urokinase plasminogen activator receptor, a marker of chronic systemic inflammation, across several datasets [Bourassa et al., 2026]. Even a replicated association can reflect several paths: PTSD may alter sleep, activity, smoking, medication use or metabolic health; prior inflammation may alter vulnerability; trauma exposure may affect both; structural adversity may continue to load the entire system.

Formally, an observed immune vector should be treated as a mixture rather than an essence. If $\mathbf{y}_{i,t} \in \mathbb{R}^p$ contains measured markers for person i at time t, a defensible observation model is

$$\mathbf{y}_{i,t} = \boldsymbol{\Lambda}\mathbf{z}_{i,t} + \boldsymbol{\Gamma}\mathbf{q}_{i,t} + \mathbf{b}_i + \boldsymbol{\varepsilon}_{i,t}, \qquad \mathbf{z}_{i,t+1} = \mathbf{A}\mathbf{z}_{i,t} + \mathbf{B}\mathbf{e}_{i,t} + \boldsymbol{\omega}_{i,t},$$

Here $\mathbf{y}_{i,t}, \mathbf{b}_i, \boldsymbol{\varepsilon}_{i,t} \in \mathbb{R}^p$; $\mathbf{z}_{i,t}, \boldsymbol{\omega}_{i,t} \in \mathbb{R}^k$; $\mathbf{q}_{i,t} \in \mathbb{R}^r$; and $\mathbf{e}_{i,t} \in \mathbb{R}^s$. Accordingly, $\boldsymbol{\Lambda} \in \mathbb{R}^{p\times k}$, $\boldsymbol{\Gamma} \in \mathbb{R}^{p\times r}$, $\mathbf{A} \in \mathbb{R}^{k\times k}$, and $\mathbf{B} \in \mathbb{R}^{k\times s}$. Marker values $\mathbf{y}_{i,t}$ must be in stated assay units or explicitly standardized; $\mathbf{z}_{i,t}$ is a latent inflammatory process; $\mathbf{q}_{i,t}$ contains measured covariates such as adiposity, infection, smoking, sleep and medication; $\mathbf{b}_i$ is stable person-level variation; and $\mathbf{e}_{i,t}$ contains trauma or contextual exposure. The vectors $\boldsymbol{\varepsilon}_{i,t}$ and $\boldsymbol{\omega}_{i,t}$ are observation and process error. Without repeated measurements and credible identification assumptions, the loading $\boldsymbol{\Lambda}$ cannot tell us whether PTSD caused the latent state, followed it or simply traveled with it. This is measurement discipline, not a claim that one linear model captures immunity.

Prospective evidence remains a decisive gap. In a cohort of women assessed before PTSD onset, inflammatory and endothelial markers did not strongly predict who subsequently developed PTSD [Sumner et al., 2018]. That null does not prove inflammation irrelevant; it blocks the easier claim that a peripheral marker is already an established vulnerability test.

Flow Hijacked uses the term neuroimmune constraint carefully. Inflammation might increase fatigue, pain, sleep disruption or the cost of exploration in some people, thereby narrowing available action. But no cytokine result diagnoses PTSD, and no immune explanation makes social danger, betrayal or meaning secondary. Biology is one interacting layer of a life, not a replacement for the life.

CORE SENTENCE

Average inflammatory differences are consistent enough to investigate and too small, heterogeneous and confounded to become a single cause, diagnosis or identity.

45

FKBP5, Epigenetics and Gene–Environment Interaction

BROADER SCIENTIFIC INFERENCE

The genome is not a trauma verdict, and the epigenome is not a hidden diary. Genes participate in regulatory systems whose activity depends on cells, tissues, developmental periods and environments. Epigenetic marks can be associated with exposure or symptoms without encoding the content of an event and without proving that the association caused a disorder.

FKBP5 has become important because its protein participates in glucocorticoid-receptor regulation. Candidate studies asked whether genetic variants modify the association between childhood trauma and later post-traumatic symptoms. A landmark study reported interactions between FKBP5 polymorphisms and childhood abuse [Binder et al., 2008]; later work proposed allele-specific, trauma-dependent demethylation at glucocorticoid-response elements as one molecular route linking developmental exposure to altered stress regulation [Klengel et al., 2013]. A meta-analysis subsequently supported gene-by-trauma interaction signals for several FKBP5 variants, while inheriting the population structure, exposure measurement and publication-bias vulnerabilities of the candidate-gene literature [Hawn et al., 2019].

The statistical claim is conditional, not deterministic. For an outcome Y_i, genotype encoding G_i, exposure E_i and covariates X_i, the conventional model is

$$g(\mathbb{E}[Y_i]) = \beta_0 + \beta_G G_i + \beta_E E_i + \beta_{GE} G_i E_i + X_i^{\mathsf{T}} \gamma.$$

Here g is the declared link function, γ contains covariate coefficients, and all predictors require explicit coding. The interaction coefficient β_{GE} asks whether an exposure–outcome association differs across the chosen genotype coding in that population. It does not imply that a person with a particular allele is destined to develop PTSD, nor that everyone without it is protected. Coding, ancestry, linkage disequilibrium, unmeasured adversity and the definition of "exposure" can alter the estimate.

Epigenome-wide studies broaden the search beyond one candidate. A 2020 meta-analysis across ten military and civilian cohorts, comprising 1,896 participants, identified PTSD-associated methylation at AHRR, a locus also entangled with smoking-related biology [Smith et al., 2020]. A 2024 meta-analysis of 23 cohorts and 5,077 participants identified additional methylation sites associated with PTSD and linked some blood methylation differences with gene expression [Katrinli et al., 2024]. Cross-cohort scale improves reliability, but blood remains blood. A methylation proportion $M_{l,k,t}$ is indexed by locus l, tissue or cell mixture k, and time t; it cannot silently stand for methylation in brain tissue, a stable lifetime mechanism or an individual clinical test.

Intergenerational findings require even stricter language. A small study reported opposite FKBP5 methylation differences in Holocaust survivors and their adult offspring relative to their respective comparison groups [Yehuda et al., 2016]. This was a significant observation, not evidence that an autobiographical traumatic memory passed through the germline. Prenatal biology, parental age, family environment, ongoing social history, ancestry, cell composition and selection remain possible pathways. "Intergenerational association" says what was observed. "Inherited trauma" claims a mechanism the design could not establish.

Biological embedding is valuable precisely when it remains dynamic and multilevel. Experience can become biologically consequential without becoming immutable. A social environment can influence sleep and endocrine state; those can influence gene regulation; regulation can influence subsequent sensitivity to context. The Flow Hijacked lens therefore treats epigenetic findings as possible traces within a coupled system, never as molecular proof that a person is permanently written by the worst thing that happened to them.

CORE SENTENCE

Gene–environment and methylation findings support conditional biological embedding, not genetic destiny, inherited autobiographical memory or an epigenetic test for PTSD.

46

Sleep, REM and Offline Processing

BROADER SCIENTIFIC INFERENCE

Sleep is not an empty interval between waking episodes. It changes memory, salience, autonomic regulation and the conditions under which safety learning can later be retrieved. In PTSD, sleep can be disturbed by hyperarousal, nightmares, pain, medication, substance use, depression or sleep-disordered breathing. Poor sleep can then make the next day harder to regulate. The relation is plausibly reciprocal, but reciprocity must be measured rather than assumed.

A systematic review found only six eligible intensive-longitudinal studies, each observing adults for seven to twenty-eight days. Shorter or poorer-quality sleep tended to precede worse next-day PTSD symptoms, while greater symptoms tended to precede nightmares or poorer sleep that night [Slavish et al., 2022]. This is suggestive temporal evidence, not proof of a self-sustaining causal loop. Most measures were self-reported, samples were small, and an omitted factor—current stress, alcohol, pain or medication—could affect both sides.

Objective and subjective channels can also diverge. In six nights of actigraphy, women with abuse-related PTSD slept more fitfully but not less long than trauma-exposed and non-traumatized controls; the PTSD group tended to underestimate duration while controls overestimated it [Friedmann et al., 2022]. Across 4,078 mattress-actigraphy sleep periods from 190 male veterans in residential treatment, researchers identified stable, decreasing and increasing duration profiles rather than one common trajectory [Miller et al., 2023]. Actigraphy estimates movement and sleep–wake timing; it does not measure sleep stages as polysomnography does.

The formal object is therefore a coupled latent process, not "hours slept." Let z_t denote sleep stage, h_t homeostatic pressure, φ_t circadian phase and x_t a waking defensive state. One schematic state model is

$$\Pr(z_{t+1} = j \mid z_t = i, h_t, \varphi_t, x_t) = \frac{\exp\left(\alpha_{ij} + b_{ij}^{\mathsf{T}}[h_t, \sin\varphi_t, \cos\varphi_t, x_t]^{\mathsf{T}}\right)}{\sum_k \exp\left(\alpha_{ik} + b_{ik}^{\mathsf{T}}[h_t, \sin\varphi_t, \cos\varphi_t, x_t]^{\mathsf{T}}\right)}.$$

The transition probabilities are dimensionless; α_{ij} is a baseline log-odds term and b_{ij} contains coefficients in inverse units of the corresponding predictors. This notation separates stage transitions from averages and lets waking state influence sleep while sleep history influences tomorrow's state. It is a conceptual model; the cited PTSD studies do not identify this full transition kernel.

REM sleep deserves particular caution. REM has been linked to emotional-memory integration and extinction, but a polysomnographic systematic review found that associations varied by sex, timing and outcome; more post-extinction REM was associated with poorer recall in female samples and better recall in male samples [Schenker et al., 2021]. A preliminary study of thirteen veterans related REM features to next-day safety learning [Straus et al., 2018]. A 2026 experiment found that late sleep enhanced extinction recall in a PTSD group [Schenker et al., 2026]. Together these findings motivate precise timing questions. They do not support "more REM is better." Continuity, sequence and context may matter as much as quantity.

Sleep also has its own disorders. Insomnia and obstructive sleep apnea can coexist, and treating PTSD does not guarantee that either resolves [El-Solh et al., 2018]. In a randomized trial, adding cognitive behavioral therapy for insomnia to Prolonged Exposure improved insomnia and quality of life more than sleep hygiene plus exposure, but did not produce superior PTSD symptom reduction [Colvonen et al., 2025]. That separation is clinically important: improving sleep may be valuable even when a global PTSD score does not move differently.

CORE SENTENCE

Sleep is a coupled regulatory process that can shape tomorrow's access to safety, but duration, fragmentation, stage, timing and subjective experience cannot be collapsed into one restorative number.

47

Nightmares as Recurrent State Entry

FLOW HIJACKED SYNTHESIS

A nightmare can be a remembered scene, an altered scene, a threat without narrative or an awakening into bodily alarm. Calling it "the trauma replaying" may fit some experiences and distort others. What nightmares share clinically is not a single symbolic content but the capacity to re-enter threat during a period that should permit reduced vigilance and restoration.

Flow Hijacked therefore treats recurrent state entry as a hypothesis about timing, not as a theory of dream meaning. Let $N_t \in \{0, 1\}$ indicate a nightmare transition during night t, and let $\mathcal{H}_t$ contain sleep stage, prior arousal, medication, breathing disturbance, recent reminders and person-specific history. A recurrent-event intensity can be written

$$\lambda_N(t \mid \mathcal{H}_t) = \lambda_0(t) \exp\left(\beta^{\mathsf{T}} X_t + b_i\right),$$

where λ_N and λ_0 are events per chosen time unit, X_t is the measured time-varying state, β contains log-hazard coefficients in inverse predictor units, and b_i is a dimensionless person-level random effect. The important empirical question is whether a nightmare changes the next-day transition structure after prior symptom persistence is modeled—not merely whether people with more nightmares also have more severe PTSD.

That distinction already changes the evidence. In daily assessments across six weeks after sexual assault, 54 women reported frequent nightmares and high early post-traumatic symptoms. Nightmares predicted the next symptom report, and symptoms predicted subsequent nightmares, before autoregressive effects were included. Once prior stability was modeled, both within-person lagged paths became nonsignificant [Boland et al., 2026]. The between-person association remained strong, meaning nightmares marked people carrying greater overall burden but did not demonstrate a short-timescale causal engine.

Physiology does not yield one nightmare signature either. In 122 trauma-exposed adults, PTSD and post-traumatic nightmares were associated with larger heart-rate responses to startling tones, while resting HRV and eyeblink electromyography were not associated with diagnosis or nightmares; non-traumatic nightmares related instead to skin conductance [Mäder et al., 2023]. Another study found that lower prior-night respiratory sinus arrhythmia and elevated re-experiencing predicted next-morning nightmare or distressing-dream endorsement [Miller et al., 2018]. These small observational findings invite replication; they do not prove that autonomic activation generates nightmares.

Longer trajectories matter. Following earthquake-exposed adolescents, researchers identified several nightmare courses—stable-low, recovering, delayed, chronic and relapsing/remitting—and the more persistent or delayed patterns were associated with depression and PTSD in young adulthood [Wang et al., 2024]. Trajectory membership is a statistical summary, not a permanent child type, and nightmares may be warning markers rather than causes.

Treatment evidence further prevents a single-mechanism story. In a 304-participant veteran trial, prazosin did not outperform placebo for distressing dreams, sleep quality or global clinical improvement at ten or twenty-six weeks [Raskind et al., 2018]. Later syntheses pooling smaller trials suggested benefit for insomnia and nightmares but not clearly for global PTSD, with very-low-to-moderate certainty and substantial room for phenotype and study differences [Lappas et al., 2024] [Mendes et al., 2025]. The right conclusion is neither "prazosin works" nor "prazosin failed," but that outcomes, populations and individual risks must remain separate.

A nightmare is not moral weakness, failed processing or proof that recovery has stopped. It is a clinically important event that can fragment sleep, make bedtime threatening and alter tomorrow's capacity. The dynamical hypothesis earns its value only if dense sleep and daytime measurement show that reducing recurrent night entries changes later transition probabilities beyond ordinary symptom persistence.

CORE SENTENCE

A nightmare may reopen a defensive state during sleep, but only temporal evidence—not metaphor—can show whether recurrent entry helps maintain next-day PTSD.

48

Bessel van der Kolk and *The Body Keeps the Score*: Influence, Evidence and Boundary

BROADER SCIENTIFIC INFERENCE

Bessel van der Kolk changed the public language of trauma. His work insisted that trauma is lived through sensation, action, relationship and regulation—not only recalled as a verbal story. His 1994 peer-reviewed review, "The body keeps the score," helped foreground memory and psychobiology [van der Kolk, 1994]. The 2014 book *The Body Keeps the Score* carried embodiment, dissociation and developmental trauma far beyond specialist circles. That influence deserves recognition. Influence, however, is not a single evidence grade, and the book is an influential trade synthesis rather than a peer-reviewed proof object.

The strongest translation of its central metaphor is supported: PTSD can involve persistent, context-sensitive changes in autonomic regulation, interoception, sensory processing, memory access and action readiness. Psychophysiological and HRV syntheses support heterogeneous brain–body differences [Pole, 2007] [Campbell et al., 2019]. A contemporary van der Kolk perspective continues to emphasize somatic symptoms and sensory dysregulation, but it presents an interpretation rather than new causal data [van der Kolk & McFarlane, 2026]. None of this demonstrates that an autobiographical episode is literally stored in muscle, fascia or an organ. The body participates in continuing regulation; it is not an anatomical archive of narrative memory.

Memory claims also separate under scrutiny. Trauma narratives can contain strong sensory detail and disturbed temporal organization, but a review found evidence for fragmentation inconclusive [O'Kearney & Perrott, 2006]. A newer meta-analytic dispute found an overall association between PTSD or acute stress and disorganization, yet the effect depended sharply on how coherence was defined: some disorganization measures differed while other organization measures were essentially null [Brewin & Field, 2024]. Independent coding studies have found no diagnostic-group difference across numerous coherence measures [Rubin et al., 2016], and fragmentation did not change consistently with treatment response in a comparison of Prolonged Exposure and sertraline [Bedard-Gilligan et al., 2017]. Trauma remembering can be sensory, involuntary and difficult to organize. It is not universally fragmented, nonverbal or devoid of semantic structure; recent fMRI work directly detected semantic encoding of both traumatic and neutral autobiographical narratives [Cisler et al., 2026].

The familiar claim that "Broca's area goes offline" is similarly too strong. Early symptom-provocation studies reported reduced left inferior-frontal activity in eight preselected physiological responders and in another sample of eight women with PTSD [Rauch et al., 1996] [Shin et al., 1999]. A study of thirty police officers found distributed task-related differences, with inferior-frontal reduction appearing within the PTSD group rather than establishing a universal between-group language mechanism [Lindauer et al., 2004]. Speech was not switched off and was not directly measured as a general capacity. Moreover, "Broca's area" contains functionally distinguishable language and multiple-demand components [Fedorenko & Blank, 2020]. A regional imaging contrast cannot be converted into an on–off theory of language.

Body-oriented treatments warrant the same fairness. A meta-analysis of 29 comparative studies reported a moderate average PTSD symptom benefit, but heterogeneity was extreme, nearly every study raised risk-of-bias concerns and interoceptive-awareness change was null [van de Kamp et al., 2023]. A yoga meta-analysis found improvement in self-reported PTSD but not clinician-rated symptoms [Nejadghaderi et al., 2024]. In a multisite trial of women veterans with military-sexual-trauma-related PTSD, trauma-sensitive yoga and Cognitive Processing Therapy both improved symptoms, and the between-treatment change contrast fell within prespecified equivalence bounds; yoga had higher completion, but this population-specific result did not establish a uniquely somatic mechanism or universal superiority [Zaccari et al., 2023]. Reviews of PTSD neurofeedback describe promising signals alongside low-quality, weakly regulated and heterogeneous evidence [Marcu et al., 2024].

The rigorous response is neither reverence nor dismissal. Van der Kolk helped many people recognize that trauma has bodily and relational consequences. The scientific obligation is to disaggregate that insight into testable claims, retain what survives, revise what does not, and never use neuroscience imagery to make a metaphor sound literal. A 2026 critical appraisal offers a useful counterweight, but it too should sharpen claim-level judgment rather than invite wholesale rejection [Scheeringa, 2026].

CORE SENTENCE

***The Body Keeps the Score* transformed how trauma is understood; its enduring scientific value lies in the questions it opened, while every mechanism and treatment claim must still earn support independently.**

PART VII

THE ALTERED TRANSITION RULES HYPOTHESIS

The central Flow Hijacked proposal concerns the rules of transition, not a permanent identity.

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Threat-State Return Asymmetry

NEW HYPOTHESIS

The central proposal of this lecture is called **Threat-State Return Asymmetry**, or **TSRA**. It is a Flow Hijacked hypothesis, not an established feature of PTSD and not a new name for hyperarousal. Its question is narrower and more demanding: after trauma, do the rules governing entry into a defensive state change differently from the rules governing return? A system may begin to enter defence with less evidence, respond more strongly to particular cues, generalise danger farther, remain activated longer, or require more support to recover. None of these changes is inevitable. They may occur in different combinations, in different people, and at different periods of the same life. The proposed novelty is this operational conjunction and its falsification programme—not ownership of older ideas about prediction, avoidance, attractors, metastability or hysteresis.

The empirical literature motivates the question without yet answering it. Intensive within-person work finds that current threat, triggers, appraisals, trauma memory and protective strategies can form different person-specific temporal networks [Hertz-Palmor et al., 2026]. Six weeks of daily observations after a hurricane revealed heterogeneous adjustment dynamics rather than one universal trajectory [Littleton et al., 2023]. Fear generalisation has prospectively predicted later post-traumatic symptoms in one firefighter cohort, but experimental fear and extinction results remain method-sensitive [Lommen et al., 2024] [Lewis et al., 2023]. At the same time, the largest multisite dynamic-connectivity study in the corpus found no robust diagnostic differences in estimated brain-state dwell or transition measures [Tomas et al., 2025]. That null result belongs inside the hypothesis, not outside it.

TSRA should therefore be represented as a profile, not hidden inside a single impressive number. Its coordinates exist only relative to a measurement protocol. For person i, context c, an explicitly chosen reference r, and a preregistered protocol, define

$$\mathcal{P} = \left(T, \Delta, p_\star, \mathbf{v}, m, \mathbf{x}_\star, \mathcal{U}, \mathbf{M}_x, \mathbf{M}_u, \mathbf{M}_y, \mathbf{M}_s\right),$$

and write

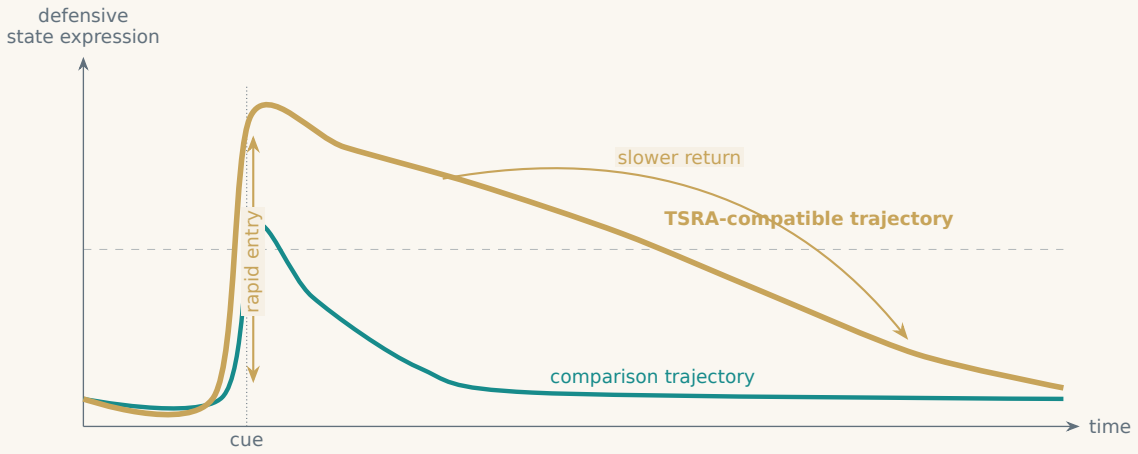
$$\boldsymbol{\Theta}_i(c;\mathcal{P}) = \begin{bmatrix} \log\left(\vartheta_r^{\uparrow}(c)/\vartheta_i^{\uparrow}(c)\right) \\ \log(G_i(c,T)/G_r(c,T)) \\ \log(\ell_i(c)/\ell_r(c)) \\ \log\left(m_{\mathrm{esc}}^{(i)}(c)/m_{\mathrm{esc}}^{(r)}(c)\right) \\ \log\left(T_{1/2}^{(i)}(c)/T_{1/2}^{(r)}(c)\right) \\ \log\left(E_{\mathrm{alt}}^{(i)}(c)/E_{\mathrm{alt}}^{(r)}(c)\right) \end{bmatrix}.$$

The log-ratio components require strictly positive quantities measured on commensurate scales. A zero, an unreachable target or a return not observed within the measurement window must be handled by a preregistered nonlog contrast or censoring rule, not by taking an undefined logarithm.

The protocol fixes the gain and control horizon T; the entry window Δ and criterion $p_\star$; cue direction $\mathbf{v}$; recovery channel m; alternative target $\mathbf{x}_\star$; admissible inputs $\mathcal{U}$; positive-definite state, input and output metric matrices $\mathbf{M}_x, \mathbf{M}_u, \mathbf{M}_y$; and a common feature metric $\mathbf{M}_s \succeq 0$. Here $\vartheta^{\uparrow}$ is the cue threshold for entry into defence; G is cue-specific gain; $\ell > 0$ is generalisation width under $\mathbf{M}_s$; m_{esc} is the median first-passage time from a defensive state D to a sufficiently safe, engaged region $\mathcal{S}$; $T_{1/2}$ is recovery half-time in channel m; and E_{alt} is a model-based cost of reaching $\mathbf{x}_\star$ under the declared horizon, constraints and metrics. Arguments inherited from $\mathcal{P}$ are suppressed on the right-hand side only for legibility. When cue amplitude cannot be calibrated, a conditional entry-hazard ratio can replace the threshold component. The reference r might be the same person in a verified safer period, a trauma-exposed participant who has recovered, or a carefully matched comparison group. Each choice answers a different question. Positive components suggest easier entry, greater transient gain, broader generalisation, longer persistence, slower recovery or lower accessibility. They must not be summed unless a preregistered measurement model justifies the weights.

This formalism changes the moral grammar. A disproportionately large response need not mean that a person is choosing danger, failing to "move on," or permanently damaged. It may mean that the system is operating under transition rules learned when misses were costly. Yet the same compassion requires precision: for someone under continuing threat, frequent defensive entry may still track the world accurately. TSRA concerns conditional response after current inputs, history, sleep, medication, pain, social safety and measurement error have been modelled—not a declaration that danger is imaginary.

The hypothesis earns its place only if entry and return can be measured separately, show reliable within-person structure, improve prediction beyond symptom severity and exposure, and change with durable recovery and functioning. If they do not, TSRA must shrink or be abandoned.



ENTRY THRESHOLD lower or less context-gated	**TRANSIENT GAIN** larger cue-specific response	**GENERALISATION** greater width
DWELL · ESCAPE longer defensive residence	**RECOVERY HALF-TIME** slower conditional return	**ALTERNATIVE ACCESS** higher control cost

Conceptual signature only: the joint entry–return asymmetry is a Flow Hijacked hypothesis, not an established PTSD mechanism.

CORE SENTENCE

TSRA proposes that trauma may alter the rules of entering defence and the rules of returning from it in different, measurable ways.

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State, Observation and Hidden Process

FLOW HIJACKED SYNTHESIS

PTSD is observed through reports, actions, physiology, sleep, attention, memory and social functioning. None of those measurements is the whole system. A racing heart may reflect threat, exertion, medication, pain or anticipation. Silence may express calm, exhaustion, strategic restraint or dissociation. A questionnaire score is clinically useful, but it does not reveal the moment-to-moment process that generated it. The mathematical task is therefore to distinguish a **latent state** from its incomplete and noisy observations.

Let the evolving latent condition of person i be $\mathbf{x}_i(t) \in \mathbb{R}^d$. Its coordinates may represent modelled threat expectation, autonomic mobilisation, contextual certainty, attentional capture, action readiness and access to social engagement; they are theoretical variables, not little objects hidden in the brain. Let $z_i(t) \in \mathcal{Z}$ be a possible latent regime, with the set $\mathcal{Z}$ learned and validated rather than imposed as "safe," "fight," "flight" or "freeze." A switching stochastic state-space model can be written

$$d\mathbf{x}_i(t) = \mathbf{f}_{z_i(t)}(\mathbf{x}_i(t), \mathbf{u}_i(t), \mathbf{c}_i(t); \boldsymbol{\theta}_i)\, dt + \mathbf{G}_{z_i(t)}(\mathbf{x}_i(t))\, d\mathbf{W}_i(t),$$

$$\Pr[z_i(t+dt) = s \mid z_i(t) = r, \mathcal{H}_t] = q^{(i)}_{rs}(\mathbf{x}_i(t), \mathbf{u}_i(t), \mathbf{c}_i(t))\, dt + o(dt),$$

$$q^{(i)}_{rs}(\cdot) \geq 0 \quad (r \neq s), \qquad q^{(i)}_{rr}(\cdot) = -\sum_{s \neq r} q^{(i)}_{rs}(\cdot),$$

$$\mathbf{y}_{ik} = \mathbf{h}(\mathbf{x}_i(t_k), z_i(t_k), \mathbf{m}_{ik}; \boldsymbol{\phi}_i) + \boldsymbol{\varepsilon}_{ik}.$$

Here $\mathbf{f}_z$ is the regime-specific nonlinear drift; $\mathbf{u}(t)$ contains time-varying inputs such as cues, safety signals, exertion or interpersonal events; $\mathbf{c}(t)$ contains context such as location, company, ongoing threat and time of day; $\boldsymbol{\theta}_i$ contains person-specific parameters; $\mathbf{W}(t)$ is a Wiener process; and $\mathbf{G}_z$ maps stochastic fluctuations into the latent coordinates. The off-diagonal intensity q_{rs} governs transition from regime r to s; the diagonal definition makes every generator row sum to zero; and $o(dt)/dt \to 0$ as $dt \to 0$. The symbol $\mathcal{H}_t$ is the measured history available at time t. At sampling time t_k, $\mathbf{y}_{ik}$ is the observed vector—perhaps experience sampling, heart period, electrodermal activity, actigraphy or task performance. The observation map $\mathbf{h}$ may be nonlinear; $\mathbf{m}_{ik}$ records measurement conditions; and $\boldsymbol{\varepsilon}_{ik}$ is measurement error.

This is a family of testable models, not a claim that one equation contains a person. A hidden Markov model makes z_t discrete and memoryless conditional on the current regime. A hidden semi-Markov model allows non-geometric dwell times. A continuous-state model may fit better if experience varies without discrete switching. Person-specific dynamic-network research demonstrates that dense sampling can reveal recurring but heterogeneous temporal organisations [Hertz-Palmor et al., 2026]. Earlier HMM work in PTSD estimated a state interpreted as difficult to disengage from, but the sample was small and the psychological label was inferred rather than directly observed [Ou et al., 2015].

The humane importance of the separation is simple. What we see is an output under conditions, not an identity. A model should be able to say, "the same outward sign can arise through different hidden processes," and then expose that claim to data. It should also allow the environment to change the process. Otherwise, a sophisticated equation merely relocates blame from character to an allegedly defective brain.

CORE SENTENCE

A measured symptom is an observation of a changing person-in-context, not the latent process itself and never the person's identity.

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The Entry Threshold

NEW HYPOTHESIS

"Triggered" is often used as though it names one event: a cue appears and a response follows. TSRA asks for a more exact description. What magnitude, combination or credibility of evidence is required before the system crosses into defence? Is entry governed mainly by the cue, by the context in which it arrives, by bodily state, or by recent history? A threshold is not a rigid line inside the brain. Under noise and uncertainty it is a probability surface.

Let $a \in \mathcal{A}_c \subseteq \mathbb{R}_+$ denote the calibrated strength of an ethically tolerable cue in context c, and let $\boldsymbol{\xi}(t)$ collect its features together with contextual danger and safety evidence. Define the first entry time

$$\tau_i^{\uparrow}(a, c) = \inf\{t > t_0 : z_i(t) = D \mid z_i(t_0) = S,\, a,\, c\}.$$

For a prespecified horizon $\Delta > 0$ and probability criterion $p_\star \in (0, 1)$, the probabilistic entry threshold is

$$\vartheta_i^{\uparrow}(c; \Delta, p_\star) = \inf\left\{a \in \mathcal{A}_c : \Pr\left(\tau_i^{\uparrow}(a, c) \le t_0 + \Delta \mid \mathcal{H}_{t_0}\right) \ge p_\star\right\}.$$

This infimum has threshold meaning only if the criterion-crossing probability is nondecreasing in a over $\mathcal{A}_c$. That monotonicity must be justified by design and checked rather than smuggled in by vocabulary; without it, $\vartheta_i^{\uparrow}$ is only the minimum observed criterion-crossing amplitude. The arrow matters: this is an entry quantity. A complementary survival formulation specifies the instantaneous transition hazard,

$$\lambda_{S\to D}^{(i)}(t) = \lambda_{0i}(t) \exp\left[\boldsymbol{\beta}_i^{\top}\boldsymbol{\xi}(t) + \boldsymbol{\gamma}_i^{\top}\mathbf{c}(t) + \boldsymbol{\eta}_i^{\top}\mathbf{x}_i(t)\right].$$

Here $\lambda_{0i}(t)$ is the person-specific baseline hazard; $\boldsymbol{\beta}_i$, $\boldsymbol{\gamma}_i$ and $\boldsymbol{\eta}_i$ weight cue features, context and current latent state. A lower $\vartheta_i^{\uparrow}$, or a larger cue-conditioned hazard after matching current state, is the entry component predicted by TSRA. Crucially, the model permits a smoke alarm to be highly responsive in one room and appropriately quiet in another. Loss of contextual gating—not sensitivity alone—is often the more consequential prediction.

Direct evidence remains partial. Laboratory and prospective studies support altered threat or safety discrimination in some samples, while a multiverse analysis found that many PTSD-versus-control extinction-retention results changed with analytic decisions and were mostly nonsignificant [Lewis et al., 2023]. One prospective study found that acute-stress reconfiguration of salience-network coupling predicted later perceived stress but not overall PTSD symptoms, illustrating why a plausible dynamic marker cannot be promoted into a disorder mechanism [Zhang et al., 2022]. Person-specific networks also show that "trigger-to-threat" coupling is prominent for some people and not the organising relation for others [Hertz-Palmor et al., 2026].

A valid experiment must ramp cue strength safely, randomise contexts, measure explicit appraisal as well as physiology and behaviour, and separate anticipatory arousal from transition. It must compare diagnosed PTSD with trauma-exposed people without PTSD and, ideally, the same people across recovery. If threshold estimates collapse after controlling sleep, current threat, medication, pain, expectancy or cue meaning, that is not a nuisance to be hidden. It is evidence that the environment or bodily starting point—not a stable altered entry rule—explains the observation.

In lived terms, the entry threshold asks why an ordinary sound can sometimes acquire extraordinary authority. It does not assume the sound is objectively trivial, nor that the response is irrational. It asks what evidence the system required at that moment, given what it had learned.

CORE SENTENCE

The TSRA entry hypothesis predicts a lower or less context-gated probability boundary for entering defence, not a universally exaggerated response.

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Cue-Specific Transient Gain

NEW HYPOTHESIS

Threshold and amplitude are different. A system can enter defence readily but respond modestly, or require a strong cue and then respond explosively. TSRA therefore separates **entry probability** from **transient gain**: how much a particular perturbation is amplified over a finite interval after baseline state and input magnitude are accounted for.

Near a reference state $\mathbf{x}_S$, a nonlinear model may be locally approximated by

$$d\delta\mathbf{x}(t) = \mathbf{A}_S\delta\mathbf{x}(t)dt + \mathbf{B}_S\delta\mathbf{u}(t)dt + \mathbf{\Sigma}_S d\mathbf{W}(t), \qquad \delta\mathbf{y}(t) = \mathbf{C}_S\delta\mathbf{x}(t) + \boldsymbol{\epsilon}(t),$$

where $\delta\mathbf{x} = \mathbf{x} - \mathbf{x}_S$, $\mathbf{A}_S$ is the local evolution operator, $\mathbf{B}_S$ maps perturbations into the state, and $\mathbf{C}_S$ maps latent changes into measured output. Before comparing gains, input and output coordinates must be placed on preregistered, commensurate scales. For positive-definite metric matrices $\mathbf{M}_u$ and $\mathbf{M}_y$, define

$$\|\mathbf{a}\|_{\mathbf{M}} := \left(\mathbf{a}^\top\mathbf{M}\mathbf{a}\right)^{1/2}, \qquad \mathbf{M}_u \succ 0, \quad \mathbf{M}_y \succ 0.$$

For an impulsive cue with feature direction $\mathbf{v}$, an output-specific finite-time gain is

$$G_{\mathbf{v}}(T) = \frac{\left\|\mathbf{C}_S e^{\mathbf{A}_S T}\mathbf{B}_S\mathbf{v}\right\|_{\mathbf{M}_y}}{\|\mathbf{v}\|_{\mathbf{M}_u}}, \qquad G_{\max}(T) = \sigma_{\max}\left(\mathbf{M}_y^{1/2}\mathbf{C}_S e^{\mathbf{A}_S T}\mathbf{B}_S\mathbf{M}_u^{-1/2}\right).$$

The weighted norm records what one unit means in each channel, $e^{\mathbf{A}_S T}$ propagates the local dynamics for time T, and $\sigma_{\max}$ is the largest singular value after whitening by the declared metrics. Without those metrics, changing measurement units alone can change the numerical gain. $G_{\mathbf{v}}(T)$ is specific to an input direction and an observed output. It might be high for a sudden approach from behind but not for a loud neutral sound, or high in heart-rate acceleration but not in reported fear. The maximum gain describes the most amplified modelled input, not necessarily any cue encountered in life.

The distinction protects against a common mistake. A high peak following a "small" external event does not establish neural amplification: the event may not be small to the person; relevant input may have been omitted; attention and expectancy may enlarge effective input; the baseline may already be elevated; measurement may saturate; or a nonlinear threshold may have been crossed. Dynamic PTSD studies report altered reconfiguration or variability in some samples, but the large ENIGMA-PGC analysis found no robust diagnostic difference in static or dynamic resting connectivity, dwell time or transition count [Tomas et al., 2025]. Large response phenomenology cannot by itself select a dynamical mechanism.

Non-normal recurrent dynamics, developed later, offer one mathematically precise route to finite-time amplification. Balanced neural-network models show that selected patterns can grow transiently while the network remains asymptotically stable [Murphy & Miller, 2009] [Hennequin et al., 2012]. Primate sensory work provides a biological bridge for balanced transient amplification [Pattadkal et al., 2024]. None of those studies establishes the operator $\mathbf{A}_S$ in PTSD. **The acquisition programme found zero direct operator-level PTSD studies estimating non-normality, singular-vector gain or pseudospectral sensitivity.**

A decisive test would fit $(\mathbf{A}_S, \mathbf{B}_S, \mathbf{C}_S)$ from training data, identify high- and low-gain cue directions without using outcome trials, and predict held-out perturbation responses. It would compare non-normal amplification against simpler accounts based on cue appraisal, baseline arousal, unstable eigenvalues, nonlinear saturation and stochastic variability. The lived meaning is not that a person is "too sensitive." It is that sensitivity has direction, timing and context—and those can be measured without turning them into blame.

CORE SENTENCE

Cue-specific gain asks which inputs are amplified, through which outputs and for how long; a large reaction alone proves none of the proposed mechanisms.

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Generalisation Width

BROADER SCIENTIFIC INFERENCE

Trauma does not teach only that one exact stimulus is dangerous. Learning generalises, because a system that waited for perfect identity would often respond too late. The scientific problem is not whether generalisation exists, but how broad it becomes, which dimensions organise it, and whether safety remains able to narrow it. In PTSD, some studies support wider perceptual, conceptual or contextual generalisation, yet the pattern is heterogeneous and cannot be assumed in every person [Lis et al., 2020] [Morey et al., 2020].

Represent a cue by a feature vector $\mathbf{s} \in \mathcal{M}$, where $\mathcal{M}$ may contain perceptual properties, semantic category, location, bodily sensation and relational meaning. Let $\boldsymbol{\mu}_D$ be a learned danger prototype. A simple generalisation kernel is

$$K_\ell(\mathbf{s}, \boldsymbol{\mu}_D) = \exp\left[-\frac{d^2_{\mathbf{M}}(\mathbf{s}, \boldsymbol{\mu}_D)}{2\ell^2}\right], \qquad d^2_{\mathbf{M}}(\mathbf{a}, \mathbf{b}) = (\mathbf{a}-\mathbf{b})^\top \mathbf{M}(\mathbf{a}-\mathbf{b}),$$

where $\mathbf{M} \succeq 0$ defines a learned pseudometric and $\ell > 0$ is the kernel width; it is a proper metric when $\mathbf{M} \succ 0$. A larger ℓ makes similarity to danger decay more slowly with distance. But ℓ is meaningful only relative to the feature space and metric. If the model omits the dimension that actually mattered—uniform, voice, smell, bodily constriction, betrayal—the estimated "overgeneralisation" may simply reveal an impoverished experiment.

The kernel can enter the defensive transition hazard while remaining gated by context:

$$\log \lambda_{S\to D}(t) = \alpha_i + \beta_i K_{\ell_i}(\mathbf{s}_t, \boldsymbol{\mu}_{D,i}) - \rho_i\, \mathcal{S}(\mathbf{c}_t) + \chi_i K_{\ell_i}(\mathbf{s}_t, \boldsymbol{\mu}_{D,i})\mathcal{S}(\mathbf{c}_t),$$

where α_i is baseline log hazard, β_i weights similarity to learned danger, ρ_i weights credible safety information $\mathcal{S}(\mathbf{c}_t)$, and the interaction coefficient χ_i tests whether context changes how cue similarity is used. TSRA predicts not merely a wider kernel but a kernel that is less effectively narrowed by valid safety context in at least some people.

This formulation joins laboratory learning to ordinary life without pretending they are equivalent. A 2024 prospective study found that fear generalisation predicted post-traumatic symptoms two years later among Dutch firefighters, strengthening temporal ordering but not establishing causation [Lommen et al., 2024]. Earlier imaging work reported bias toward generalising fear associations across cues and involvement beyond a single "fear centre," including visual, insular, thalamic and locus-coeruleus-related systems [Morey et al., 2015]. Other experiments and analytic multiverses constrain universal accounts of deficient acquisition or extinction [Lewis et al., 2023] [Pöhlchen et al., 2020].

Generalisation can also be semantic and social. "One person was unsafe" can become "authority is unsafe"; "my body surged" can become "my body cannot be trusted." Those are not interchangeable processes, and a single Gaussian kernel is only a disciplined starting model. Mixture kernels, anisotropic metrics and hierarchical Bayesian models may be needed to learn which dimensions widen and which remain precise. The humane objective is not to persuade someone that all situations are safe. It is to discover whether the system can again use real differences between situations.

CORE SENTENCE

Generalisation width measures how far danger learning spreads through a meaningful feature space and how effectively credible context can narrow it.

54

Dwell Time, Escape Time and Sticky States

FLOW HIJACKED SYNTHESIS

Peak reactivity captures the loudest moment. It can miss the more exhausting fact: how long the system remains organised around defence after the cue has passed. TSRA names this persistence without presuming its cause. A state may appear "sticky" because its internal dynamics return slowly, because it is repeatedly re-entered, because the environment continues to supply danger, or because the measurement cannot resolve rapid switching. These explanations demand different responses.

Suppose a defensive regime D begins at time t_0. Define the first-passage time to a prespecified safe-engagement region $\mathcal{S} \subset \mathbb{R}^d$ as

$$\tau_{D\to\mathcal{S}} = \inf\{s > 0 : \mathbf{x}(t_0 + s) \in \mathcal{S} \mid z(t_0) = D\}.$$

Its survival function and escape hazard are

$$S_D(t \mid \mathcal{H}_{t_0}) = \Pr(\tau_{D\to\mathcal{S}} > t \mid \mathcal{H}_{t_0}), \qquad h_{D\to\mathcal{S}}(t) = -\frac{d}{dt}\log S_D(t).$$

The median escape time m_{esc} satisfies $S_D(m_{\text{esc}}) = 1/2$, when such a time exists. A hidden Markov model implies geometric or exponential dwell distributions; a hidden semi-Markov model estimates a more flexible duration law. That distinction matters. If the data show a heavy right tail, forcing memoryless transitions can manufacture a misleading state architecture.

Persistence must also be separated from recurrence. Let $N_D(t_0, t_1)$ count entries into D during an interval. Two people can spend the same total time in defence while one has a single slow recovery and the other repeatedly returns after new cues. The first suggests altered relaxation; the second may reflect input frequency or rapid re-entry. Long-term PTSD research documents remission, chronicity and recurrence, but definitions and schedules vary enough that recurrence could not be cleanly pooled in one systematic review [Brooks & Greenberg, 2024]. A broader long-course review likewise found substantial variability rather than a single chronic pathway [Steinert et al., 2015].

PTSD-specific dynamic evidence is suggestive but incomplete. Daily symptom networks during conflict identify lagged relations among startle, affect, blame and avoidance, yet conditional associations do not identify state escape [Greene et al., 2018]. A newer person-specific study found multiple temporal formulations rather than one network [Hertz-Palmor et al., 2026]. A 2026 minimal mathematical model can reproduce long recovery times, bistability-like trajectories and "ghost" slowing from an autocatalytic symptom process, but its parameter is not a validated individual mechanism [Grosskopf et al., 2026].

The experiment TSRA needs begins when a perturbation ends. It samples densely enough to estimate the whole return distribution; records fresh cues, social events and bodily inputs; and defines safe engagement through functioning as well as symptom reduction. Censoring must be explicit when observation stops before return. The reference region $\mathcal{S}$ must be person- and context-sensitive: quiet immobility is not necessarily safety, and active engagement is not necessarily absence of distress.

In lived experience, "the danger is over" and "my system has returned" can be two different times. Mathematics can respect that interval without declaring it permanent.

CORE SENTENCE

A sticky state is an empirical claim about the distribution of return times after inputs are accounted for, not a synonym for severe symptoms.

55

Recovery Half-Time

NEW HYPOTHESIS

Recovery is usually evaluated over weeks or months by change in a symptom total. TSRA adds a shorter-timescale quantity: after a bounded disturbance, how quickly do particular dimensions move back toward a person's own safe baseline? The proposal is not to reduce recovery to a stopwatch. It is to make return kinetics visible, because peak activation and duration may carry different information.

Let $y_m(t)$ be outcome channel m—for example, subjective current threat, heart period, electrodermal activity, attentional narrowing or capacity to resume a chosen task. Let $\mu^S_{im}(c)$ be person i's context-specific reference level, estimated across credible safe periods rather than from one quiet minute. If a cue ends at t_0, define the excess response

$$r_{im}(t) = \left| \mathbb{E}\left[y_{im}(t) \mid \mathcal{H}_{t_0} \right] - \mu^S_{im}(c_t) \right|.$$

The recovery half-time is then

$$T^{(m)}_{1/2,i} = \inf\left\{ \tau > 0 : r_{im}(t_0 + \tau) \le \tfrac{1}{2} r_{im}(t_0) \right\}.$$

If $r_{im}(t_0+\tau) = r_{im}(t_0)e^{-\kappa_{im}\tau}$, then $T^{(m)}_{1/2,i} = \log 2/\kappa_{im}$. Here $\kappa_{im} > 0$ is the channel-specific relaxation rate. Real recovery may be biphasic, oscillatory, interrupted or never reach half within the observation window; forcing a single exponential would erase precisely the dynamics of interest. A more flexible model uses a mixture $r(t) = \sum_j a_j e^{-\kappa_j t}$, where a_j and κ_j are component amplitudes and rates, a Gaussian process, or a first-passage distribution. The superscript m is essential: physiology, felt safety and functioning may return on different clocks.

Evidence for coupled short-term processes exists, but it does not yet validate this metric. A systematic review of daily studies found preliminary bidirectional associations between poor sleep and next-day PTSD symptoms, based on only six short intensive-longitudinal samples [Slavish et al., 2022]. In a 2026 post-assault EMA study, nightmare–symptom lagged associations disappeared after autoregressive stability was included, a reminder that apparent feedback can be ordinary persistence [Boland et al., 2026]. Over treatment sessions, symptoms and functioning showed reciprocal, time-varying relations rather than one simple direction [Benfer et al., 2024]. Longitudinal imaging has also reported sequential changes in different amygdala connections across recovery, but those observations do not measure momentary return kinetics [Yoon et al., 2017].

A TSRA study should estimate half-times after repeated, standardised and naturalistic perturbations, with uncertainty intervals and censored trials. It should test reliability across days; distinguish removal of the original cue from arrival of new inputs; and ask whether shortened half-time predicts later functioning beyond baseline symptom severity and peak response. Treatment sensitivity would strengthen the construct only if change is durable and linked to life outside the laboratory.

The lived translation is gentle but important. Someone may know, cognitively, that a moment has passed while breathing, attention, muscle tone, images and social availability return at different rates. That lag is not a failure of insight. Nor is faster always better: a person in real danger may need sustained mobilisation. The quantity becomes clinically meaningful only when context and chosen functioning are part of the definition.

CORE SENTENCE

Recovery half-time measures how different parts of a system return after perturbation, while refusing to equate speed with virtue or symptom change with complete recovery.

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Alternative-State Accessibility and Controllability

FLOW HIJACKED SYNTHESIS

Recovery is more than occupying a less distressed point. It may involve regaining access to states that trauma made difficult to reach: sleep, curiosity, social presence, concentration, play, grief without alarm, or the ability to imagine a future. TSRA calls this **alternative-state accessibility**. Control theory supplies exact questions about reachability, but its language must be handled carefully. A person is not a machine to be driven into compliance.

For a controlled nonlinear stochastic system,

$$d\mathbf{x}(t) = \big[\mathbf{f}(\mathbf{x}(t), \mathbf{c}(t)) + \mathbf{B}(\mathbf{x}(t), \mathbf{c}(t))\mathbf{u}(t)\big]dt + \mathbf{\Sigma}(\mathbf{x}(t))d\mathbf{W}(t),$$

the deterministic skeleton's reachable set from $\mathbf{x}_0$ over horizon T is

$$\mathcal{R}_T(\mathbf{x}_0) = \{\mathbf{x}_T : \exists\, \mathbf{u} \in \mathcal{U} \text{ such that } \mathbf{x}(0) = \mathbf{x}_0,\ \mathbf{x}(T) = \mathbf{x}_T\}\,,$$

where $\mathcal{U}$ is the set of feasible, safe and acceptable inputs. For the stochastic system, reachability instead concerns the probability of entering a target neighbourhood. In a local linear approximation $\dot{\mathbf{x}} = \mathbf{A}\mathbf{x} + \mathbf{B}\mathbf{u}$, let $\mathbf{M}_u \succ 0$ be the preregistered input-cost metric. The corresponding finite-horizon controllability Gramian is

$$\mathbf{W}_{c,\mathbf{M}_u}(T) = \int_0^T e^{\mathbf{A}(T-\tau)}\mathbf{B}\mathbf{M}_u^{-1}\mathbf{B}^\top e^{\mathbf{A}^\top(T-\tau)}d\tau.$$

For a reachable target $\mathbf{x}_T$, the unconstrained minimum of $\int_0^T \mathbf{u}(t)^\top \mathbf{M}_u \mathbf{u}(t)dt$ is

$$E_{\min}^{(\mathbf{M}_u)} = \left(\mathbf{x}_T - e^{\mathbf{A}T}\mathbf{x}_0\right)^\top \mathbf{W}_{c,\mathbf{M}_u}(T)^\dagger \left(\mathbf{x}_T - e^{\mathbf{A}T}\mathbf{x}_0\right),$$

where † denotes the Moore–Penrose pseudoinverse. Small eigenvalues of $\mathbf{W}_{c,\mathbf{M}_u}$ identify directions that the model considers hard to reach under the declared input units and costs. In lived language, the same effort can open one state while barely moving another.

These equations are bridge mathematics. Structural-connectome studies have mapped putative control roles, but results depend heavily on normalization, topology and model choice [Gu et al., 2015] [Karrer et al., 2020]. Reanalyses warn that popular controllability metrics can track simple degree and can appear in biologically implausible null networks [Tu et al., 2018]. Stronger perturbational bridges exist: diffusion-informed predictions have shared variance with intracranial stimulation responses in epilepsy, and TMS–EEG work has estimated state-dependent controllable directions in healthy motor states [Stiso et al., 2019] [Shikauchi et al., 2026]. None validates a PTSD control-energy biomarker.

For TSRA, $\mathbf{u}(t)$ is not merely stimulation. It may encode therapy operations, sleep restoration, medication, movement, breathing, a trusted other, environmental safety or self-chosen action. These inputs differ in consent, meaning, timescale and biological pathway; adding them as columns of one matrix does not make them interchangeable. A target must also be chosen with the person, not defined as stillness or statistical normality. Constraints, adverse effects and effort belong in the cost functional.

The hypothesis is that durable recovery may expand $\mathcal{R}_T$, reduce the cost of reaching valued states, or create new context-sensitive routes. It fails if estimated accessibility is unreliable, reduces to symptom severity or network degree, does not predict actual responses to held-out perturbations, or changes without improved functioning.

CORE SENTENCE

Controllability is useful here only as a test of whether valued alternative states become reachable—not as a metaphor for controlling people.

57

Fast–Slow Coupling and Hysteresis

NEW HYPOTHESIS

A sudden sound unfolds in milliseconds; a night of fragmented sleep alters the next day; months of avoidance reshape what is encountered; social safety may change over years. PTSD cannot be understood on one clock. Fast–slow systems provide a language for how rapid defensive responses can be governed by slower variables that carry history.

Let $\mathbf{x}(t)$ contain fast threat, autonomic and action-readiness variables, while $\mathbf{s}(t)$ contains slower quantities such as sleep debt, learned precision, endocrine or immune context, avoidance-conditioned opportunity, and reliable social safety. A generic stochastic fast–slow model is

$$d\mathbf{x} = \mathbf{f}(\mathbf{x}, \mathbf{s}, \mathbf{u}, \mathbf{c})dt + \mathbf{\Sigma}_x d\mathbf{W}_x(t), \qquad d\mathbf{s} = \varepsilon\, \mathbf{g}(\mathbf{x}, \mathbf{s}, \mathbf{u}, \mathbf{c})dt + \sqrt{\varepsilon}\, \mathbf{\Sigma}_s d\mathbf{W}_s(t), \quad 0 < \varepsilon \ll 1.$$

The drift functions $\mathbf{f}$ and $\mathbf{g}$ govern fast and slow evolution; $\mathbf{\Sigma}_x$ and $\mathbf{\Sigma}_s$ map their stochastic fluctuations. The small parameter ε states that $\mathbf{s}$ usually changes more slowly than $\mathbf{x}$. It does not assign one biological substrate to either vector. For a temporarily fixed $\mathbf{s}$, the fast subsystem may have one stable response, several stable branches, or a long "ghost" transient near a vanished equilibrium. Slow movement of $\mathbf{s}$ can therefore change the response to the same cue without any contradiction in the person.

Hysteresis is a stronger claim than persistence. Suppose $q(t)$ is an experimentally controlled threat-evidence parameter. During a slow upward sweep, entry into defence occurs at $q_\uparrow$; during a downward sweep, return occurs at $q_\downarrow$. A loop exists when

$$\mathcal{H} = q_\uparrow - q_\downarrow > 0,$$

so that after entry, threat evidence must fall below the original entry value before the prior state is recovered. In a stochastic system, let the random crossing amplitudes be $Q_\uparrow$ and $Q_\downarrow$. Two preregistrable distributional estimands are

$$\mathcal{H}_{0.5} = \mathrm{med}(Q_\uparrow) - \mathrm{med}(Q_\downarrow), \qquad p_{\mathcal{H}} = \Pr(Q_\uparrow > Q_\downarrow).$$

The experiment must specify whether evidence for a loop means $\mathcal{H}_{0.5} > 0$, $p_{\mathcal{H}} > 1/2$, or another identified functional, with uncertainty intervals. Order, habituation, expectancy and fatigue must be modelled. TSRA would predict altered entry and exit distributions, but no direct PTSD experiment in the corpus has demonstrated this loop.

A 2026 PTSD-specific minimal model shows how autocatalytic symptom dynamics can generate bistability, broad recovery times and ghost-like slowing, but aggregate trajectory resemblance cannot identify the true generating mechanism [Grosskopf et al., 2026]. Bridge work shows that acute stress in healthy adults can increase network integration and reduce transition variability [Wang et al., 2022]. That direction may be adaptive in the short term; therefore low flexibility alone cannot diagnose pathology. A 2026 opinion connects predictive coding with restoration of metastability while explicitly acknowledging the shortage of direct PTSD metastability tests [Kotler et al., 2026].

The decisive experiment would present safely bounded, counterbalanced rising and falling evidence, preferably across sleep conditions and recovery, while sampling experience, action and autonomic physiology. It would compare a hysteretic fast–slow model with a non-hysteretic model containing adaptation, habituation and expectation. If entry and exit paths coincide after those alternatives are fitted, hysteresis is not supported. If ongoing danger fully explains the slow variable, the claim should move from autonomous persistence to accurate environmental tracking.

The lived meaning is familiar without being proof: the conditions that start alarm need not be the conditions that end it. Mathematics becomes humane when it makes that asymmetry testable rather than turning it into destiny.

CORE SENTENCE

Fast–slow coupling explains how history can shape present response; hysteresis is the stricter, still-unproven prediction that entry and return follow different paths.

PART VIII

STATES, AMPLIFICATION AND A MODEL THAT MUST BE ABLE TO FAIL

A serious model must explain amplification and persistence, and it must specify how it could be wrong.

58

From Symptom Scores to State-Space Models

BROADER SCIENTIFIC INFERENCE

A symptom score answers an important question: how much of a defined clinical pattern is present over a stated period? It is not designed to answer which process changed first, what prompted a transition, or why two people with the same total are moving in opposite directions. If $\mathbf{y}(t) = (y_1(t), \dots, y_p(t))^\top$ is a vector of measured symptoms, a total score is approximately a projection

$$S(t) = \mathbf{w}^\top \mathbf{y}(t),$$

with scoring weights $\mathbf{w}$. The map from $\mathbf{y}$ to S is many-to-one. High intrusion with low numbing and low intrusion with high numbing may produce the same total. A rising score and a falling score can pass through the same value. This is not a defect in clinical scales; it is a limit on the questions a scalar can answer.

A state-space model retains temporal order and separates latent evolution from measurement:

$$\mathbf{x}_{k+1} = \mathbf{F}_{z_k}\mathbf{x}_k + \mathbf{B}_{z_k}\mathbf{u}_k + \boldsymbol{\eta}_k, \qquad \mathbf{y}_k = \mathbf{H}_{z_k}\mathbf{x}_k + \boldsymbol{\varepsilon}_k,$$

$$\Pr(z_{k+1} = s \mid z_k = r, \mathbf{x}_k, \mathbf{u}_k) = P_{rs}(\mathbf{x}_k, \mathbf{u}_k).$$

Here $\mathbf{x}_k$ is a continuous latent vector, z_k a possible discrete regime, $\mathbf{F}_z$ the regime-specific dynamics, $\mathbf{B}_z$ the input map, $\mathbf{H}_z$ the observation map, and $(\boldsymbol{\eta}_k, \boldsymbol{\varepsilon}_k)$ process and measurement noise. An HMM removes the continuous layer; a switching linear dynamical system retains both; dynamic structural equation modelling estimates lagged relations among repeatedly measured variables. Each is a hypothesis about time, not simply a more technical analysis.

Direct PTSD studies show both promise and restraint. Thirty-day smartphone networks during conflict found lagged symptom relations that differed from contemporaneous and between-person networks [Greene et al., 2018]. A 2024 EMA network found group-level prominence of hypervigilance alongside marked person-level heterogeneity [Stefanovic et al., 2024]. Person-specific models in 2026 recovered several recurring formulations rather than a single PTSD network [Hertz-Palmor et al., 2026]. Earlier HMM work estimated difficulty disengaging from an inferred negative-mood state, but the sample and external validation were limited [Ou et al., 2015]. Most importantly, a 30-site analysis of 1,035 trauma-exposed participants found no robust PTSD-group differences in dynamic resting-state connectivity, dwell time or transition count [Tomas et al., 2025].

Moving to state space therefore does not guarantee discovery. The number of states can be imposed by the analyst; scanner drift can masquerade as transition; irregular sampling can distort lags; and group averages can invent a network that fits no individual. A good model must predict held-out observations, remain stable under reasonable preprocessing, distinguish person-specific from shared structure, and outperform simpler autoregressive models. Latent regimes should be named only after independent behavioural or experiential validation.

The human advantage is not that mathematics sees a hidden "true self." It is that temporal models can preserve change. They can distinguish a difficult hour from a permanent trait and ask whether access, sequence or recovery is changing before a monthly total moves.

CORE SENTENCE

State-space models can recover timing and hidden structure that totals discard, but every inferred state remains a model awaiting validation.

59

Attractors and Basins: Useful Mathematics, Unproven Ontology

FLOW HIJACKED SYNTHESIS

The word "attractor" is compelling because it gives shape to persistence. In mathematics, however, it has a stricter meaning than "a state that is hard to leave." For a deterministic flow $\mathbf{\Phi}_t$ generated by $\dot{\mathbf{x}} = \mathbf{f}(\mathbf{x})$, an attracting invariant set $\mathcal{A}$ has a basin

$$\mathcal{B}(\mathcal{A}) = \left\{ \mathbf{x}_0 : \lim_{t\to\infty} \operatorname{dist}(\mathbf{\Phi}_t(\mathbf{x}_0), \mathcal{A}) = 0 \right\}.$$

$\mathcal{A}$ can be a fixed point, cycle or more complicated set. Its basin is the collection of initial conditions converging toward it. Neither a high symptom mean nor repeated occupancy of a clustered fMRI pattern demonstrates invariance, attraction or a basin boundary.

"Depth" requires still more assumptions. If a stochastic system has gradient drift,

$$d\mathbf{x} = -\nabla V(\mathbf{x})dt + \sigma d\mathbf{W}(t),$$

with scalar potential V, isotropic noise amplitude σ, and suitable boundary conditions, then its stationary density is proportional to

$$p_\infty(\mathbf{x}) \propto \exp\left[-\frac{2V(\mathbf{x})}{\sigma^2}\right].$$

In a small-noise limit, escape time can grow approximately exponentially with a barrier ΔV. But brain–body–environment dynamics are generally driven, history-dependent and non-equilibrium. A unique scalar potential may not exist; different noise covariances and coordinates alter the apparent landscape. Saying "the PTSD basin is deeper" is therefore not a poetic substitute for estimating perturbation response and escape.

There are genuine reasons to test attractor models. A small MEG study reported reduced local variability and a constrained neural repertoire in combat-related PTSD, but explicitly did not measure basin geometry [Mišić et al., 2016]. A 2026 study using energy-landscape language found less frequent but more stable anomalous estimated states in a small PTSD sample; its cross-sectional clusters do not establish causal attractors [Wu et al., 2026]. A deliberately minimal PTSD model generated bistability, slow recovery and ghost transients consistent with aggregate courses [Grosskopf et al., 2026]. It shows that such dynamics are possible, not that its equations are uniquely true. The large ENIGMA dynamic-connectivity null further blocks any universal state-trapping claim [Tomas et al., 2025].

An attractor account becomes scientific when it predicts convergence from multiple initial conditions, perturbation-dependent return, separable basins, transition rates under known noise, and parameter change with recovery. It must beat alternatives: continuing threat, repeated cue input, ordinary autoregression, latent subgroups, sleep-mediated persistence, avoidance learning and measurement artefact. It must also allow no attractor at all.

As a lived metaphor, a basin can express the effort required to move when familiar routes seem to fold back toward alarm. As an empirical statement, it is unproven. Flow Hijacked retains the image only under that visible boundary.

CORE SENTENCE

Attractor and basin language is mathematically useful only when convergence, boundaries and escape are tested; in PTSD it remains a model, not an established ontology.

60

Multistability and Metastable Flexibility

FLOW HIJACKED SYNTHESIS

Multistability means that the same governing system can support more than one stable long-run pattern. Metastability describes configurations that persist long enough to matter yet remain capable of transition. These ideas are attractive for PTSD because human experience can alternate among vigilance, engagement, sleep, numbing, grief and dissociation. But variety in experience does not itself prove multiple attractors, and fewer measured transitions do not automatically mean pathology.

For estimated regimes $z_t \in \{1, \dots, K\}$, a continuous-time Markov approximation uses a generator $\mathbf{Q}$, whose off-diagonal entries $q_{rs} \geq 0$ are transition intensities and whose rows sum to zero. The transition matrix over lag Δ is

$$\mathbf{P}(\Delta) = e^{\mathbf{Q}\Delta}, \qquad \boldsymbol{\pi}^{\top}\mathbf{Q} = \mathbf{0}^{\top}, \qquad \pi_r \geq 0 \;\; (r = 1, \dots, K), \qquad \sum_{r=1}^{K} \pi_r = 1,$$

where $\boldsymbol{\pi}$ is a stationary occupancy distribution when it exists. In the Markov case, the expected dwell time in a nonabsorbing state r with $q_{rr} < 0$ is $-1/q_{rr}$; an absorbing state has $q_{rr} = 0$ and infinite dwell under the model. Slow nonzero eigenmodes of $\mathbf{Q}$ indicate long mixing times. A nearly decomposable $\mathbf{P}$ contains sets with high internal transition probability and low probability of leaving—one operational meaning of metastability. Hidden semi-Markov models should replace this formulation when dwell distributions have memory.

"Flexibility" must be decomposed into repertoire size, occupancy balance, transition diversity, context appropriateness and capacity to return. More transitions are not always better: rapid chaotic switching can be disabling, while sustained concentration or restorative sleep is valuable stability. In healthy adults, acute stress has been reported to increase network integration and reduce transition variability [Wang et al., 2022]. That finding makes an essential point: the same direction of change can be adaptive over minutes and costly if it persists indiscriminately for months.

PTSD evidence is mixed. A cross-sectional MEG study reported a constrained repertoire [Mišić et al., 2016], and a small 2026 fMRI study reported more stable anomalous states [Wu et al., 2026]. Treatment research has associated effective cognitive therapy with changes in time spent in particular default-mode subnetworks [Charquero-Ballester et al., 2022]. Yet the large multisite ENIGMA-PGC study did not find robust diagnostic differences in state dwell or transition counts [Tomas et al., 2025]. A 2026 opinion proposes restored metastability as a trauma framework while acknowledging that direct PTSD testing is sparse [Kotler et al., 2026].

TSRA therefore predicts **context-sensitive metastable flexibility**, not maximum entropy or perpetual motion. Under credible safety, the system should be able to enter and sustain engagement, and to leave defence without excessive delay. Under genuine threat, stable defence may be appropriate. A valid study would predefine regimes, validate them across modalities, quantify uncertainty in state assignment, and test whether transition structure predicts functioning beyond mean symptoms. If a continuous one-dimensional severity model predicts held-out data just as well, the multistable account should yield.

CORE SENTENCE

Healthy flexibility is not constant switching; it is reliable access to the right stability and the right transition for the present context.

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Entropy, Complexity and Recurrence

BROADER SCIENTIFIC INFERENCE

Entropy and complexity are often used as scientific-sounding synonyms for freedom, health or disorder. They are none of those things by themselves. They quantify properties of a representation at a chosen scale. A highly random signal can have high entropy and little meaningful organisation; a stable restorative state can have low entropy and high value. PTSD cannot be inferred from the direction of one complexity measure.

For regime occupancies π_r, state entropy is

$$H(Z) = -\sum_{r=1}^{K} \pi_r \log \pi_r.$$

For a discrete Markov chain with transitions P_{rs}, the entropy rate is

$$h_Z = -\frac{1}{\Delta} \sum_{r=1}^{K} \pi_r \sum_{s=1}^{K} P_{rs} \log P_{rs},$$

with $0 \log 0 = 0$ and sampling interval Δ. H measures occupancy diversity; h_Z measures uncertainty in the next transition. Two systems can share H while one follows a rigid sequence and the other switches unpredictably.

Recurrence analysis asks a different question. Given reconstructed states $\mathbf{x}_i$, define

$$R_{ij}(\epsilon) = \mathbf{1}\left\{\|\mathbf{x}_i - \mathbf{x}_j\|_{\mathbf{M}} \le \epsilon\right\}, \qquad \mathrm{RR}(\epsilon) = \frac{1}{N^2} \sum_{i,j=1}^{N} R_{ij}(\epsilon),$$

where ϵ is a recurrence radius, $\mathbf{M}$ a metric, $\mathbf{1}$ the indicator function, and N the number of reconstructed samples. Recurrence rate RR measures how often the trajectory revisits nearby regions. Diagonal-line statistics can quantify repeated sequences, but results depend on embedding dimension, delay, nonstationarity, artefact removal and the arbitrary scale ϵ. Sample entropy similarly estimates the negative logarithm of the conditional probability that matching sequences of length m remain matched at $m + 1$; it is estimator- and data-length-sensitive.

Small direct studies illustrate both possibility and limitation. An older EEG study found lower correlation-dimension estimates in PTSD across several channels, but used short recordings and multiple comparisons [Chae et al., 2004]. A 2023 resting-fMRI study reported lower entropy in several regions only in PTSD comorbid with major depression, making PTSD-specific interpretation impossible [Fu et al., 2023]. A 2026 MEG study found dimensional associations between post-traumatic stress severity, scale-free exponents and interhemispheric coupling, but did not establish criticality or a diagnostic biomarker [Popescu et al., 2026].

The proper TSRA question is not “Does PTSD lower complexity?” It is whether multiscale entropy or recurrence adds reliable information about entry, dwell and recovery, conditional on context and symptom level. A preregistered analysis should test several justified timescales, correct multiplicity, use phase-randomised and autocorrelation-preserving surrogates, and validate predictions out of sample. A measure that distinguishes groups but cannot predict a person’s transitions may remain scientifically interesting while being clinically unusable.

CORE SENTENCE

Entropy, complexity and recurrence describe a signal at a chosen scale; their clinical meaning comes only from context, prediction and validation.

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Effective Connectivity and Dynamic Causal Models

BROADER SCIENTIFIC INFERENCE

Functional connectivity asks which signals covary. Effective connectivity asks which directed influences, under a specified generative model, best explain the observations. The distinction is valuable because TSRA concerns transition rules, not correlation alone. It is also dangerous when "effective" is misheard as proven biological causation.

In bilinear dynamic causal modelling, latent neural activity $\mathbf{x}(t)$ may be written

$$\dot{\mathbf{x}}(t) = \left(\mathbf{A} + \sum_{j=1}^{m} u_j(t) \mathbf{B}^{(j)} \right) \mathbf{x}(t) + \mathbf{C}\mathbf{u}(t) + \boldsymbol{\omega}(t),$$

$$\mathbf{y}(t) = \mathbf{h}_{\text{obs}}(\mathbf{x}(t), \boldsymbol{\vartheta}_h) + \boldsymbol{\varepsilon}(t).$$

$\mathbf{A}$ contains baseline directed coupling, $\mathbf{B}^{(j)}$ the modulation of coupling by experimental input u_j, $\mathbf{C}$ direct driving inputs, and $\boldsymbol{\omega}$ endogenous fluctuations; m is the number of modelled inputs. The observation function $\mathbf{h}_{\text{obs}}$, with parameters $\boldsymbol{\vartheta}_h$, maps neural states into BOLD or electrophysiological data, while $\boldsymbol{\varepsilon}$ is observation error. Bayesian inversion estimates a posterior over parameters and model evidence. Conclusions therefore depend on the regions included, model family, priors, input timing and observation model.

PTSD studies have used this framework to challenge a one-direction "top-down failure" story. One resting-state stochastic DCM study reported predominantly bottom-up amygdala/PAG-to-vmPFC coupling in non-dissociative PTSD and a different, more top-down pattern in a dissociative subtype [Nicholson et al., 2017]. A task reanalysis using subliminal trauma cues favoured bidirectional PAG–default-mode models and reported stronger PAG-to-cortical effective coupling and cue modulation in PTSD [Terpou et al., 2020]. A method-development study identified frontal, insular and hippocampal disruption foci in soldiers with trauma, but derived biological and classifier claims in the same sample [Rangaprakash et al., 2018]. These are model-based group results, not direct recordings of causal messages moving between named regions.

For TSRA, a stronger design would fit separate pre-entry, defensive and recovery epochs; allow context to modulate $\mathbf{A}$; and predict held-out responses to controlled perturbations. Competing model spaces should include bottom-up, top-down, reciprocal, common-input and observation-confound accounts. Posterior predictive checks should ask whether the inferred couplings reproduce response timing, not merely whether one candidate has the highest relative evidence among a narrow set.

Even successful DCM does not make PTSD a circuit diagram. The estimated state depends on scale, and autonomic, sensory and social inputs can sit outside the imaged network. Effective connectivity is most useful when it narrows a causal hypothesis for later perturbation. It is least useful when arrows on a brain image are treated as a verdict about a person. Its strongest role here is to turn a broad network story into rival, quantitatively explicit pathways that a later intervention can distinguish.

CORE SENTENCE

Effective connectivity is directed influence inside an explicit generative model; it becomes causal evidence only through adequate alternatives and successful perturbational prediction.

63

Non-Normal Operators and Non-Orthogonal Modes

NEW HYPOTHESIS

Non-normality is the most mathematically specific—and most evidentially restricted—mechanism considered in this lecture. A linear operator $\mathbf{A} \in \mathbb{C}^{n\times n}$ is normal when it commutes with its adjoint:

$$\mathbf{A}\mathbf{A}^* = \mathbf{A}^*\mathbf{A}.$$

If this equality fails, $\mathbf{A}$ is non-normal. Its eigenvectors need not be orthogonal. As a result, eigenvalues can correctly predict eventual decay while badly underestimating what happens first. Consider

$$\mathbf{A} = \begin{bmatrix} -\alpha & K \\ 0 & -\alpha \end{bmatrix}, \qquad \alpha > 0, \quad K \neq 0,$$

whose two eigenvalues are both $-\alpha$. The system is asymptotically stable, yet

$$e^{\mathbf{A}t} = e^{-\alpha t}\begin{bmatrix} 1 & Kt \\ 0 & 1 \end{bmatrix}.$$

An initial perturbation in the second coordinate generates a first-coordinate response $Kte^{-\alpha t}$, which can grow substantially before it decays. This defective Jordan block has only one ordinary eigenvector; the off-diagonal K is directed feed-forward coupling that makes the operator non-normal. Stability and short-time amplification are therefore different questions.

Under a chosen Euclidean state metric, an indicator of possible instantaneous growth is the numerical abscissa

$$\omega(\mathbf{A}) = \lambda_{\max}\left(\frac{\mathbf{A} + \mathbf{A}^*}{2}\right).$$

Here $\lambda_j(\mathbf{A})$ are the eigenvalues and Re takes their real parts. Even when $\max_j \operatorname{Re}\lambda_j(\mathbf{A}) < 0$, a positive $\omega(\mathbf{A})$ permits some perturbations to grow initially. Neural-network theory has established stable non-normal amplification in balanced recurrent systems and shown that structured inputs can be selectively amplified [Murphy & Miller, 2009] [Hennequin et al., 2012]. Later theory demonstrates nonlinear transient amplification with short-term plasticity [Wu & Zenke, 2021], and primate work supplies a bridge from models to transient sensory amplification [Pattadkal et al., 2024].

Now the boundary that must remain impossible to miss: **as of 6 September 2026, the research programme located zero eligible PTSD studies that estimate operator non-normality from patient data, test non-orthogonal modes, calculate input-specific finite-time gain, or analyse PTSD pseudospectra.** PTSD is not known to "be a non-normal system." Non-normality is an optional mechanistic subhypothesis for the rapid-entry or high-gain component of TSRA.

Nor would finding a non-normal fitted operator settle the matter. Estimated $\mathbf{A}$ changes under observation scale and state coordinates; noise, omitted input, nonlinear thresholds and time variation can imitate transients; and ill-conditioned eigenvectors make inference fragile. Because normality is not invariant under arbitrary non-unitary coordinate changes, the latent metric must be justified by the observation model or biophysical constraints. A credible result would require identifiable state-space models, uncertainty on the operator, eigenvalue-matched normal surrogates, held-out perturbation predictions and independent replication.

The lived relevance is conditional but humane. A stable system can briefly produce a large response without being globally unstable. If this mechanism were supported, it would offer a precise alternative to interpreting disproportionate reactivity as weakness. Until then, the equation is a question, not an answer.

CORE SENTENCE

Non-normality can make stable dynamics transiently amplify selected inputs, but there is currently no direct operator-level evidence that PTSD does so.

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Transient Amplification and Pseudospectral Sensitivity

NEW HYPOTHESIS

Eigenvalues describe asymptotic modes of a fixed linear operator. They do not fully describe finite-time response, input direction or sensitivity to small model perturbations. Those missing quantities are exactly why non-normality is relevant to TSRA—and why it must be tested with more than a dramatic response curve.

All gain and sensitivity statements require a fixed coordinate geometry. Using the preregistered positive-definite metrics from $\mathcal{P}$, define whitened operators

$$\widetilde{\mathbf{A}} = \mathbf{M}_x^{1/2}\mathbf{A}\mathbf{M}_x^{-1/2}, \qquad \widetilde{\mathbf{B}} = \mathbf{M}_x^{1/2}\mathbf{B}\mathbf{M}_u^{-1/2}, \qquad \widetilde{\mathbf{C}} = \mathbf{M}_y^{1/2}\mathbf{C}\mathbf{M}_x^{-1/2}.$$

For $\dot{\mathbf{x}} = \mathbf{A}\mathbf{x}$, the maximum state amplification at horizon t is then

$$G_{\mathbf{M}_x}(t) = \sup_{\mathbf{x}_0 \neq \mathbf{0}} \frac{\|e^{\mathbf{A}t}\mathbf{x}_0\|_{\mathbf{M}_x}}{\|\mathbf{x}_0\|_{\mathbf{M}_x}} = \sigma_{\max}(e^{\widetilde{\mathbf{A}}t}).$$

With an input map $\mathbf{B}$ and readout $\mathbf{C}$, cue-specific gain becomes

$$G_{\mathrm{io}}(t) = \sigma_{\max}\left(\widetilde{\mathbf{C}}e^{\widetilde{\mathbf{A}}t}\widetilde{\mathbf{B}}\right).$$

The corresponding right singular vector specifies the most amplified direction in whitened input coordinates; the left singular vector specifies the whitened output pattern at time t. Transforming them back restores the declared input and output units. A claim about cue specificity should prospectively predict both, not fit them after observing the response.

Pseudospectra quantify operator sensitivity. For $\epsilon > 0$,

$$\Lambda_\epsilon(\widetilde{\mathbf{A}}) = \left\{z \in \mathbb{C} : \|(z\mathbf{I} - \widetilde{\mathbf{A}})^{-1}\|_2 > \epsilon^{-1}\right\} \cup \Lambda(\widetilde{\mathbf{A}}).$$

Here $\mathbf{I}$ is the identity, z is a complex spectral parameter, and $\Lambda(\widetilde{\mathbf{A}})$ is the ordinary spectrum. Equivalently, $z \in \Lambda_\epsilon(\widetilde{\mathbf{A}})$ if z is an eigenvalue of $\widetilde{\mathbf{A}} + \mathbf{E}$ for some unstructured perturbation $\|\mathbf{E}\|_2 < \epsilon$ in the whitened coordinates. A stable spectrum accompanied by pseudospectral contours extending far toward instability signals that small operator errors or physiological changes could strongly alter transient behaviour under that declared perturbation class. The resolvent

$$\widetilde{\mathbf{H}}(z) = \widetilde{\mathbf{C}}(z\mathbf{I} - \widetilde{\mathbf{A}})^{-1}\widetilde{\mathbf{B}}$$

adds input and output structure, revealing which frequencies and channels are amplified. Classical work showed why stable eigenvalues can fail to predict transient growth in non-normal physical systems [Trefethen et al., 1993]; neural theory later formalised input-selective transient coding and amplification [Bondanelli & Ostojic, 2020] [Hennequin et al., 2012]. These are bridge results, not PTSD findings.

A PTSD test would estimate $(\mathbf{A}, \mathbf{B}, \mathbf{C})$ from training sessions using dense EEG or MEG plus autonomic data, preregister the metrics and perturbation class, propagate full parameter uncertainty, and preregister cue directions predicted to have high or low gain. It would compare actual held-out peaks, time-to-peak and decay with eigenvalue-matched normal models, nonlinear threshold models and input-only models. If pseudospectral sensitivity merely reflects estimation noise or arbitrary rescaling, or if predicted high-gain cues do not outperform matched controls, the non-normal hypothesis fails.

The phrase “a small cue caused a huge response” is insufficient because external magnitude is not internal input norm. Meaning, context, expectation and interoception help construct $\mathbf{Bu}$. Pseudospectral analysis can become useful only after that input is measured honestly. Its promise is precision: not that the system is fragile in every direction, but that particular perturbations may be amplified over particular windows. Its danger is turning elegant mathematics into retrospective mythology.

CORE SENTENCE

Pseudospectra and singular vectors make transient amplification testable by predicting which perturbations grow, where they appear and when they decay.

65

Network Controllability and Control Energy

FLOW HIJACKED SYNTHESIS

Accessibility asks whether an alternative state can be reached. Network controllability asks whether a specified model, with specified inputs, can move between states. In a discrete linear time-invariant system

$$\mathbf{x}_{k+1} = \mathbf{A}\mathbf{x}_k + \mathbf{B}\mathbf{u}_k,$$

complete state controllability requires

$$\operatorname{rank}\begin{bmatrix}\mathbf{B} & \mathbf{AB} & \mathbf{A}^2\mathbf{B} & \cdots & \mathbf{A}^{n-1}\mathbf{B}\end{bmatrix} = n.$$

This is a binary theorem under exact assumptions, not evidence that a brain or a life is practically steerable. Near-singular controllability can demand impossible energy. For continuous dynamics, the Gramian $\mathbf{W}_c(T)$ determines the unconstrained minimum-energy solution, but its smallest eigenvalues can make target directions prohibitively costly. A more realistic objective is

$$J[\mathbf{u}] = \int_0^T \left[(\mathbf{x}(t) - \mathbf{x}_\star(t))^\top \mathbf{Q}(\mathbf{x}(t) - \mathbf{x}_\star(t)) + \mathbf{u}(t)^\top \mathbf{R}\mathbf{u}(t)\right] dt + \Phi(\mathbf{x}(T)),$$

where $\mathbf{x}_\star$ is a desired trajectory, $\mathbf{Q} \succeq 0$ penalises deviation, $\mathbf{R} \succ 0$ penalises input burden, and Φ is terminal cost. In clinical translation, burden must include adverse effects, distress, effort, access, consent and interference with valued life—not voltage or mathematical norm alone.

Network neuroscience provides useful bridges and sharp warnings. Structural-connectome models introduced average, modal and boundary controllability, but these quantities are highly sensitive to network construction and normalization [Gu et al., 2015] [Karrer et al., 2020]. Critical reanalysis found no meaningful single-region controllability in common formulations and showed that degree-like relations can survive in unrealistic null graphs [Tu et al., 2018]. A reproducible pipeline now supports stronger null-model practice [Parkes et al., 2024]. Human studies have connected modelled lower-cost transitions with more frequent brain-state transitions, but model energy is not identical to metabolic expenditure [Ceballos et al., 2025]. Perturbational studies using intracranial stimulation and TMS–EEG are encouraging bridges, not PTSD validation [Stiso et al., 2019] [Shikauchi et al., 2026].

For treatment, the relevant question is not “Which node controls PTSD?” It is whether a person-specific model predicts that a safe, acceptable input at a particular time will increase access to a valued state—and whether the observed transition matches that prediction. Therapy, medication, sleep, social support and neuromodulation act through different pathways and timescales; they cannot be treated as interchangeable columns of $\mathbf{B}$. The target may also need to be a distribution over flexible states rather than one fixed endpoint.

TSRA would be strengthened if recovery reduced validated control cost specifically for safe engagement and if this change predicted functioning beyond symptom averages. It would be weakened if controllability reduces to node degree, fails perturbational prediction, or changes without lived benefit.

CORE SENTENCE

Mathematical controllability is not clinical control; its value lies in predicting safe, consented routes toward states a person has reason to value.

66

Identifiability, Competing Models and Falsification

NEW HYPOTHESIS

A serious model must risk losing. TSRA is not protected by the fact that persistence feels asymmetric, nor by a state-space diagram that can be redrawn after every result. Its parameters must be identifiable, its predictions must survive held-out data, and simpler models must be allowed to win.

Even an exact linear input–output record does not uniquely reveal latent coordinates. For any invertible matrix $\mathbf{T}$, the transformation

$$\tilde{\mathbf{x}} = \mathbf{T}\mathbf{x}, \qquad \widetilde{\mathbf{A}} = \mathbf{T}\mathbf{A}\mathbf{T}^{-1}, \qquad \widetilde{\mathbf{B}} = \mathbf{T}\mathbf{B}, \qquad \widetilde{\mathbf{C}} = \mathbf{C}\mathbf{T}^{-1}$$

produces the same input–output behaviour because

$$\mathbf{C}e^{\mathbf{A}t}\mathbf{B} = \widetilde{\mathbf{C}}e^{\widetilde{\mathbf{A}}t}\widetilde{\mathbf{B}}.$$

Latent axes are therefore not automatically unique biological entities. Structural identifiability asks whether distinct parameters can generate identical ideal observations; practical identifiability asks whether finite noisy data can distinguish them. Sparse sampling, unknown input, process noise, observation error and person-level heterogeneity can make entry threshold, slow recovery and repeated re-triggering observationally equivalent.

The required test is a model tournament. A symptom-autoregressive baseline M_0 should compete with an input-driven linear model M_1, a nonlinear threshold model M_2, a switching or semi-Markov model M_3, the separable entry–return TSRA model M_4, and a non-normal extension M_5. Comparison should use preregistered held-out log predictive density, calibration, posterior predictive checks and site-level external validation—not fit alone. Complexity penalties or hierarchical priors should prevent a flexible TSRA model from "winning" merely because it has more ways to imitate data.

The evidence warns against easy victory. Fear-learning conclusions can change across reasonable analytic universes [Lewis et al., 2023]. A large dynamic-connectivity consortium found null diagnostic-group results that smaller studies could not override by narrative force [Tomas et al., 2025]. PTSD prediction from electronic health records looked much stronger when the target was a circular EHR label than when it was a semistructured interview [Crow et al., 2025]. Person-specific temporal models are promising, but their clusters still require independent validation [Hertz-Palmor et al., 2026].

TSRA should be weakened or rejected if entry and return parameters are unreliable; if their apparent asymmetry disappears after current threat, cue frequency, sleep, medication, baseline arousal and measurement error are included; if M_4 adds no out-of-sample prediction beyond M_0–M_3; if parameter change fails to mediate durable functioning; or if results do not replicate across trauma types and cultures. The non-normal extension should be rejected if eigenvalue-matched normal or nonlinear models predict perturbations equally well, if pseudospectra are dominated by estimation uncertainty, or if high-gain cue directions fail prospectively. An autonomous-attractor account should yield if persistence disappears once continuing input is modelled.

Failure would still teach us something. It might show that PTSD persistence is better explained by ongoing danger, ordinary learning, sleep disruption, social conditions, latent heterogeneity or measurement timescale. Flow Hijacked is not committed to one mechanism. It is committed to asking why return is difficult in a form precise enough for reality to answer back.

CORE SENTENCE

TSRA deserves the centre of the lecture only if it predicts more than simpler models and can be decisively reduced, revised or rejected by data.

PART IX

MORE THAN FEAR

Trauma can alter self, trust, development and social possibility without reducing them to fear circuitry.

67

Hyperarousal and Dissociation as Possible Regimes

NEW HYPOTHESIS

The familiar picture of PTSD is a system turned up too high: scanning, startled, unable to sleep, ready to move. That picture is real for some people, at some times. It is not the whole phenomenology. Others describe derealisation, depersonalisation, altered time, emotional distance, or a loss of access to an integrated sense of "I am here, now." And one person may move between these patterns. If we place all of them on a single line called *severity*, we risk mistaking different organisations of experience for different amounts of the same thing.

There is empirical reason to preserve this distinction. Latent-class analyses have identified high-PTSD groups distinguished by additional dissociative symptoms, helping motivate the dissociative subtype, although such classes depend on the sample, items and statistical model [Wolf et al., 2012] [Lanius et al., 2012]. A very large adolescent study found substantial overlap between self-report dissociative profiles of probable PTSD and probable Complex PTSD; that overlap is informative, but neither an algorithm nor a latent profile is a clinical diagnosis [Bi et al., 2026]. In a pilot brain–body study, PTSD and its dissociative subtype showed contrasting correlations between heart-rate variability and brainstem–limbic connectivity. That is compatible with differently organised modes, not proof of two neural kinds [Thome et al., 2022]. Persistent derealisation two weeks after acute trauma also predicted worse symptoms at three months in the AURORA cohort, beyond initial symptom burden, while its imaging subset remained much smaller than the full observational sample [Lebois et al., 2022].

Flow Hijacked therefore proposes a two-dimensional minimum representation rather than a severity line:

$$\mathbf{z}_t = \begin{bmatrix} a_t \\ \iota_t \end{bmatrix} \in \mathcal{Z}, \qquad a_t := \text{mobilisation/arousal}, \quad \iota_t := \text{experiential integration and present anchoring.}$$

High a_t with preserved ι_t is not the same state as high a_t with collapsing integration; low outward movement with low ι_t is not simply "less arousal." The observable symptom vector $\mathbf{y}_t$ is only a noisy projection, $\mathbf{y}_t = h(\mathbf{z}_t, \mathbf{c}_t) + \boldsymbol{\varepsilon}_t$, where $\mathbf{c}_t$ denotes context. Different latent states can therefore produce similar totals, and similar latent states can look different in different settings.

This is a hypothesis about possible regimes, not a finding that PTSD contains two attractors. Existing subtype studies are mainly between-person and cross-sectional; they cannot show within-person switching, dwell time, hysteresis or metastability. A preregistered script-imagery study in 71 people with PTSD is especially instructive: dissociation was not linked to the predicted bodily immobility, facial immobility or staring, while autonomic relations were nonlinear and some experiential predictions failed [Danböck et al., 2024]. The result does not destroy regime thinking. It tells us what an honest regime model must do: predict transitions and counterintuitive nulls better than a continuous-severity model, in dense measurements within the same person.

CORE SENTENCE

A single symptom total can conceal radically different ways of being unable to return.

68

Mobilisation, Freeze and Shutdown Without a Ladder Myth

BROADER SCIENTIFIC INFERENCE

Threat response is often narrated as a staircase: first social engagement, then fight or flight, then freeze, then shutdown. It is memorable. It is also too orderly for the evidence. Real defensive behaviour is assembled from partially separable processes—orientation, autonomic mobilisation, motor inhibition, attention, analgesia, action selection, memory and subjective presence. They can co-occur, uncouple, reverse rapidly, or become difficult to read from the outside. Stillness can accompany intense sympathetic mobilisation; dissociation can occur without visible immobility; movement can coexist with profound disconnection.

A ladder imposes a total ordering,

$$s_1 \prec s_2 \prec \cdots \prec s_K,$$

as though every organism must traverse the same states in the same direction. A more defensible representation is a product space,

$$\mathcal{S} = \mathcal{M}_{\text{motor}} \times \mathcal{A}_{\text{autonomic}} \times \mathcal{I}_{\text{integration}} \times \mathcal{C}_{\text{context}},$$

in which a response is a coordinate $\mathbf{s}_t = (m_t, a_t, \iota_t, c_t)$, not a rung. This notation does not establish the true physiology. It makes the empirical demand explicit: a theory must predict which combinations occur, under which cues, with what transition probabilities, and with what recovery path.

Two people may therefore share the same outward posture while occupying very different internal configurations; one person may also reach similar stillness through different routes on different occasions. Behaviour alone cannot identify the latent state.

The available PTSD evidence supports that caution. In the preregistered trauma-script study described above, acute dissociation showed an inverted-U relation with heart rate and higher nonspecific skin-conductance fluctuation and high-frequency HRV, but not the proposed motor and facial immobility markers. Nor did the study confirm a simple equation between dissociation and emotional numbing [Danböck et al., 2024]. A separate pilot found opposite directions of HRV–connectivity association in PTSD and dissociative PTSD [Thome et al., 2022]. Persistent derealisation has prospective significance in a large post-trauma cohort, yet it still does not identify a mandatory autonomic sequence [Lebois et al., 2022].

Popular polyvagal language can offer a memorable vocabulary for felt shifts in connection, mobilisation and collapse. This lecture will not convert that vocabulary into a verified phylogenetic ladder. Meta-analysis supports lower resting parasympathetic HRV indices on average in PTSD, but HRV is affected by respiration, posture, medication, fitness, sleep and measurement protocol; it does not directly identify a named vagal pathway or a person's latent state [Schneider & Schwerdtfeger, 2020].

This matters clinically and morally. "Shutdown" should not be read as surrender, weakness or a primitive endpoint. "Mobilisation" should not be read as greater courage or greater pathology. A response that looks unavailable may have been the least dangerous action in the environment that trained it. Conversely, a compelling evolutionary story is not evidence that every present reaction remains optimal. Function depends on present context: whether escape was possible, whether another person was safe, whether the threat was controllable, and whether the defensive configuration can release when circumstances change.

The scientific alternative to the ladder myth is not vagueness. It is a harder, testable account: measure multiple channels at once; distinguish state from trait; sample transitions densely; and compare competing models out of sample. Until that work exists, "mobilise," "freeze" and "shutdown" are useful descriptive families—not a universal chronology and not an anatomical destiny.

CORE SENTENCE

The nervous system does not owe our theory a neat staircase.

69

Complex PTSD and Disturbances in Self-Organisation

BROADER SCIENTIFIC INFERENCE

The diagnostic distinction has already been defined. The deeper question here is what disturbances in self-organisation add to the dynamics. Affect regulation, negative self-concept and relational difficulty may interact with core PTSD, but they need not rise and fall together. Hyperactivation and hypoactivation can alternate. A threat memory can soften while shame remains. A person may regain occupational function before trust becomes possible. "Complex" should therefore not collapse these courses into one higher point on a severity line.

The measurement literature supports taking the domain seriously while resisting premature ontology. The International Trauma Questionnaire has substantial psychometric support, but its classifications remain self-report algorithms rather than clinician-made diagnoses [Cloitre et al., 2018]. A 2026 meta-analytic confirmatory factor analysis spanning 57 studies and 43,066 participants found that the intended higher-order organisation fit adequately, yet a correlated seven-factor model fit better; it also highlighted the distinction between affective hyperactivation and hypoactivation and found limited reliability in some subscales [Kindred et al., 2026]. Earlier latent-profile work identified separable PTSD-like and Complex-PTSD-like patterns, but profile membership depended on the sample and indicators [Cloitre et al., 2013].

To move from structure to process, let

$$\boldsymbol{\eta}_t = \begin{bmatrix} p_t & r_t^+ & r_t^- & n_t & \ell_t \end{bmatrix}^\top, \qquad \boldsymbol{\eta}_{t+1} = \mathbf{F}(\boldsymbol{\eta}_t, \mathbf{c}_t) + \boldsymbol{\xi}_t,$$

where p_t represents core PTSD burden; r_t^+ and r_t^- affective hyperactivation and hypoactivation; n_t negative self-concept; ℓ_t relational access; and $\mathbf{c}_t$ context. A cross-sectional factor solution estimates covariance among components. It does not identify the transition function $\mathbf{F}$, the direction of coupling, or its local Jacobian $\mathbf{J}_t = \partial \mathbf{F} / \partial \boldsymbol{\eta}$. Calling these components a dynamical regime would therefore outrun the evidence.

Longitudinal results show why the missing dynamics matter. In two convenience samples, baseline disturbances in self-organisation predicted later core PTSD symptoms, while relationships involving dissociation and psychotic symptoms were inconsistent [Fung et al., 2025]. A five-year adolescent study observed 21 pathways among probable PTSD, probable Complex PTSD and no-disorder classifications, making one-wave identity claims untenable [Kairyte et al., 2026]. Adverse childhood experiences are associated with both diagnoses, and more strongly with Complex PTSD on average, but do not make either outcome inevitable [Li et al., 2026]. Developmental Trauma Disorder remains a distinct proposed construct with promising but insufficiently independent validation [Morelli & Villodas, 2022].

Nor does the diagnosis dictate one universal treatment sequence. A 2026 synthesis found few consistent differences between phase-based and non-phase-based approaches [Lee et al., 2026]. A trial in women with childhood-abuse-related complex presentations found benefit from both DBT-PTSD and Cognitive Processing Therapy, with an advantage for DBT-PTSD in that particular population [Bohus et al., 2020]. Stabilisation may be essential for one person and unnecessary delay for another. The configuration guides inquiry; it does not issue a single command. Its purpose is precision without reducing a life to a category.

CORE SENTENCE

Complex PTSD names an expanded pattern of suffering; it does not turn complexity into destiny.

70

Moral Injury Is Not a Synonym for PTSD

BROADER SCIENTIFIC INFERENCE

Fear is not the only way an event can remain alive. A person may survive what happened and still be unable to reconcile what they did, failed to do, witnessed, or experienced as betrayal by someone or an institution on which they depended. The phrase *potentially morally injurious event* names an exposure; *moral injury* names a proposed pattern of moral-emotional, relational and spiritual consequences. Neither term is interchangeable with PTSD, depression, grief, guilt, shame or suicidality.

Systematic reviews find that moral-injury-related constructs are associated with a range of mental-health and behavioural outcomes, including PTSD symptoms, but definitions, instruments and populations vary considerably [Hall et al., 2022] [Williamson et al., 2018]. In a large veteran sample, potentially morally injurious experiences were common among people screening positive for PTSD or depression, yet this overlap did not make the constructs identical [Norman et al., 2022]. A prospective Israeli combatant cohort that began before enlistment linked predeployment characteristics, combat exposure, betrayal-related experiences, guilt and shame with later post-traumatic stress symptoms. Because most links remained observational and self-reported, the study supports temporal architecture more than a single causal chain [Zerach et al., 2023]. In the same programme, ethical preparation was associated with lower later PTSD and other psychiatric symptoms, but it was not a randomised intervention [Zerach et al., 2023].

The distinction matters because the implied questions differ. In a predominantly threat-based formulation, the system asks: *Is this cue dangerous now?* In moral injury, the unresolved question may be: *What does this event mean about me, about us, or about the authority I trusted?* Fear extinction alone may therefore leave the moral contradiction untouched. Conversely, guilt or outrage after a morally consequential event is not automatically a disorder. It can be intelligible, reality-based and socially informative. Clinical concern arises from the pattern, persistence, impairment, rigidity and surrounding risk—not from the mere presence of moral pain.

Flow Hijacked treats moral injury as a possible change in the system's evaluative rules. The relevant state variables may include threat, responsibility, agency, belonging, legitimacy and the anticipated possibility of repair. That is a synthesis, not a demonstrated computational mechanism. Its test is whether moral variables add prospective explanatory value after exposure severity, baseline symptoms, depression and social context are modelled—and whether interventions that credibly address responsibility, compassion, restitution, institutional response or meaning change outcomes through those variables.

There is also a danger in romanticising the category. "Moral injury" can become an elegant label placed over grief, political disagreement, occupational stress or ordinary remorse. It can also shift attention from material repair to the individual's coping. The scientific task is to specify the event, appraisal, actor, relationship and outcome. The humane task is to hear moral pain without forcing it into a fear disorder simply because PTSD is the better-known language.

CORE SENTENCE

Fear asks whether the world is dangerous; moral injury may ask whether the self, the group or the trusted world is still morally inhabitable.

71

Betrayal, Shame and the Architecture of Trust

FLOW HIJACKED SYNTHESIS

Trust is not a sentimental extra added after the "real" neurobiology. It is a working estimate of whether another person, institution or environment will respond as expected when vulnerability becomes costly. Betrayal can therefore do two things at once: create pain in the present and alter the credibility of later safety signals. Reassurance from the same source that failed may carry less evidential weight, even when its words are correct.

Shame and guilt also require separation. Guilt can concern an action—*I did something wrong*—whereas shame often generalises toward the self—*there is something wrong with me*. In practice, instruments and experiences do not always respect that clean distinction. Meta-analytic evidence shows a moderate association between shame and post-traumatic stress symptoms, not a universal pathway or proof that shame causes PTSD [López-Castro et al., 2019]. A larger three-level meta-analysis likewise found meaningful associations for shame and guilt while showing that construct and measurement choices matter [Shi et al., 2021]. Prospective work around betrayal provides stronger temporal leverage but still not experimental causality. In a pre-October-7 panel, betrayal-related appraisals predicted later probable PTSD and depression after adjustment for baseline screening status and exposure; subsequent reports from the same panel linked early betrayal with one-year symptom burden [Levi-Belz et al., 2024] [Levi-Belz et al., 2026]. These are related analyses, not independent replications.

A minimal formal account treats trust in actor j, context c, at time t as a posterior expectation:

$$\tau_{j,c}(t) := \mathbb{E}\big[\theta_{j,c} \mid \mathcal{H}_t\big], \qquad \tau_{j,c}(t+1) = \Pi_{[0,1]}\Big(\tau_{j,c}(t) + \alpha^{+}_{j,c}[\delta_t]_{+} + \alpha^{-}_{j,c}[\delta_t]_{-}\Big),$$

where $\theta_{j,c}$ is the actor's latent reliability, $\mathcal{H}_t$ the history of encounters, δ_t a reliability prediction error, $[u]_{+} = \max(u, 0)$, $[u]_{-} = \min(u, 0)$, and $\Pi_{[0,1]}$ projection onto a probability scale. The Flow Hijacked hypothesis is not that these parameters have already been estimated in PTSD. It is that severe betrayal may create asymmetric updating—large or durable negative learning, narrow generalisation of positive evidence, or strong context specificity—so that trust recovers by repeated credible experience rather than instruction.

Shame can intensify this architecture by changing who is treated as the unreliable agent. If the model assigns failure globally to the self, social withdrawal may reduce exposure to disconfirming relationships. If it assigns failure globally to others, vigilance may make ambiguity feel like further evidence. Both are plausible feedbacks; neither should be presumed for an individual.

Repair, then, cannot mean demanding trust. It means making reliability observable: consistency, consent, accountability, truthful limits and enough time for positive evidence to become believable. That is a moral principle and a scientific prediction. It should be tested, not merely admired.

CORE SENTENCE

Trust is not restored by being requested; it is restored when safety becomes credible enough to update the model.

72

Identity, Meaning and the Social Self

FLOW HIJACKED SYNTHESIS

Trauma can change more than what a person remembers. It can alter the questions through which experience is organised: Who am I now? Which roles remain possible? What kind of world is this? What future can still be inhabited? These are not decorations around a fear circuit. They influence attention, action, affiliation, responsibility and the interpretation of bodily states. At the same time, identity language must not swallow diagnosis. Not every change in identity is PTSD, and no laboratory measure in this corpus reads a person's meaning directly from the brain.

Complex PTSD research gives one empirical foothold. Negative self-concept and relational disturbance form part of the ICD-11 disturbances-in-self-organisation domain, with substantial but measurement-dependent support [Cloitre et al., 2018] [Kindred et al., 2026]. In two longitudinal convenience samples, baseline disturbances in self-organisation predicted later core PTSD symptoms, while other cross-lagged relationships among dissociation and psychotic symptoms were inconsistent [Fung et al., 2025]. Moral-injury reviews add evidence that transgression and betrayal appraisals covary with distress across military and civilian settings [Hall et al., 2022]. Qualitative work can illuminate the lived architecture without estimating prevalence: a selected Nova sample whose participants were substance-exposed and receiving psychological support described individual and collective recovery as interacting, and body-recovery volunteers described meaning and commemoration as both burden and resource [Simon et al., 2025] [Cohen-Zada, 2026].

Together, these literatures justify taking self and meaning seriously. They do not justify reading identity directly from symptom scales, assuming that every person experiences identity rupture, or converting a qualitative theme into a population mechanism.

Flow Hijacked represents identity and meaning as slow variables that can shape fast state transitions:

$$\mathbf{x}_{t+1} = F(\mathbf{x}_t, \mathbf{u}_t, \mathbf{c}_t; \boldsymbol{\phi}_t) + \boldsymbol{\omega}_t, \qquad \boldsymbol{\phi}_{t+1} = \boldsymbol{\phi}_t + \varepsilon\, G(\mathbf{x}_t, \mathbf{u}_t, \mathbf{r}_t), \quad 0 < \varepsilon \ll 1.$$

Here $\mathbf{x}_t$ is the fast brain–body–action state; $\mathbf{u}_t$ is current input; $\mathbf{c}_t$ is context; and $\boldsymbol{\phi}_t$ represents slower assumptions about self, others, agency and future. The vector $\mathbf{r}_t \in \mathbb{R}^h$ contains measured relational and repair evidence that may enter slow learning—for example, reliable response, recognition, restitution or co-regulation. The small parameter ε marks a slower timescale, not immutability. This fast–slow formulation is a Flow Hijacked synthesis. The studies above do not establish its equations or parameters.

The formulation clarifies a humane paradox. An identity organised around vigilance, self-sufficiency or emotional distance may preserve coherence after a world becomes unreliable. Later, the same organisation may narrow relationships and futures. Calling it "maladaptive" too quickly forgets what it solved. Calling it permanent forgets that slow variables still change through action, relationship, language, moral repair and new experience.

Meaning-making is therefore neither a cure nor a compulsory silver lining. A person may recover without constructing a grand lesson. Another may need a story that can hold rupture without letting rupture become the whole self. The relevant outcome is not inspirational language; it is whether more truthful and liveable models permit more choices, relationships and futures.

CORE SENTENCE

Trauma may not erase the self; it may recruit the self into keeping danger intelligible.

73

Social Ecology and Co-Regulation

BROADER SCIENTIFIC INFERENCE

A nervous system never learns safety alone. It learns inside homes, neighbourhoods, institutions, workplaces, treatment rooms and cultures. Other people provide information about whether danger is near, whether distress can be shared, whether action will be supported, and whether help will still exist tomorrow. "Social support" is therefore not merely a pleasant buffer placed beside an individual disorder. It can be an input to the threat model—and an outcome damaged by the disorder.

The strongest evidence is reciprocal rather than one-directional. A meta-analysis of longitudinal studies, including 32,402 participants, found that social support predicted later PTSD symptoms and PTSD symptoms predicted later support, with small average cross-lagged associations after prior levels were controlled [Wang et al., 2021]. A five-wave AURORA analysis of 2,943 recently trauma-exposed participants likewise found that emotional support and PCL-5 symptoms predicted one another across much of the following year. Emotional support was most strongly related to subsequent negative-mood/cognition symptoms, while avoidance was the symptom cluster most strongly related to subsequently lower perceived support [Santos et al., 2026]. During Prolonged Exposure, higher support was associated with greater symptom reduction, but that association alone cannot tell us whether support caused improvement, improvement made support accessible, or both [Price et al., 2018]. Neighbourhood resources have also been associated prospectively with post-trauma trajectories and reward reactivity, extending context beyond the intimate dyad without proving neural mediation [Webb et al., 2024].

A coupled model captures the problem more honestly than a one-way "buffer" arrow:

$$\begin{bmatrix} p_{t+1} \\ s_{t+1} \end{bmatrix} = \begin{bmatrix} f_p(p_t, \mathbf{e}_t) - \beta s_t \\ f_s(s_t, \mathbf{q}_t) - \gamma p_t \end{bmatrix} + \begin{bmatrix} \epsilon_t^{(p)} \\ \epsilon_t^{(s)} \end{bmatrix},$$

where p_t is symptom burden, s_t is accessible and trusted support, $\mathbf{e}_t$ represents exposure and material conditions, and $\mathbf{q}_t$ represents relationship quality and opportunity. The coefficients β and γ are not established universal constants. The equation makes a testable claim: support and symptoms may form a feedback system, so cross-sectional correlation cannot identify which lever moved first.

It also allows support to fail, arrive too late, or become unusable when it is conditional, unsafe or supplied by a source the person cannot trust.

Co-regulation should be used with equal care. A reliable other may change breathing, attention, action choice and the meaning of an ambiguous cue. Shared routines can lower uncertainty. Institutions can make safety materially true—or fail to do so. Yet the current literature rarely measures two people's physiology, behaviour and context densely enough to establish a specific co-regulatory mechanism. "Connection heals" is humane but scientifically underspecified.

Social ecology also prevents individual blame. A person cannot regulate their way out of unsafe housing, institutional betrayal, isolation, discrimination or repeated exposure. Nor does receiving support guarantee recovery. The better question is whether the environment supplies credible, usable conditions under which alternative states become accessible—and whether the person can receive them without coercion.

CORE SENTENCE

Safety is partly social: the system learns not only from the cue, but from whether anyone reliable remains when the cue arrives.

74

Development, Sensitive Periods and Parent–Child Coupling

BROADER SCIENTIFIC INFERENCE

A child is not a smaller adult with fewer words. Development changes what can be predicted, remembered, named, regulated and borrowed from another person. It changes the meaning of separation, the available action repertoire, and the degree to which a caregiver's state becomes part of the child's evidence about the world. This creates sensitivity, not destiny. Plasticity can amplify exposure; it also keeps later learning possible.

Across 64 youth studies, a meta-analysis found that peri-traumatic and post-traumatic factors—including appraisal, family functioning and social support—were generally more informative than many distal pre-trauma variables, although estimates varied by design and trauma type [Trickey et al., 2012]. A longitudinal study of 170 young people exposed to violence found that altered early threat discrimination statistically mediated later increases in PTSD symptoms over two years. It supports developmental learning as a candidate pathway, not a complete causal explanation [Machlin et al., 2024]. At the structural-brain level, a 2026 ENIGMA mega-analysis of 3,711 participants found small age- and sex-specific maltreatment associations and no significant paediatric effects after correction, an important boundary against deterministic "trauma changes the child's brain" rhetoric [Wang et al., 2026].

Parent and child trajectories can also be coupled over long periods. After the Wenchuan earthquake, maternal PTSD-symptom trajectories predicted children's mental health ten years later [Chen et al., 2020]. A systematic review of war-related trauma and relationships in the first three years of life found only 11 heterogeneous studies, enough to establish importance but not one direction or mechanism [Chasson et al., 2026]. The relationship can carry safety, dysregulation, language, routine and access to resources; parent and child may also share the same danger and losses.

The same exposure can mean something different at age four, fourteen or forty. Development alters the available concepts, dependencies and control—not simply the numerical magnitude of a common adult response.

A minimal coupled system is

$$\mathbf{x}_{t+1}^{(c)} = F_c\left(\mathbf{x}_t^{(c)}, \mathbf{x}_t^{(p)}, \mathbf{e}_t; \boldsymbol{\theta}_c(a_t)\right) + \boldsymbol{\omega}_t^{(c)},$$
$$\mathbf{x}_{t+1}^{(p)} = F_p\left(\mathbf{x}_t^{(p)}, \mathbf{x}_t^{(c)}, \mathbf{e}_t; \boldsymbol{\theta}_p\right) + \boldsymbol{\omega}_t^{(p)},$$

where $\mathbf{x}_t^{(c)}$ and $\mathbf{x}_t^{(p)}$ are child and parent states, $\mathbf{e}_t$ is shared environment, and $\boldsymbol{\theta}_c(a_t)$ changes with developmental age a_t. The equation explicitly allows bidirectionality and common causes. It forbids the lazy inference that a correlation between parent and child symptoms identifies parental fault.

Sensitive-period claims require prospective evidence that timing changes susceptibility beyond cumulative exposure, and they must preserve later plasticity. Assessment must also be age-valid: caregiver report, child report, behaviour and physiology are not interchangeable. Development belongs in the causal model because the system is changing while it learns—not because childhood writes an irreversible sentence.

CORE SENTENCE

Child and caregiver systems can be coupled without making the caregiver the cause or the child's future fixed.

75

Resilience and Post-Traumatic Growth Without Romance

BROADER SCIENTIFIC INFERENCE

Resilience is often spoken of as a possession: something strong people have and others lack. Longitudinal science offers a better, kinder definition. Resilience is an observed course in a specified domain over a specified interval under specified conditions. A person may work, parent or connect while still having nightmares. Another may have low symptoms and little access to meaning or safety. No single axis can decide who has "recovered well."

Reviews of post-trauma trajectories consistently find heterogeneity—stable-low symptoms, recovery, delayed difficulty and persistent difficulty—but the number and apparent prevalence of classes change with sampling, attrition, measurement schedule and mixture-model assumptions [Galatzer-Levy et al., 2018]. Reanalysis work has shown that estimates of a dominant resilience trajectory can shrink when less restrictive models are used [Infurna & Luthar, 2016]. A latent class is therefore a summary of data under a model, not a resilient human type. It should never become a moral benchmark against which a person with persistent symptoms is judged.

Post-traumatic growth requires an equally careful noun. People can report changed priorities, deeper relationships, spiritual change or a stronger sense of possibility after trauma. Those reports can be meaningful. They are not proof that the event was beneficial, nor are they necessarily the same as prospectively measured positive change. In a study with pre-event and post-event measures, perceived growth was largely unrelated to actual positive change and was associated with distress and positive reinterpretation [Frazier et al., 2009]. A meta-analysis linked social support with reported growth, but much of the literature was cross-sectional and self-reported [Ning et al., 2023]. A systematic review found recurring joint patterns of post-traumatic stress and growth, underscoring that the two can coexist rather than sit at opposite ends of one scale [Fletcher et al., 2023].

Flow Hijacked therefore separates at least four outcomes: symptom course, functioning, accessible relationships and future possibility. Improvement in one may precede, enable or fail to produce improvement in another. Reported growth may be a truthful account of meaning-making even when symptoms remain. It may also be an effort to preserve coherence. Only a genuine pre-event measure can establish change from a prior level, and even then causation by trauma is not automatically identified.

Resilience is also conditional on resources. A stable course supported by housing, income, care, community and physical safety cannot be compared morally with a course unfolding amid repeated danger and loss. Context is part of the trajectory, not noise around it.

The absence of romance does not mean the absence of hope. It means hope is no longer purchased by praising the event or grading the survivor. Recovery can include grief. Adaptation can include continuing vulnerability. Growth, when a person names it, belongs to that person—not to a theory that needs trauma to have a gift inside it.

CORE SENTENCE

No person owes trauma a transformation; growth does not make the wound worthwhile, and persistent pain is not failed resilience.

PART X

OCTOBER 7 AND THE SCIENCE OF CONTINUING THREAT

October 7 offers urgent naturalistic evidence under continuing threat; it does not define PTSD as a whole.

76

A Scientific Case Study, Not a Symbol

BROADER SCIENTIFIC INFERENCE

October 7, 2023 and the prolonged period that followed belong in this lecture for a precise scientific reason. They created an unusually consequential naturalistic setting in which severe direct exposure, bereavement, captivity, displacement, military service, caregiving, repeated media exposure and continuing uncertainty could be studied across time. A few research programmes had measured the same people before the attack. Others already held long-running wearable or population data. Those designs can reveal change that a one-time survey cannot.

They still do not turn history into an experiment. Exposure was not randomised. The event was followed by war, displacement, reserve mobilisation, economic disruption, hostage uncertainty, later escalations and repeated personal losses. The counterfactual—what each person's course would have been without these events—cannot be observed directly. Attrition, online-panel coverage, self-selection and shared measurement methods remain consequential. Privacy and consent are especially delicate for small survivor, hostage and bereaved communities.

The strongest panel evidence shows why the case matters. One national cohort measured probable PTSD, depression and anxiety several weeks before the attack and repeatedly afterward, later extending to six waves over 27 months [Levi-Belz et al., 2024] [Levi-Belz et al., 2026]. A separate sample had observations in 2018 and 2022 before two post-attack waves, permitting longer-horizon trajectory modelling [Levin et al., 2025]. A three-year smartwatch programme captured abrupt changes in reported stress, mood, sleep and activity around the event, though neither the devices nor the later PCL-5 screening constitute a PTSD diagnosis [Yamin et al., 2025]. Another population cohort followed probable PTSD, probable Complex PTSD and prolonged-grief patterns during ongoing trauma and found that these outcomes did not move together [Levin et al., 2026].

These studies make October 7 a powerful test of the lecture's central question: how do prior state, exposure topology, loss, sleep, bodily regulation, trust and social context shape entry into and return from defensive states? They also expose a limit in the question. For many people, the environment had not returned to stable safety. "Post-traumatic" can mark diagnostic chronology without claiming that threat, uncertainty or bereavement had ended.

This Part therefore refuses three shortcuts. It will not use the event as political rhetoric. It will not convert Israel into one traumatised organism or assign a national diagnosis. And it will not treat this literature as the universal template for PTSD. The evidence is one substantial case study within a much larger science of interpersonal violence, accidents, disasters, combat, captivity, displacement and other traumas. Its value lies in what its designs permit us to ask—and in the discipline with which we state what they cannot answer.

Its placement matters as well. The general science has already established the questions and evidential boundaries; the case study now tests their usefulness. The event is not being used to generate a theory immune from comparison with other traumas.

CORE SENTENCE

October 7 is scientifically illuminating when treated as evidence in context, not as a symbol asked to carry an entire theory of PTSD.

77

The Discipline of Diagnostic Language

EMPIRICAL RESULT

Words such as *trauma*, *stress* and *PTSD* are often used as if intensity alone connects them. Scientific language requires a different rule: the noun must match the measurement. Trauma exposure is an event criterion, not a disorder. Acute stress describes an early response or a distinct diagnosis when its full criteria are assessed. Post-traumatic stress symptoms, or PTSS, are dimensional. A validated questionnaire cutoff may indicate probable or screen-positive PTSD. Clinician-diagnosed PTSD requires an appropriate diagnostic assessment; an administrative code is a further, different form of ascertainment.

In the 182-publication October 7 dossier assembled for this lecture, 100 records concern adjacent trauma responses, grief, distress, adaptation or functioning without establishing PTSD; 52 report PTSS severity; 29 use questionnaire-defined probable PTSD; and one uses administrative ICD ascertainment. No located population study established PTSD prevalence through clinician-administered diagnostic interviews. That distribution does not demote the other 181 papers. It specifies what each can be called.

Examples make the distinction concrete. The national pre-event panel reported *probable PTSD* from self-report thresholds, not 710 clinical diagnoses [Levi-Belz et al., 2024]. Nova trajectory studies used the PCL-5 to model symptom courses or screen-positive status, again not clinician diagnosis [Simon et al., 2026] [Magal et al., 2026]. A study of displaced adults validated an acute-stress symptom scale; it did not estimate PTSD [Gilbar, 2025]. Work on hostage families measured health, sleep and retrospective change without a PTSD instrument [Livne et al., 2024]. Studies of children released from captivity and adult released hostages used qualitative public testimonies to understand experience and coping, not diagnostic prevalence [Katz et al., 2024] [Levkovich et al., 2025]. Prolonged grief, depression and anxiety may coexist with PTSD, but their presence cannot be counted as PTSD by proxy [Levin et al., 2026] [Groweiss et al., 2026].

The single administrative record is instructive in the opposite direction. A Clalit cohort identified PTSD through ICD-10 coding while studying post-event benzodiazepine prescribing. An administrative diagnosis is closer to clinical care than a symptom cutoff, but it may miss untreated cases, encode access patterns and differ from a structured interview [Rahamim et al., 2026]. The Clinician-Administered PTSD Scale remains an anchor for diagnostic assessment, yet even a strong instrument does not erase sampling or timing questions [Blake et al., 1995].

This precision is not pedantry. Inflated diagnostic language can pathologise normal responses to extraordinary or continuing danger, distort service planning, and erase grief or moral injury by renaming it. Language that is too restrictive can also hide serious impairment. The remedy is not to minimise; it is to report the instrument, threshold, window, population and function, then reserve the strongest noun for the strongest ascertainment.

The same rule applies to change. Moving below a screening cutoff is not necessarily recovery, remaining above it is not necessarily chronic diagnosed PTSD, and a higher group mean does not diagnose any individual. Function and lived need must be reported alongside categories.

CORE SENTENCE

Distress is not a diagnosis, a cutoff is not an interview, and grief is not PTSD by another name.

78

What Pre-October-7 Baselines Can and Cannot Show

EMPIRICAL RESULT

A pre-event baseline is rare in trauma research because disasters do not announce themselves to investigators. When the same people have been measured before an event, we can distinguish newly observed change from burden that was already present. That is a major advantage over asking people afterward to remember how they used to feel. It is not, however, a complete causal counterfactual.

Let $Y_i(t_0)$ be participant i's measured outcome before the event and $Y_i(t_1)$ the observed outcome afterward. The within-person change is

$$\Delta_i = Y_i(t_1) - Y_i(t_0).$$

The event-specific causal effect would instead compare the observed future with an unobserved future under no event,

$$\tau_i(t_1) = Y_i^{(1)}(t_1) - Y_i^{(0)}(t_1).$$

Because $Y_i^{(0)}(t_1)$ is missing, it does not follow that $\Delta_i = \tau_i(t_1)$ unless assumptions about secular change, measurement invariance, co-occurring exposures and attrition are defensible. A baseline anchors the starting point; it does not manufacture the missing future.

The best-known October 7 panel measured the same adults in August 2023 and again five to six weeks after the attack. Probable PTSD rose from 16.2% to 29.8%, while probable depression and anxiety also rose [Levi-Belz et al., 2024]. Later analyses examined Jewish and Arab participants, betrayal, bereavement, exposure groups and 27-month trajectories [Groweiss et al., 2024] [Levi-Belz et al., 2024] [Levi-Belz et al., 2025] [Levi-Belz et al., 2026]. These papers provide different questions and time windows, but they substantially reuse one Panel4All cohort. Their sample sizes must not be added, and publication count is not replication count.

A separate quasi-representative Jewish sample contributed observations from 2018 and 2022 before the post-attack waves. Its long baseline helps distinguish persistent, vulnerable and recovering symptom courses, while the long gaps, different prior traumas and mixture-model choices complicate causal attribution [Levin et al., 2025]. Other baselines answer narrower questions. One prospective study measured support and emotional variables before October 7 but introduced the event-specific post-traumatic measure afterward, so it cannot be called a pre-event PTSD baseline [Barel et al., 2025]. Sleep research combined genuinely pre-event population data with retrospectively recalled sleep in other subsamples; those evidential levels should not be merged [Zak et al., 2025]. Long-running smartwatch data provide objective temporal continuity, but later screening outcomes and volunteer-device selection impose different limits [Yamin et al., 2025].

Even a perfectly timed baseline can drift in meaning if the instrument is not invariant across conditions. An item about vigilance may signify disproportionate reactivity before an event and realistic monitoring during active threat. Attrition can selectively remove the most burdened or least reachable participants, while regression to the mean can mimic recovery after an acute peak.

The hierarchy is therefore clear: same-person, prospectively measured, construct-matched baselines are strongest; pre-event adjacent outcomes and objective traces are valuable but narrower; repeated post-event measures establish course; recalled "before" states are vulnerable to reconstruction; cross-sectional snapshots establish burden, not change. None alone identifies mechanism.

CORE SENTENCE

A baseline tells us where a person was; it does not reveal the counterfactual future they would otherwise have lived.

79

Exposure Gradients and Heterogeneous Trajectories

EMPIRICAL RESULT

The most defensible conclusion from the October 7 trajectory literature is not that everyone recovered, nor that a nation remained permanently traumatised. It is that trajectories differed, and that exposure topology mattered. Direct attack, loss of a close person, captivity-related uncertainty, displacement, reserve service, income loss and repeated indirect exposure are not interchangeable doses of one substance. Each changes a different set of demands and resources.

In the six-wave pre-event panel, probable PTSD rose sharply at one month and attenuated substantially on average by 27 months, while direct exposure and Arab ethnicity predicted higher trajectories. Depression and anxiety had partly different predictors, and reported post-traumatic growth rose while nontrivial symptoms remained [Levi-Belz et al., 2026]. A related analysis divided participants into direct exposure, bereavement, reserve duty and indirect exposure. Average burden declined, but reserve-duty participants had the highest probable-disorder rates and symptom levels at both follow-ups, with minimal improvement, whereas indirectly exposed participants showed significant symptom reduction [Levi-Belz et al., 2025]. Bereavement analyses found higher probable PTSD, depression and anxiety nine months after the attack and less acute-wave decline than among non-bereaved participants [Levi-Belz et al., 2025]. These results are informative but derive from overlapping members of the same panel; they are not three independent confirmations.

A different long-baseline Jewish sample identified four symptom trajectories—stable-low, recovery, vulnerability and chronic-high—across observations beginning in 2018. The majority occupied the stable-low class in that particular model, while childhood trauma and war exposure differentiated other courses [Levin et al., 2025]. Among Nova survivors, a selected two-wave cohort produced a much larger proportion of high or worsening PCL-5 trajectories [Simon et al., 2026]. That contrast is not a contradiction to be averaged away. The Nova sample represented unusually direct exposure, had no pre-event symptom baseline, used variable assessment windows, and was vulnerable to selection and attrition. It cannot estimate a national course.

Trajectory models are commonly written as finite mixtures:

$$p(\mathbf{y}_{i,1:T}) = \sum_{k=1}^{K} \pi_k \, p(\mathbf{y}_{i,1:T} \mid z_i = k, \boldsymbol{\theta}_k), \qquad \sum_{k=1}^{K} \pi_k = 1.$$

Here z_i is an unobserved class, π_k its estimated proportion, and $\boldsymbol{\theta}_k$ its trajectory parameters. The number K, membership and proportions depend on observation times, missingness, distributional assumptions and candidate models. A class is not an attractor, a biological essence or a fixed prognosis.

Exposure gradients therefore change conditional probabilities, not destinies. They should guide targeted support and better study design, not create a hierarchy of legitimate suffering. The most useful next models will allow time-varying exposures and states, preserve individual trajectories beneath group averages, and test whether context and resources alter entry and return—not merely whether one group's mean score is higher.

They must also permit exposure category to change: an indirectly affected participant may later be bereaved, displaced or mobilised. A label assigned at baseline can otherwise conceal the very sequence that shapes the trajectory.

CORE SENTENCE

Exposure changes the odds and the terrain; it does not write one trajectory for every person who was there.

80

Displacement, Uncertainty and Continuous Traumatic Stress

BROADER SCIENTIFIC INFERENCE

Displacement is often entered into a model as a binary covariate: evacuated or not. Lived displacement is a bundle. It can include prior proximity to attack, loss of home and routine, crowded or temporary housing, family separation, interrupted work and schooling, weakened community ties, financial loss, and uncertainty about return. It may also move a person away from immediate physical danger. A higher symptom score among displaced people cannot reveal which pathway mattered—or whether different pathways acted in opposite directions.

In a four-wave cohort of young adults living in northern and southern conflict zones, traumatic loss, displacement and income loss were associated with persistently higher anxiety, depression, PTSD-screen and betrayal scores over a year. The first observation occurred months after October 7, so the study establishes longitudinal association under continuing exposure, not change from a pre-event baseline [Amsalem et al., 2026]. A study of 480 displaced residents from one southern city linked acute-stress symptoms with direct and family exposure and lower support; it validated a symptom scale rather than diagnosing acute stress disorder or PTSD [Gilbar, 2025]. A cross-sectional comparison found higher probable PTSD and lower functioning and trust among Jewish evacuees, but exposure differences and displacement were inseparable [Bitton et al., 2026]. Among adolescents, displacement was associated with more depression and stress, while the anxiety difference was null; no PTSD measure was used [Segal et al., 2026]. Nulls matter because displacement does not have one inevitable psychological signature.

Continuing threat changes the logic of "persistence." Let D_t denote credible current danger, C_t the context available to the person, and S_t their defensive state. The relevant transition law is conditional:

$$\Pr(S_{t+1} = s' \mid S_t = s, D_t, C_t, H_t),$$

where H_t is the person's history. A high probability of remaining vigilant while D_t remains high is not, by itself, evidence of a pathological return rule. TSRA becomes clinically informative only if current danger, real control and available safety are modelled—and if the defensive policy remains excessively generalised, inflexible or impairing relative to those conditions.

Research following a later wartime escalation illustrates the point: evacuated participants reported more continuous-traumatic-stress response, depression and anxiety, and peritraumatic dissociation was a strong predictor across outcomes. Yet the sample was online, predominantly female and already embedded in continuing threat [Harwood-Gross et al., 2026]. The construct *continuous traumatic stress* describes adaptation while threat recurs or remains anticipated. It is not synonymous with ICD-11 Complex PTSD, and it should not be inferred solely because calendar time has passed.

Nor does it erase individual variation: people living under the same declared threat can have profoundly different proximity, control, shelter, loss and physiological burden.

The word *post* can mark time since an initiating event. It must not smuggle in a safety condition that people do not inhabit. Scientific compassion begins by asking whether the alarm is responding to memory, to the present, or to both.

CORE SENTENCE

When danger is still arriving, return cannot be judged as though safety were already available.

81

Children, Families and Jewish and Arab Communities

BROADER SCIENTIFIC INFERENCE

Children and communities are often described with the very categories that need explanation. “Youth,” “family,” “Jewish” and “Arab” are not mechanisms. Within each are different ages, languages, locations, losses, prior adversities, economic conditions, service access, exposure patterns and relationships to institutions. Research that treats the label as the cause risks turning an unequal environment into an intrinsic group property.

The Jewish–Arab evidence after October 7 is a clear warning. In a prospective national panel with pre-event measurements, Arab participants had higher adjusted odds of probable PTSD, depression and anxiety than Jewish participants at the early follow-up [Groweiss et al., 2024]. The longer six-wave analysis from the same cohort also associated Arab ethnicity with a higher probable-PTSD trajectory [Levi-Belz et al., 2026]. But a separate representative cross-sectional survey found broadly similar probable PTSD rates across its Arab and Jewish respondents [Mayer et al., 2024]. Differences in timing, baseline control, exposure, sampling, language, trust, resources and service barriers can account for some divergence. The evidence does not support one timeless ethnic-risk statement. A Bedouin adolescent study measured exposure, distress, coping, sense of coherence and community resilience—not PTSD—and found substantial within-group patterning by sex and coping [Al-Said & Braun-Lewensohn, 2024]. It should remain evidence about those measured constructs.

Youth studies require the same restraint. A digital survey of 744 adolescents found a high questionnaire-defined probable-PTSD threshold and associations with direct impact, economic deterioration, news exposure, resilience and family support. It was cross-sectional and nonprobability-sampled, so its percentage is not a population diagnosis rate [Refaeli et al., 2025]. In 198 adolescents studied with questionnaires, ecological momentary assessment and nightly diaries, fatigue and avoidant coping were risk correlates while in-person interaction was protective; PTSD was not measured [Haham et al., 2026]. Displaced adolescents showed higher depression and stress but no anxiety difference in another cross-sectional study [Segal et al., 2026].

Family studies show coupling without assigning blame. In a treatment-seeking sample of 220 child–parent dyads, maternal and child post-traumatic symptoms were associated differently by child age and exposure; the high screening rates cannot be generalised beyond the referred sample, and cross-sectional mediation cannot establish direction [Rachamim et al., 2025]. Among mothers of children aged five to eighteen, partner reserve deployment was associated with child difficulty only when parental burnout was high, suggesting conditional rather than uniform family effects [Keleynikov et al., 2026]. A perinatal matched-control survey linked partner deployment with probable maternal depression and attachment difficulty, with lower support as a statistical mediator; infant outcomes and causal mediation were not established [Allouche-Kam et al., 2025].

The correct scientific unit is therefore not ethnicity or family status alone. It is a developing person in a family, language, exposure history, material setting and changing threat environment. The correct ethical stance is equally clear: family coupling is not family fault, and community difference is not community essence.

CORE SENTENCE

A demographic label is not a mechanism; the mechanism lives in exposure, development, resources, relationships and actual safety.

82

Survivors, Bereaved Families, Soldiers, Responders, Health Workers and Media Exposure

BROADER SCIENTIFIC INFERENCE

There is no single population called “those affected by October 7.” The research becomes more honest when each group is represented by its exposure, outcome, design and missing evidence—not arranged in a competition over whose suffering counts most.

Among Nova festival survivors, an early help-seeking study of 123 people examined acute stress symptoms, anxiety, depression and peritraumatic dissociation; it did not diagnose PTSD, excluded several severely affected groups, and could not establish causal effects of pre-event substance use [Nacasch et al., 2024]. Later work found heterogeneous PCL-5 trajectories in a selected two-wave sample and circadian instability in a prospective wearable cohort, but both used screening outcomes and neither had pre-event PTSD assessment [Simon et al., 2026] [Magal et al., 2026]. These studies are unusually valuable for direct mass-trauma exposure and still leave major questions about representativeness and long-term clinical course.

Captivity and ambiguous loss require different evidence. In 26 older adults from Gaza-envelope kibbutzim, released former hostages had higher grief than displaced non-captive survivors, while group differences in PTSD, disturbances in self-organisation, depression and anxiety were null. The sample was far too small for reassurance or prevalence inference [Hoffman et al., 2026]. Interviews with hostage relatives mapped private trauma, public struggle, solidarity, impaired functioning and continuing bonds without producing symptom rates [Yehene et al., 2025]. Media interviews representing 18 released children documented experiences of kidnapping, captivity and release but were repeated, edited and not diagnostic assessments [Katz et al., 2024]. Public testimonies from released adults revealed coping through relationships, routines, imagination and preserved agency; survivorship and media selection prevent claims of efficacy [Levkovich et al., 2025]. Bereavement studies with pre-event data show higher probable PTSD, depression and anxiety over time, but grief must not be reduced to those outcomes [Levi-Belz et al., 2025].

Military evidence is similarly differentiated. In 806 help-seeking reservists, hand-to-hand combat, exposure to remains and perceived responsibility for noncombatant death had different associations with probable PTSD symptom patterns; the male-dominated clinical sample was cross-sectional [Shelef et al., 2025]. Fifteen reservists with pre-existing PTSD described hyper-awareness as useful in the field and return home as difficult. That qualitative finding illustrates context dependence, not a general benefit of re-enlistment [Harwood-Gross et al., 2025].

First responders and care workers occupy a shared-trauma ecology: they may be exposed personally while responding professionally. A first-month study linked traumatic stress and resilience differently according to responder status and active engagement, without diagnosing PTSD [Saar-Ashkenazy et al., 2024]. Interviews with body-recovery volunteers described sensory overload, moral conflict, support and commemoration [Cohen-Zada, 2026]. Mental-health workers reported anxiety, acute stress, media-related secondary trauma, resilience and growth; therapists treating bereaved and hostage families reported more secondary traumatic stress and anxiety, but neither study established PTSD prevalence [Dahan et al., 2024] [Shklarski & Latzer, 2025]. Studies of hospital staff and clinicians mainly measured wellbeing, resilience and burnout, not PTSD [Cohen & Sudarsky, 2024].

Finally, media exposure crosses all these groups. Greater news or social-media exposure is repeatedly associated with anxiety or PTSS [Kaim & Bodas, 2024] [Enav et al., 2025] [Yamin et al., 2025]. The causal direction remains unresolved: exposure may amplify distress, distress may drive checking, and both may form a loop. No one should infer equivalence between viewing media and surviving direct attack.

Taken together, the literature supports a person–exposure–time matrix, not one national score. It also reveals the missing studies: large clinician-assessed prospective cohorts of direct survivors, released hostages, bereaved families, responders and minority communities, built with privacy, consent and cohort independence at their centre.

CORE SENTENCE

There is no single aftermath; the honest unit is a person in a particular exposure, relationship and time—not a nation converted into one score.

PART XI

TREATMENT: ESTABLISHED CARE AND THE FRONTIER

Established treatment outranks novelty, while frontier work remains worth testing carefully.

83

Treatment Outcome Is Not the Same as Mechanism

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE

A treatment can work before we know exactly how it works. That sentence is not a retreat from science; it is one of science's safeguards. In PTSD research, efficacy is established by an outcome contrast: people allocated to one condition improve more, on average, than people allocated to a credible comparison. Mechanism is a harder claim. It asks whether a specified process changed because of treatment, whether that change preceded clinical improvement, whether manipulating that process changed the outcome, and whether plausible alternatives were ruled out. A fall in symptoms after exposure does not by itself prove fear erasure. Improvement after Cognitive Processing Therapy does not prove that belief change was the sole active ingredient. Benefit from the full EMDR protocol does not establish that bilateral stimulation uniquely caused it.

The outcome itself is not one number. Let the patient-important outcome vector be

$$\mathbf{Y}_i(t) = \begin{bmatrix} S_{\text{tot},i}(t) & D_{\text{dx},i}(t) & F_i(t) & Q_i(t) & B_{\text{tx},i}(t) \end{bmatrix}^\top ,$$

where S_{tot} is symptom severity, D_{dx} diagnostic status, F functioning, Q quality of life, and B_{tx} treatment burden or harm for person i at time t. A trial may show a favorable average treatment effect on S_{tot} while leaving durability, daily functioning, or rare harms uncertain. Network meta-analyses support several psychotherapies, yet relative rankings become less secure when comparisons are active rather than wait-list based and when follow-up evidence thins [Yunitri et al., 2023] [Mavranezouli et al., 2020]. Session-level work also shows that symptoms and functioning can influence one another in different temporal directions, which cautions against treating symptom reduction as a complete account of recovery [Benfer et al., 2024].

Causal mechanism requires still more. In the expression below, Y denotes one prespecified scalar component of $\mathbf{Y}$—or a clinically justified scalar utility $Y = \mathbf{a}^\top \mathbf{Y}$, with $\mathbf{a}$ fixed before analysis—not the entire vector. Component-specific effects must still be reported rather than silently collapsed into one favorable average. If A denotes treatment and M a proposed mediator, then the total effect for that scalar estimand can be written in potential-outcome notation as

$$\text{TE} = \mathbb{E}[Y(1, M(1)) - Y(0, M(0))] .$$

Decomposing this into a mediated path and a direct path requires assumptions about confounding, timing, measurement error, and the meaning of hypothetical cross-world quantities. In ordinary language: observing that M and Y move together is not enough. Both might reflect expectancy, therapeutic alliance, repeated approach, spontaneous change, or a third process. Small mechanistic samples make the problem sharper. A longitudinal fMRI study found changes in occupancy of large-scale network states after successful cognitive therapy, an important direct bridge to dynamics; however, the recovered subgroup was small, and the finding does not prove that network-state change caused recovery [Charquero-Ballester et al., 2022]. Likewise, target engagement without clinical superiority is evidence that an intervention reached something, not that it helped enough.

This distinction protects patients from two symmetrical errors. We should not withhold an effective treatment because its mechanism remains incomplete. We should also not market an elegant mechanism as though it were an established treatment. Throughout this Part, every intervention will therefore be judged on separate axes: trial design, comparator, sample size, symptom and functional effects, completion, durability, adverse events, independent replication, and regulatory status. Mechanism will be discussed, but never allowed to borrow certainty from outcome.

CORE SENTENCE

A treatment effect tells us that an intervention changed an outcome; it does not, by itself, tell us which part changed the system or why.

84

What Guidelines Agree On

EMPIRICAL RESULT · AUTHORS' INTERPRETATION

Across serious guidelines, the center of gravity is clear even when the wording differs: offer an individual, manualized, trauma-focused psychotherapy when it is clinically appropriate, available, and acceptable to the person. The 2023 VA/DoD guideline gives its strongest psychotherapy recommendation to Prolonged Exposure, Cognitive Processing Therapy, and EMDR. The 2025 American Psychological Association guideline strongly recommends CPT, PE, and trauma-focused CBT, while placing EMDR in a conditional category. NICE's 2018 guideline also centers trauma-focused psychological treatment, with age, timing, preference, and context shaping the choice. These differences in grade are not a contest in which one guideline has discovered the single winner. Grading systems weigh certainty, comparators, feasibility, and values differently, and every document has an evidence-search closing date. A systematic review of fourteen international guidelines found broad convergence on CBT and substantial variation in recency and in nightmare recommendations [Martin et al., 2021].

The evidence base behind that consensus is broad rather than immaculate. A 2023 network meta-analysis covering 98 randomized trials and 5,567 participants found benefit across CPT, EMDR, cognitive therapy, NET, PE, CBT, and present-centered therapy, while comparative rankings remained uncertain [Yunitri et al., 2023]. A more recent individual-participant-data meta-analysis found trauma-focused CBT superior to inactive comparisons but not significantly different from active psychological treatments; only a subset of eligible trial data was available, and moderator findings were limited [Wright et al., 2025]. The responsible conclusion is neither "all therapies are the same" nor "one brand is universally best." It is that several well-specified treatments work, average differences among bona fide treatments are often modest or uncertain, and fit matters.

Guidelines also agree, in broad form, on what should follow that first sentence. Treatment choice is shared rather than imposed. Diagnosis, current safety, medical and psychiatric comorbidity, dissociation, substance use, previous treatment, developmental stage, language, cultural meaning, clinician competence, session burden, and the person's informed preference all belong in the decision. Present-centered therapy and Written Exposure Therapy can be credible alternatives when a first-line trauma-focused protocol is declined, unavailable, or poorly matched. Medication is reasonable when it is preferred or clinically indicated, but psychotherapy usually receives priority when both are feasible. For children and adolescents, developmentally adapted trauma-focused CBT has the strongest general position; adult evidence cannot simply be miniaturized [Lofthouse et al., 2026].

Agreement has edges. The VA/DoD evidence review searched through May 2022, so its "insufficient evidence" judgments cannot adjudicate trials published in 2024–2026. NICE's 2018 date must be visible for the same reason. Conversely, a new trial does not instantly overturn a guideline: synthesis, replication, harms, applicability, and implementation still matter. Guideline recommendations are population-level starting points, not instructions to persist indefinitely with an approach that is not helping.

The practical rule is simple but not mechanical. Begin with the best-supported options. Explain them without coercion. Measure symptoms, functioning, sleep, risk, and burden. Reassess when improvement stalls. Preserve a route to another evidence-based treatment rather than turning nonresponse into a judgment about the person. Established care outranks novelty, but evidence-based care also includes the humility to change course.

CORE SENTENCE

Guidelines offer a strong starting map—trauma-focused psychotherapy first when feasible—but good care still requires preference, context, measurement, and a route to change course.

85

Prolonged Exposure

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Prolonged Exposure is built around a humane but demanding proposition: carefully approaching safe reminders can create learning that avoidance has prevented. In-vivo exposure addresses places, activities, objects, or situations that are avoided despite being sufficiently safe in the present. Imaginal exposure returns, with structure and support, to the trauma memory. The purpose is not to force disclosure, overwhelm the person, or prove courage. Nor is it to delete what happened. Contemporary learning accounts treat extinction as new, context-sensitive learning that can compete with older threat predictions.

The clinical evidence is among the strongest in PTSD treatment. A meta-analysis of 65 articles and 4,929 participants found large average benefits against wait-list or treatment as usual, smaller benefits against non-trauma-focused active controls, and little average difference from other trauma-focused treatments or medication; available follow-up effects were generally stable [McLean et al., 2022]. In a head-to-head trial of 916 U.S. veterans, both PE and CPT produced substantial improvement. PE's average advantage on clinician-rated symptoms was statistically detectable but not clinically meaningful, and dropout was higher, an unusually clear reminder that efficacy and acceptability are different dimensions [Schnurr et al., 2022]. A trial including people with psychotic disorders found PE and EMDR both superior to waiting list, with benefit maintained to six months and no evidence that a universal stabilization phase was required [van den Berg et al., 2015]. These data argue against blanket exclusion, not against individualized safeguards.

PE is often described as habituation: distress rises, then falls. That can occur, but within-session fear reduction is not the only plausible engine and should not become a performance test. A person can learn, "I can remember and remain here," "this cue is not the event," or "anxiety can change without escape" even when distress does not trace a smooth downward curve. The more defensible mechanism is a family of updates involving expectancy violation, contextual discrimination, self-efficacy, memory elaboration, and weakened negative reinforcement of avoidance. Which components carry the effect, for whom, remains incompletely resolved.

Delivery can be adapted without assuming that more intensity is always better. Massed and standard PE schedules remained clinically effective at twelve months in a noninferiority follow-up [Dell et al., 2023]. Another randomized trial of compressed formats found meaningful gains in both arms, with some differences in maintenance [Peterson et al., 2023]. A 2026 meta-analysis reported an average completion rate of 71.8% across guideline-recommended individual trauma-focused treatments and suggested that completed dose and personalized scheduling may matter, while acknowledging that much of the dose evidence is not randomized [Leithner et al., 2026].

The clinical boundary matters most. Exposure should be collaborative, paced, and directed toward avoided situations that are safe enough to approach—not toward danger that remains real. Risk, substance withdrawal, medical instability, dissociation, sleep deprivation, and the person's consent can alter timing and format. Temporary distress is not the same as injury, yet it deserves preparation and monitoring. Dropout is not proof that PE is harmful, and completion is not proof that it was right for this person. PE is a powerful route to new learning, not a moral test and not the only road back.

CORE SENTENCE

Prolonged Exposure works by making safe approach and new learning possible; it is neither forced reliving nor erasure, and its strength does not make it the right format for everyone.

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Cognitive Processing Therapy

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Trauma can change more than predictions of physical danger. It can leave conclusions that feel less like thoughts than laws: "I should have known," "I caused what happened," "nobody can be trusted," "power is always dangerous," "the future is closed." Cognitive Processing Therapy calls these stuck points, not because the person is stubborn, but because an explanation forged under shock, coercion, hindsight, or betrayal can continue organizing attention, emotion, and action. CPT examines those conclusions through structured writing, questioning, and cognitive practice across domains such as safety, trust, power and control, esteem, and intimacy.

The treatment has a mature evidence base. A meta-analysis of eleven studies with 1,130 adults reported large advantages over inactive controls—Hedges' $g = 1.24$ after treatment and $g = 0.90$ at follow-up—while advantages over active treatments were smaller and were not clearly retained at follow-up [Asmundson et al., 2019]. The large 916-participant veteran trial comparing CPT with PE found substantial improvement in both groups. PE showed a small mean advantage that did not reach clinical importance; the result supports two effective choices rather than a league-table victory [Schnurr et al., 2022]. The broader network evidence likewise places CPT among several effective psychotherapies while leaving fine-grained rankings uncertain [Yunitri et al., 2023].

CPT's explanatory appeal is obvious: if an overgeneralized appraisal keeps generating danger, shame, or guilt, changing that appraisal should loosen the loop. But efficacy does not prove that formal cognitive restructuring is the unique mediator. Beliefs may change because the person repeatedly approaches memories and meanings; symptoms may fall before a belief measure detects change; therapeutic relationship, practice, expectancy, and behavioral re-engagement may contribute. A secondary session-level analysis of the same 916-participant veteran parent trial described above found different patterns of symptom change in CPT and PE, suggesting process heterogeneity, not an independent efficacy replication or a single common clock [Moshier et al., 2024]. In a small 2026 trial analysis of people with comorbid PTSD and major depression, within-person reductions in core PTSD symptoms predicted subsequent reductions in depressive clusters, whereas the reverse paths were not significant; the finding is clinically interesting but too small to become a universal sequence [Kline et al., 2026].

In Flow Hijacked terms, CPT may reduce the precision of threat-laden or morally condemning priors that keep reinterpreting ambiguous evidence in one direction. That is a scientific synthesis, not a demonstrated operator inside the brain. If b_t denotes a belief distribution at session t, CPT could be represented as improving the calibration of updating,

$$b_{t+1}(z) \propto p(e_t \mid z)^{\lambda_t} b_t(z),$$

where z is a hypothesis about self or world, e_t is new evidence, and λ_t is the effective weight given to that evidence. The clinical meaning is not "think positively." It is learning to let present evidence count, while refusing to rewrite responsibility in ways that excuse perpetrators or blame survivors.

CPT can be delivered individually or in groups and in standard or intensive schedules. It still requires attention to language, literacy, culture, moral injury, dissociation, and ongoing danger. Some people prefer its explicit work with meaning; others find experiential exposure or EMDR more acceptable. A formulation should help choose, not rank character. The aim is not to win an argument with a traumatized mind. It is to restore enough epistemic room for a more accurate and less imprisoning account of what happened and what remains possible.

CORE SENTENCE

CPT does not ask a person to deny the trauma; it helps separate what happened from the global rules about self, trust, blame, and future that the trauma taught too well.

87

EMDR

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Eye Movement Desensitization and Reprocessing is a structured, multi-phase treatment in which trauma-related material is activated while attention is alternated, commonly through guided eye movements. It includes history taking, preparation, assessment, processing, installation, body scan, closure, and reevaluation. That whole protocol—not an isolated pair of moving fingers—is what clinical trials usually test. This distinction allows two truths to stand together: EMDR is an evidence-based PTSD treatment, and the unique causal role and neurobiological story of bilateral stimulation remain less certain than the clinical efficacy of the package.

An individual-participant-data meta-analysis identified fifteen eligible randomized trials but obtained eight datasets totaling 346 people. It found no statistically significant differences between EMDR and other psychological therapies in symptom reduction, response, remission, or dropout [Wright et al., 2024]. This supports comparative efficacy; it does not demonstrate superiority. In the 155-participant trial among people with psychotic disorders, EMDR and PE both outperformed waiting list, with gains maintained through six months [van den Berg et al., 2015]. A 2025 randomized study of 159 patients with personality disorder—some with and some without PTSD at baseline—extended evidence into a population often excluded from trials: symptoms fell relative to wait-list, and loss of diagnosis was reported within the initially diagnosed subgroup [Hafkemeijer et al., 2025]. Across the wider network literature, EMDR appears among effective therapies, while certainty about rankings and long-term comparisons remains limited [Yunitri et al., 2023].

Why might dual attention matter? Working-memory accounts propose that holding an image while performing another demanding task could alter vividness and emotionality. Orienting-response, interhemispheric, REM-like, and reconsolidation accounts have also been proposed. The component literature does not license one settled explanation of the whole protocol, and "it mimics REM" is not an established clinical mechanism. Any contribution of a component could depend on task difficulty, memory properties, expectancy, therapist guidance, and timing. Protocol efficacy cannot be back-filled as proof of every mechanism attached to the protocol.

The lived work is also easily caricatured. EMDR does not require the memory to be a perfectly fragmented, nonverbal object waiting for bilateral stimulation to unlock it. Trauma memories are heterogeneous. Nor should bodily sensations during processing be described as literal trauma stored in tissue. They are real components of an embodied memory-and-prediction system, but the metaphor must not outrun evidence. Preparation is important; it should not become an indefinite gatekeeping phase unsupported by individualized risk.

Guidelines grade EMDR differently. VA/DoD places it with PE and CPT in its strongest recommendation; APA's 2025 guideline recommends it conditionally; NICE recommends it for indicated adults. That is a difference in evidence appraisal, not a finding that the treatment changes ontological status at a border. The practical choice turns on preference, access to a trained clinician, comorbidity, tolerability, and response. Adverse-event reporting across psychotherapy trials remains less systematic than it should be, so "no unusual signal" is not equivalent to exhaustive safety knowledge.

EMDR deserves neither mystification nor dismissal. Its strongest claim is already substantial: as a complete, trained protocol, it can reduce PTSD symptoms and help many people. Scientific respect means allowing that claim to remain strong without making the mechanism more certain, more exotic, or more exclusive than the evidence permits.

CORE SENTENCE

EMDR is a credible evidence-based treatment as a full protocol; its clinical value is clearer than any single story about why bilateral stimulation works.

88

Written, Narrative and Phase-Based Approaches

EMPIRICAL RESULT · AUTHORS' INTERPRETATION

The choice is not simply between three long protocols. Written Exposure Therapy, Narrative Exposure Therapy, and phase-based approaches ask different practical questions: Can an effective intervention require fewer sessions? Can repeated traumatic experiences be placed within an autobiographical timeline? Does strengthening emotion and relationship skills before trauma processing add value for people with complex histories? The answers are promising, but not interchangeable.

Written Exposure Therapy is deliberately spare: usually five structured sessions, with sustained writing about the trauma and its meaning, little therapist-directed cognitive restructuring, and no extensive between-session exposure assignments. Three head-to-head trials make WET more than a convenient idea. In 126 adults, it met a prespecified noninferiority criterion against twelve-session CPT and had fewer dropouts [Sloan et al., 2018]. In 169 service members, WET was again noninferior to CPT; completion was 76.5% compared with 54.8% [Sloan et al., 2022]. In 178 veterans, it was noninferior to PE through the primary assessment, with dropout of 12.5% versus 35.6%, and outcomes followed through thirty weeks [Sloan et al., 2023]. Those are meaningful access and burden findings. They do not prove that five sessions are universally sufficient.

Noninferiority has exact logic. If lower symptom scores are better and $\Delta_{\text{NI}} > 0$ is the largest clinically acceptable loss, the test is

$$H_0 : \mu_{\text{WET}} - \mu_{\text{control}} \geq \Delta_{\text{NI}}, \qquad H_1 : \mu_{\text{WET}} - \mu_{\text{control}} < \Delta_{\text{NI}}.$$

Success depends on a justified margin, retention, assay sensitivity, and missing-data assumptions—not on a nonsignificant difference. WET's lower session burden may be a genuine clinical advantage, especially where time, travel, work, caregiving, or workforce scarcity obstruct care.

Narrative Exposure Therapy constructs a chronological lifeline in which multiple traumatic and nontraumatic experiences are located and narrated. It has particular relevance in refugee, war-affected, and multiple-trauma settings. A 2019 synthesis of sixteen randomized trials involving 947 participants found large within-group changes and benefit over nonactive comparators, but not over combined active comparators [Lely et al., 2019]. A later meta-analysis of eighteen randomized trials estimated a moderate between-group effect, $g \approx -0.57$, and a higher probability of loss of diagnosis, yet those advantages attenuated or disappeared after publication-bias adjustment [Wei & Chen, 2021]. Humanitarian importance should sharpen, not relax, the standard of evidence; cultural adaptation, interpreters, continuing threat, and practical safety deserve direct study.

STAIR—Skills Training in Affective and Interpersonal Regulation—aims to strengthen emotional and relational capacities before or alongside trauma processing. An influential 104-participant childhood-abuse trial found advantages for a STAIR-plus-exposure sequence on selected outcomes [Cloitre et al., 2010]. Yet a 121-participant trial found STAIR plus EMDR no better than immediate EMDR, with slightly higher dropout [van Vliet et al., 2021]. In 2026, a session-level trial analysis found no superior early emotion-regulation improvement from STAIR over immediate PE and no mediation of later PTSD change [Oprel et al., 2026]; a contemporary meta-analysis found few robust differences between phase-based and non-phase-based care amid limited heterogeneous trials [Lee et al., 2026].

The humane conclusion is not that stabilization is useless. Skills work can be valuable, and some people want or need it. The evidence does not support making it a universal tollgate that every person with complex trauma must pass before receiving trauma-focused treatment. Briefness, narration, and sequencing should be selected for the person and context, then judged by outcomes—not elevated into doctrines.

CORE SENTENCE

Brief, narrative, and skills-based routes can widen access and fit, but none should become a universal shortcut or a universal gate before trauma-focused care.

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Nightmares, Insomnia and Sleep-Focused Treatment

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE

Night does not merely reveal daytime PTSD. For some people, sleep is one of the places where the defensive regime repeatedly rebuilds itself: difficulty settling, abrupt awakenings, recurrent dreams, fear of sleep, and the next day's exhausted vigilance. Treating sleep as an adjunctive target is therefore not cosmetic. It may improve suffering and functioning even when the effect on the full PTSD syndrome is smaller.

The first discipline is to separate outcomes. Nightmare frequency, nightmare intensity, insomnia severity, objective sleep, daytime impairment, and clinician-rated PTSD are related but not identical. A 2024 network meta-analysis of twenty-four randomized trials with 1,647 participants found favorable estimates for CBT for insomnia on sleep quality and for imagery rehearsal therapy on nightmare severity; the reported standardized mean difference for IRT on nightmares was about -0.65, while networks and follow-up remained heterogeneous [Huang et al., 2024]. In a 2025 randomized trial of ninety-four veterans with diagnosed PTSD and insomnia, adding CBT-I before and alongside PE improved insomnia and quality of life more than sleep hygiene plus PE, but did not produce superior PTSD symptom reduction [Colvonen et al., 2025]. That is not a failed result. It is evidence that sleep and PTSD outcomes can dissociate.

Imagery rehearsal invites the person, while awake, to alter a recurrent nightmare's script and practice the new imagery. It does not require interpreting a dream as a hidden message, and it does not establish that the trauma memory itself has been reconsolidated. In a small thirty-one-person veteran trial, adding IRT to group CBT-I did not outperform CBT-I alone; both groups improved on several self-reports, objective actigraphy did not differ, and residual sleep difficulty remained [Prguda et al., 2023]. The intervention remains plausible and guideline-compatible, but the incremental effect is not guaranteed.

Prazosin illustrates why averages and subgroups must not be collapsed. A large multicenter trial randomized 304 veterans and found no advantage over placebo for distressing dreams, sleep quality, or global change at ten or twenty-six weeks [Raskind et al., 2018]. A 2025 meta-analysis of ten trials and 648 participants nevertheless found pooled benefits for nightmares and insomnia, around SMD $= -0.64$ and -0.65, while the global PTSD effect was nonsignificant [Mendes et al., 2025]. The VA/DoD guideline consequently suggests prazosin for PTSD-associated nightmares, not for global PTSD. Blood pressure, orthostasis, dizziness, fainting risk, and other medication interactions belong in the decision.

A 2026 trial of dronabinol in 171 adults reported a moderate improvement in nightmare severity. Adverse events occurred in 89.7% on dronabinol and 77.1% on placebo, while serious adverse events occurred in seven active-arm participants and none receiving placebo; long-term efficacy and safety remain unresolved [Roepke et al., 2026]. That single compound-specific result cannot be converted into evidence for cannabis as a class. A 2026 scoping review found that only one of seven cannabinoid RCTs showed clear clinical superiority, while favorable observational reports were highly vulnerable to bias [Aviram et al., 2026].

Sleep care also requires ordinary medicine: assess obstructive sleep apnea, circadian disruption, substances, medications, pain, and environmental interruption. A nightmare intervention is not a replacement for comprehensive PTSD care when intrusion, avoidance, altered mood and cognition, hyperarousal, or impaired function continue. But helping a person sleep can be a legitimate endpoint in its own right. A night with less fear is not merely a proxy; it is part of a life returning.

CORE SENTENCE

Sleep-focused treatment need not cure all of PTSD to matter; nightmares, insomnia, daytime function, and global PTSD should be measured separately and treated honestly.

90

Pharmacotherapy: Modest Averages and Individual Decisions

EMPIRICAL RESULT · AUTHORS' INTERPRETATION

Medication can help PTSD, but its average effects are modest and its role should not be inflated by familiarity. As of 6 September 2026, sertraline and paroxetine remained the only medications with a specific U.S. FDA indication for adult PTSD, as summarized in the VA clinician medication guide. Venlafaxine was guideline-supported when medication was chosen but remained off-label for PTSD in the United States. The current Cochrane review, updating the 2022 publication, covered sixty-six randomized trials and 7,442 participants and found that SSRIs increased treatment response relative to placebo—about 58% versus 35% in pooled data—with moderate-certainty evidence [Williams et al., 2026]. Another synthesis spanning 115 pharmacological studies estimated a small pooled SSRI symptom effect, SMD $= -0.28$ with a 95% confidence interval from -0.39 to -0.17 [Hoskins et al., 2021]. Response definitions, trial duration, and missing data differ, so neither number is an individual's forecast.

SSRIs and venlafaxine may reduce PTSD symptoms and can also address depression or anxiety. They do not erase a trauma memory, and benefit usually develops over weeks. Adverse effects can include gastrointestinal disturbance, activation or sedation, sleep change, sexual dysfunction, and discontinuation symptoms; age, pregnancy, bipolar-spectrum risk, suicidality, interactions, and medical conditions alter the decision. Medication choice, dose, continuation, tapering, and combination with psychotherapy require individualized clinical care.

Negative evidence belongs at the center, not in small print. In a six-month multisite trial that randomized 296 people with chronic military-related PTSD and included 247 in the primary analysis, risperidone augmentation did not significantly improve clinician-rated PTSD and produced more weight gain, fatigue, somnolence, and hypersalivation [Krystal et al., 2011]. A systematic review found no convincing PTSD efficacy for benzodiazepines and raised concerns about dependence, cognition, accidents, prognosis, and interference with psychotherapy [Guina et al., 2015]. VA/DoD recommends against benzodiazepines and cannabis or cannabis derivatives for treating PTSD. Acute sedation or relief is not the same outcome as durable recovery, and abrupt discontinuation can itself be dangerous.

Recent development programs show why the totality of evidence matters. Two trials of brexpiprazole plus sertraline reported clinician-rated advantages of roughly five to six CAPS-5 points in samples of 416 and 321 participants [Davis et al., 2025] [Hobart et al., 2025]. A larger fixed-dose phase 3 trial of 553 participants was null [Davis et al., 2025]. In 2025 an FDA advisory committee voted 10–1 that efficacy had not been established, and the sponsors subsequently reported a Complete Response Letter; the combination was not approved for PTSD by the cutoff. Selecting only the published positive trials would tell the wrong story.

Other compounds remain investigational. A 2025 phase 2b trial of the $\alpha 7$ nicotinic receptor modulator BNC210 randomized 182 people and reported a CAPS-5 difference of -4.03, $d = 0.40$, $p = .048$; adverse-event withdrawals were more frequent on active drug, and confirmatory replication was still needed [Papapetropoulos et al., 2025]. A borderline positive trial is an invitation to test, not a new standard of care.

The right contrast is not "natural" versus "medical," or psychotherapy versus medication as identities. It is expected benefit, burden, uncertainty, preference, comorbidity, access, and what will be measured next. Some people benefit substantially from medication; some do not; some prefer it; some cannot tolerate it. Modest average effects can be deeply meaningful to an individual, provided the average is not sold as certainty and nonresponse is noticed in time.

CORE SENTENCE

Medication is a legitimate, preference-sensitive route with modest average benefits; careful prescribing means honoring individual gain while keeping null trials, adverse effects, and alternatives in view.

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Nonresponse, Dropout, Adverse Events and Access

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE

Evidence-based does not mean universally effective, universally completed, or universally available. A 2024 meta-analysis of eighty-six randomized trials with 7,894 participants estimated that 39.2% remained nonresponsive after first-line psychological treatment. Heterogeneity was high, half the studies were judged at high risk of bias, and definitions differed [Semmlinger et al., 2024]. The number therefore cannot be placed over a person as destiny. It does establish a moral and scientific obligation: we need plans for what happens when the first treatment does not work.

"Nonresponse" itself can hide several different events. The treatment may have been inaccessible, culturally or practically mismatched, delivered with insufficient fidelity, stopped because life became unmanageable, or completed without sufficient benefit. The outcome threshold may be too crude. Symptoms may improve while sleep or functioning remains impaired; depression may lift while intrusion persists; diagnosis may be lost without a felt return to life. A 2026 review found major inconsistency across fifty-five studies in definitions of treatment-resistant PTSD and argued for repeated reassessment of delivery, adherence, comorbidity, and outcome rather than premature assignment to a fixed refractory type [Al-Shamali et al., 2026].

Dropout is equally ambiguous. Leaving can reflect burden, avoidance, symptom activation, work and caregiving, transport, cost, poor alliance, poor fit, improvement, or harm. It should neither be dismissed as patient failure nor automatically counted as treatment toxicity. Intent-to-treat and completer effects answer different questions. If $Y_i(a)$ is person i's outcome under assignment a and $C_i(a)$ indicates completion, then

$$\theta_{\text{ITT}} = \mathbb{E}[Y(1) - Y(0)]\,, \qquad \theta_{\text{comp}} = \mathbb{E}[Y(1) - Y(0) \mid C(1) = C(0) = 1]\,.$$

The second quantity concerns an unobserved principal stratum and cannot be recovered by simply deleting people who left. Per-protocol success can look large because the people able to continue are selected by circumstances and response.

Harms are harder to estimate than benefits because psychotherapy trials have often measured them inconsistently. A 2026 pediatric review located seventy-two RCTs with 5,677 participants, but only six reported PTSD deterioration and nineteen reported adverse events. Available estimates did not indicate excess harm and were reassuring for trauma-focused CBT, yet sparse reporting meant that uncommon events were not ruled out [Hoppen et al., 2026]. In adults, temporary distress during trauma processing is expected; persistent deterioration, suicidality, dissociation, substance escalation, sleep collapse, or loss of function require active monitoring rather than being normalized as proof that treatment is "working."

Access is part of effectiveness. A systematic review of CPT and PE implementation found that training plus audit and feedback improved clinician attitudes and availability but did not produce consistent routine use; referral complexity, perceived inflexibility, competing needs, and patient complexity remained barriers [Ackland et al., 2023]. A 2026 review of measurement-based PTSD care found only fifteen eligible studies, all U.S.-based and eleven veteran-focused, with obstacles including irregular measures, technology problems, and poor explanation of why monitoring matters [Al-Shamali et al., 2026]. Asking a score without sharing its meaning is data extraction, not collaborative care.

The humane response to nonresponse is curiosity with structure. Verify the diagnosis and present danger. Ask what changed, what did not, and what the treatment cost. Examine fidelity without blaming either party. Address sleep, substance use, pain, moral injury, dissociation, depression, safety, and social conditions. Then adapt, switch, combine, or pause according to evidence and preference. A treatment can fail a person; that does not mean the person failed treatment.

CORE SENTENCE

Nonresponse is a signal to reassess the route, the fit, the delivery, and the outcome—not a verdict that the person is unreachable.

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TMS, Theta-Burst, tDCS and Neurofeedback

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Neuromodulation is attractive because it appears to touch the system directly. But "TMS for PTSD" is not one intervention. Target, coil, laterality, frequency, intensity, number and spacing of sessions, concurrent task, medication, symptom measure, and comparator all change the experiment. Deep TMS, intermittent theta-burst stimulation, accelerated schedules, transcranial direct-current stimulation, EEG neurofeedback, and real-time fMRI feedback should not be merged into a technological class effect.

For repetitive TMS, the most rigorous summaries remain cautious. A 2024 Cochrane review contained thirteen randomized trials with 577 participants; its primary clinician-rated estimate came from only three studies and 99 participants and was small and nonsignificant, SMD $= -0.14$ with a 95% confidence interval from -0.54 to 0.27 [Brown et al., 2024]. A 2026 meta-analysis of sixteen trials and 786 participants found large pooled self-report changes but nonsignificant clinician-interview effects, wide prediction intervals, sparse durability, and low certainty [Rodríguez-Drincourt-de-Elízaga et al., 2026]. Another 2026 combat-related synthesis showed the familiar gap between large uncontrolled within-group change and an imprecise sham-controlled clinician-rated difference [Virto-Farfan et al., 2026]. Protocol-specific signals still matter: a seventy-five-person iTBS trial favored active protocols over sham [Yuan et al., 2023], and a 119-person trial adding navigated TMS to residential PE reported modest clinician-rated benefit that now requires independent replication outside that intensive setting [Fox et al., 2026].

Two 2026 FDA device decisions must be stated exactly. On 8 May, FDA granted De Novo classification DEN250013 to Modius Spero, a Class II prescription home-use device for symptoms associated with PTSD in adults aged 22 and older, to be used with a comprehensive treatment plan rather than as stand-alone therapy. A twelve-week, double-blind sham-controlled trial randomized 383 adults described as having diagnosed, at least moderately severe PTSD. PCL-5 scores fell 23.5 points with vestibular-system stimulation and 12.1 with sham; discontinuation was equal, and 9% reported mild dizziness with no group difference [Colvonen et al., 2026]. The primary endpoint was self-report, the manufacturer sponsored and funded the investigator-initiated study and helped design it, and durability beyond twelve weeks remains unestablished.

On 3 June 2026, FDA cleared the MeRT system through 510(k), K260402, as an adjunctive device for adults with PTSD. Its pivotal summary described 158 randomized participants and standardized effects of approximately -0.32 on PCL-5 and -0.51 on CAPS-5. The intention-to-treat population comprised 77 active and 81 sham participants; 84% completed treatment, and no serious adverse events were reported. Clearance is not drug approval, not evidence of superiority to first-line psychotherapy, and not proof that EEG-personalized alpha frequency is the active ingredient: the trial tested the bundled system rather than personalization against fixed-frequency TMS. Follow-up analyses were post hoc, and independent durability data remained limited at the cutoff.

tDCS evidence is thinner. In a 2024 trial of fifty-four predominantly male participants, tDCS paired with virtual-reality exposure produced a self-rated PTSD advantage at one month, about $d = -0.82$, but no quality-of-life advantage; blinding, pairing, and generalizability constrain the claim [Wout-Frank et al., 2024]. A positive combined intervention does not show what stimulation alone contributed.

Neurofeedback shifts the language from stimulation to learned self-regulation, yet the evidential problem is similar. A thirty-eight-person EEG-neurofeedback trial reported effects around $d = 0.77$ after treatment and 0.75 at three months [Nicholson et al., 2023]. A 2025 meta-analysis concluded that moderate-to-large estimates came mainly from passive-control studies and rated certainty very low to low [Berman et al., 2025]. Real-time fMRI feedback can demonstrate target engagement: in a double-blind twenty-five-person trial, participants learned aspects of amygdala regulation, but symptoms were not clearly superior to control feedback [Zhao et al., 2023]. Target engagement is necessary for some mechanistic claims; it is not sufficient for clinical benefit.

Across these technologies, common adverse effects are usually transient headache, scalp discomfort, tingling, or fatigue, while seizure risk with TMS is rare under appropriate protocols. Small studies cannot characterize rare harms well. Cost, repeated visits, device quality, operator training, blinding integrity, and access also belong in the benefit-risk equation. Neuromodulation may eventually change when and how corrective learning becomes possible. For now, the strongest statement is protocol-specific: some signals are promising, two devices have PTSD-related U.S. marketing authorization with different indications and evidence bases, and no frontier device has displaced established trauma-focused care.

CORE SENTENCE

Neuromodulation should be judged protocol by protocol—by sham-controlled clinical outcomes, durability, harms, and replication—not by the persuasive aura of acting directly on the brain.

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Ketamine, MDMA and Psychedelic Frontiers

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Fast-acting and experience-altering compounds have reopened a serious question: can a temporary change in plasticity, fear, affect, or interpersonal openness make therapeutic updating more possible? The question deserves rigorous study. It does not license a single "psychedelic treatment" category, because ketamine, MDMA, psilocybin, and methylone differ pharmacologically, experientially, procedurally, and evidentially. The therapy, preparation, setting, expectancy, masking, and post-session support may be part of the intervention rather than scenery.

Ketamine evidence is mixed. In a thirty-person active-controlled repeated-infusion trial, ketamine produced a CAPS advantage of 11.88 points over midazolam, with response in 67% versus 20% and an effect around $d = 1.13$; among responders, median time to loss of response was only 27.5 days [Feder et al., 2021]. A much larger multisite dose-ranging trial in 158 veterans and active-duty personnel found no PTSD-specific benefit, although the standard dose improved depression [Abdallah et al., 2022]. Dissociation, blood-pressure elevation, nausea, acute perceptual effects, and misuse potential require supervised delivery. Ketamine is not FDA approved for PTSD. Intranasal esketamine's approval for certain depressive conditions does not transfer to PTSD.

"Ketamine-assisted psychotherapy" adds another uncertainty: which component, sequence, and interaction matter? An open-label 2025 study combining ketamine with WET enrolled fourteen people and analyzed thirteen; large within-person change and 69% response were encouraging, but no control condition could separate drug, writing treatment, expectancy, attention, or spontaneous change [Feder et al., 2025]. The often-invoked "plasticity window" is a plausible bridge hypothesis until timing and interaction are directly manipulated.

MDMA-assisted therapy has the strongest psychedelic-adjacent PTSD trial program and the most important regulatory caution. MAPP1 randomized ninety people with severe PTSD and reported a large advantage for MDMA with manualized therapy [Mitchell et al., 2021]. MAPP2 randomized 104 people; mean CAPS-5 change was -23.7 versus -14.8, approximately $d = 0.7$, with a disability effect around $d = 0.4$. Seven participants had a severe treatment-emergent adverse event—five in the MDMA-assisted arm and two in the placebo-with-therapy arm—but the paper reported no serious treatment-emergent adverse events or deaths [Mitchell et al., 2023]. Functional unblinding is difficult when subjective drug effects are conspicuous; expectancy, therapist conduct, the psychotherapy component, sponsor-network dependence, rare harms, and controlled long-term durability remained concerns. FDA issued a Complete Response Letter on 8 August 2024 and did not approve the application. A stakeholder reported resubmission in August 2026, but no FDA approval had occurred by 6 September 2026. Resubmission is a procedural event, not new efficacy evidence.

For psilocybin, direct PTSD evidence remained very early. A 2026 open-label phase 2 study of twenty-two participants reported roughly thirty-point CAPS-5 reductions at weeks four and twelve. Without randomization or control, regression, expectancy, selection, and clinician contact cannot be separated. There were 117 treatment-emergent adverse events, mostly on dosing day, with no serious events or withdrawals reported [McGowan et al., 2026]. Evidence from depression cannot be imported as PTSD efficacy. A distinct investigational entactogen, TSND-201 or methylone, showed a positive six-week signal in a sixty-five-person randomized phase 2 trial: the least-squares mean difference was -9.64 CAPS-5 points (90% confidence interval, -16.48 to -2.80; $p = .01$), favoring TSND-201. It was administered without psychotherapy, although sessions were monitored nondirectively by mental-health professionals. Headache, appetite reduction, nausea, dizziness, blood-pressure increase, dry mouth, and insomnia were reported [Jones et al., 2026]. Replication and longer safety follow-up are essential.

These approaches may eventually help people not reached by current care. Their study must include active comparators, expectancy measurement, masking assessment, independent replication, functional outcomes, long follow-up, and factorial designs capable of separating drug, psychotherapy, and interaction. Hope is allowed. Evidential shortcuts are not.

CORE SENTENCE

Altered-state and rapid-plasticity treatments may open possibilities, but published promise, mechanism stories, and regulatory approval are three different things.

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Stellate Ganglion Block, Propranolol and D-Cycloserine

EMPIRICAL RESULT · AUTHORS' INTERPRETATION · BROADER SCIENTIFIC INFERENCE

Three interventions illustrate three different attempts to intervene at leverage points: reduce sympathetic output, disrupt adrenergic memory reconsolidation, or amplify learning during exposure. Each begins with a coherent mechanism. None has earned first-line status.

Stellate ganglion block injects local anesthetic near the cervical sympathetic chain, with the aim of reducing persistent hyperarousal. A multisite sham-controlled trial of 113 active-duty service members—80% of whom met CAPS-5 diagnostic criteria, with the remainder at a symptom threshold—reported an eight-week CAPS change of -12.6 after SGB versus -6.1 after sham [Olmsted et al., 2020]. Yet a 2016 randomized double-blind trial was negative [Hanling et al., 2016], and a 2026 pilot adding SGB to CPT found no significant treatment interaction in thirty-nine participants [Held et al., 2026]. Procedural risks include vascular or nerve injury, intravascular injection and seizure, pneumothorax, infection, and temporary Horner syndrome. A reduction in hyperarousal would be valuable, but it is not automatically full-disorder recovery. Credible sham, diagnostic rigor, longer follow-up, and independent replication remain necessary.

Propranolol paired with trauma-memory reactivation attempts to weaken adrenergic restabilization after a retrieved memory becomes labile. A sixty-person randomized trial reported an approximately 11.5-point symptom advantage for propranolol before reactivation [Brunet et al., 2018]. A later sixty-six-person trial found that both reactivation groups improved but detected no drug-specific advantage through three months [Roullet et al., 2021]. A seven-study 2025 meta-analysis, which mixed prevention and treatment paradigms, reported a statistically significant pooled improvement ($Z = 2.32$, $p = .02$, $I^2 = 0\%$) and characterized the evidence as preliminary [Li et al., 2025]. That estimate and the conflicting direct reactivation trials do not establish a robust general treatment effect. Reactivation itself, repeated narration, expectancy, or context may account for change. Most clinical studies infer memory destabilization; they do not measure it. Bradycardia, hypotension, and bronchospasm risk make medical screening more than a formality.

D-cycloserine is a partial NMDA-receptor agonist intended to enhance learning near an exposure session. The appealing phrase is "extinction enhancer," but the biology is less obedient. If the learning update is idealized as

$$\Delta w_t = \eta_t \, \delta_t, \qquad \delta_t = r_t - \hat{r}_t,$$

then w_t is an associative weight, δ_t is prediction error, and η_t is the learning rate. Increasing η_t does not specify what is learned. A well-designed session that violates danger expectations might be strengthened; an overwhelming or prematurely ended session could strengthen threat or escape learning. Pharmacological enhancement is conditional on the experience it enhances.

The clinical results fit that warning. In a 156-person virtual-reality exposure trial, D-cycloserine did not improve the primary PTSD outcome; exposure gains persisted, but secondary signals appeared dependent on session learning [Rothbaum et al., 2014]. A 192-person randomized study also found no primary augmentation advantage [Difede et al., 2022]. A transdiagnostic meta-analysis across anxiety disorders, obsessive-compulsive disorder, and PTSD estimated a small, nonsignificant overall effect of about $g = 0.15$ [McGuire et al., 2017]. The same 156-person study found that alprazolam during exposure was associated with a higher PTSD diagnosis rate at three months than placebo, a sobering signal that immediate anxiolysis and durable corrective learning can diverge [Rothbaum et al., 2014].

Mechanistic elegance is valuable because it generates tests. It becomes dangerous when it substitutes for them. These interventions should be evaluated by incremental benefit over credible care, not by whether their biological story sounds precise. If their effect depends on arousal state, prediction error, timing, or responder phenotype, those conditions must be prospectively measured and validated.

CORE SENTENCE

A plausible leverage point is not yet a reliable treatment; sympathetic blockade, reconsolidation interference, and learning enhancement must each survive controlled tests of what actually changes.

95

Virtual Reality, Digital Care, AI and Ecological Intervention

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE · NEW HYPOTHESIS

Technology can change where treatment happens, what can be measured, and when support arrives. It does not repeal the need for a diagnosis, a credible mechanism, a comparison condition, privacy, human responsibility, or evidence of benefit. ”Digital” is a delivery property, not a treatment mechanism.

Virtual-reality exposure can present controllable multisensory cues when imaginal access is difficult. The active scientific idea remains exposure and new learning, not immersion itself. Randomized comparisons have not established general superiority over standard exposure [McLay et al., 2017], while a 2022 meta-analysis supported symptom improvement but found heterogeneity in comparators, hardware, content, and study size [Heo & Park, 2022]. Cybersickness, overactivation, dissociation, cost, and the possibility that a standardized virtual world fits the developer better than the patient all matter. The question is whether VR improves reach, engagement, or learning for a specified person—not whether it looks more advanced.

Selected guided digital programs have a stronger direct evidence base than autonomous PTSD apps, although that does not establish guidance itself as the active ingredient. An umbrella review of eleven meta-analyses found benefit for CBT-based digital interventions, though durability and the added value of guidance remained uncertain [Tng et al., 2024]. A 2025 randomized trial in ninety-five Chinese adults meeting full or subthreshold PTSD criteria found that clinician-guided online WET outperformed minimal contact at one month, with clinician-rated and self-report effects around $d = -0.74$ and -0.79. Improvement persisted within the intervention group to six months, but the controlled comparison ended at one month [Li et al., 2025]. Two 2026 pragmatic trials of a self-guided intervention for Syrian refugees—selected for distress and functional impairment rather than diagnosed PTSD, with PTSD symptoms a secondary short-form outcome—were null in intention-to-treat analyses, with dropout of roughly 86.3% and 82.1% [Burchert et al., 2026]. Scalability without use is not effectiveness. Language, culture, data cost, housing, safety, and trust are causal parts of implementation.

Direct evidence for autonomous, trauma-focused AI treatment in a diagnosed PTSD cohort remained absent at the cutoff. A 2026 three-arm trial randomized 995 psychologically distressed Israeli university students to a conversational AI platform, face-to-face group therapy, or waiting list. PTSD was not an inclusion diagnosis, and self-reported PCL-5 symptoms did not differ among groups after twelve weeks or three months [Shoshani et al., 2026]. Earlier publications described possible generative-AI use cases or compared decisions on clinical vignettes; they were not patient-outcome trials [Held et al., 2025] [Wislocki et al., 2025]. AI may assist psychoeducation, translation, documentation, supervision, triage, or clinician decision support. Autonomous trauma processing is a much stronger claim. Hallucination, privacy loss, bias, manipulative attachment, false reassurance, and failure during crisis require explicit governance, consent, audit trails, human oversight, and escalation routes.

Ecological momentary assessment and wearables offer a different possibility: observe state close to life rather than reconstructing a week from memory. A hyperarousal-event study reached a participant-separated AUC $= 0.70$, but used self-reported event labels during unusual multiday cycling events [Sadeghi et al., 2022]. In a 1,618-person emergency-department cohort, watch-only discrimination of probable PTSD was near chance, AUC $= 0.56$, and did not improve the survey model [Cakmak et al., 2021]. Prediction is not prevention. A just-in-time intervention can be written as a policy

$$A_t \sim \pi_\theta(\cdot \mid \mathcal{H}_t), \qquad \mathcal{H}_t = \{\mathbf{y}_{0:t}, \mathbf{c}_{0:t}, A_{0:t-1}\},$$

where $\mathbf{y}_{0:t}$ contains the observed symptom and physiological measurements, $\mathcal{H}_t$ is the person’s observed history of those measurements, context, and prior support, and A_t is the prompt or intervention offered now. The therapeutic question is not classifier accuracy alone. It is the proximal causal effect of offering A_t given history $\mathcal{H}_t$, balanced against false alarms, missed events, interruption, battery burden, and surveillance.

This is a Flow Hijacked hypothesis: a low-burden intervention delivered before a defensive transition fully develops may sometimes require less control energy than an intervention delivered at its peak. It demands micro-randomized trials, patient-important outcomes, and the option to turn sensing off. Continuous monitoring can itself intensify vigilance. A system designed to restore freedom must not make the person feel watched by their own treatment.

CORE SENTENCE

Technology earns a place in PTSD care when it improves learning, access, timing, or choice—not when novelty disguises attrition, weak evidence, or surveillance.

96

Precision Treatment and Adaptive Sequencing

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE

Precision care is often imagined as a baseline scan that selects the perfect therapy. The more realistic near-term version is humbler and more useful: make a good first choice, measure what happens, and adapt before ineffective care becomes inertia. Baseline moderators may help, but repeated response data often contain more information than a single prediction made before treatment begins.

Formally, a dynamic treatment regime is a sequence $d = (d_1, \dots, d_T)$ in which

$$A_t = d_t(\mathcal{H}_t), \qquad V(d) = \mathbb{E}\left[\sum_{t=0}^{T} \gamma^t r_t \,\middle|\, d\right],$$

where $\mathcal{H}_t$ contains the person's observed history up to decision time t, A_t is the next treatment action, r_t combines benefit, function, burden, and harm, and $0 < \gamma \leq 1$ determines how strongly durable outcomes are valued. A Sequential Multiple Assignment Randomized Trial, or SMART, can compare decision rules by re-randomizing people at defined response points. It tests a sequence, not merely a product.

A 2025 pragmatic trial gives rare direct evidence. Seven hundred primary-care patients were assigned to sequences beginning with an SSRI or WET. Initial strategies did not differ significantly overall. Among 122 of 295 people assigned to an SSRI who met the trial's nonresponse criterion, switching to venlafaxine produced a 9.2-point PCL-5 reduction compared with 2.3 points after WET augmentation; the adjusted between-group difference was 10.19 points, with a 95% confidence interval from 4.97 to 15.41 [Fortney et al., 2025]. This is not a universal rule that medication switching beats psychotherapy. It is a sequence-specific result in a pragmatic population, and treatment engagement and fidelity were imperfect. It demonstrates why the second decision deserves randomization rather than intuition.

Another 2026 SMART enrolled 477 hematopoietic-cell-transplant survivors with probable or subthreshold cancer-related post-traumatic stress. A self-guided app did not initially outperform usual care; among nonresponders, sequences escalating to therapist-delivered CBT produced stronger and more sustained improvement than app coaching [Smith et al., 2026]. The lesson is not "apps fail." It is that a low-intensity entry point may be useful only when the system reliably recognizes nonresponse and opens the higher-intensity route.

Prediction algorithms remain earlier. A personalized-advantage analysis in ninety-two people with complex PTSD retrospectively estimated a large clinician-rated advantage for model-matched PE or STAIR, about $d = 0.83$, but used many candidate predictors in a small dataset and lacked prospective external validation [Frost et al., 2025]. In intensive CPT programs, continuously updated models reached roughly $R^2 = 0.62$ midway through treatment in a training sample of 362 and validation sample of 108, yet complex machine-learning methods did not outperform linear mixed models [Smith & Held, 2023]. For webSTAIR, machine learning and baseline-severity-only models explained similar variance in 189 veterans [Hallenbeck et al., 2024]. Complexity is not accuracy.

Prediction is not prescription. Prognostic models estimate outcome risk under observed care; treatment-selection models must estimate a treatment-by-person interaction. Clinical utility then requires a prospective trial showing that using the model improves outcomes over good shared decision-making, without worsening inequity. Calibration across trauma types, languages, sex and gender, race and ethnicity, comorbidity, ongoing threat, and service settings must be tested. A precision system should reveal uncertainty, allow refusal, and make switching easier. Its purpose is not to declare what kind of patient someone is. It is to reduce time spent on a route that is not opening.

CORE SENTENCE

The most credible precision care is adaptive and revisable: choose carefully, measure together, and change the sequence before nonresponse hardens into abandonment.

97

Treatment as a Dynamical Control Problem

FLOW HIJACKED SYNTHESIS · NEW HYPOTHESIS

We can now return to the central Flow Hijacked question. If PTSD involves altered rules for entering, sustaining, and leaving defensive states, treatment may be understood—not literally reduced—as an effort to change those rules. Control here never means controlling the person. It means helping a person regain influence over a system that has become too easily recruited by threat and too slow to release them.

Let $\mathbf{x}_t \in \mathbb{R}^n$ denote a latent brain-body-person state, $\mathbf{c}_t$ the context, $\mathbf{u}_t = \mathbf{u}(t) \in \mathbb{R}^m$ a treatment input, and $\mathbf{W}_t \in \mathbb{R}^r$ a standard Wiener process. A continuous-time model is

$$d\mathbf{x}_t = \mathbf{f}_\theta(\mathbf{x}_t, \mathbf{c}_t)\, dt + \mathbf{B}_\theta(\mathbf{x}_t, \mathbf{c}_t)\mathbf{u}_t\, dt + \mathbf{\Sigma}_\theta(\mathbf{x}_t, \mathbf{c}_t)\, d\mathbf{W}_t.$$

The vector field $\mathbf{f}_\theta$ describes untreated tendencies; $\mathbf{B}_\theta(\mathbf{x}_t, \mathbf{c}_t) \in \mathbb{R}^{n\times m}$ describes where an intervention has leverage; and $\mathbf{\Sigma}_\theta(\mathbf{x}_t, \mathbf{c}_t) \in \mathbb{R}^{n\times r}$ maps stochastic variation into the latent coordinates. Threat-State Return Asymmetry predicts that, for some people and contexts, treatment response should be visible not only as a lower mean symptom score but as a higher entry threshold, smaller cue-specific transient gain, narrower generalization, shorter defensive dwell time, faster recovery, and greater access to safe engagement.

A treatment policy could be defined by

$$\mathbf{u}^\star(\cdot) = \underset{\mathbf{u}(\cdot)\in\mathcal{U}_{[0,T]}}{\arg\min}\, \mathbb{E}\left[\Phi(\mathbf{x}_T) + \int_0^T \left(q(\mathbf{x}_t, \mathbf{c}_t) + \tfrac{1}{2}\mathbf{u}(t)^\top \mathbf{R}\mathbf{u}(t) + \lambda h(\mathbf{u}(t))\right) dt\right].$$

Here $\mathcal{U}_{[0,T]}$ is the set of inputs that are progressively measurable with respect to the observed-data filtration and satisfy prespecified dose, timing, consent, stopping, and safety constraints. The function q penalizes suffering and lost function; $\mathbf{R} \in \mathbb{R}^{m\times m}$ with $\mathbf{R} \succ 0$ represents treatment burden; $h \geq 0$ represents adverse-effect burden or soft constraint violations; $\lambda \geq 0$ weights that burden; and Φ values durable future accessibility. The optimal path is therefore not the largest possible intervention. It is the least burdensome safe sequence that produces durable life-relevant change.

Different treatments suggest different control hypotheses. PE may raise transition thresholds and widen access to safe contexts through approach and inhibitory learning. CPT may reduce the precision of threat, blame, or shame priors that feed the state back into itself. Sleep treatment may reduce overnight re-entry and improve next-day recovery. Medication may lower background gain or comorbid load. Neuromodulation may temporarily change state accessibility; timing it to corrective learning may matter more than stimulation alone. Ketamine or entactogen-assisted work may open a time-limited learning window. Adaptive care may observe response and change the input rather than repeating what is not working. These are hypotheses, not established mappings.

There are direct anchors, but they are still bridges. Successful cognitive therapy has been associated with changed occupancy of large-scale brain-network states in a small longitudinal sample [Charquero-Ballester et al., 2022]. EEG neurofeedback studies report neural and symptom change, yet rigorous active-control evidence remains thin [Nicholson et al., 2023]. fMRI-guided TMS changed amygdala threat reactivity without immediate symptom superiority [van Rooij et al., 2026]—exactly the kind of target-engagement/clinical-outcome dissociation a control model must explain. A two-person implanted closed-loop study showed that amygdala-theta-triggered stimulation was technically feasible, not that it was efficacious [Gill et al., 2023]. Adaptive sequencing has clinical evidence without measuring neural controllability [Fortney et al., 2025].

No trial has yet shown that PTSD is a single attractor, directly estimated basin depth, or demonstrated restored controllability as the cause of recovery. The model earns its place only if it predicts more than metaphor: dense pre/post state measurements should reveal altered transition matrices beyond symptom averages; change should generalize across real contexts; dynamics should mediate function; and rival models should be tested. If those predictions fail, the language must be revised or abandoned.

The promise is not that mathematics makes treatment impersonal. It can make our obligations clearer. We must measure where the system goes, how quickly it returns, what the intervention costs, and whether more of life has become reachable. The person is not the plant in a controller diagram. The person is the one whose possible futures the model is required to serve.

CORE SENTENCE

In the Flow Hijacked hypothesis, treatment succeeds when it does more than suppress symptoms—it changes the transition rules so safer, fuller states become easier to enter and easier to remain in.

PART XII

RECOVERY AS REOPENED POSSIBILITY

Recovery may begin when other states and futures become reachable again.

98

Recovery Is Not Forgetting

BROADER SCIENTIFIC INFERENCE

Recovery is often described as though the task were to erase an event, silence every alarm, and restore an earlier version of the self. That is neither a defensible account of memory nor a humane standard for a life. A person can remember clearly and suffer less. A body can still become activated and recover more readily. A difficult anniversary can remain difficult without governing the year around it. Recovery is therefore not the absence of every reaction. It is an altered relation among memory, present context, action, and time.

Clinical studies usually operationalise recovery through a reduction in symptoms, loss of diagnostic status, remission, or improved functioning. These outcomes overlap, but they are not interchangeable. A questionnaire may fall below a threshold while sleep, intimacy, work, or trust remain impaired; conversely, a person may still report symptoms while rebuilding a life of meaning and participation. Session-level analyses also show that symptoms and functioning influence one another in complex, time-dependent ways rather than moving as a single quantity [Benfer et al., 2024]. A responsible lecture must therefore resist turning one score into a verdict on the whole person.

Nor does improvement guarantee a permanently monotone path. A systematic review of PTSD recurrence found that studies used such inconsistent definitions, intervals, and denominators that a pooled recurrence estimate was not justified [Brooks & Greenberg, 2024]. That uncertainty matters. It means we should neither promise that distress can never return nor mistake a later activation for the erasure of all previous progress. Re-emergence under a new loss, threat, illness, legal process, or public reminder may reflect a changed context interacting with a still-plastic system. It is information to understand, not proof of personal failure.

For people whose danger continues, the word recovery requires further care. It cannot mean learning that an unsafe environment is safe. It may mean increasing discrimination among kinds and moments of danger, protecting sleep where possible, recovering choice within constraint, reducing unnecessary carry-over, and sustaining connection without denying reality. The target is not indiscriminate calm. It is more accurate and more flexible regulation.

In the Flow Hijacked frame, recovery is best treated as renewed access. The traumatic event need not become insignificant. Instead, defensive states cease to possess exclusive rights over attention, prediction, physiology, and action. Memory becomes one source of evidence among others; the present acquires weight again; futures that were once computationally inaccessible become thinkable and livable. That is a more demanding definition than forgetting, but also a more possible one.

CORE SENTENCE

Recovery does not require the past to disappear; it requires the present and the future to become influential again.

99

Recovery Trajectories and Flexibility

EMPIRICAL RESULT · BROADER SCIENTIFIC INFERENCE

There is no single post-traumatic trajectory. Prospective cohorts repeatedly identify heterogeneous courses: persistently low symptoms, early elevation followed by decline, slower recovery, delayed worsening, and chronic high symptom burden. In the Jerusalem Trauma Outreach and Prevention Study, one modelling solution identified rapid-remitting, slow-remitting, and non-remitting symptom trajectories across fifteen months [Galatzer-Levy et al., 2013]. A six-year injury cohort likewise found several long-term patterns rather than one inevitable progression [Bryant et al., 2015]. These findings are important because they replace fatalism with variation. They do not, however, license a new mythology in which every individual belongs to a fixed hidden type.

Trajectory classes are statistical summaries, not natural species. Their number and apparent prevalence can change with the scale used, assessment schedule, missing-data assumptions, sample composition, and model specification. A review and statistical evaluation showed how sensitive resilience and dysfunction classes can be to analytic choices [Galatzer-Levy et al., 2018]. Machine-learning work has reproduced broad trajectory structure across two injury samples while failing to transport the clinically crucial distinction between remitting and non-remitting courses reliably [Tomas et al., 2022]. The science supports heterogeneity more strongly than it supports precise early assignment.

Flexibility offers a different question. Instead of asking only, “Which curve will this person follow?”, we can ask how sensitively the system changes policy when the evidence changes. Can attention widen after the cue is evaluated? Can arousal rise for protection and then fall? Can a person select different regulatory strategies across situations rather than using one response everywhere? Performance-based work has reported reduced flexibility in the selection of emotion-regulation strategies in high post-traumatic stress groups, but this remains one component of a much larger construct [Fine et al., 2023]. Resilience itself is outcome-, domain-, and time-specific: functioning at work can coexist with severe sleep disturbance, and outward stability can coexist with physiological or relational cost [Bonanno, 2021].

This distinction changes how we listen to recovery. A falling average symptom score is valuable, but it may conceal brittle stability. A person who functions only by eliminating uncertainty, avoiding all reminders, or maintaining continuous activation may appear well until circumstances change. Conversely, an uneven course may contain genuine adaptation if episodes shorten, recovery accelerates, choice expands, and important activities return. Variability is not automatically dysregulation; stability is not automatically health. The meaning depends on context, scale, and consequence.

The most scientifically cautious conclusion is also the most hopeful: neither acute distress nor an early risk score is destiny. Early burden matters, and some courses are painfully persistent, but trajectories are shaped across time by exposure, biology, treatment, sleep, resources, relationships, and new learning. Recovery is better understood as an evolving capacity for context-sensitive change than as membership in a “resilient” category.

CORE SENTENCE

The strongest trajectory finding is not that people fall into fixed types, but that post-traumatic courses are heterogeneous and remain open to change.

100

Safety Learning in Real Worlds

BROADER SCIENTIFIC INFERENCE

Laboratory extinction is deliberately spare: a cue that once predicts an aversive outcome is presented repeatedly without that outcome. The procedure reveals something essential—new safety learning can coexist with older threat learning—but a real life is never a quiet laboratory chamber. Streets, bodies, voices, news alerts, smells, dates, authority figures, and relationships arrive in combinations. The relevant question is not whether fear can fall in one setting. It is whether learning can travel across settings without becoming dangerously indiscriminate.

Reviews of fear-extinction research provide a serious mechanistic rationale, but the empirical pattern is not uniform [Lokshina et al., 2023]. A multiverse analysis found that PTSD-versus-trauma-exposed-control results for extinction retention changed with defensible analytic decisions and were mostly nonsignificant [Lewis et al., 2023]. Just as importantly, a large preregistered 2026 analysis reported no fear-conditioning or extinction differences between trauma-exposed people with and without clinically diagnosed PTSD on its selected measures [Davidson et al., 2026]. A serious model must hold all of these results. Laboratory learning alterations may characterise some people, tasks, phases, or measurement channels; they are not a universal essence of PTSD.

Safety in the world is learned through discrimination. The organism must discover not only that "nothing bad happened," but which cue, in which place, with which person, under which conditions, now carries a different probability. A treatment room can provide powerful new evidence, yet renewal outside that room is possible because extinction memories are context-sensitive. This is one reason practice across settings, times, internal states, and forms of action may matter. The aim is not maximal exposure to everything. It is sufficiently precise contact with what has been avoided, at a dose and pace that permits observation, updating, and agency.

Behavior supplies evidence too. Approaching a valued place, remaining present during a tolerable rise in arousal, testing a feared prediction, or accepting support can create outcomes unavailable under total avoidance. But context must remain sovereign. If a cue genuinely predicts danger, protective action is not a cognitive error. If social power is unsafe, trust cannot be prescribed as an internal exercise. Safety learning becomes credible when the environment participates—through predictability, consent, material protection, accountable institutions, and reliable people.

Flow Hijacked therefore treats generalisation as a two-sided problem. Threat may spread too broadly, but "safety" can also be generalised recklessly by outsiders who do not bear the risk. Recovery requires calibrated boundaries: narrower false alarms without blunting warranted defense; broader access to ordinary life without demanding naïveté. The measurable outcome is not fearlessness. It is a better match between response and present evidence, followed by a more reliable return when mobilisation is no longer needed.

CORE SENTENCE

Real-world safety learning is not the command to feel safe; it is the gradual recovery of accurate discrimination, agency, and return.

101

Sleep, Body and Social Ecology as Conditions of Return

BROADER SCIENTIFIC INFERENCE

Psychological treatment does not occur in an empty nervous system. It occurs in a body that has slept or not slept, eaten or not eaten, moved or remained braced, been accompanied or isolated, and encountered a world that may be stable or repeatedly disruptive. These conditions do not reduce PTSD to lifestyle. They change the background against which learning, attention, memory, and regulation operate.

Sleep is especially important because it is both an outcome and a possible contributor. Nightmares and fragmentation can repeatedly return the organism to defensive states; insufficient or unstable sleep can impair attention and emotion regulation the following day. Following mass trauma, wearable-derived circadian instability—particularly instability in sleep timing—prospectively predicted later PTSD symptom burden beyond initial symptoms in one study, but the design was observational and lacked a pre-trauma sensor baseline [Magal et al., 2026]. That result is a promising risk signal, not proof that stabilising a wearable metric will prevent PTSD.

Autonomic measures deserve the same disciplined interpretation. A 2026 meta-analysis found lower average vagally mediated heart-rate variability in PTSD, yet the magnitude depended strongly on whether recordings lasted five minutes or twenty-four hours [Tucker et al., 2026]. In a naturalistic ambulatory study, groups did not differ in mean physiology; within the PTSD group, reminders coincided with faster heart rate, while other autonomic channels did not follow the expected pattern [Wisco et al., 2025]. There is no single "traumatised body number." Bodily regulation is dynamic, task-dependent, and shaped by medication, health, respiration, movement, age, and measurement protocol.

Relationships are not merely comforting additions to an otherwise individual mechanism. A longitudinal meta-analysis found reciprocal prediction: lower support preceded greater PTSD symptoms, and greater symptoms also preceded erosion or loss of perceived support [Wang et al., 2021]. A later multiwave trauma cohort similarly reported bidirectional links between emotional support and symptoms [Santos et al., 2026]. This pattern is consistent with a reciprocal feedback loop, but observational cross-lagged associations do not by themselves establish one. Withdrawal, irritability, shame, or avoidance may make support harder to receive precisely when it is most needed; unreliable or blaming environments may then confirm that connection is unsafe.

The practical implication is not a universal prescription for sleep hygiene, breathing, exercise, or social contact. Each can help, fail, or become another demand. The implication is structural: treatments work inside an ecology. Reducing night-time state entry, making bodily signals less ambiguous, restoring daily rhythms, and building relationships that respect consent may lower the control effort required for change. None substitutes for trauma-focused care when that care is indicated. Together, they can make new learning more available and more likely to survive outside the clinic.

CORE SENTENCE

Recovery is learned by a person, but the conditions that permit return are distributed across sleep, body, relationship, and world.

102

Wearables and Early Warning Without Surveillance Fantasy

AUTHORS' INTERPRETATION

A watch can record movement, heart rate, sleep intervals, and sometimes electrodermal activity. A phone can sample location, voice, screen use, and momentary reports. These technologies invite a compelling idea: detect the approach of a defensive episode before it becomes overwhelming, then offer support at the right moment. The idea is scientifically plausible. Its present clinical maturity is easy to exaggerate.

A 2025 systematic review and meta-analysis of passively collected data found that only wake after sleep onset and the relative amplitude of physical activity showed statistically significant pooled associations with PTSD symptoms, and both associations were small [Zhu et al., 2025]. Studies used different devices, features, windows, populations, and outcomes, with little external validation. A three-day ambulatory study likewise showed that mean physiology did not cleanly distinguish PTSD from control groups [Wisco et al., 2025]. Even a strong group association would not by itself establish an individual alarm threshold.

Intensive self-report can add context that sensors lack. A momentary ten-item version of the PCL-5 showed encouraging within- and between-person reliability over six weeks in a sample of women who presented for emergency care after sexual assault [Patrick et al., 2026]. In principle, jointly modelling reports and sensors could distinguish "heart rate rose because I climbed stairs" from "heart rate rose while a reminder narrowed my options." But the tool measured momentary symptoms; it did not turn passive data into diagnosis or prove that an automated intervention improves outcome.

The October 7 literature offers an unusually relevant naturalistic example. A study combining survey and smartwatch data reported early changes in stress, mood, activity, and sleep among people at risk after the attacks, alongside associations with heavy media exposure [Yamin et al., 2025]. Its value lies partly in temporal resolution. Its limits—self-selection, attrition, device algorithms, screening rather than diagnostic interviews, and a singular continuing-threat context—must travel with the result.

The greatest danger is surveillance fantasy: the belief that more data necessarily produces more truth and better care. Electronic-health-record models can appear accurate when they predict a clinical label partly created by patterns of healthcare use, then perform substantially worse against interview-based PTSD [Crow et al., 2025]. Wearables can similarly encode occupation, poverty, caregiving, disability, battery habits, and access to safe places. False alarms can intensify vigilance; missed alarms can create false reassurance. Employers, insurers, militaries, schools, or states could misuse data gathered under the language of care.

A defensible system would be opt-in, minimal, transparent, locally processed where possible, revocable, and governed by the person rather than an institution. It would report uncertainty, be validated across groups and devices, and demonstrate benefit in prospective trials before making clinical claims. The best near-term role may be collaborative pattern discovery—helping a person and clinician notice rhythms—rather than invisible automated judgment.

CORE SENTENCE

A sensor may help reveal a pattern, but it must never be permitted to turn uncertainty into surveillance or a person into a risk score.

103

Treatment Response as Changed Transition Structure

FLOW HIJACKED SYNTHESIS

Symptom reduction tells us that something changed; it does not, by itself, tell us what changed. The Flow Hijacked proposal asks whether successful treatment can sometimes be described more precisely as a change in transition structure: fewer unnecessary entries into defensive states, lower amplification after entry, shorter residence, easier access to alternative actions, and greater dependence on current context. This is a synthesis, not an established common mechanism of psychotherapy.

Let the latent state of the brain–body–person system be $\mathbf{x}_t \in \mathbb{R}^n$, the current context be $\mathbf{c}_t$, and the treatment input be $\mathbf{u}_t$. A controlled stochastic model may be written

$$d\mathbf{x}_t = \mathbf{f}_{\boldsymbol{\theta}(t)}(\mathbf{x}_t, \mathbf{c}_t)\, dt + \mathbf{B}_{\boldsymbol{\theta}(t)}(\mathbf{x}_t)\mathbf{u}_t\, dt + \boldsymbol{\Sigma}_{\boldsymbol{\theta}(t)}(\mathbf{x}_t)\, d\mathbf{W}_t,$$

where $\boldsymbol{\theta}(t)$ contains parameters governing transition thresholds, coupling, noise, and contextual sensitivity. Treatment need not push the state directly from "ill" to "well." It may instead change $\boldsymbol{\theta}$: altering what future inputs do. Exposure may generate disconfirmatory evidence under tolerable activation; cognitive processing may revise meanings and policies; sleep treatment may change the background conditions for consolidation; medication may alter arousal or learning conditions; social repair may make a previously unavailable action safe enough to attempt.

For a defensive set $\mathcal{D}$ and an engaged set $\mathcal{E}$, define first-passage times

$$\tau_{\mathcal{D}\to\mathcal{E}} = \inf\{s > 0 : \mathbf{x}_{t+s} \in \mathcal{E} \mid \mathbf{x}_t \in \mathcal{D}\}, \qquad \tau_{\mathcal{E}\to\mathcal{D}} = \inf\{s > 0 : \mathbf{x}_{t+s} \in \mathcal{D} \mid \mathbf{x}_t \in \mathcal{E}\}.$$

A TSRA-compatible response would predict that treatment changes their conditional distributions, not merely a global symptom mean: after non-dangerous cues, $\tau_{\mathcal{D}\to\mathcal{E}}$ becomes shorter or less variable, while unwarranted transition into $\mathcal{D}$ becomes less probable. Crucially, warranted defensive transitions should remain available.

There are early empirical hints that within-treatment processes can be temporally ordered. In one psychotherapy analysis, changes in PTSD symptoms and functioning influenced one another differently across sessions [Benfer et al., 2024]. In a small trial of comorbid PTSD and depression, within-person reductions in core PTSD symptoms preceded later reductions in depressive clusters, while the reverse paths were not significant [Kline et al., 2026]. A secondary EMA analysis used differential equations and eigen-decomposition to suggest that two suicide-focused interventions embedded in massed CPT produced different patterns of affective coupling [Bryan et al., 2026]. None directly validates TSRA or estimates the proposed state transitions. Together they show that treatment process can be studied through time and coupling—providing a methodological bridge toward transition tests—rather than only as a pre–post difference.

CORE SENTENCE

Treatment may succeed not only by lowering distress, but by changing what the system does next.

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A Direct TSRA Test Protocol

NEW HYPOTHESIS

Threat-State Return Asymmetry must be testable at the level where it makes its claim: within-person transitions under changing context. A cross-sectional comparison of mean amygdala activation cannot establish it. Neither can a single symptom score. A minimally adequate study would combine controlled perturbations, dense real-world sampling, prospective follow-up, and an explicitly comparative generative model; a decisive result would still require independent replication.

Recruit at least three preregistered groups: people with clinician-diagnosed PTSD; trauma-exposed people without PTSD; and demographically matched people without criterion-level trauma exposure. Where ethically and scientifically possible, stratify rather than erase trauma type, time since exposure, dissociative features, medication, sleep disturbance, and continuing threat. Repeat measurement before, during, and after an evidence-based treatment, with an untreated or active-comparison condition when ethical. The experiment should include threat and safety cues, ambiguous cues, context switches, extinction and return-of-fear tests, and low-burden recovery periods. EMA should sample cue, appraisal, action, symptom state, perceived control, social context, and time-to-recovery; sensors may supplement these reports but never replace them.

Let $z_t \in \{1, \dots, K\}$ denote a latent regime, $\mathbf{x}_t \in \mathbb{R}^d$ the continuous latent state within a regime, and $\mathbf{y}_t$ the multimodal observations. One hierarchical switching state-space specification is

$$
\begin{aligned}
z_{t+1} &\sim \text{Categorical}\left(\boldsymbol{\pi}_{z_t}(\mathbf{q}_t; \boldsymbol{\theta}_i)\right), \\
\mathbf{x}_{t+1} &= \mathbf{A}_i^{(z_t)} \mathbf{x}_t + \mathbf{G}_i^{(z_t)} \mathbf{q}_t + \boldsymbol{\eta}_t, \\
\mathbf{y}_t &= \mathbf{C}_i^{(z_t)} \mathbf{x}_t + \boldsymbol{\epsilon}_t,
\end{aligned}
$$

where $\mathbf{q}_t$ contains external cues and measured context, $\boldsymbol{\theta}_i$ gives participant-specific transition parameters, and the noise terms have preregistered distributions. TSRA predicts a structured difference rather than generic dysregulation: relative to appropriate comparison groups, safe or ambiguous cues should produce some combination of lower entry thresholds, higher transient gain, wider generalisation, longer defensive dwell, and greater control energy for return. These parameters should predict prospective impairment and within-person recovery better than baseline symptom severity alone.

The confirmatory analysis must compare TSRA against simpler rivals: an overall arousal model, a static severity model, a memory-specific model, and flexible nonmechanistic predictors. Model selection should use held-out participants and held-out contexts, calibration as well as discrimination, posterior predictive checks, and external replication. State labels must be derived without naming every high-arousal cluster "threat." Measurement invariance across language, sex, age, culture, and devices must be tested. Missingness should be modelled because disengagement may itself depend on state.

The strongest intervention test asks whether change in TSRA parameters mediates durable clinical and functional improvement—and whether manipulating a proposed parameter changes the predicted transition. A beautiful fit is not enough. The protocol earns explanatory force only if its latent quantities remain identifiable, generalise, and respond selectively to perturbation.

CORE SENTENCE

TSRA becomes science only when its transition parameters outperform simpler explanations and survive prospective, out-of-sample tests.

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What Would Falsify the Flow Hijacked Model

NEW HYPOTHESIS

A hypothesis protected from failure is a metaphor wearing laboratory language. Flow Hijacked must therefore say in advance what evidence would weaken or defeat its central proposal. TSRA would be challenged if well-powered, preregistered studies repeatedly found no reliable entry–return asymmetry after measurement error, continuing threat, trauma type, medication, sleep, and comorbidity were addressed. It would be challenged if the parameters could not be identified from realistic data, if they changed radically with arbitrary modelling choices, or if they failed to replicate across tasks and daily life.

It would also be weakened if a simpler model did the work. Suppose baseline severity, general negative affect, or one nonspecific persistence parameter predicted future symptoms and treatment response as well as a full transition model. In that case, the additional geometry would not have earned its complexity. Formally, for a TSRA model $\mathcal{M}_T$ and a simpler rival $\mathcal{M}_0$, the relevant standard is prospective held-out predictive advantage, with flexibility controlled during model fitting by preregistered hierarchical priors or regularisation:

$$\Delta_{T0} = \mathbb{E}_{(\mathbf{y},\mathbf{c})\sim p_{\text{new}}}[\log p(\mathbf{y} \mid \mathbf{c}, \mathcal{M}_T) - \log p(\mathbf{y} \mid \mathbf{c}, \mathcal{M}_0)] .$$

This display is an expected log predictive-density difference on genuinely new observations; it is not, by itself, an algebraic parameter-count penalty. Model flexibility must be constrained without access to the test observations and audited with calibration, posterior predictive checks and repeated external validation.

If $\Delta_{T0} \leq 0$ across independent cohorts and relevant outcomes, TSRA should be simplified or abandoned, not rescued by increasingly private interpretations.

Current null findings already constrain the story. A large ENIGMA-PGC analysis found no robust PTSD-group differences in static connectivity, dynamic connectivity, dwell time, or transition count using its resting-state method [Tomas et al., 2025]. Another large analysis located dynamic effects mainly in PTSD with mild traumatic brain injury and in interactions with age and anxiety, warning against generalising a universal PTSD signature [Dwulit et al., 2026]. A preregistered fear-learning study found no diagnostic-group differences on its selected conditioning and extinction measures [Davidson et al., 2026]. These do not falsify all conditional, task-evoked, autonomic, social, or individual-level dynamics. They do falsify careless claims that altered dynamics or extinction deficits are already universal facts.

Non-normal amplification has an even sharper burden. To support that subhypothesis directly, PTSD data would need to identify a local effective operator, demonstrate non-orthogonal modes or pseudospectral sensitivity, predict input-specific transient growth, and replicate the result. Large responses to small cues are not sufficient: nonlinear thresholds, unmeasured cue meaning, high baseline gain, or ordinary recurrent feedback can produce similar observations. If these alternatives explain the data more parsimoniously, the non-normal bridge must remain outside the PTSD mechanism claim.

Finally, treatment must be able to disconfirm mechanism. If symptoms improve while proposed transition parameters remain unchanged, or parameters change without functional benefit, the asserted pathway is wrong or incomplete. The purpose of falsification is not to weaken compassion. It prevents a person's suffering from being colonised by a theory that refuses to listen.

CORE SENTENCE

The Flow Hijacked model deserves a place in science only if it can lose to the data.

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A Research Programme for the Next Decade

FLOW HIJACKED SYNTHESIS

The next decade should move PTSD research from isolated snapshots toward coordinated measurement of change. This does not require attaching a sensor to every person or placing every mechanism inside one impossible study. It requires a sequence of studies in which scales and methods constrain one another.

First, build prospective cohorts with genuine pre-trauma information wherever exposure can be studied ethically without anticipating harm: emergency and healthcare workers, military and rescue personnel, high-risk occupations, and population panels already collecting mental-health, sleep, and social data. When unexpected collective trauma occurs, preserve the distinction between a pre-event measure collected for another purpose and a purpose-built PTSD baseline. Repeated clinician interviews, functioning, sleep, medication, physical health, loss, ongoing exposure, and social conditions should accompany symptom screens. The 2026 individual-participant-data meta-analysis of acute-care cohorts shows the value of harmonisation while also illustrating that early predictors remain associations rather than maintenance mechanisms [Ratanatharathorn et al., 2026].

Second, connect laboratory perturbations to daily life. The same participant can complete context-shifted safety-learning tasks, ambulatory reports after naturally occurring reminders, and follow-up assessments of work, intimacy, parenting, and participation. Sampling should be dense around transitions but sparse enough to remain livable. The analysis must distinguish within-person dynamics from between-person differences and separate observation error from process noise.

Third, treat intervention as system identification. Randomised treatments provide controlled inputs. Rather than adding exploratory biomarkers after the fact, investigators should preregister which transition parameter a treatment is expected to change, when it should change, and what functional consequence should follow. Null target-engagement trials are especially informative: pharmacologically increasing a candidate biological signal without improving neural or clinical outcomes can rule out a specific augmentation prediction and weaken an attractive causal shortcut [Tansey et al., 2026]. Measurement-based care can support adaptive decisions, but a 2026 review found a small, predominantly US veteran implementation literature and substantial practical barriers [Al-Shamali et al., 2026].

Fourth, develop models at several levels. A deliberately minimal 2026 model reproduced broad chronic and recovering courses and a threshold-like treatment prediction, but its aggregate fit cannot identify a unique individual mechanism [Grosskopf et al., 2026]. Minimal models should meet richly measured cohorts; neural effective-connectivity models should meet autonomic and behavioral data; social and institutional variables should enter as causes, not decorative covariates. Competing model classes—switching, continuous nonlinear, reinforcement-learning, predictive-processing, network, and conventional longitudinal models—should be compared on the same held-out outcomes.

Finally, build governance into method. Participants must retain meaningful control over intensive data. Cross-cultural and bilingual measurement should be designed before recruitment. Samples should include children, older adults, displaced people, civilians, combatants, survivors of interpersonal violence, and communities living under continuing threat without treating any group as interchangeable. Results, code, preprocessing decisions, and null findings should be shared when privacy permits. The aim is not a universal equation of trauma. It is a cumulative science precise enough to discover where different equations are needed.

CORE SENTENCE

The next advance is more likely to come from linked, falsifiable studies of change across person, context, treatment, and time than from one more dramatic biomarker.

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Terminology Key

BROADER SCIENTIFIC INFERENCE

Language is part of scientific control. In this lecture, a **potentially traumatic event** refers to exposure of the kind relevant to diagnostic criteria; it does not presume a disorder. **Post-traumatic stress symptoms**, or PTSS, are dimensions of experience and behavior that can be measured without establishing diagnosis. **Probable PTSD** means that a screening or self-report algorithm crossed a specified threshold. **Diagnosed PTSD** requires the relevant diagnostic procedure and criteria. These terms must not be exchanged for rhetorical force.

A **state** is a configuration of variables over a chosen interval; it is not a personality or identity. A **latent state** is inferred from observations rather than measured directly. A **transition** is a change in state or in the probability of occupying another state. **Dwell time** is the duration spent in a defined state; **first-passage** or **escape time** is the time until a specified destination is reached. A system can show long dwell without possessing a literal anatomical "basin."

An **attractor** is a set toward which nearby trajectories converge under a mathematical dynamical system. A **basin of attraction** is the set of initial conditions that converge toward that attractor. In this lecture, attractor language is a model proposal unless a study directly estimates the required dynamics. **Metastability** describes temporary persistence with eventual transition; it does not simply mean flexibility. **Hysteresis** means that the current state depends on the path by which conditions were reached, so entry and exit can occur at different parameter values. **Bifurcation** is a qualitative change in dynamical behavior as a parameter varies.

Transient gain is the magnitude by which a perturbation is amplified over a finite interval. A **non-normal operator** does not commute with its adjoint; its eigenvectors need not be orthogonal, allowing transient growth even when all eigenmodes are asymptotically stable. A **pseudospectrum** describes how eigenvalues may shift under perturbation and can reveal sensitivity invisible to the ordinary spectrum. These are exact mathematical ideas. Their use in PTSD remains a bridge hypothesis, not a diagnosis.

Controllability asks whether admissible inputs can move a system among states; **control energy** quantifies the input cost under a particular model. Neither term implies that a person should be controlled. **Prediction error** is the discrepancy between an expected and observed outcome. **Precision** is the estimated reliability assigned to a signal or prediction, not mere confidence. **Active inference** describes action partly as sampling or changing observations under a generative model; it remains one theoretical framework among competitors.

The recurring dynamical notation is also fixed: $\mathbf{x}_t$ is latent state, $\mathbf{y}_t$ observation, $\mathbf{c}_t$ measured context, and $\mathcal{H}_t$ observed history. The symbols D and S label discrete defensive and sufficiently safe regimes when a regime model is used; $\mathcal{D}$ and $\mathcal{E}$ label defensive and engaged regions of a continuous state space. Diagnosis indicators, outcome components and experimental trial labels used in other sections are local quantities and must not be substituted for these dynamical objects.

Finally, **Threat-State Return Asymmetry (TSRA)** is the Flow Hijacked hypothesis that, in at least some PTSD presentations, trauma alters conditional transition rules so defensive states are entered more readily, broadly, or strongly than they are left once present evidence no longer warrants them. It is operationalised through entry threshold, cue-specific gain, generalisation width, dwell or escape time, recovery half-time, and access to alternative states. It is not a synonym for PTSD, and it must be allowed to fail.

CORE SENTENCE

Precise language keeps a useful model from silently becoming a fact, a diagnosis, or a definition of a person.

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Academic Conclusion

FLOW HIJACKED SYNTHESIS

The empirical literature has not established a single lesion, molecule, memory mechanism, network state, autonomic signature, or learning deficit that defines all PTSD. It instead supports a heterogeneous disorder in which alterations have been observed across threat and safety learning, contextual memory, attention, appraisal, sleep, autonomic and endocrine function, brain-network interaction, behavior, identity, and social ecology. Effects vary by task, population, trauma, developmental stage, comorbidity, time, and method. Strong null findings and strategies that achieve biological target engagement without clinical benefit are not peripheral inconveniences; they define the boundary of what can responsibly be claimed.

Established trauma-focused psychotherapies have the strongest overall evidence and remain the clinical reference point, although average efficacy does not eliminate nonresponse, dropout, adverse effects, access barriers, or individual preference. An individual-participant-data meta-analysis found trauma-focused cognitive-behavioral therapies superior to inactive comparisons while not demonstrating broad superiority over active treatments [Wright et al., 2025]. Frontier interventions may expand options, but novelty, biological vividness, and regulatory attention are not substitutes for independent replication, durability, safety, and real-world effectiveness.

The Flow Hijacked contribution is a proposed level of integration. PTSD may be studied as a brain–body–person–environment system whose learned rules of prediction, amplification, transition, and recovery have been altered by experience. Threat-State Return Asymmetry makes that proposal more specific: in some people and contexts, defensive states may become easier to enter than to leave. This asymmetry could arise through several nonexclusive mechanisms—broader threat generalisation, altered contextual weighting, reinforced avoidance, sleep disruption, autonomic feedback, social loss, or changes in network coupling. It need not imply one common biological substrate.

Mathematically, this is a claim about conditional dynamics, not about an immutable trait. For a defensive region $\mathcal{D}$, an engaged region $\mathcal{E}$, a matched horizon Δ, and a context vector $\mathbf{c}$, define

$$p_i^{\mathcal{E}\to\mathcal{D}}(\mathbf{c},\Delta) = \mathbb{P}\big(\mathbf{x}_{i,t+\Delta} \in \mathcal{D} \mid \mathbf{x}_{i,t} \in \mathcal{E},\, \mathbf{c}_{i,t} = \mathbf{c}\big)\,,$$
$$p_i^{\mathcal{D}\to\mathcal{E}}(\mathbf{c},\Delta) = \mathbb{P}\big(\mathbf{x}_{i,t+\Delta} \in \mathcal{E} \mid \mathbf{x}_{i,t} \in \mathcal{D},\, \mathbf{c}_{i,t} = \mathbf{c}\big)\,.$$

For probabilities strictly between zero and one, a signed matched-horizon contrast is

$$\mathcal{A}_i^{\mathrm{ER}}(\mathbf{c},\Delta) = \operatorname{logit} p_i^{\mathcal{E}\to\mathcal{D}}(\mathbf{c},\Delta) - \operatorname{logit} p_i^{\mathcal{D}\to\mathcal{E}}(\mathbf{c},\Delta), \qquad \operatorname{logit}(p) = \log\frac{p}{1-p}.$$

A positive value means that, under this particular operational definition, entry is more probable than return across the same horizon and context. Boundary probabilities require a preregistered estimator or must be reported without an undefined logit. This contrast is not the whole TSRA profile; it is one directly estimable summary. The scientific task is to estimate such quantities, relate them to lived function, compare them with simpler models, and test whether effective treatment changes them. The ethical task is to remember that no latent state contains the whole person and no population average predicts one life with certainty.

This framework changes the organising question. Instead of asking only where trauma is stored, we ask what makes a protective response recur, amplify, persist, generalise, or release. Instead of equating recovery with quiet, we ask whether the system can discriminate, transition, and regain options. Instead of protecting the model, we specify its competitors and failure conditions. The result is not a completed theory of PTSD. It is a disciplined research programme built to join mechanism with time, context, treatment, and humane reality.

CORE SENTENCE

PTSD may be understood most fruitfully not as one broken component, but as altered conditional dynamics that remain heterogeneous, treatable, and open to falsification.

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Humane Closing — The World Does Not Have to Feel Safe All at Once

FLOW HIJACKED SYNTHESIS

There is a particular cruelty in asking a person whose protection has become exhausting to simply relax. The instruction ignores what the system has learned, what it may still be facing, and how much intelligence can be contained in vigilance. It also places the burden of proof on the person: trust first, sleep first, stop avoiding first, believe the danger is over. But recovery rarely begins with a verdict delivered from outside.

It can begin more locally. One room becomes less difficult. One night contains a longer interval of sleep. A sound still startles, but the body returns before the day is lost. A memory arrives and remains a memory rather than becoming the whole present. A boundary is set without shame. A conversation survives discomfort. Help becomes slightly more reachable. None of these moments is small to a system that has learned to organise around danger.

The Flow Hijacked lens does not say that the person is a machine, nor that an equation can encompass grief, betrayal, pain, courage, or meaning. It says something narrower and, perhaps, kinder: patterns that feel total may have structure. What has structure can be observed. What can be observed can sometimes be influenced. And what has been learned under unbearable conditions is not therefore a permanent identity.

Treatment is not a moral test. A person does not fail exposure, cognitive processing, EMDR, medication, sleep treatment, or any other intervention. An intervention may be poorly timed, inaccessible, culturally mismatched, intolerable, incomplete, or simply insufficient for this person at this moment. Care can be adjusted. Goals can be renegotiated. Safety can be built materially as well as internally. Recovery can include grief and anger; it need not perform gratitude or growth.

Nor must the entire world feel safe. No nervous system owes the world indiscriminate trust. The meaningful shift may be from "danger is everywhere and return is unavailable" toward "danger can be located, responses can change, and some places, people, actions, and futures can become possible." The widening may be slow and nonlinear. There may be setbacks. A reopened future does not invalidate what happened; it refuses to let what happened hold exclusive authorship over what happens next.

This lecture began with a protective system that could not easily leave the state it had learned. It ends not with a command to leave, but with a scientific and human possibility: the rules of return are not necessarily fixed. Sometimes the first evidence of recovery is not calm. It is choice.

CORE SENTENCE

The world does not have to feel safe all at once for one more future to become reachable.

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GUIDELINE AND REGULATORY RECORDS · NOT PEER-REVIEWED EVIDENCE

R1. U.S. Department of Veterans Affairs and Department of Defense (2023-06). VA/DoD Clinical Practice Guideline for Management of Posttraumatic Stress Disorder and Acute Stress Disorder. Current official guideline located for this programme; systematic evidence review searched through 2022-05-04. official record

R2. National Institute for Health and Care Excellence (2018-12-05). Post-traumatic stress disorder (NG116). Published guideline retained as an established-treatment reference; its age must remain visible. official record

R3. U.S. Food and Drug Administration (2026-06-03). K260402 — Magnetic e-Resonance Therapy (MeRT) System 510(k) clearance. FDA-cleared Class II device as an adjunctive treatment for adults with PTSD. official record

R4. U.S. Food and Drug Administration (2024-08-08). Complete Response Letter for NDA 215455 (midomafetamine/MDMA-assisted therapy). Not FDA approved for PTSD. The 2024 application received a Complete Response Letter. official record

R5. Multidisciplinary Association for Psychedelic Studies (2026-08-10). Statement concerning reported 2026 resubmission of MDMA-assisted therapy application. Sponsor/advocacy-adjacent report of resubmission; no FDA approval found by 2026-09-06. official record

R6. U.S. Food and Drug Administration (2025-07-18). Psychopharmacologic Drugs Advisory Committee meeting materials — brexpiprazole plus sertraline for PTSD. The advisory committee voted 10–1 that the submitted evidence did not establish efficacy; the PTSD indication was not approved. official record

R7. Otsuka and Lundbeck (2025-09-20). Complete Response Letter announcement for brexpiprazole plus sertraline supplemental application. Company-confirmed Complete Response Letter; brexpiprazole was not approved for PTSD. official record

R8. American Psychological Association (2025). Clinical Practice Guideline for the Treatment of Posttraumatic Stress Disorder in Adults. Current APA adult PTSD guideline page located at the cutoff. official record

R9. U.S. Department of Veterans Affairs, National Center for PTSD (current at 2026-09-06). Overview of Psychotherapy for PTSD. PE, CPT and EMDR are described as the treatments with the strongest recommendation; WET, Ehlers' CT and PCT as suggested alternatives. official record

R10. U.S. Department of Veterans Affairs, National Center for PTSD (current at 2026-09-06). Clinician's Guide to Medications for PTSD. Sertraline and paroxetine are the only medications with an FDA-approved PTSD indication; venlafaxine is guideline-supported off-label. official record

R11. US Food and Drug Administration (2020-11). De Novo classification request for NightWare Kit (DEN200033). Class II prescription adjunctive device under 21 CFR 882.5705; product code QMZ. official record

R12. U.S. Food and Drug Administration (2026-05-08). DEN250013 — Modius Spero De Novo classification order. FDA granted De Novo Class II classification for a prescription, home-use transcranial nerve stimulation device for PTSD-associated symptoms in adults aged 22 and older, used with a comprehensive treatment plan under a healthcare professional; it is not intended as stand-alone therapy or to alter usual care. official record

INFLUENTIAL CONTEXTUAL WORK — NOT COUNTED AS PEER-REVIEWED EVIDENCE

Bessel van der Kolk (2014). *The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma*. Its cultural and clinical influence is discussed in Section 48; mechanistic and treatment claims are rebuilt from peer-reviewed evidence.

FLOW HIJACKED · LECTURE 50

THE WORLD DOES NOT HAVE TO FEEL SAFE ALL AT ONCE

Sometimes the first evidence of recovery is not calm. It is choice.