

Internal State and Extinction Durability in Retrieval Control

Gil Raites

1 Motivation and Gap

Fear extinction and exposure-based learning can reduce visible distress before the older threat association has been durably updated. A subject can appear regulated during an extinction-like experience while the older threat association remains retrievable later. The mechanistic problem is the difference between suppressing threat expression during the session and changing the association that controls later retrieval.

The central question is how an experimentally imposed internal state changes which process controls behavior: threat-expression gating or durable remodeling of the association that controls later retrieval. A state that dampens fear output may reduce freezing, avoidance or distress during the learning window. The same state may also weaken prediction error, extinction encoding, retrieval access or structural remodeling needed for extinction to hold across delay, context change and internal-state change.

The experiment varies the internal state present during learning and retrieval. Increased inhibitory tone can reduce immediate fear expression by gating threat output during extinction. A plasticity-support state can change fronto-limbic regulation and neuroimmune remodeling conditions. Mixed state conditions can produce mixed effects because one state may dampen prediction error while another supports longer-timescale plasticity.

Calm behavior can be read as improvement before the source of that calm has been identified. A mechanistic account has to determine whether reduced fear expression comes from durable updating, state-bound safety retrieval or temporary expression suppression. The validation strategy separates those cases through behavioral timing, neural retrieval organization and structural remodeling. Behavioral readouts test expression and timing. Neural readouts test engram access and CA1/MEC-CA1 retrieval organization. Structural readouts test dendritic spine remodeling, microglial state and state-matched retrieval stability.

The proposal therefore treats reduced fear expression during the altered state as an incomplete measurement. The decisive measurement is later safety retrieval across conditions where the original threat association could regain behavioral control.

2 Background

Fear extinction is an active learning process in which a prior threat association loses behavioral dominance through new inhibitory learning and structural remodeling. The original threat trace can persist after extinction. A competing safety trace gains behavioral control when retrieval access, context generalization and circuit remodeling support later use.

Acute suppression and durable updating differ at the level of trace remodeling. Acute suppression can reduce fear output before the threat trace has been remodeled. GABAergic inhibition can gate fear output and select safety-biased circuit states during that window. Durable extinction also requires synaptic re-weighting, spine remodeling, latent-cause revision and circuit reorganization that reduce the older threat trace's later behavioral control.

Retrieval determines whether the safety trace can control later behavior. Extinction can be encoded while later access remains context-bound. External context, internal state, time delay and the induced learning state can all act as retrieval cues. If extinction-like learning occurs under a narrow internal state, later retrieval may be strongest when that internal state returns. A later state change can reduce access to the extinction trace and return behavior to the older threat response.

CA1 ensemble dynamics provide a readout for state-bound retrieval. CA1 supports contextual gating, pattern separation, ensemble reactivation and temporal sequence organization. CA1 population activity can indicate whether retrieval is organized around the fear-conditioned context, the extinction context or a mismatch between training state and retrieval state.

MEC-hippocampal coordination routes contextual input before CA1 resolves the retrieval state. MEC and entorhinal inputs provide spatial and contextual scaffolding that helps CA1 distinguish danger contexts from safety contexts. If the induced internal state changes inhibitory coordination, oscillatory mode or information routing, the retrieval context can be reorganized even when the external environment has not changed.

The structural remodeling pathway runs through microglial contact with fear engram neurons, dendritic spine regulation and complement-

linked synapse selection. Microglial contact can support transient silencing at the soma and longer-timescale dendritic remodeling through synapse engulfment. C1q and C3 can mark synapses for pruning or reorganization. IL-10-linked anti-inflammatory signaling may preserve dendritic spine density and cognitive plasticity, while pro-inflammatory activation may weaken the synaptic environment needed for extinction consolidation.

Plasticity-support states introduce a conditional remodeling pathway. BDNF-linked plasticity, vmPFC-amygdala regulation, hippocampal-amygdala connectivity and safety-memory consolidation may improve under those conditions. The evidence also requires caution because plasticity effects can vary with timing, duration, baseline state and context dependence. Plasticity support should therefore be treated as a conditional modifier with timing and context constraints.

The working model separates four layers of durability: expression control, retrieval access, context routing and structural remodeling. A learning-state manipulation may alter any one of these layers, so the experimental design must distinguish which layer is changing.

3 Research Goal and Hypothesis

The hypothesis tests how the internal state present during extinction-like experience redistributes control across acute threat suppression, safety-memory consolidation and durable updating of older threat associations.

$$B_t = \sigma(\alpha F_t - \beta S_t R_t + \gamma I_t)$$

In this expression, B_t is observed fear behavior, F_t is the original threat trace, S_t is the extinction or safety trace, R_t is retrieval access to the safety trace and I_t is acute inhibitory tone. Lower fear behavior can come from expression gating through I_t or from safety retrieval through $S_t R_t$. Those outcomes have different implications for later relapse.

Elevated inhibitory tone may reduce immediate threat expression and weaken the encoding, consolidation or later retrieval of extinction learning. Safety learning may become tied to a specific internal state, which leaves the extinction trace unable to control behavior across contexts.

Plasticity-support states may change the direction of this effect by supporting fronto-limbic regulation, synaptic remodeling or microglia-linked recovery of plasticity. Plasticity support should improve durable extinction only when extinction learning generalizes across retrieval conditions.

Neuroimmune state may change extinction durability by altering dendritic spine maintenance and pruning programs. The same behavioral protocol may produce different outcomes when microglia-linked remodeling supports synaptic stabilization or drives excessive synaptic loss.

Mixed internal-state conditions may produce interaction effects across expression gating and plasticity support. Acute inhibitory tone may dampen fear expression, while plasticity-support states may support longer-timescale remodeling. The combined state may reduce distress during extinction and still produce state-bound, incomplete or timing-dependent extinction unless the behavioral protocol preserves prediction error, extinction-session engagement and retrieval across contexts.

The central prediction is a dissociation between fear reduction during training and safety retrieval after the state changes. A manipulated internal state may lower freezing or distress during extinction while leaving latent-cause structure, engram reactivation, CA1/MEC-CA1 organization or synaptic architecture insufficiently updated. Durable updating should therefore be defined by stable retrieval across delay, external context change and internal-state change. Reduced fear expression during training alone does not establish that update.

4 Proposed Experimental Approach

The experiment would pair contextual fear conditioning with extinction training under controlled internal-state conditions. The core design would compare baseline state, elevated inhibitory tone during extinction, plasticity-support state and combined modulation state. Retrieval would be tested in matched and unmatched internal states.

The behavioral design should separate acquisition, consolidation, retrieval and generalization. The retrieval matrix should include four conditions: same context with same internal state, same context with changed internal state, changed context with same internal state and

changed context with changed internal state. The retrieval matrix separates environmental renewal from internal-state retrieval failure.

$$R_t = \exp\left(-\frac{\|z_{\text{train}} - z_{\text{retrieval}}\|^2}{2\sigma_z^2}\right)$$

Here, z_{train} is the internal state during extinction learning and $z_{\text{retrieval}}$ is the internal state during later retrieval. Same-context and changed-context tests should be paired with same-state and changed-state tests because retrieval access may depend on the distance between learning state and retrieval state.

The main behavioral readouts should be grouped into freezing measures, latency measures and recovery measures. Freezing measures include total amount, onset and offset. Latency and recovery measures include avoidance persistence, approach latency, response recovery time and trial-to-trial change. Total freezing is necessary, but timing measures show how quickly the system resolves competition between threat and safety traces. A subject may show low freezing with delayed safety retrieval, persistent avoidance or fast threat-response onset.

Fear engram neurons would be labeled during conditioning and assayed after extinction and retrieval. Readouts would include cFos or related activity tagging, fear-engram reactivation, extinction-engram reactivation, microglial recruitment to engram somata, microglial contact with dendrites, spine density and complement-associated remodeling. Behavior, engram access and structural remodeling would therefore function as separate validation axes. The combined behavioral, engram and structural measures distinguish temporary silencing of the fear trace from remodeled structural support for retrieval.

CA1 activity would be recorded during extinction training, rest or offline consolidation and each retrieval phase. Ensemble analyses would compare reactivation of fear-conditioned, extinction-conditioned and context-specific population patterns. Temporal analyses would compare sequence replay, population-state trajectories and timing alignment with amygdala and prefrontal readouts.

MEC-CA1 coordination would be recorded or perturbed during extinction and retrieval. The experiment should compare entorhinal input signatures, CA1 population states, context discrimination, amygdala-prefrontal alignment and state-dependent connectivity across matched and mismatched retrieval conditions. The MEC-CA1 comparison would test whether renewal reflects failed context discrimination, disrupted entorhinal input to CA1 or poor alignment between hippocampal context signals and fear-control circuits.

The plasticity-support arm would contrast baseline state, elevated inhibitory tone and plasticity-support state. Readouts would include vmPFC-amygdala coupling, hippocampal-amygdala connectivity, BDNF-linked plasticity markers and recall after delay or context shift. This readout tests whether plasticity support broadens extinction stability or strengthens extinction inside the trained context.

The neuroimmune arm would measure microglial activation state, cytokine profile, dendritic spine density and synaptic pruning markers after extinction and retrieval. Readouts would include IL-10-linked anti-inflammatory signaling, MEF2C-related microglial programs, complement or Fc-gamma receptor pathway markers and hippocampal spine preservation. This readout tests whether a remodeling-permissive immune-synaptic state supports durable extinction.

The combination-state arm would contrast isolated inhibitory modulation, isolated plasticity support and combined modulation. The design would track acute freezing reduction, extinction-session engagement, delayed recall, renewal after context change and state-matched retrieval. This readout tests how combined state changes extinction learning conditions, especially cases where distress falls and the safety trace stays weak, narrow or difficult to retrieve.

5 Anticipated Outcomes and Significance

The primary validating outcome is a behavioral flip: elevated inhibitory tone reduces visible fear during extinction, then fear returns during delayed recall, internal-state change or external-context change. A training-session reduction followed by later return of fear supports the distinction between expression control during training and weak retrieval of the extinction trace later.

$$D_t = S_t R_t M_t$$

Here, D_t is durable updating, S_t is the safety trace, R_t is access to that trace and M_t is the remodeling-permissive state. Durable updating requires the safety trace, access to the safety trace and structural remodeling support. Low fear during training is not enough.

The strongest validation pattern combines low fear with neural and structural evidence for safety-trace control. Low fear during retrieval should be accompanied by extinction-linked cFos or engram reactivation, reduced fear-engram dominance and organized CA1 extinction-context activity. The same result should show stable MEC-CA1 context routing, adaptive spine remodeling and a remodeling-permissive microglial state. Durable updating should hold across delay, context change and internal-state change.

The retrieval condition diagnoses the source of relapse. Fear after internal-state change would support state-dependent retrieval. Fear after external-context change would support contextual renewal. Fear across both changes would indicate weak generalization across the retrieval landscape.

CA1 validation tests the ensemble state associated with relapse or safety retrieval. Relapse should reactivate fear-conditioned sequences or threat-associated ensembles. Successful extinction retrieval should show extinction-context or safety-associated CA1 organization. If behavior and CA1 activity diverge, the model would need to separate hippocampal context readout from downstream amygdala or prefrontal output control.

MEC-CA1 validation tests context routing during renewal and freezing. Renewal should appear with disrupted MEC-CA1 coordination, altered entorhinal input signatures, weakened context separation or state-dependent reconfiguration of hippocampal-entorhinal activity. Context-generalized extinction retrieval should involve coordinated MEC-hippocampal routing that allows CA1 to index the current context as safe across delay, context change and internal-state change.

Behavioral timing tests response structure alongside total freezing. The model is supported if altered state reduces total freezing during extinction while later tests show delayed safety retrieval, persistent avoidance, faster threat-response onset or renewed freezing after context or state change.

The main refuting case comes from inhibitory tone itself. Inhibitory modulation can support extinction under specific timing conditions. If strategically timed inhibitory modulation improves extinction engagement while preserving delayed recall and context-generalized retrieval, the model should treat inhibitory tone as timing-dependent. This qualification prevents the paper from treating expression dampening as uniformly disruptive.

The plasticity-support validation path expects improved extinction recall, reduced renewal or stabilized safety retrieval across context changes. If plasticity support improves only the trained context, the result indicates that durable updating still depends on retrieval generalization. If plasticity support produces mixed outcomes across timing, duration or baseline state, the model should treat that support as a protocol-dependent modifier.

The neuroimmune validation path expects extinction durability to track anti-inflammatory signaling, spine preservation or precise synaptic pruning. If fear expression drops while microglial activation, spine loss or weak remodeling persists, the result supports the distinction between acute regulation and durable updating. If extinction stays durable despite disrupted microglial markers, the model would need to place neuroimmune state downstream of stress history or separate it from retrieval control.

Timing of state modulation may determine whether the system enters momentary stabilization, state-bound learning or durable extinction across contexts. A clinically useful model should distinguish calm behavior from durable updating. Medication effects belong in this model only as one possible route into the internal-state conditions tested here: inhibitory tone, plasticity tone, retrieval access and remodeling environment. The experimental model should validate across behavior, engram activity, CA1 and MEC-CA1 retrieval organization, microglial remodeling and state-matched retrieval.

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