

# The Auditory Disruption of Mnemonic Architectures: Mechanisms of Interference, Destabilization, and Physiological Impairment

## Abstract

The fragility of human memory is nowhere more apparent than in its susceptibility to auditory interference. While the auditory system serves as a critical channel for language acquisition and environmental monitoring, its obligatory processing nature renders it a potent disruptor of cognitive stability. This report provides an exhaustive analysis of the mechanisms by which auditory tones, noise, and speech disrupt memory across three distinct temporal phases: the immediate maintenance of information in **Working Memory (WM)**, the **Consolidation** of traces during sleep, and the **Reconsolidation** of retrieved long-term memories. We synthesize evidence from over one hundred disparate studies to delineate the specific acoustic properties—such as "changing-state" characteristics, deviance, and infrasonic frequency—that facilitate this disruption. Unlike visual distraction, which can be gated by eyelid closure or aversion, auditory input enjoys privileged access to neural processing centers. This report details how random auditory stimulation stunts the traveling waves of slow-wave sleep, how changing-state tones crash the serial ordering mechanisms of the phonological loop, and how infrasound induces neuroinflammatory cascades that physically degrade hippocampal integrity. We further critically evaluate the efficacy of "binaural beats," revealing a landscape of inconsistent results where intended entrainment often manifests as cognitive load and fatigue.

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## 1. Introduction: The Obligatory Nature of Auditory Processing

Memory is not a unitary store but a dynamic set of processes involving encoding, maintenance, consolidation, and retrieval. Each of these stages is vulnerable to interference, yet the auditory modality presents a unique challenge to mnemonic stability. Evolutionary biology posits that the auditory system functions as an "early warning system," remaining active even during sleep or focused attention to detect environmental threats. This survival mechanism, however, comes at a cognitive cost: the inability to completely "close one's ears" results in a continuous stream of sensory data that the brain must filter, process, or inhibit.

The query "Can you use auditory tones to disrupt memory?" touches upon a fundamental

tension in cognitive neuroscience: the competition between top-down goal maintenance (remembering) and bottom-up sensory processing (hearing). The literature reveals that disruption is not merely a function of loudness (intensity) but is intricately tied to the *structure* of the sound and the *state* of the memory.

In the waking state, working memory is disrupted not by noise per se, but by acoustic variation—the "Changing-State Effect" [1, 2]. In the sleeping brain, consolidation is disrupted not by waking the subject, but by altering the spatiotemporal trajectory of slow waves via random stimulation [3]. In the retrieval phase, long-term memories are disrupted by "prediction error" signals introduced by auditory deviants [4, 5]. Finally, at the physiological level, low-frequency infrasound can bypass conscious perception entirely to inflict cellular damage on the hippocampus [6].

This report is structured to guide the reader through these distinct domains of disruption, moving from the psychological interference of working memory to the physiological disruption of sleep and neural health.

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## 2. Working Memory Interference: The Irrelevant Sound Effect (ISE)

Working Memory (WM) refers to the limited-capacity system responsible for the temporary storage and manipulation of information. It is the workspace of the mind, essential for reasoning, decision-making, and behavior guidance. A robust phenomenon known as the **Irrelevant Sound Effect (ISE)** demonstrates that task-irrelevant sound—even when participants are explicitly instructed to ignore it—significantly impairs performance on serial recall tasks [1, 7].

### 2.1 Theoretical Frameworks of Interference

To understand how tones disrupt memory, one must first understand the architecture of the system they are disrupting. Early interpretations relied on Baddeley's model of the **Phonological Loop**, a sub-component of working memory comprising a phonological store (the "inner ear") and an articulatory rehearsal process (the "inner voice") [8].

#### 2.1.1 The Phonological Loop and Speech Specificity

Initially, it was believed that the ISE was specific to speech. The theory posited that irrelevant speech gained obligatory access to the phonological store, where it corrupted the phonological traces of the visually presented items (e.g., digits or letters) being rehearsed [8, 9]. Because both the to-be-remembered items and the irrelevant background noise shared a phonological code, they interfered with one another. This "speech-specific" view suggested

that non-speech sounds, such as tones, would be benign.

### 2.1.2 The Changing-State Hypothesis

Subsequent research dismantled the speech-specific view, replacing it with the **Changing-State Hypothesis**. This framework argues that the critical factor for disruption is not the semantic or phonological content of the sound, but its acoustic variation over time [2, 10].

The auditory system functions as a change detector. When it encounters a stream of sound that varies in pitch, timbre, or tempo (a "changing state"), it automatically segments this stream into auditory objects and processes their order. This **obligatory order processing** competes with the serial order processing required to maintain the sequence of visual items in working memory [11].

- **Steady-State Tones:** A sequence of repeated tones (e.g., "C, C, C, C") contains no order information because there is no change. Consequently, the brain does not engage order-processing mechanisms, and no interference occurs [12].
- **Changing-State Tones:** A sequence of varying tones (e.g., "C, G, E, B") compels the brain to register the transition from one pitch to another. This generates a stream of order cues that conflicts with the rehearsal of the visual list "7, 2, 9, 4" [13].

The magnitude of disruption caused by changing-state tones is remarkably similar to that caused by speech, suggesting that speech is disruptive not because it is language, but because it is acoustically complex and constantly changing [13].

## 2.2 The Deviant Effect: Disruption via Attentional Capture

While the changing-state effect explains interference through structural conflict (process-based), a second mechanism drives disruption: **Attentional Capture**, often referred to as the **Deviant Effect**.

The deviant effect occurs when a rare or unexpected auditory event violates the prevailing auditory context. For example, in a sequence of repeated standard tones (e.g., "beep beep beep"), the insertion of a different tone ("boop") or a burst of white noise causes a momentary orienting response [5].

### 2.2.1 Acoustic vs. Categorical Deviance

Research distinguishes between types of deviance:

- **Acoustic Deviance:** A change in the physical properties of the sound, such as frequency or intensity.
- **Categorical Deviance:** A change in the *type* of sound, such as a spoken word appearing in a sequence of tones.

Studies indicate that categorical deviants are often more disruptive than simple acoustic

deviants [5]. This suggests that the "surprise" signal is weighted by the semantic or structural distance between the standard and the deviant.

### 2.2.2 Habituation Dynamics

A critical distinction between the Changing-State Effect and the Deviant Effect lies in habituation. The Changing-State effect is resistant to habituation; the structural conflict between auditory and visual order processing persists as long as the sound varies. In contrast, the Deviant Effect diminishes over time. As the participant becomes accustomed to the presence of random deviants, the orienting response weakens, and the disruption to memory subsides [5, 14]. This suggests that the Deviant Effect is driven by a novelty detection mechanism (likely the Mismatch Negativity system, discussed in Section 6) that updates its predictive model.

## 2.3 The Tonal Loop: Material-Specific Interference

The interference effects of auditory tones are particularly pronounced when the working memory task itself involves tonal information. This has led to the proposal of a **Tonal Loop**, analogous to the Phonological Loop, dedicated to the retention of pitch and musical information [15].

### 2.3.1 Musicians vs. Non-Musicians

Experimental designs comparing musicians and non-musicians have revealed fascinating dissociations that support the existence of specialized memory stores.

- **Verbal Recall:** Irrelevant speech disrupts the recall of verbal items (digits/letters) but has a reduced effect on the recall of tonal sequences.
- **Tonal Recall:** Irrelevant music or changing tones significantly disrupts the recall of tonal sequences but has little effect on verbal recall [15, 16].

Crucially, irrelevant music disrupts tonal recall in *both* musicians and non-musicians. While musicians generally possess a higher baseline capacity for tonal memory (likely due to better encoding strategies), they are not immune to interference. The varying pitch contours of irrelevant music gain obligatory access to the tonal rehearsal loop, overwriting the pitch traces of the to-be-remembered melody [15].

This finding has significant implications for the user's query: if one wishes to disrupt a specific type of memory, the auditory disruptor should be matched to the content. To disrupt the memorization of a speech, play changing-state speech or tones. To disrupt the memorization of a melody or prosody, play changing-state music.

## 2.4 The Role of Intensity

For decades, it was assumed that the ISE was independent of intensity—that a whisper was as disruptive as a shout, provided it was audible. Recent pre-registered replication studies have

challenged this dogma. Results now indicate that both steady-state and changing-state sequences presented at higher intensities (e.g., **75 dB**) are significantly more disruptive than those presented at lower intensities (e.g., **45 dB**) [17].

This finding suggests a "general arousal" or "energetic masking" component to auditory disruption. High-intensity sound may degrade the signal-to-noise ratio of internal neural representations or simply induce a higher cognitive load through the effort required to inhibit the loud stimulus.

## 2.5 Developmental Trajectories

The susceptibility to auditory disruption appears to be a fundamental feature of the human cognitive architecture, present from childhood through adulthood. Studies comparing children (8–10 years), adolescents (11–12 years), and adults found that serial recall was similarly impaired by classroom noise in all age groups [7].

However, the *mechanism* may shift slightly with age. Children, possessing less developed inhibitory control (prefrontal cortex maturation), have historically shown evidence of greater susceptibility to attentional capture [18]. Yet, the core ISE—the structural clash of order processing—remains robust across the lifespan, indicating it is an unavoidable consequence of how the human brain organizes temporal information.

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## 3. Disruption of Memory Consolidation During Sleep

While working memory interference is a transient phenomenon, auditory stimulation during sleep can alter the permanent storage of information. Sleep is the critical window for **system memory consolidation**, the process by which labile memory traces in the hippocampus are transferred to the neocortex for long-term storage. This process relies on a precise choreography of neural oscillations: **Slow Waves (SWs)**, **Sleep Spindles**, and **Sharp-Wave Ripples**. Disruption of this choreography by auditory tones can abort the consolidation process.

### 3.1 The Physiology of Consolidation

During Non-Rapid Eye Movement (NREM) sleep, the brain generates high-amplitude Slow Oscillations (< 1 Hz). These oscillations orchestrate the timing of faster events:

1. **Slow Oscillation (SO):** A global wave that synchronizes large cortical areas.
2. **Sleep Spindles (10–16 Hz):** Thalamocortical bursts nested in the "up-state" of the SO, associated with synaptic plasticity.
3. **Hippocampal Ripples:** High-frequency bursts associated with the replay of specific memory traces.

The precise coupling of these three events (SO-Spindle-Ripple) is essential. The SO provides the "temporal frame," the spindle provides the "plasticity environment," and the ripple carries the "data."

## 3.2 Random Auditory Stimulation: The Mechanism of Disruption

The timing of auditory stimulation determines whether it acts as a medicine or a poison for memory.

- **Closed-Loop (Phase-Locked) Stimulation:** When pink noise pulses are delivered exactly at the peak of the SO "up-state," they enhance the wave amplitude and boost consolidation [19].
- **Open-Loop (Random) Stimulation:** When tones are delivered randomly, without regard for the brain's phase, they act as potent disruptors.

### 3.2.1 Stunting the Traveling Wave

A landmark study detailed in bioRxiv (2025) provides the most granular mechanism of disruption to date: the alteration of slow-wave traveling dynamics [3].

Slow waves are not stationary standing waves; they are traveling waves that propagate anterior-to-posterior (or vice versa) across the cortical mantle. This propagation is thought to facilitate the information transfer across distant brain regions.

The study found that random auditory stimulation "stunts" these traveling profiles:

- **Trajectory Length:** The distance the wave travels across the scalp is significantly shortened.
- **Spatial Spread:** The number of electrodes recruited into the wave decreases.
- **Frontal Impact:** The disruption is most severe in frontal regions, critical for declarative memory integration [3].

By physically interrupting the propagation of the wave, random tones uncouple the distant brain regions that need to communicate to consolidate a memory.

### 3.2.2 Reduction in Slow-Wave Sleep (SWS)

Beyond the dynamics of individual waves, random stimulation alters the macro-architecture of sleep. It leads to a marked decrease in the total time spent in **Slow-Wave Sleep (N3)** and a concurrent increase in lighter **N2 sleep** [3, 20]. Crucially, this reduction in low-frequency power persists even in the silent intervals *between* the tones, suggesting that the stimulation triggers a sustained shift in the neural environment—a "vigilance" state that prevents the brain from sinking into the depths required for optimal consolidation.

## 3.3 Differential Impact on Memory Types

The disruption caused by random auditory stimulation is specific to **declarative memory** (facts, events, figural pairs). In the studies reviewed, procedural memory (skills, motor tasks)

remained relatively intact despite the stimulation [3]. This aligns with the theory that declarative memory is heavily dependent on the hippocampal-neocortical dialogue mediated by slow waves, whereas procedural memory relies more on local synaptic changes in motor cortices or REM sleep.

### 3.4 The White Noise Debate: Masking vs. Disruption

In the context of sleep hygiene, "white noise" and "pink noise" are often touted as beneficial. The literature presents a complex, often contradictory picture [21, 22].

- **The Masking Hypothesis:** Continuous noise raises the auditory threshold, preventing sudden environmental sounds (sirens, door slams) from triggering an arousal response. By masking these "deviants," continuous noise preserves sleep continuity.
- **Stochastic Resonance:** Some evidence suggests that **pink noise** (which has a 1/f power spectrum closer to natural brain rhythms) might facilitate sleep stability via stochastic resonance [23, 24].
- **Disruptive Potential:** However, if the noise is too loud, or if it lacks the specific 1/f spectrum (e.g., harsh white noise), it can fragment sleep architecture. A systematic review noted that while subjective reports are often positive, objective measures of sleep quality (polysomnography) are mixed, and potential negative effects on hearing health and dependency exist [21].

Table 3.1: Impact of Auditory Stimulation on Sleep Physiology

Stimulation Type	Timing	Physiological Effect	Memory Consequence
Closed-Loop Pink Noise	Phase-locked to SO Up-State	Enhances SO amplitude; increases Spindle density.	Enhances Declarative Consolidation [19]
Random Tones	Asynchronous (Open-Loop)	Stunts traveling waves; reduces SWS duration; increases N2.	Disrupts Declarative Consolidation [3]
Continuous White Noise	Constant	Increases auditory threshold; masks environmental deviants.	Neutral/Variable (Prevents arousal) [21]

<b>Infrasound</b>	Chronic Exposure	Induces cortisol release; micro-arousals.	<b>Disrupts</b> Sleep Architecture & Recovery [25]
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## 4. Disruption of Memory Reconsolidation: The "Anti-Memory" Tone

Memory is not a "read-only" file. When a consolidated memory is retrieved (reactivated), it enters a temporary labile state where it must be "re-saved" or **reconsolidated**. During this window—typically lasting up to 6 hours—the memory is vulnerable to modification or erasure. While pharmacological blockades (e.g., propranolol) are well-known, **auditory distraction** serves as a potent behavioral intervention to disrupt this process.

### 4.1 The Reconsolidation Window and Prediction Error

The standard paradigm for studying reconsolidation involves Pavlovian Fear Conditioning. A subject (human or rodent) learns to associate a Tone (CS) with an unpleasant stimulus (US, e.g., shock). Later, presenting the Tone alone elicits a fear response (freezing).

To trigger reconsolidation, the memory must not only be retrieved but **destabilized**. This requires **Prediction Error**—a violation of expectation [4]. If the tone is presented and the expected shock *does not* occur (or occurs in a different timing/magnitude), the brain detects a mismatch and opens the molecular window to update the memory trace.

### 4.2 Auditory Distraction as an Interference Agent

Once the memory is labile, it requires cognitive resources to restabilize. If these resources are diverted by a competing task or stimulus, the reconsolidation fails.

- **The "Wash Bottle" Distractor:** In animal models, a sudden, irrelevant auditory/tactile stimulus (such as the sound and puff of a wash bottle) administered *during* the reactivation session (specifically after 1 minute of exposure) effectively disrupted the reconsolidation of a fear memory [26]. The animals tested days later showed significantly reduced freezing, indicating the fear memory had been weakened or erased.
- **Mechanism:** The distractor acts as a "resource thief." The amygdala and hippocampus are engaged in processing the prediction error of the fear memory. The sudden auditory distractor triggers a massive orienting response, hijacking the protein synthesis machinery or attentional resources needed to "save" the updated fear trace. The result is a failure to reconsolidate, and the trace decays [26].

### 4.3 Wake Targeted Memory Reactivation (TMR): A Double-Edged



## Sword

Targeted Memory Reactivation (TMR) usually refers to playing cues during sleep to boost memory. However, **Wake TMR**—playing the cues while the subject is awake—can have the opposite effect if paired with interference.

- **The Tetris Study:** In a study involving vocabulary learning, participants heard the auditory cues associated with the words while playing a demanding visuospatial game (Tetris) during a rest period. Unlike the sleep condition (which improved memory), this wakeful reactivation paired with cognitive load led to a **disruption** of the memory traces [27].
  - **Interpretation:** The auditory cues successfully reactivated the memories, rendering them labile. However, because the brain was busy playing Tetris, it could not reconsolidate them. The labile traces were left "hanging" and subsequently degraded—a phenomenon termed "reactivation-induced forgetting."
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## 5. Binaural Beats and Neural Entrainment: Modulation or Myth?

The commercial market is flooded with "Binaural Beat" audio tracks promising to enhance memory, focus, and relaxation. The premise is **brainwave entrainment**: presenting two slightly different frequencies (e.g., 400 Hz and 440 Hz) to each ear creates a perceived "beat" at the difference frequency (40 Hz). The theory assumes the brain will synchronize its firing to this 40 Hz rhythm (Gamma), inducing a state of high focus. However, scientific scrutiny reveals a landscape where these tones can often act as disruptors.

### 5.1 Mechanisms of Entrainment (Frequency Following Response)

The brain does exhibit a **Frequency Following Response (FFR)** to rhythmic auditory stimuli. Neural populations in the auditory cortex synchronize to the envelope of the sound [28, 29]. However, entraining the *auditory cortex* is not the same as entraining the *hippocampus* or the *prefrontal cortex* required for memory.

### 5.2 Evidence of Negative Effects and Disruption

Contrary to the enhancement claims, several rigorous studies report negative outcomes:

- **Impaired Serial Recall:** Immediate recall memory was found to be significantly **decreased** in participants listening to binaural beats compared to control conditions in some experiments [30].
- **Null Results:** A large number of studies report no significant difference between binaural beats and silence, suggesting that for many individuals, the "beat" is simply irrelevant noise that fails to modulate cognition [31, 32].

- **Cognitive Load Theory:** Processing the illusion of a binaural beat (which requires the integration of inputs from both ears in the superior olivary complex) consumes cognitive resources. If the primary task is difficult (e.g., a high-load N-back task), the addition of binaural beats acts as a secondary task, stealing resources and reducing performance [33, 34].

## 5.3 Frequency Mismatch

The disruption is often a result of **frequency mismatch**.

- **Theta/Delta Beats:** Low-frequency beats (Theta/Delta) are associated with sleep and drowsiness. Playing these frequencies during a task that requires vigilance (Working Memory) can induce fatigue and lower arousal, directly impairing performance [35].
- **Gamma/Beta Beats:** While sometimes helpful for vigilance, high-frequency beats can induce anxiety in sensitive individuals, which is also detrimental to memory performance [31].

**Table 5.1: Reported Effects of Binaural Beats on Memory**

Beat Frequency	Intended Effect	Reported Outcome in Literature	Mechanism of Failure/Success
<b>Delta (1-4 Hz)</b>	Sleep / Deep Relaxation	<b>Impairment</b> of active WM tasks; Increased fatigue.	Mismatch with task demands (Vigilance vs. Drowsiness) [35].
<b>Theta (4-8 Hz)</b>	Meditation / Memory	<b>Mixed/Null.</b> Some impairment in verbal recall.	Cognitive load of processing the beat [30].
<b>Beta (13-30 Hz)</b>	Focus / Alertness	<b>Variable.</b> Some improvement in vigilance; Anxiety in others.	Increases arousal, but effect is inconsistent [36].
<b>Gamma (40 Hz)</b>	Binding / Insight	<b>Mixed.</b> Some boost in N-back; Null in others.	Entrainment reliability is low in many subjects [32].

## 6. Infrasound: Pathological Disruption of Memory Hardware

Moving beyond the audible spectrum (20 Hz - 20 kHz), **infrasound** (< 20 Hz) represents a form of auditory stimulation with documented deleterious effects on memory, primarily mediated by physiological stress and structural damage rather than cognitive interference. This is relevant to industrial environments, wind turbines, and urban noise pollution.

### 6.1 Evidence from Animal Models

Studies utilizing rat models exposed to infrasound (e.g., **16 Hz at 130 dB**) have consistently shown significant impairment in **spatial learning and memory** (Morris Water Maze tasks) [6, 37]. The mechanisms identified are severe and structural:

- **Hippocampal Apoptosis:** Infrasound exposure induces oxidative stress and upregulation of pro-inflammatory cytokines (**IL-1 $\beta$** , **TNF- $\alpha$** ) specifically in the hippocampus [6, 38].
- **Glial Activation:** It triggers the activation of astrocytes and microglia. Specifically, it causes the opening of **Connexin 43 (Cx43)** hemichannels in astrocytes. This opening leads to an excessive, uncontrolled release of glutamate and ATP into the extracellular space.
- **Excitotoxicity:** The flood of glutamate leads to excitotoxicity, damaging neurons and impairing the synaptic plasticity (LTP) required for memory formation [6].
- **Neurogenesis Inhibition:** Infrasound has been shown to inhibit adult neurogenesis in the dentate gyrus and downregulate the **BDNF-TrkB** signaling pathway, effectively halting the brain's ability to generate new memory hardware [37].

### 6.2 Human Implications: Vibroacoustic Disease

While human studies rarely expose subjects to the high intensities used in animal models (130 dB), there is evidence that low-frequency noise (LFN) from industrial sources can cause **Vibroacoustic Disease (VAD)**. Symptoms include mental fatigue, lack of concentration, and reduced cognitive performance [25, 39].

- **Cognitive Load:** Even at lower decibels, LFN is processed by the brain. It acts as a constant stressor, elevating cortisol levels. High cortisol is known to be neurotoxic to the hippocampus, providing a pathway for chronic LFN to impair memory over time [40].
- **Fatigue:** The constant processing of low-frequency vibration accelerates the onset of mental fatigue, indirectly impairing working memory and vigilance [39].

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## 7. Neural Mechanisms of Auditory Disruption (Synthesis)

To synthesize the diverse findings across WM, Sleep, and Infrasound, we can identify common neural mechanisms that allow sound to disrupt memory.

## 7.1 Mismatch Negativity (MMN) and Resource Depletion

The brain continuously generates an internal probabilistic model of the auditory environment. When an incoming sound violates this model (a deviant), the auditory cortex generates a **Mismatch Negativity (MMN)** response [41, 42].

- **The Interrupt Signal:** The MMN serves as an involuntary "interrupt" signal. It triggers an attention switch from the internal task (memory rehearsal) to the external environment.
- **Frontal Lobe Engagement:** The MMN has a frontal component associated with the re-allocation of attention. Frequent elicitation of MMN (in a chaotic auditory environment) drains the limited resources of the prefrontal central executive. With the executive occupied by processing noise, the maintenance of memory traces falters [43].

## 7.2 Oscillatory Desynchronization

Memory relies on the precise coupling of brain rhythms (e.g., Theta-Gamma coupling for WM, Slow Wave-Spindle coupling for sleep).

- **Interference:** External auditory rhythms that do not match the brain's internal processing frequency can disrupt this coupling. Random tones during sleep dissociate the spindle from the slow wave [3].
- **Alpha Suppression:** Unexpected sounds trigger a desynchronization of Alpha rhythms (which usually inhibit irrelevant regions). This break in Alpha suppression allows "noise" to flood the processing centers, degrading the fidelity of the memory trace [44].

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## 8. Conclusion

The query "Can you use auditory tones to disrupt memory?" finds a resounding affirmative in the scientific literature. The disruption is not a unitary event but a sophisticated interference with the specific machinery of memory at various stages:

1. **In the Waking State: Changing-state tones** disrupt the serial ordering mechanisms of Working Memory, while **auditory deviants** hijack attention via the MMN response. This disruption is robust, affecting both musicians and non-musicians, and persists across the lifespan.
2. **In the Sleeping State: Random (open-loop) auditory stimulation** acts as a physiological disruptor, stunting the traveling slow waves essential for the dialogue between the hippocampus and neocortex. This effectively prevents the consolidation of declarative memories.
3. **In the Retrieval State: Auditory distractors** presented during the fragile window of reconsolidation can block the restabilization of memory traces, functioning as a

behavioral "erase" button for fear memories.

4. **In the Long Term: Infrasound** acts as a physical stressor, inducing neuroinflammation and excitotoxicity that degrades the structural integrity of the hippocampus.

Conversely, steady-state sounds (white noise) or precisely timed (closed-loop) stimulation can have neutral or beneficial effects. Thus, sound is a double-edged sword: a potential enhancer of neural rhythms when synchronized, but a potent weapon of disruption when random, changing, or structurally conflicting.

The implications for this are profound, spanning from the design of educational environments (where changing-state noise must be minimized) to the treatment of PTSD (where auditory disruption during reconsolidation offers therapeutic hope), and the regulation of industrial noise (where infrasound poses a silent threat to neural health).