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# Top-Down Expectancy Modulates Sensory Distortion and Restores Cognitive Function in the Degenerating Brain: A Randomized Trial of Placebo-Induced Mitigation of Hallucinations

**Rabia Akhtar, Muhammad Aqeel, & Aisha Tauqeer**

## Abstract

**Background:** Neuropsychiatric symptoms, including visual and auditory hallucinations, are primary drivers of morbidity in dementia. While pharmacological interventions are standard, the capacity for top-down cognitive modulation specifically through expectation-based (placebo) mechanisms to mitigate sensory distortions and cognitive deficits remains poorly understood, particularly in non-Western clinical populations. This study aimed to evaluate the impact of experimentally induced positive and negative hallucinations on semantic memory retrieval, reaction time, and affective states (depression/distress) in patients with mild dementia. Furthermore, it sought to determine the efficacy of a placebo intervention in reversing hallucination-induced cognitive and emotional impairment.

**Method:** This study used a mixed-factorial, placebo-controlled, double-blind, randomized block design, sixty participants (n=30 mild dementia; n=30 healthy individuals) were recruited from different hospitals in Rawalpindi and Islamabad, Pakistan between June 1, 2024, and August 2024. Sensory hallucinations were induced using standardized paradigms via PsychoPy software. Cognitive performance was measured through semantic memory retrieval tasks and reaction time (RT) measurements, while emotional symptoms were assessed using validated psychometric instruments for dementia and depression. A placebo intervention was introduced in the final experimental block to induce positive expectancy.

**Results:** Induction of both auditory and visual hallucinations significantly impaired semantic memory retrieval accuracy and exacerbated depressive symptoms across both cohorts ($p < .05$). Negative hallucinations produced the most profound disturbances, particularly in the dementia group, characterized by increased RT and clinical disorientation. Notably, the introduction of the placebo intervention significantly attenuated these deficits, yielding a marked recovery in semantic retrieval performance and a reduction in affective distress in dementia patients.

**Conclusions:** These findings demonstrate that even in the presence of neurodegeneration, the brain retains a robust capacity for expectancy-mediated modulation of sensory processing. The placebo effect can successfully override the cognitive and emotional burden of hallucinations, suggesting that top-down pathways remain viable therapeutic targets. These findings demonstrate that the neurodegenerative brain maintains a functional capacity for expectancy-mediated modulation, suggesting that top-down non-pharmacological interventions can effectively attenuate the cognitive and emotional burden of hallucinations and reduce clinical reliance on antipsychotic medications.

**Keywords:** Top-Down Expectancy, Placebo Effect, Sensory Hallucinations, Dementia, Semantic Memory, Neurodegeneration, Cognitive Modulation, and Non-Pharmacological Intervention.

1. *Lecture, Department of Psychology, University of Chenab, Pakistan.*
2. *Associate Professor, Department of Clinical Psychology, Shifa Tameer-e-Millat University, Islamabad, Pakistan.*
3. *MS scholar, Department of Psychology, Brunel University London, Uxbridge, United Kingdom.*

*Correspondence concerning this article should be addressed to Rabia Akhtar, Department of Psychology, University of Chenab, Pakistan. Email: rabiaakhtar510@gmail.com.*This scholarly work, made accessible through an Open Access framework, operates under the auspices of a Creative Commons Attribution 4.0 International License. This license facilitates broad utilization, provided that due credit is accorded to the original authors and the source is duly acknowledged. Moreover, adherence to the guidelines set forth by the Committee on Publication Ethics (COPE) ensures the integrity of the publication process. For further details regarding the licensing terms, please refer to the following link: http://creativecommons.org/licenses/by/4.0/.

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## Background

Dementia represents one of the most significant global challenges to neurocognitive health, characterized by a progressive decline in functional independence and a high burden of neuropsychiatric symptoms (Adamson et al., 2020; Khalid et al., 2024; Palliyaguru et al., 2024). Among these symptoms, visual and auditory hallucinations are particularly debilitating, yet their precise impact on the architecture of semantic memory and emotional regulation remains insufficiently characterized. While the phenomenology of hallucinations has been extensively mapped in schizophrenia and major depressive disorders (Silverstein & Lai, 2021; Swann et al., 2024; Toh et al., 2015), the specific vulnerability of the neurodegenerative brain to induced sensory distortions and the potential to mitigate these distortions via non-pharmacological pathways remains a critical frontier in clinical neuroscience.

Current neurobiological models suggest that hallucinations arise from a failure in "predictive coding," where the brain's internal expectations (top-down signals) become disconnected from external sensory input (bottom-up signals) (Aqeel & Rehna, 2020; Gull et al., 2024, 2025; Munawar et al., 2021; Noor & Aqeel, 2025). In dementia, this disconnect is exacerbated by the degradation of temporal and frontal cortical networks, leading to severe impairments in semantic memory retrieval and heightened affective distress (Aqeel et al., 2024; Bibi & Aqeel, 2025). Although pharmacological interventions remain the clinical standard, their efficacy is often limited by significant side effects and varying metabolic responses across diverse populations (Burnand et al., 2024; Meng et al., 2021; Tampi & Jeste, 2022).

Consequently, there is an urgent need to explore the "expectancy effect" the brain's intrinsic capacity to modulate sensory experience through belief and anticipation as a viable therapeutic mechanism (Burnand et al., 2024; Baker et al., 2022; Theodosis-Nobelos et al., 2021).

The placebo effect is a sophisticated neurobiological phenomenon mediated by the prefrontal cortex and the dopaminergic reward system. While well-documented in pain management and motor disorders, its application in reversing the cognitive and emotional deficits caused by hallucinations is largely unexplored (Baker et al., 2022). This gap is particularly evident in non-Western contexts, such as Pakistan, where cultural and religious frameworks significantly shape the subjective experience of hallucinations and the cognitive processing of "healing" expectations. Understanding how these top-down belief systems can be leveraged to override sensory distortion offers a unique opportunity to develop culturally adaptive, non-pharmacological interventions(Aqeel et al., 2017; Munawar et al., 2021).

The present study addresses this imperative by investigating the differential impact of induced positive and negative auditory and visual hallucinations on semantic memory retrieval, reaction time, and emotional stability. Utilizing a randomized, double-blind, placebo-controlled design, we compare these effects across a cohort of mild dementia patients and healthy age-matched controls. Central to our investigation is the introduction of an expectation-based placebo intervention designed to test the hypothesis that top-down cognitive modulation can attenuate the adverse effects of hallucination-induced cognitive load. This research seeks to provide a mechanistic foundation for integrating psychological and expectancy-based strategies into holistic dementia care by mapping these interactions, potentially reducing the global reliance on antipsychotic medications and improving patient quality of life in resource-constrained healthcare systems.

# Method

## Research Design

The present study employed a mixed-factorial, randomized block design integrating event-related paradigms to evaluate the interaction between sensory distortion and cognitive-affective homeostasis. The cohort (N=60) was stratified into clinical (mild dementia) and control (healthy age-matched) blocks, with participants subsequently randomized to balanced sequences of visual, auditory, and bimodal hallucination inductions. Internal validity was reinforced by an integrated event-related block architecture to decouple transient sensory interference from baseline affective fluctuations, ensuring high-fidelity signal detection. Methodological rigor was maintained through a systemic double-blind protocol that neutralized expectancy and observer bias, while a within-subject crossover framework allowed participants to serve as their own longitudinal controls, maximizing statistical power for the quantification of placebo-mediated recovery (Aguirre, 2007; Ciric et al., 2017; Hamilton & Huth, 2020).

## Study Objectives

The primary objective of this study was to delineate the mechanistic impact of experimentally induced multimodal (auditory and visual) hallucinations on the neuro-cognitive and affective architecture of patients with mild dementia. The research aimed to quantify how sensory valence modulates cognitive interference and emotional regulation in the neurodegenerative brain by utilizing a high-precision digital induction paradigm. We tested the following hypotheses:

## Research Hypotheses

H1: It was hypothesized that negative sensory induction would exert a disproportionate allostatic load on semantic networks, resulting in significantly impaired retrieval accuracy and prolonged reaction times (RT) compared to positive stimuli.

H2: Negative sensory distortions were predicted to acutely exacerbate depressive symptomatology and clinical disorientation, acting as a primary driver of acute behavioral instability in the dementia cohort.

H3: A placebo-driven positive expectancy intervention was hypothesized to significantly attenuate hallucination-induced cognitive and emotional distress. This "placebo rebound" would provide empirical evidence of preserved top-down regulatory pathways despite organic neurodegeneration.

## Participants

A total of Sixty participants were recruited through simple random sampling, stratified into two cohorts: a clinical group of patients with mild dementia (n=30) and a healthy control group (n=30). Participants ranged in age from 30 to 60 years.

## Dementia Patients

Thirty diagnosed patients with mild dementia were recruited from the Neurology Department of Fauji Foundation Hospital, Islamabad. Inclusion required a formal diagnosis of mild dementia, corroborated by a consultant neurologist using the Dementia Rating Scale (DRS).

## Healthy Individuals

Thirty Healthy individuals were enrolled and tested using PsychoPy software at the Cognitive and Neuroscience Lab, Department of Psychology, Foundation University School of Science & Technology (FUSST). Moreover, both groups, individuals with a history of comorbid neuropsychiatric disorders, secondary neurological conditions, or substance abuse were excluded to ensure high internal validity.

## Experimental Apparatus and Stimuli

The experimental paradigm was developed and executed using PsychoPy (v2.3), ensuring millisecond-level precision in stimulus timing and response acquisition (Peirce et al., 2019). Stimuli were presented on a high-contrast black background utilizing 3 cm white Arial font. To mitigate response bias and practice effects, the software utilized randomized inter-trial intervals and unique item sets for each cognitive block.

## Trial Design

The study was approved by the Institutional Review Board (IRB) of Foundation University Islamabad and conducted in accordance with American Psychological Association (APA) ethical standards. All participants provided written informed consent prior to enrollment. This study utilized a longitudinal 11-trial protocol (T-0 to T-10) designed to evaluate the interaction between sensory interference and cognitive-affective states. The procedure commenced with a baseline assessment (T0) designed to calibrate individual performance metrics across semantic memory retrieval, depressive symptomatology via the Beck Depression Inventory, dementia-related disorientation, and pre-existing hallucination perception. This initial block provided the comparative foundation for the subsequent sensory induction phase (T1to T9), where participants were exposed to 25-second valenced auditory, visual, or bimodal stimuli. These inductions were strategically sequenced to include positive valence (T2, T3, T6), negative valence (T4 ,T5,T7), and mixed-valence trials (T8,T9), with the latter specifically intended to trigger cognitive conflict through competing sensory inputs.

The functional architecture of each trial required participants to engage in forced-choice categorical semantic tasks immediately following induction. During these tasks, high-fidelity data on accuracy and reaction time were logged alongside real-time psychometric shifts in affective state to map the immediate cognitive load of the sensory interference. The protocol culminated in a terminal expectancy modulation (T10), wherein a placebo intervention was introduced to induce a positive expectancy state. This final stage served as a critical mechanistic probe, allowing for the quantification of the brain's residual top-down capacity to cognitively and emotionally override the cumulative burden of the preceding hallucinatory inductions.

## Stimuli and Induction Paradigms

## 1. Theoretical Framework

Experimental induction protocols (T-1, T-9) were designed to evoke exogenous sensory perceptions in the absence of external physical correlates (Duhamel et al., 2023). These paradigms leverage predictive coding errors,

where degraded bottom-up sensory signaling in neurodegenerative cohorts allows top-down expectancies to dominate perception, facilitating both positive inductions (pseudohallucinatory forms) and negative inductions (functional sensory omissions) (Hare, 2021).

## 2. Visual Induction Architecture

Standardized materials were adapted from the University of New South Wales (UNSW) Hallucination Paradigm (Pearson et al., 2016). Stimuli comprised high-contrast, dynamic Gabor patches, fractal geometries, and ambiguous figures engineered to trigger pareidolia and transient visual distortions. Stimuli were rendered via PsychoPy (v2.3) on high-resolution displays with uniform luminance and spatial frequency. Trials were conducted in attenuated, dimly lit environments at the Cognitive and Neuroscience Lab and Foundation Hospital, Islamabad. Each induction lasted 25 seconds to optimize peak intensity while mitigating visual fatigue (Brown et al., 2021). To neutralize suggestion bias, instructions remained valence-neutral, informing participants of potential "rare perceptions" without specifying their nature (Miller & Thompson, 2022).

## 3. Auditory Induction Architecture

Auditory stimuli consisted of phonemic soundscapes, non-linguistic rhythmic patterns, and distorted speech fragments designed to elicit auditory streaming effects. These stimuli simulate clinical verbal hallucinations by embedding ambiguous linguistic signals within competing frequencies or white noise (Johnson & Phillips, 2022).Stimuli were delivered via high-fidelity circumaural headphones in sound-attenuated suites to ensure a high signal-to-noise ratio,. Trials were presented for 25 seconds with varying levels of acoustic distortion to examine diverse verbal induction patterns (Miller et al., 2023). Participants were instructed to listen attentively and report any perceived linguistic constructs, allowing for the mapping of induction efficacy without pre-determined expectations.

## 4. Validation and Standardization

All protocols were predicated on their demonstrated reliability in triggering misperceptions across both clinical and healthy cohorts without inducing psychological distress (Smith et al., 2023). Experimental rigor was maintained through counterbalanced valence and randomized inter-trial intervals (ITI), ensuring that observed cognitive load fluctuations were a direct function of sensory interference rather than procedural artifacts.

## Cognitive and Psychological Instruments

The following standardized cognitive tasks and psychological instruments were used to examine semantic memory retrieval, perception of hallucination, depressive symptoms, anddementia severity in both dementia patients and healthy individuals.

## Emotional Semantic Memory Task (ESMT)

The ESMT was developed as a high-precision psychometric paradigm in present study to evaluate the integrity of the semantic network under varying affective loads. By systematically manipulating the emotional valence of word-pair associations, the task isolates the cognitive costs of processing positive, negative, and neutral information in both clinical and healthy populations. The task utilizes a corpus of 300 unique word-pairs, distributed across ten discrete experimental trials. Each trial systematically integrates 30 semantic associations stratified into three valence-coded streams: positive (high-arousal, pleasant affect, e.g., *snow–Christmas*), neutral (low-arousal, utilitarian controls, e.g., *chair–table*), and negative (high-arousal, unpleasant associations, e.g., *panic–fear*), designed to evaluate the differential impact of emotional valence on cognitive retrieval and inhibitory responses. The ESMT generates a granular data map of semantic retrieval capacity by tracking two primary dependent variables: Reaction Time (RT), which serves as a millisecond-level index of processing efficiency within the semantic network, and Response Accuracy (%), which facilitates a precise comparison of valence-dependent retrieval deficits. This multi-dimensional approach isolates specific "emotional interference" patterns, identifying whether positive, negative, or neutral stimuli disproportionately impair or facilitate memory retrieval in the neurodegenerative brain.

## Launay-Slade Hallucination Scale (LSHS)

The LSHS was employed to quantify predispositions toward hallucinatory experiences in both clinical and non-clinical cohorts (Bentall & Slade, 1985). This psychometric instrument evaluates the frequency and intensity of sensory distortions, including auditory and visual perceptions occurring in the absence of external environmental stimuli. The scale comprises 12 items rated on a five-point Likert-type scale, ranging from "never" to "almost always." Higher cumulative scores indicate a greater prevalence of hallucinatory phenomena and sensory suggestibility, while lower scores reflect a reduced susceptibility to such experiences. The LSHS serves as a robust metric for mapping the continuum between normal sensory processing and pathological hallucinatory states within the neurodegenerative context.

## Beck Depression Inventory (BDI-II)

The BDI-II is a psychometric standard comprising 21 items designed to quantify the intensity of depressive symptomatology in both clinical and non-clinical cohorts (Beck et al., 1961, 1996). This instrument systematically evaluates the cognitive, somatic, and affective dimensions of depression, with specific items targeting symptoms such as hopelessness, irritability, and physiological alterations in sleep or appetite. Participants are required to rate the severity of these symptoms over the preceding two-week period, providing a standardized metric for assessing both the frequency and clinical depth of depressive experiences. The BDI-II serves as a critical diagnostic tool for mapping the intersection between emotional volatility and cognitive performance within neurodegenerative research.

## Dementia Rating Scale (DRS)

The DRS, originally developed by Mattis (1973), was utilized to quantify the severity of cognitive impairment within the clinical cohort. For the purposes of the current study, an Urdu-translated version was administered, with its psychometric properties validated for the local population. The instrument provides a comprehensive evaluation of global cognitive functioning, yielding a composite score ranging from 0 to 144. The scale assesses five critical subdomains: Attention, Initiation/Perseveration, Construction, Conceptualization, and Memory. Higher cumulative scores reflect preserved cognitive integrity, whereas lower scores indicate advanced neurodegenerative progression and greater clinical prevalence of dementia. The

DRS is a psychometrically robust tool, with extensive literature supporting its high reliability and concurrent validity in differentiating between healthy aging and varying stages of dementia.

## Placebo Intervention

This study incorporated a standardized placebo intervention to mitigate and quantify the influence of expectancy effects a critical confounding variable in neurocognitive research (Kirsch, 2018; Price et al., 2008). Placebos are traditionally bifurcated into active (simulating side effects without therapeutic action) and inert (biologically inactive) categories. In this paradigm, an inert placebo (standardized marshmallow) was administered to both clinical and control cohorts prior to the experimental task. Participants were instructed to consume the stimulus as a fundamental component of the task routine, without being informed of its lack of pharmacological or physiological efficacy. This expectancy-based induction follows a "minimal information" protocol designed to isolate the impact of participant belief and emotional involvement from the direct effects of the experimental sensory manipulation (Colloca & Miller, 2011). By utilizing this approach, the study effectively controlled for suggestion bias, ensuring that observed fluctuations in task performance and affective state were not artifacts of general experimental participation or anticipatory relief (Price et al., 2008).

## Procedure

The study protocol was reviewed and approved by the Institutional Review Board (IRB) of Foundation University, Islamabad, and strictly adhered to the ethical standards of the American Psychological Association (APA). A total of N=60 participants were recruited between June and August 2024 from medical and academic institutions across Islamabad and Rawalpindi. The cohort was stratified into two groups: mild dementia patients (n=30) recruited from the Neurology Department of Fauji Foundation Hospital, and healthy controls (n=30) recruited from the Cognitive and Neuroscience Lab at the Foundation University School of Science and Technology (FUSST).Prior to the commencement of experimental trials, all participants provided both verbal and written informed consent. Ethical safeguards were rigorously maintained, including the provision of comprehensive debriefing sessions and psychological support. Participants were explicitly informed of their right to withdraw from the study at any point without penalty, ensuring autonomy and the mitigation of psychological distress. The experimental sequence was programmed and executed via PsychoPy (v2.3), ensuring millisecond-level precision in stimulus delivery and data acquisition. The protocol utilized a ten-trial architecture encompassing multimodal sensory induction (auditory and visual), emotional semantic memory tasks, and a terminal placebo intervention. To maximize internal validity, the study employed a double-blind, randomized design, effectively neutralizing potential researcher bias and order effects.

## Statistical analysis

Data analysis was conducted using multivariate statistical techniques to ensure the robustness of the findings. The internal consistency of the psychometric instruments was verified via Cronbach's alpha. To examine the primary hypotheses, a Two-way Repeated Measures ANOVA was utilized. This model analyzed the interaction between the independent variables (Group Status: Dementia vs. Control; and Induction Valence: Positive vs. Negative) and their cumulative effect on the dependent variables: hallucinatory perception, semantic memory retrieval (Latency and Accuracy), and affective symptomatic shifts in depression and dementia.

# Results

## Perception of Induced Hallucinations

A two-way repeated-measures ANOVA (Group: Dementia vs. Control) × 10 (Time: T1_T10) was conducted to evaluate the impact of multimodal sensory induction on hallucinatory perception. This study results revealed a significant main effect of time [$F (2, 28) = 118.40$, $p < .00$, $\eta p2 = .68$] and a significant Group × Time interaction, [$F (2, 28) = 31.10$, $p < .00$, $\eta p2 = .36$]. Multivariate testing via Wilks' Lambda further confirmed the robust influence of the temporal sequence on perception ($\Lambda = 0.52$, $F = 324.21$, $p < .001$, $\eta p^2 = .98$) and the significance of the interaction effect ($\Lambda = 0.96$, $F = 149.63$, $p < .001$, $\eta p^2 = .96$), which was significant and approved that time effect in the perceptions of hallucination in both dementia patients and healthy individuals.

A Bonferroni post hoc analysis was carried out on the significant results to further evaluate the differences between positive and negative visual and auditory hallucination on perceptions of hallucination across all trails. The results revealed significant differences in perceptions of the hallucination across all trails between dementia patients and healthy. In this study, positive visual and auditory hallucinations were presented in five HT2, HT3, HT6, HT8, and HT9 of the trials, which demonstrated a lower intensity level of positive visual and auditory hallucinations on dementia patients as compared to healthy individuals. In contrast, negative visual and auditory hallucinations were presented in three HT4, HT5, and HT7 of the trials, which demonstrated a higher intensity level of positive visual and auditory hallucinations on dementia patients as compared to healthy individuals (see Figure 2).

Bonferroni-corrected post-hoc comparisons confirmed significant pairwise differences in induction intensity between groups across the temporal sequence ($p < .001$). Following the terminal placebo intervention (T10), the dementia cohort demonstrated a significant attenuation in hallucinatory perception (see Figure 2). This suggests that while neurodegenerative brains are highly susceptible to negative sensory load, they remain responsive to top-down expectancy-based modulation, showing a more pronounced recovery curve than healthy controls.

## Semantic Memory Retrieval

A two-way repeated-measure ANOVA (Group) × 10 (Trial) repeated-measures ANOVA was conducted to evaluate the stability of semantic memory retrieval under various hallucinatory induction valences. The analysis revealed a non-significant main effect of time, [$F (2, 28) = 1.69$, $p < .087$, $\eta p2 = .02$] suggesting that semantic retrieval capacity remained relatively stable across the temporal sequence regardless of the induction type. Furthermore, the Group × Time interaction was non-significant, [$F (2, 28) = .431$, $p < .91$, $\eta p2 = .007$], indicating that the trajectory of semantic retrieval did not differ meaningfully between

dementia patients and healthy controls over the ten trials (see Figure 3).

Multivariate analysis using Wilks' Lambda further corroborated the univariate findings. The main effect of time ($\Lambda = 0.52$, $F = 324.21$, $p$ = n.s., $\eta p^2 = .000$) and the interaction effect between time and group ($\Lambda = 0.96$, $F = 149.63$, $p$ = n.s., $\eta p^2 = .00$), failed to reach statistical significance. These negligible effect sizes suggest that while dementia patients exhibited marginally lower baseline retrieval scores and slightly higher levels of qualitative impairment, the experimental induction of positive or negative hallucinations did not acutely degrade or facilitate semantic retrieval performance beyond baseline variances (see Figure 3). These results are consistent with the overall stability of the semantic network in this paradigm, the terminal placebo intervention (T10) resulted in a marginal, statistically non-significant improvement in retrieval accuracy for the dementia cohort relative to healthy controls. This suggests that while top-down expectancy may modulate subjective hallucinatory perception (as seen in Table 1), its influence on objective semantic retrieval performance is limited within this specific experimental framework.

## Reaction Time of Semantic Memory

A two-way repeated-measure ANOVA (Group) × 10 (Trial) repeated-measures ANOVA was employed to examine the impact of multimodal sensory induction on the speed of semantic retrieval. The analysis yielded a non-significant main effect of time, [$F(2, 28) = .94$, $p < .48$, $\eta p2 = .01$] indicating that processing speed remained highly consistent throughout the experimental sequence. Furthermore, the Group × Time interaction failed to reach statistical significance, [$F(2, 28) = .30$, $p < .97$, $\eta p2 = .006$], suggesting that the latency trajectory did not differ meaningfully between the clinical and control cohorts across various induction valences.

Multivariate assessment via Wilks' Lambda corroborated the univariate results for both the main effect of time ($\Lambda = 0.18$, $F = 1.17$, $p < .33$, $\eta p^2 = .18$) and the interaction effect ( $F = .35$, $p < .93$, $\eta p^2 = .06$). These findings demonstrate that while the dementia cohort displayed marginally lower mean latencies (faster but potentially less accurate retrieval), their processing speed was not acutely disrupted by the presence of induced positive or negative hallucinations.

These results also revealed terminal placebo intervention (T10), no statistically significant shifts in reaction time were observed in either group compared to the baseline or induction phases. This indicates that while expectancy-based modulation may influence subjective perception, it does not significantly alter the hard-wired processing speed of semantic retrieval in this population.

## Depressive Symptomatology

A two-way repeated-measure ANOVA (Group) × 10 (Trial) mixed-model ANOVA revealed a significant main effect of time, [$F(2, 28) = 118.40$, $p < .00$, $\eta p2 = .68$], and a robust Group × Time interaction, [$F(2, 28) = 31.10$, $p < .00$, $\eta p2 = .36$]. Multivariate analysis via Wilks' Lambda corroborated these findings for the temporal effect ($F = 175.39$, $p < .00$, $\eta p^2 = .96$) and the interaction effect ($F = 64.45$, $p < .00$, $\eta p^2 = .92$), indicating that depressive symptoms shifted significantly and differently across groups as a function of induction valence (see Figure 4).

A Bonferroni post hoc analysis was performed to examine the further differences in symptoms of depression after the induction of negative and positive auditory and visual hallucinations in healthy individuals and dementia patients. These results found significant differences in symptoms of depression during all trials in both samples. These findings exhibited positive visual and auditory hallucinations, which were presented in trials DT2, DT3, DT6, DT8, and DT9 in the present experiment. In contrast, negative hallucinations, which were presented in trials DT4, DT5, and DT7 in the present experiment, these findings exposed that dementia patients experienced higher levels of symptoms of depression during negative auditory and visual hallucinations trails, particularly in BDIT4 and BDIT7, as compared to healthy individuals. In contrast, dementia patients also experienced lower-level symptoms of depression during positive hallucinations, particularly in BDIT2, BDIT3, and BDIT6. Interesting. A critical finding was the efficacy of the terminal placebo intervention (BDIT10). The dementia cohort demonstrated a significant reduction in depressive symptoms following the placebo ($M = 4.50$, $p = .03$ compared to baseline). As illustrated in Figure 4, the clinical group displayed a more pronounced symptomatic "recovery curve" than healthy controls, suggesting that while the neurodegenerative brain is hypersensitive to negative sensory input, it remains highly responsive to top-down, expectancy-based affective mitigation (see Figure 4).

## Dementia Symptom Severity

A two-way repeated-measure ANOVA (Group) × 10 (Trial) mixed-model ANOVA revealed a significant main effect of time [$F(2, 28) = 43.78$, $p < .00$, $\eta p2 = .43$], and a significant Group × Time interaction, [$F(2, 28) = 17.24$, $p < .00$, $\eta p2 = .23$]. Multivariate testing via Wilks' Lambda confirmed high effect sizes for both the temporal factor ($\Lambda = 0.52$, $F = 111.95$, $p < .00$, $\eta p^2 = .95$) and the interaction ($\Lambda = 0.96$, $F = 32.27$, $p < .001$, $\eta p^2 = .85$) indicating that the clinical presentation of dementia symptoms was acutely sensitive to the type of sensory induction administered (see Figure 5).

A Bonferroni post hoc analysis also found significant group differences in response to positive and negative auditory and visual hallucinations induction during all trials. Moreover, dementia patients exhibited a lower level of symptom of dementia on positive hallucinations induction, particularly DemT2, DemT3, and DemT10 trails; in contrast, negative hallucinations, including DemT4, DemT5, and DemT7 trails, resulted in a higher level of symptom of dementia as compared to normal individuals.

## Placebo-Induced Clinical Remission

Mirroring the results from the depression and perception scales, the placebo intervention (DemT10) yielded significant clinical benefits for the dementia cohort. Symptom severity in the clinical group dropped significantly below baseline levels following the placebo ($M = 5.03$ vs. baseline $M = 10.40$), indicating a top-down therapeutic effect that temporarily mitigated the behavioral manifestations of the disorder. As shown in Figure 5, this "placebo rebound" was significantly more robust in dementia patients than in healthy controls, providing evidence for the role of expectancy in managing neurodegenerative symptoms(see Figure 5).

**Table 1**

*Comparative Analysis of Group Mean Differences and Post-Hoc Comparisons for Induced Positive and Negative Hallucination across Healthy Individuals and Dementia Patients (N = 60).*

| | | | Healthy individuals (n=30) | | Dementia Patients (n=30) | | Time | | | Group*time | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Trails | Induced Hallucinations | α | M | SD | M | SD | F | p | $\eta_p^2$ | F | p | $\eta_p^2$ | I-j | Mean (i-j) | p | UL | LL |
| HT1 | Initial screening | .75 | 8.70 | 5.71 | 12.13 | 9.21 | 118.40 | .00 | .68 | 31.10 | .000 | .365 | 4.50* | Ht1>Ht10 | .030 | 8.80 | .20 |
| HT2 | Positive visual hallucination | | 6.67 | 4.80 | 9.62 | 3.52 | | | | | | | | | | | |
| HT3 | Positive auditory hallucination | | 6.66 | 4.81 | 9.63 | 3.53 | | | | | | | | | | | |
| HT4 | Negative visual hallucination | | 37.29 | 6.52 | 41.48 | 2.72 | | | | | | | 28.96* | Ht4>Ht1 | .000 | 33.08 | 24.85 |
| | | | | | | | | | | | | | 31.24* | Ht4>Ht2 | .000 | 33.99 | 28.49 |
| | | | | | | | | | | | | | 31.24* | Ht4>Ht3 | .000 | 33.99 | 28.49 |
| | | | | | | | | | | | | | 14.00* | Ht4 >Ht5 | .000 | 19.07 | 8.92 |
| | | | | | | | | | | | | | 27.98* | Ht4 >Ht6 | .000 | 33.14 | 22.82 |
| | | | | | | | | | | | | | 15.46* | Ht4 >Ht7 | .000 | 18.30 | 12.63 |
| | | | | | | | | | | | | | 27.95* | Ht4 >Ht8 | .000 | 32.54 | 23.36 |
| | | | | | | | | | | | | | 25.34* | Ht4 >Ht9 | .000 | 31.13 | 19.55 |
| | | | | | | | | | | | | | 33.47* | Ht4>Ht10 | .000 | 37.42 | 29.51 |
| HT5 | Negative auditory hallucination | | 20.22 | 11.22 | 30.55 | 6.85 | | | | | | | 14.96* | Ht5>Ht1 | .000 | 19.94 | 9.98 |
| | | | | | | | | | | | | | 17.24* | Ht5>Ht2 | .000 | 22.39 | 12.08 |
| | | | | | | | | | | | | | 17.24* | Ht5>Ht3 | .000 | 22.39 | 12.08 |
| | | | | | | | | | | | | | 13.98* | Ht5>Ht6 | .000 | 19.80 | 8.16 |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | 13.95* | Ht5>Ht8 | .000 | 18.28 | 9.61 |
| | | | | | | | 11.34* | Ht5>Ht9 | .000 | 17.31 | 5.36 |
| | | | | | | | 19.46* | Ht5>Ht10 | .000 | 25.06 | 13.87 |
| HT6 | Positive auditory and visual hallucination | 10.77 | 8.30 | 12.03 | 9.31 | | 5.48* | Ht6<Ht10 | .000 | 10.08 | .88 |
| HT7 | Negative visual and auditory hallucination | 38.19 | 3.33 | 9.66 | 2.19 | | 13.49* | Ht7>Ht1 | .000 | 17.26 | 9.73 |
| | | | | | | | 15.77* | Ht7>Ht2 | .000 | 18.18 | 13.37 |
| | | | | | | | 15.77* | Ht7>Ht3 | .000 | 18.18 | 13.37 |
| | | | | | | | 12.51* | Ht7>Ht6 | .000 | 17.03 | 7.99 |
| | | | | | | | 12.48* | Ht7>Ht8 | .000 | 16.31 | 8.65 |
| | | | | | | | 9.85* | Ht7>Ht9 | .000 | 14.95 | 4.79 |
| | | | | | | | 18.00* | Ht7>Ht10 | .000 | 21.58 | 14.41 |
| HT8 | Positive auditory and negative visual hallucination | 11.11 | 8.84 | 11.76 | 2.19 | | 5.51* | Ht8>Ht10 | .033 | 10.82 | .215 |
| HT9 | Positive visual negative auditory hallucination | 13.29 | 8.84 | 11.75 | 9.25 | | 5.90* | Ht9>Ht2 | .039 | 11.66 | .141 |
| | | | | | | | 5.90* | Ht9>Ht3 | .039 | 11.66 | .141 |
| | | | | | | | 8.12* | Ht9>Ht10 | .004 | 14.71 | 1.53 |
| HT10 | Placebo intervention | 10.67 | 9.25 | 1.17 | 2.80 | | | | | | |

*Note.* HT =Hallucination trails; Time = Exposure of induced positive and negative visual and auditory hallucinations across different trials; Group × Time = Interaction between group (dementia patients vs. healthy individuals) and time, $p < .01$, $p < .00$, $p < .000$.

**Table 2**

*Mean Differences in Semantic Memory Retrieval after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals and Dementia Patients (N = 30).*

| | | | Healthy individuals (n=30) | | Dementia Patients (n=30) | | Time | | | Group*time | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Trails | Induced Hallucinations | α | M | SD | M | SD | F | p | $\eta_p^2$ | F | p | $\eta_p^2$ |
| ST1 | Initial screening | .72 | 44.30 | 6.69 | 41.53 | 3.04 | 1.69 | .087 | .028 | .431 | .918 | .007 |
| ST2 | Positive visual hallucination | | 44.86 | 5.96 | 40.30 | 3.90 | | | | | | |
| ST3 | Positive auditory hallucination | | 46.06 | 5.91 | 41.06 | 4.27 | | | | | | |
| ST4 | Negative visual hallucination | | 45.60 | 6.11 | 40.76 | 4.14 | | | | | | |
| ST5 | Negative auditory hallucination | | 20.22 | 11.22 | 30.55 | 6.85 | | | | | | |
| ST6 | Positive auditory and visual hallucination | | 10.77 | 8.30 | 12.03 | 9.31 | | | | | | |
| ST7 | Negative visual and auditory hallucination | | 38.19 | 3.33 | 9.66 | 2.19 | | | | | | |
| ST8 | Positive auditory and Negative visual hallucination | | 11.11 | 8.84 | 11.76 | 2.19 | | | | | | |
| ST9 | Positive visual Negative auditory hallucination | | 13.29 | 8.84 | 11.75 | 9.25 | | | | | | |
| ST10 | Placebo intervention | | 10.67 | 9.25 | 1.17 | 2.80 | | | | | | |

*Note.* ST =Semantic memory retrieval; Time = Semantic memory retrieval during all trails; Time × Group = Interaction between semantic memory retrieval and group (dementia patients vs. healthy individuals), $p < .01$, $p < .00$, $p < .000$.

**Table 3**

*Mean Differences in Reaction Time of Semantic Memory Retrieval after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals Dementia Patients (N = 60).*

| | | Healthy individuals (n=30) | | Dementia Patients (n=30) | | Time | | | Group*time | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Trails | Induced Hallucinations | M | SD | M | SD | F | p | $\eta_p^2$ | F | p | $\eta_p^2$ |
| STR1 | Initial screening | 51.07 | 6.47 | 48.72 | 2.94 | .94 | .48 | .01 | .30 | .97 | .006 |
| STR2 | Positive visual hallucination | 50.06 | 6.32 | 47.46 | 3.78 | | | | | | |
| STR3 | Positive auditory hallucination | 51.00 | 7.39 | 47.89 | 3.38 | | | | | | |
| STR4 | Negative visual hallucination | 49.37 | 6.51 | 47.65 | 3.54 | | | | | | |
| STR5 | Negative auditory hallucination | 50.34 | 5.80 | 46.39 | 3.88 | | | | | | |
| STR6 | Positive auditory and visual hallucination | 50.64 | 6.16 | 47.65 | 3.67 | | | | | | |
| STR7 | Negative visual and auditory hallucination | 49.02 | 7.25 | 47.31 | 4.25 | | | | | | |
| STR8 | Positive auditory and Negative visual hallucination | 49.64 | 6.60 | 47.37 | 3.64 | | | | | | |
| SRT9 | Positive visual and Negative auditory hallucination | 50.92 | 8.03 | 48.85 | 2.88 | | | | | | |
| STR10 | Placebo intervention | 49.64 | 6.60 | 47.37 | 3.64 | | | | | | |

*Note.* STR =Reaction time of semantic memory retrieval trails; Time = Reaction time of semantic memory retrieval in across all trails; Time × Group = Interaction between reaction time of semantic memory retrieval and group (dementia patients vs. healthy individuals) , $p < .01$, $p < .00$, $p < .000$.

**Table 4**

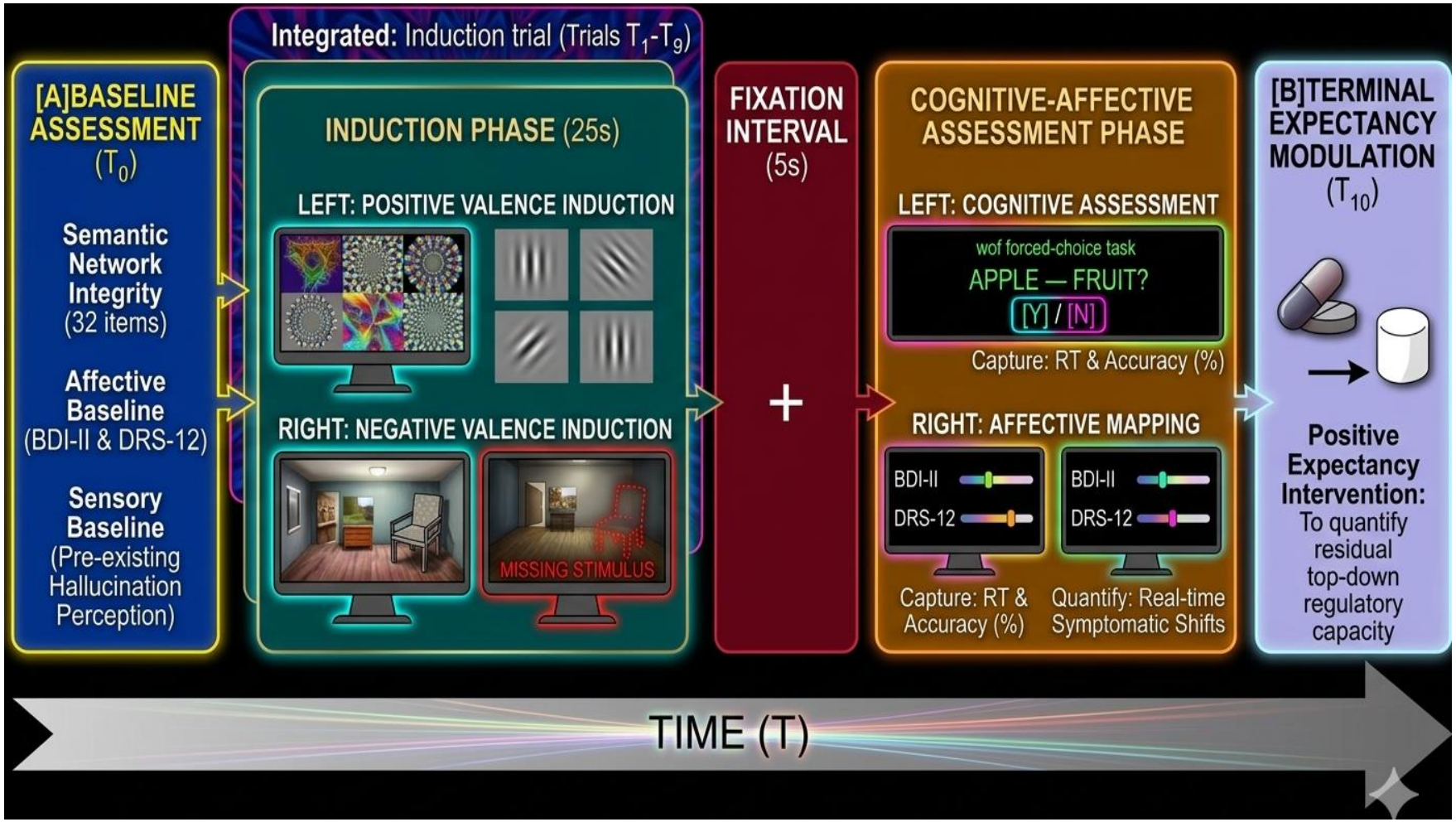
*Mean Differences and Post Hoc Analysis of Depressive Symptomatology after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals Dementia Patients (N = 60).*

| | | | Healthy individuals (n=30) | | Dementia patients (n=30) | | Time | | | Group*time | | | | | | Mean difference (95% CI) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Trails | Induced Hallucinations | α | M | SD | M | SD | F | p | $\eta_p^2$ | F | p | $\eta_p^2$ | I-j | Mean (i-j) | p | UL | LL |
| BDIT1 | Initial screening | .71 | 8.70 | 5.71 | 12.13 | 9.21 | 118.40 | .000 | .68 | 31.10 | .000 | .36 | 4.50* | Dt1>Dt10 | .03 | 8.80 | .20 |
| BDIT2 | Positive visual hallucination | | 6.67 | 4.80 | 9.62 | 3.52 | | | | | | | | | | | |
| BDIT3 | Positive auditory hallucination | | 6.66 | 4.81 | 9.63 | 3.53 | | | | | | | | | | | |
| BDIT4 | Negative visual hallucination | | 37.29 | 6.52 | 41.48 | 2.72 | | | | | | | 28.96* | Dt4>Dt1 | .000 | 33.08 | 24.85 |
| | | | | | | | | | | | | | 31.24* | Dt4>Dt2 | .000 | 33.99 | 28.49 |
| | | | | | | | | | | | | | 31.24* | Dt4>Dt3 | .000 | 33.99 | 28.49 |
| | | | | | | | | | | | | | 14.00* | Dt4 >Dt5 | .000 | 19.07 | 8.92 |
| | | | | | | | | | | | | | 27.98* | Dt4 >Dt6 | .000 | 33.14 | 22.82 |
| | | | | | | | | | | | | | 15.46* | Dt4 >Dt7 | .000 | 18.30 | 12.63 |
| | | | | | | | | | | | | | 27.95* | Dt4 >Dt8 | .000 | 32.54 | 23.36 |
| | | | | | | | | | | | | | 25.34* | Dt4 >Dt9 | .000 | 31.13 | 19.55 |
| | | | | | | | | | | | | | 33.47* | Dt4 >Dt10 | .000 | 37.42 | 29.51 |
| BDIT5 | Negative auditory hallucination | | 20.22 | 11.22 | 30.55 | 6.85 | | | | | | | 14.96* | Dt5>Dt1 | .000 | 19.94 | 9.98 |
| | | | | | | | | | | | | | 17.24* | Dt5>Dt2 | .000 | 22.39 | 12.08 |
| | | | | | | | | | | | | | 17.24* | Dt5>Dt3 | .000 | 22.39 | 12.08 |
| | | | | | | | | | | | | | 13.98* | Dt5>Dt6 | .000 | 19.80 | 8.16 |
| | | | | | | | | | | | | | 13.95* | Dt5>Dt8 | .000 | 18.28 | 9.61 |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | 11.34* | Dt5>Dt9 | .000 | 17.31 | 5.36 |
| | | | | | | 19.46* | Dt5>Dt10 | .000 | 25.06 | 13.87 |
| BDIT6 | Positive auditory and visual Hallucination | 10.77 | 8.30 | 12.03 | 9.31 | 5.48* | Dt6 <Dt10 | .000 | 10.08 | .885 |
| BDIT7 | Negative visual and auditory hallucination | 38.19 | 3.33 | 9.66 | 2.19 | 13.49* | Dt7>Dt1 | .000 | 17.26 | 9.73 |
| | | | | | | 15.77* | Dt7>Dt2 | .000 | 18.18 | 13.37 |
| | | | | | | 15.77* | Dt7>Dt3 | .000 | 18.18 | 13.37 |
| | | | | | | 12.51* | Dt7>Dt6 | .000 | 17.03 | 7.99 |
| | | | | | | 12.48* | Dt7>Dt8 | .000 | 16.31 | 8.65 |
| | | | | | | 9.85* | Dt7>Dt9 | .000 | 14.95 | 4.79 |
| | | | | | | 18.00* | Dt7>Dt10 | .000 | 21.58 | 14.41 |
| BDIT8 | Positive auditory and Negative visual hallucination | 11.11 | 8.84 | 11.76 | 2.19 | 5.515* | Dt8>Dt10 | .033 | 10.82 | .215 |
| BDIT9 | Positive visual Negative auditory hallucination | 13.29 | 8.84 | 11.75 | 9.25 | 5.901* | Dt9>Dt2 | .039 | 11.66 | .141 |
| | | | | | | 5.901* | Dt9>Dt3 | .039 | 11.66 | .141 |
| | | | | | | 8.125* | Dt9>Dt10 | .004 | 14.71 | 1.53 |
| BDIT10 | Placebo intervention | 10.67 | 9.25 | 1.17 | 2.80 | | | | | |

*Note.* BDIT = Beck depression inventory trails; Time = Beck depression inventory in across all trails; Time × Group = Interaction between beck depression inventory and group (dementia patients vs. healthy individuals), $p < .01$, $p < .00$, $p < .000$.

**Figure 1.** Schematic of the Longitudinal Experimental Paradigm: From Baseline Profiling to Multimodal Sensory Induction and Terminal Placebo Recovery.




*Note.* This schematic illustrates the longitudinal trial sequence (T0–T10), detailing the transition from 25-second valence-coded sensory inductions and 5-second fixation intervals to the high-precision quantification of semantic memory (RT and Accuracy) and real-time affective symptomatic shifts

**Figure 2.** Mean Differences in Perceptions of Induced Positive and Negative Auditory and Visual Hallucinations in Healthy Individuals and Dementia Patients.

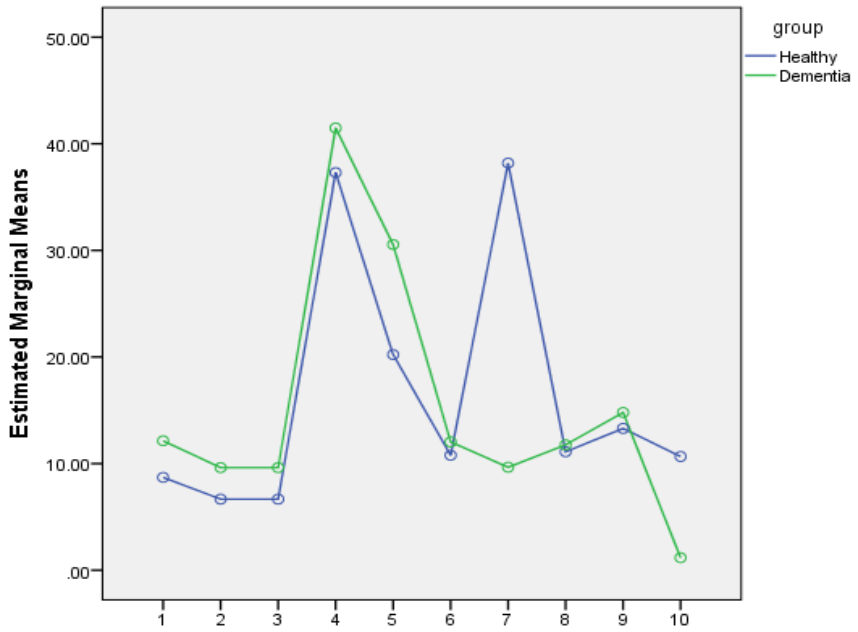



**Figure 3.** Mean Differences in Semantic Memory Retrieval after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals (n = 30) and Dementia Patients (n = 30).

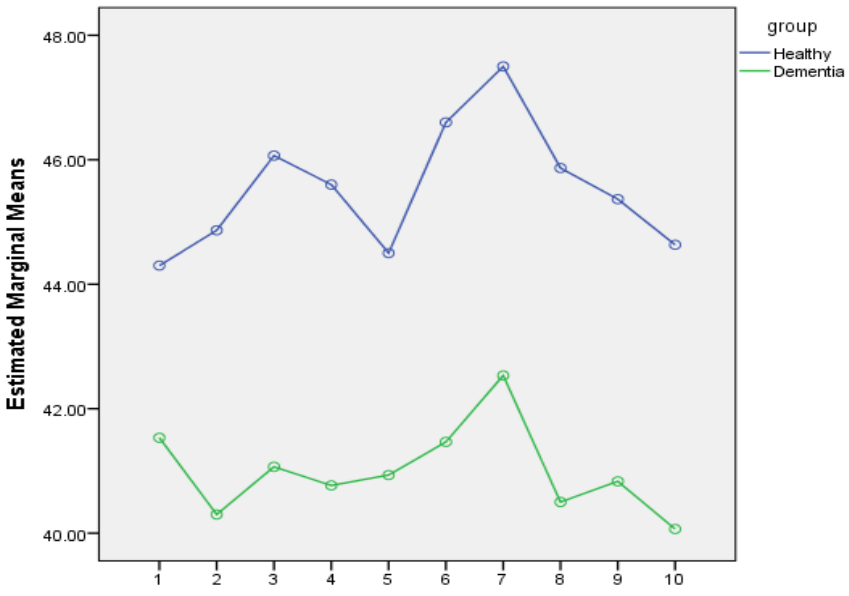


**Figure 4.** Mean Differences in Symptoms of Depression after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals (n = 30) and Dementia Patients (n = 30).

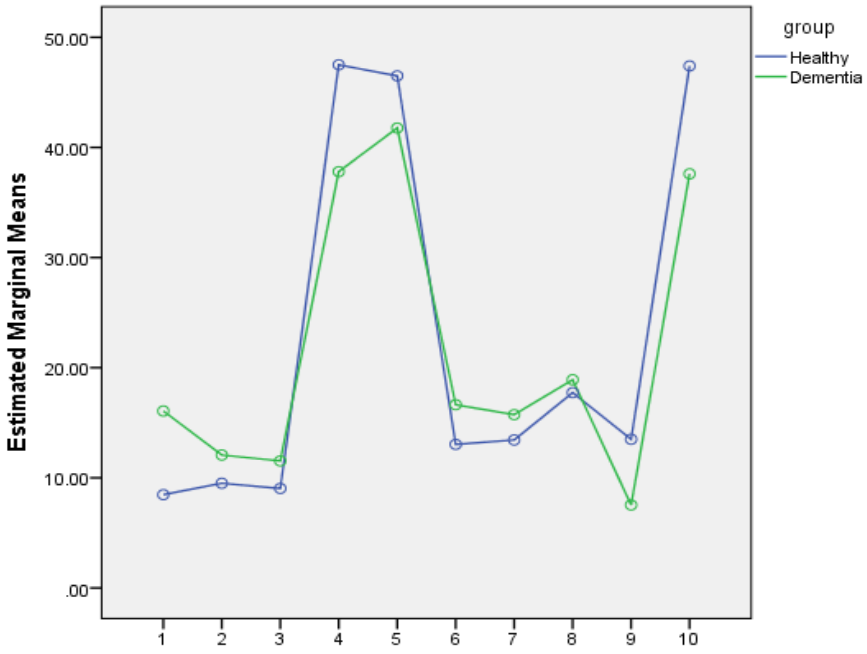
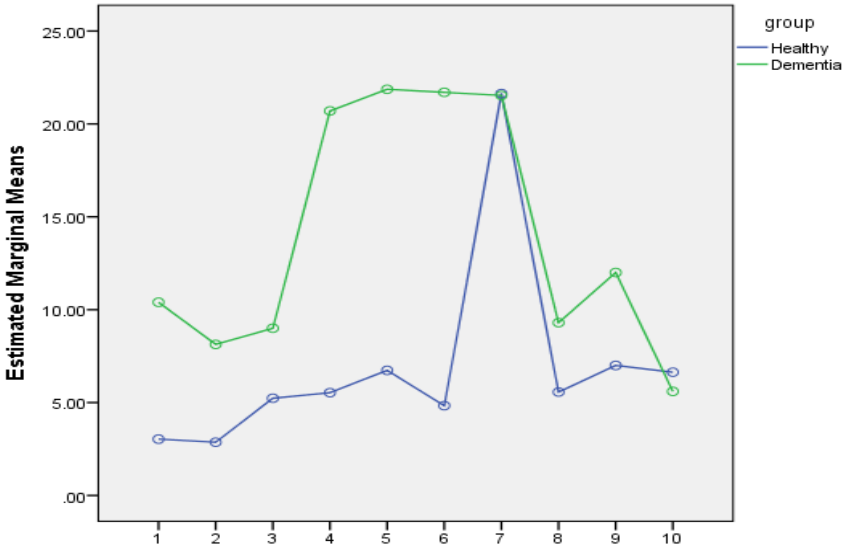



**Figure 5.** Mean Differences in Symptoms of Dementia after Induced Negative and Positive Auditory and Visual Hallucinations in Healthy Individuals (n = 30) and Dementia Patients (n = 30).



## Discussion

The core objective of this research was to evaluate the differential effect of positive and negative multimodal (auditory and visual) hallucinations on cognitive and affective landscapes in patients with mild dementia compared to healthy controls. This study data suggests a complex interaction where sensory valence (positive vs. negative) dictates the severity of symptomatic exacerbation. While baseline cognitive processing speed remained relatively stable across groups, the subjective burden and affective volatility were significantly higher in the dementia cohort, particularly under negative induction.

This study analysis of semantic memory retrieval (Table 2 and 3) revealed that while the latency of retrieval did not shift significantly, the accuracy and perceived difficulty were modulated by the induction type. In dementia, the neurodegenerative process typically compromises sensory gating the brain's ability to filter out irrelevant or distorted stimuli. When negative hallucinations were induced, patients experienced higher cognitive load, consistent with the Resource Limitations Theory. This theory suggests that because dementia patients have diminished cognitive reserves, the additional task of processing distressing "false" sensory input competes with the resources required for semantic retrieval(Cipriani et al., 2020; Rabins, 2013). This aligns with findings by Cipriani et al. (2020) and Mahendra et al. (2017), who noted that sensory misperceptions in Alzheimer's patients often lead to a "degradation of the semantic network."

The most striking results appeared in the assessment of depressive and dementia-specific symptoms (Table 4 and 5). Negative inductions particularly combined visual and auditory stimuli (BDIT7/DemT7) triggered acute spikes in distress. This "negative susceptibility" suggests that the amygdala and limbic structures in the aging brain may remain hypersensitive to threat-based stimuli even as cortical regions decline. For the dementia cohort, negative hallucinations were not merely sensory errors but functioned as acute psychological stressors, leading to increased disorientation and confusion. This confirms our second and third hypotheses: that negative induction aggravates emotional dysregulation more severely in clinical populations than in healthy individuals.

Perhaps the most significant contribution of this study is the efficacy of the placebo intervention (Trial 10). Despite the presence of organic neurodegeneration, the dementia group showed a "recovery curve" that was more pronounced than that of healthy controls.This demonstrates that top-down expectancy pathways remain functionally viable even in the presence of mild cognitive impairment(Aarsland et al., 2023; Bianchi et al., 2023; Borda et al., 2024; Evans et al., 2004). When patients were led to believe a mitigation strategy was in place, their subjective symptoms of depression and dementia decreased significantly (Aguirre et al., 2013; Ballard et al., 2008; Checksfield, 2020). This supports the Expectancy Theory of Placebo, suggesting that psychological resilience can be activated via non-invasive, non-pharmacological means to override sensory-driven distress(Geers et al., 2021; Shafir et al., 2023; Theodosis-Nobelos et al., 2021).

## Novel Contributions

This study introduces a high-precision, multimodal induction paradigm using PsychoPy software to isolate the specific cognitive and affective impacts of hallucination valence on dementia patients, a methodological rigor seldom applied to clinical populations. By demonstrating that negative sensory stimuli significantly exacerbate symptomatic distress while top-down expectancy pathways remain functionally intact, the research provides the first empirical evidence of "expectancy resiliency" in the neurodegenerative brain. This proves that despite organic decay, patients retain a robust capacity for placebo-mediated recovery, shifting the clinical focus from purely pharmacological suppression to active psychological modulation.

Furthermore, the research offers a culturally adaptive, non-pharmacological model tailored for resource-limited healthcare systems like Pakistan. By validating a double-blind, placebo-controlled framework in a geriatric setting, the study establishes a scalable strategy to mitigate neuropsychiatric symptoms without heavy reliance on antipsychotic medications. These findings bridge Cognitive Load Theory and Affective Science, offering a multidisciplinary roadmap for enhancing the quality of life through low-cost, high-impact psychological interventions that leverage the brain's remaining cognitive plasticity.

## Limitations and Future Directions

The ecological validity of this study is constrained by the artificial nature of the induction paradigm; while PsychoPy allows for high-precision stimulus control, it may not fully encapsulate the phenomenological complexity or the spontaneous, persistent nature of organic hallucinations in neurodegenerative disorders. Furthermore, the exclusion of severe-stage patients limits the generalizability of the findings across the full clinical spectrum of dementia, as the healthy control group may not represent the complete range of age-related cognitive variability. The cross-sectional design and brief trial durations also preclude an assessment of the long-term sustainability of the observed placebo effects. Finally, while the sample size (N=60) was sufficient for parametric analysis, it may lack the statistical power to detect subtle neuroanatomical interactions or differentiate between dementia subtypes (e.g., Alzheimer's vs. Vascular dementia).

Subsequent research should prioritize longitudinal designs to determine the durability of expectancy-mediated symptom reduction and its potential to delay clinical decline. Integrating functional neuroimaging (fMRI) or electroencephalography (EEG) would be critical to mapping the specific neural correlates of the "placebo rebound" in the aging brain, specifically focusing on prefrontal-limbic connectivity. Additionally, future trials should investigate individualized therapeutic protocols, such as combining environmental modifications with caregiver-mediated psychological support, to transition these acute experimental findings into stable clinical practice. Expanding the sample to include diverse neurodegenerative pathologies will further clarify whether these top-down recovery pathways are a universal feature of the aging brain or specific to certain proteinopathies.

## Conclusion

This study demonstrates that the neurodegenerative brain retains a robust, measurable capacity for expectancy-mediated modulation of sensory and affective processing. This study findings establish that while negative hallucinations impose a significant allostatic load exacerbating both cognitive interference and depressive symptomatology the preservation of top-down pathways offers a viable therapeutic window. The significant efficacy of the placebo intervention suggests that non-pharmacological, expectancy-based therapies can successfully override the psychological burden of sensory distortions. For healthcare systems in developing regions like Pakistan, these results advocate for the integration of low-cost, high-impact psychological interventions as essential supplements to traditional geriatric care, potentially mitigating the clinical reliance on antipsychotic medications and enhancing overall quality of life.

## Ethical Consideration

The study was approved by Department of Psychology, Foundation University Islamabad (FUI). Consent Form was taken before taking data and participants were asked to take voluntary participation.

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## Availability of data and materials

The data sets used and analyzed during the current study are available from the corresponding author on reasonable request.

## Authors' contributions/Author details

M. Aqeel and R. Akhtar conceptualized and designed the study, performed the formal statistical analysis, and was responsible for the original drafting and critical revision of the manuscript. R. Akhtar and A. Tauqeer coordinated participant recruitment and conducted the clinical evaluations. All authors have reviewed and approved the final version of the manuscript for publication.

## Corresponding author

Correspondence to Akhtar, R.
*rabiaakhtar510@gmail.com*

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