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RESEARCH ARTICLE

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Utility of the pareidolia test in mild cognitive impairment with Lewy bodies and Alzheimer's disease

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Abstract

Objectives: Previous research has identified that dementia with Lewy bodies (DLB) has abnormal pareidolic responses which are associated with severity of visual hallucinations (VH), and the pareidolia test accurately classifies DLB with VH. We aimed to assess whether these findings would also be evident at the earlier stage of mild cognitive impairment (MCI) with Lewy bodies (MCI-LB) in comparison to MCI due to AD (MCI-AD) and cognitively healthy comparators.

Methods: One-hundred and thirty-seven subjects were assessed prospectively in a longitudinal study with a mean follow-up of 1.2 years (max = 3.7): 63 MCI-LB (22% with VH) and 40 MCI-AD according to current research diagnostic criteria, and 34 healthy comparators. The pareidolia test was administered annually as a repeated measure.

Results: Probable MCI-LB had an estimated pareidolia rate 1.2–6.7 times higher than MCI-AD. Pareidolia rates were not associated with concurrent VH, but had a weak association with total score on the North East Visual Hallucinations Inventory. The pareidolia test was not an accurate classifier of either MCI-LB (Area under curve (AUC) = 0.61), or VH (AUC = 0.56). There was poor sensitivity when differentiating MCI-LB from controls (41%) or MCI-AD (27%), though specificity was better (91% and 89%, respectively).

Conclusions: Whilst pareidolic responses are specifically more frequent in MCI-LB than MCI-AD, sensitivity of the pareidolia test is poorer than in DLB, with fewer patients manifesting VH at the earlier MCI stage. However, the high specificity and ease of use may make it useful in specialist clinics where imaging biomarkers are not available.

KEYWORDS
dementia with Lewy bodies, mild cognitive impairment, pareidolia, visual hallucinations

Key Points

- Pareidolia responses to ambiguous visual stimuli may be a surrogate for visual hallucinations
- Pareidolias are more common in dementia with Lewy bodies than in Alzheimer's disease (AD)
- We found an increased rate of pareidolias in mild cognitive impairment (MCI) with Lewy bodies than in AD or healthy comparators
- Misperceptions in the pareidolia test are reasonably specific to MCI with Lewy bodies, but these may lack sensitivity at early stages

1 | BACKGROUND

Visual hallucinations (VH) are a feature of several psychiatric, neurological and ophthalmological disorders.[1] In dementia, VHs are particularly associated with the clinical syndrome of dementia with Lewy bodies (DLB) with an estimated prevalence of 55%–78%.[2] Complex VH, characteristically of well-formed images of people or animals, are one of the core clinical features differentiating clinically-suspected Lewy body aetiology from the competing diagnosis of Alzheimer's disease (AD) in both dementia[3] and mild cognitive impairment (MCI).[4]

VH may be context-dependent, and therefore difficult to quantify in clinical or research settings; these are often assessed through clinical interview after self-report by the patient, or report of this apparent phenomenon by an informant. This may limit the detection of VHs in the absence of insight by the patient, or unavailability of an informant. Comparable visual illusory phenomena (pareidolias, misperceptions of meaningful forms within ambiguous or visually-noisy stimuli) which may be elicited on demand in an experimental setting have therefore been proposed as proxies of VH.

These pareidolic misidentifications have been shown to be more common in DLB than in AD or healthy controls, and to be positively correlated with the frequency of VH.[5] This research suggested that human and animal faces and bodies were the most common illusions in these tasks, supporting a phenomenological link to DLB-associated complex VH. A simplified pareidolia test, where participants report the presence or absence of illusory faces amongst visual noise scenes, has similarly shown to be reliable in discriminating DLB from AD (sensitivity of 81% and specificity of 92%). Pareidolia responses were more common in DLB than AD and healthy controls, were more common within cases of DLB with clinically-judged VH, and positively correlated with neuropsychiatric inventory (NPI) hallucination (of any sensory modality) scores.[6]

While the pareidolia test shows apparent utility in discriminating hallucinations and DLB at the dementia stage, this utility has not yet been demonstrated in the prodromal stages of cognitive decline of MCI with Lewy bodies (MCI-LB) when cognitive impairments have begun to manifest. While neuropsychiatric symptoms, including VHs, may be present at this stage, they may be less common than in DLB while the full clinical syndrome is still emerging.[7,8]

We therefore aimed to test the rate of pareidolic misidentifications in MCI-LB in contrast to MCI due to AD (MCI-AD) and age-matched healthy control subjects using the noise pareidolia test, and to consider the utility of this test in detecting clinically-judged complex VH and MCI-LB. Based on the above findings from the dementia literature, our hypotheses to test were: (1) MCI-LB patients would produce more pareidolic responses than MCI-AD or controls; (2) pareidolic responses would be more common in clinically-judged visual hallucinators than non-hallucinators, and correlate with severity of hallucinations; (3) the pareidolia test would acceptably classify clinically-judged visual hallucinators, and MCI-LB cases.

2 | METHODS

2.1 | Participants

2.1.1 | Patients

Recruitment for this longitudinal cohort has been described in depth previously.[9] Briefly, participants over 60 years old were recruited from memory services, neurology and geriatric medical clinics in North East England. Prospective participants provided informed consent before undergoing more detailed screening by a research study medical doctor, and magnetic resonance (MRI) brain imaging. Those with possible frontotemporal or vascular aetiologies, parkinsonism preceding onset of cognitive symptoms by more than one year, dementia, or absence of objective cognitive impairment at screening were excluded. Inclusion criteria were age $\geq$60 years, and diagnosis of MCI at screening in accordance with NIA-AA criteria; concern about, and objective evidence of decline in cognition with maintained ability to function independently,[10] requiring a Clinical Dementia Rating (CDR) no higher than 0.5.

2.1.2 | Controls

Healthy participants were recruited from families of patients and local research involvement services and similarly screened as with patients undergoing medical review, neurological examination, MRI

brain imaging, and comprehensive neurocognitive assessment. Inclusion criteria were being age ≥60 years and cognitively healthy, with no known brain disease and a CDR of 0.

All participants, both patients and controls, were required to be medically stable on study entry. Local deprivation was calculated for each participant from the 2019 English Indices of Multiple Deprivation (IMD); IMD scores are divided at country-wide deciles so that a rank of one corresponds to living within one of the 10% most deprived neighbourhoods in England, and a rank of 10 being within the 10% least deprived neighbourhoods.[11]

2.2 | Design

Participants were assessed annually in a longitudinal design with repeated clinical interview with patient and informant (where available), medical review, and neurocognitive assessment. Differential clinical diagnoses (as below) were reviewed annually based on clinical interview, medical review, and imaging findings.

2.3 | Procedure

2.3.1 | Clinical assessment and imaging

Participants underwent a detailed clinical assessment at baseline including physical and neurological examination, and at annual follow-up visits. Informants were also interviewed if available to provide further information. Interviews included the Geriatric Depression Scale, Clinician Assessment of Fluctuations, Dementia Cognitive Fluctuations Scale, Neuropsychiatric Inventory (NPI) for informants, North-East Visual Hallucinations Inventory (NEVHI) for patients, Unified Parkinson's Disease Rating Scale—Part III, and Mayo Sleep Questionnaire. Instrumental Activities of Daily Living were rated by the informant (blind to cause, and therefore sensitive to non-cognitive causes of dependence), and the CDR was completed by the clinical assessor based on this interview.

^{123}I-FP-CIT SPECT imaging was offered to all participants at baseline as previously detailed,[12] and repeated at one year follow-up. Images were visually rated as normal or abnormal by a five-person consensus panel of FP-CIT imaging experts, blind to clinical information. ^{123}I-mIBG cardiac sympathetic innervation imaging (cardiac mIBG) was also offered to all participants at baseline; delayed images (taken ~ 4 h post-injection with medium energy collimators) were quantified with a heart:mediastinum ratio cut-off of <1.86 considered abnormal based on local data from healthy controls.[13]

2.4 | Clinical diagnosis and differential classification

A three-person consensus panel of experienced old age psychiatrists (AJT, PCD, JPT) independently reviewed clinical research notes provided from the clinical interview and assessment annually to confirm the presence of all-cause MCI according to NIA-AA criteria[10] at baseline.

Each panel member also independently rated the presence or absence of each of the four core clinical features of DLB (parkinsonism, REM sleep behaviour disorder, fluctuating cognition, and complex VHs) based on research clinical notes and blind to imaging results. Core clinical features and imaging results were then incorporated along with clinical diagnosis to classify patients as MCI-AD (MCI, with no core clinical features of DLB, normal FP-CIT and mIBG imaging), possible MCI-LB (MCI with either one core clinical feature of DLB and normal imaging, or no core clinical features with abnormal FP-CIT and/or mIBG imaging), or probable MCI-LB (MCI with two or more core clinical features of DLB, or one clinical feature with one or more imaging abnormalities). These diagnoses were therefore consistent with current guidelines for classification of MCI-LB in research settings.[4]

Diagnoses and classifications were repeated and updated after each follow-up assessment. In the case that participants were seen to have lost functional independence at follow-up assessment, all-cause criteria for dementia were considered.[14] No further follow-up was undertaken after diagnosis of dementia.

2.5 | Neurocognitive assessment

A detailed neurocognitive assessment battery was administered to all participants separately from the clinical review, including the Addenbrooke's Cognitive Examination—Revised (ACE-R) as a test of global cognitive function from which Mini Mental State Examination (MMSE) was derived, the National Adult Reading Test (NART) was administered at baseline as an estimator of premorbid function. Additional assessments were administered, but not considered for this work, having being detailed elsewhere.[8]

2.6 | Pareidolia test

The 40-item visual noise pareidolia test[6] was administered at baseline and repeated at annual follow-up in the same manner: forty black-and-white visual noise images were presented sequentially on laminated cards. Individually differing human face images were presented within the noise in eight of these stimuli, and the remaining 32 contained only visual noise. After being shown three example stimuli to become acquainted with the task (two with faces, one without), participants were allowed up to 30 s to view each of the 40 test pages and asked to report if they did, or did not, see a face in each image. The test administrator recorded responses, out of view and without feedback or correction to the participant, as either correct (correctly identifying a face which was present, or correctly identifying a non-face stimulus), missed (missing a face which was present), or a pareidolia (where the participant identified a face as being present in a noise-only image). When providing ambiguous responses (e.g., 'maybe'), participants were prompted to provide either a 'yes' or

'no' answer. As in previous studies, the count of pareidolia responses was the outcome of interest.

2.7 | Analysis

To assess group differences in the production of pareidolia responses, incorporating repeat assessments over time to maximise data availability and account for any time trends (e.g., increased pareidolia rates as MCI progressed), a generalised linear mixed model with log link function was estimated using the *lme4* package for *R* software. Model fit was assessed by the Akaike Information Criterion (AIC). Subject-specific random intercept and time slope were included, as were relevant covariates by block entry, with continuous variables centred to a meaningful reference value, mean integer or median to aid in intercept interpretation: time since baseline assessment (in years), presence of visual impairment reported at health screening, global cognitive function (ACE-R score, time-varying; centre at 84), premorbid function (NART estimated IQ; centre at 100), gender (female as reference), age (centre at 75 years), education (years in education, centre at 13), and local deprivation (IMD rank, centre at 5). Up to third-degree polynomials were assessed for all continuous variables to allow for non-linear effects. Diagnostic group (Model 1) and VH presence (time-varying, where applicable) as rated by the clinical panel (Model 2) were included as hypothesis-testing fixed effects, with group × time interactions included when supported by improvements in model fit.

Significance was considered as $p < 0.05$ for hypothesis-testing effects, after controlling for relevant covariates.

To assess the sensitivity and specificity of the pareidolia task in classifying a) clinically-judged VHs and b) MCI-LB (possible or probable), receiver operating characteristic (ROC) curves were plotted, and the area under the ROC curve (AUC) derived with the *plotROC* package for *R* software. Sensitivity and specificity of this test in differentiating MCI-LB were assessed using cut-offs previously identified from the dementia stages: ≥ 5 pareidolia errors (vs. MCI-AD) or ≥3 errors (vs. controls).[6] Classification analyses made use of baseline test data only, to eliminate the influence of further decline on pareidolia error rates.

3 | RESULTS

3.1 | Baseline characteristics

One-hundred and three MCI patients and 34 healthy controls were available for inclusion (Figure 1). A median of two observations (baseline and 1-year follow-up) were available for each participant (Mean follow-up time = 1.2 years, SD = 0.99, max = 3.7 years). Demographics and baseline task performance are presented in Table 1. Consistent with the respective dementia syndromes, there was a gender disparity between MCI-AD and MCI-LB groups, with the former being predominantly female, and the latter predominantly male. VH were not as prevalent in MCI-LB as previously observed in DLB, being identified in 14 out of 64 cases (22%) at baseline at this earlier stage.

3.2 | Pareidolia analysis

Second-degree polynomials (linear and quadratic terms) were supported for the fixed effect of time only. No interactions with diagnosis were supported, and the resulting best-fitting models are presented in Table 2, with covariate effects in Table S1. Model 1 assessed diagnostic group differences in rate of producing pareidolia responses. The expected pareidolia count at the intercept (reference group: MCI-AD) was 0.79 (95% CI: 0.38–1.67). Healthy controls did not significantly differ in their test performance from MCI-AD, with expected pareidolia counts of 0.60. Probable MCI-LB were significantly more likely than MCI-AD to falsely perceive faces within noise stimuli (1.22 to 6.69 times the rate of pareidolias: expected count of 2.26). While possible MCI-LB had a similar point estimate to probable MCI-LB, there was more uncertainty in this estimate, and so this was not significantly different from MCI-AD (0.97 to 7.13 times the rate of pareidolias: expected count of 2.07). There was a slight positive growth in pareidolia response rates over time initially, though this was attenuated by the quadratic term over longer time periods, which may reflect the exclusion of dementia cases after follow-up.

These associations remained after controlling for the presence or absence of clinically-judged complex VHs at baseline or follow-up as a time-varying predictor (Model 2), which was not a significant predictor of pareidolic responses. The variance inflation factor for each component was low (all < 2 across both models), suggesting there was little collinearity between predictors.

In both models the marginal R^2 was relatively low compared to the conditional R^2, suggesting that much of the variance in this measure could be attributed to individual-level differences in task performance. This is supported by the expected pareidolia values being low, even in MCI-LB groups, compared to the true observed range of pareidolia responses produced at baseline (see Table 1).

Repeated-measure correlations found no significant association between pareidolia responses and NPI-measured hallucinations score as rated by informants (r [71] = 0.03, $p = 0.782$), but did support a weak positive correlation between pareidolia production and total score on the NEVHI as rated by patients (r [120] = 0.22, $p = 0.017$).

3.3 | Classification analysis

Despite broad group differences in pareidolia response rates, the pareidolia test was found to have poor utility in classifying both hallucinating MCI cases specifically (AUC = 0.56), and MCI-LB (AUC = 0.61) in general (see Figure S1). Using cut-offs identified from the dementia stage,[6] the noise pareidolia test had a sensitivity of 27% (95% CI: 16%–40%) and specificity of 89% (75%–97%) in

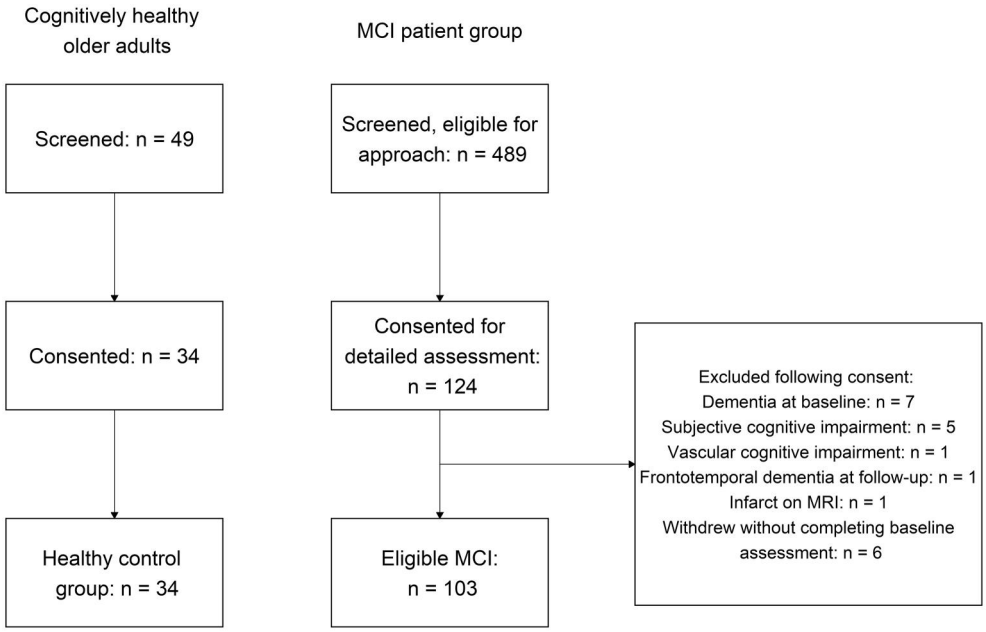

FIGURE 1 Recruitment flowchart for healthy controls and mild cognitive impairment (MCI) groups

TABLE 1 Baseline characteristics and task performance

	Control (n = 34)	MCI-AD (n = 40)	Possible MCI-LB (n = 20)	Probable MCI-LB (n = 43)	p Value
Female gender	10 (29.4%)	23 (57.5%)	9 (45.0%)	7 (16.3%)	<0.001[a]
Age	74.2 (7.45)	76.2 (7.54)	74.1 (7.95)	74.9 (6.36)	0.598[b]
Years in education	14 [8.5, 24]	11 [10, 20]	11 [9, 25]	11 [9, 21]	0.007[c]
Local deprivation decile rank	6.5 [1, 10]	6 [1, 10]	3.5 [1, 10]	5 [1, 10]	0.078[c]
Instrumental activities of daily living	-	8 [2, 8]	7 [3, 8]	6 [4, 8]	0.012[c]
National Adult Reading Test Estimated Full-Scale IQ	114 (8.64)	109 (12.3)	102 (11.4)	108 (9.54)	<0.001[b]
Mini Mental State Examination	28.5 (1.13)	26.9 (2.05)	26.0 (2.97)	26.4 (2.47)	<0.001[b]
Addenbrooke's Cognitive Examination—revised, total	92.7 (4.3)	82.4 (8.3)	78.0 (11.3)	83.0 (9.2)	<0.001[b]
Neuropsychiatric Inventory—Total	-	5 [0, 34]	3 [0, 44]	15 [0, 52]	0.011[c]
Neuropsychiatric Inventory—Hallucinations	-	0 [0, 1]	0 [0, 4]	0 [0, 8]	0.006[c]
North-East Visual Hallucinations Inventory	0 [0, 3]	0 [0, 7]	1 [0, 15]	0 [0, 16]	<0.001[c]
Pareidolia task—Pareidolias	0 [0, 5]	1 [0, 16]	2 [0, 20]	2 [0, 14]	0.007[c]
Pareidolia task—Misses	0 [0, 1]	0 [0, 3]	0 [0, 3]	0 [0, 2]	0.371[c]
Visual hallucinations	0 (0%)	0 (0%)	4 (20.0%)	10 (23.3%)	<0.001[a]
Any visual impairment	2 (5.9%)	3 (7.5%)	2 (10.0%)	2 (4.7%)	0.868[a]

Note: Mean (SD), Median [Range], or Count (%).

Abbreviations: AD, Alzheimer's disease; LB, Lewis bodies; MCI, mild cognitive impairment.

[a]Chi-square test.

[b]ANOVA.

[c]Kruskal-Wallis test.

TABLE 2 Generalised linear mixed models estimating pareidolia response production differences between diagnostic groups (Model 1) and hallucinators (Model 2). Intercept as expected count, fixed effects as incidence rate ratio

	Model 1.			Model 2.		
Fixed effects (reference group/value)	**Incidence rate ratio[a]**	**95% CI**	***p***	**Incidence rate ratio**	**95% CI**	***p***
Intercept (MCI-AD)	0.79	0.38–1.67	0.542	0.79	0.38–1.67	0.537
Healthy Control (vs. MCI-AD)	0.76	0.30–1.92	0.557	0.77	0.30–1.96	0.584
Possible MCI-LB (vs. MCI-AD)	2.62	0.97–7.13	0.059	2.83	1.02–7.85	0.046
Probable MCI-LB (vs. MCI-AD)	2.86	1.22–6.69	0.016	3.05	1.27–7.35	0.013
Time, linear term	1.58	1.01–2.45	0.043	1.55	1.00–2.41	0.051
$Time^2$, quadratic term	0.79	0.69–0.91	0.001	0.80	0.69–0.92	0.002
Visual hallucinations present (vs. Absent)	-	-	-	0.67	0.28–1.58	0.357
Controlling for: visual impairment, gender, global cognitive function[b], premorbid intelligence[c], age, education, and deprivation (see Table S1).						
Random effects						
σ^2	0.84			0.84		
Observations	271			271		
Marginal R^2/Conditional R^2	0.211/0.796			0.208/0.799		

Abbreviations: AD, Alzheimer's disease; LB, Lewis bodies; MCI, mild cognitive impairment.

[a]Baseline expected error rate at intercept, incidence rate ratio for all other effects.

[b]Addenbrooke's Cognitive Examination—Revised.

[c]National Adult Reading Test—Estimated Full-Scale IQ.

differentiating MCI-LB from MCI-AD (cut-off ≥ 5 pareidolia responses), and sensitivity of 41% (28%–55%) and specificity of 91% (76%–98%) in differentiating MCI-LB from healthy controls (cut-off ≥ 3).

4 | DISCUSSION

4.1 | Summary of aims and findings

We aimed to assess if differences in performance on the noise pareidolia test observed between DLB and AD would also be present in the respective MCI stages of these.

We found only limited support for our hypotheses; probable MCI-LB were found to make more pareidolic mispercetions when completing this test than MCI-AD and controls, consistent with previous findings in DLB[6] but this association was not clearly found in the possible MCI-LB group.

There was no clear association between rates of pareidolic misperceptions and the presence of complex VHs as assessed by an expert clinical panel, contrary to the hypothesis. No association was found between pareidolia rates and hallucination severity (of any sensory modality) assessed by the NPI, but a weak association was found more specifically with simple and complex VH severity as measured by the NEVHI.

Finally, the utility of the noise pareidolia test in classifying either MCI-LB or clinical VHs was not supported; while the noise pareidolia test was able to differentiate MCI-LB from MCI-AD or healthy controls with good specificity, cut-off values from DLB had low sensitivity when applied to MCI-LB.

4.2 | Interpretation

These results partially extend previous findings to suggest that differences in the experience of pareidolias between DLB and AD[5,6] may already be apparent at the MCI stages of these aetiologies, with a higher rate of pareidolic responses in MCI-LB, though this was limited to the most diagnostically clear sub-group of probable MCI-LB. However, there was considerable subject-level variability in the reporting of perceived faces not attributable to any considered variable. Consequently, the noise pareidolia test had less predictive value in classifying either a Lewy body syndrome, or VHs in MCI, in comparison to the dementia stage.[6] However, as a simple assessment with relatively low time cost and good specificity for MCI-LB, the pareidolia test may have value as an accessible early screening test for suspected MCI-LB in settings where more accurate but costly markers such as FP-CIT or mIBG imaging are not available.

VH were much less common in this MCI-LB sample than is typical in DLB (22% vs. 55%–78%),[2] and pareidolias also occurred at higher rates in previous studies than in our own (mean of 3.5 in MCI-LB vs. 7.3 in DLB)[6] which may account for the limited utility of the noise pareidolia task in classifying these and MCI-LB. As our MCI patients were in the prodromal stage it remains likely that their clinical symptoms will continue to develop with more VH emerging closer to the onset of, and during, dementia. Pareidolic misidentifications may

precede the eventual clinical manifestation of VH as a simple form of visual illusion comparable to the visual perceptual dysfunctions and progressive decline commonly observed in DLB[15,16] and MCI-LB,[17] which have been shown to predict the eventual onset of VHs in DLB.[18] Future work may consider whether pareidolia rates in non-hallucinating MCI predict the eventual emergence of complex VH by the onset of dementia; the prospective identification of complex VHs may have value in clinical and research settings due to the previously-reported association between this particular clinical feature of DLB and progressive cognitive decline in MCI.[19]

Several factors could account for both individual- and group differences in the production of pareidolia responses; previous research has suggested that an increased reliance upon prior knowledge in discrimination of ambiguous visual imagery may mediate the associations between Lewy body disorders and VHs.[20] When approaching the pareidolia test with a clearly defined objective (to discriminate faces from noise) some individuals, and particularly those with MCI-LB may therefore place relatively more weighting on this prior expectation, therefore increasing the rate of misperceptions even in the absence of clinically manifest complex VHs.

The high variability in rates of pareidolias even within MCI-AD could suggest that, despite uniformity in instructions, individuals may still vary in their understanding and approach to the test; some participants may favour a false positive-minimising strategy (only reporting unambiguous perception of faces), while others may favour a false negative-minimising strategy (reporting faces in the absence of a true misperception to avoid missing one). With the diagnostic effect sizes being relatively small in the MCI stages, individual-level factors such as these may contribute to the limited classification utility in prodromal DLB.

While functional independence was highly variable at baseline in the MCI group with some particularly low IADL scores, these were assessed to include all contributions to functional dependence, including motor impairment (previously found to be correlated with baseline instrumental activities of daily living (IADL) scores in MCI-LB, while cognitive scores were not)[17] and social or cultural factors (e.g., the patient's contributions to housework were limited even prior to onset of any cognitive impairment). Despite some low IADL scores, all patients were judged to have MCI at baseline as evidenced by a CDR <1.

4.3 | Strengths and limitations

These data include a moderately-sized cohort with detailed clinical assessment and imaging for aetiological classification. We have made use of flexible modelling approaches to incorporate repeated measures to appropriately account for individual-level effects, and controlled for several anticipated confounding variables.

However, considerable variability was observed in this sample which was not explained by fixed effects. While we controlled for visual impairment reported at medical review, no objective measures of visual acuity were available, though previous research found no association between visual acuity and pareidolia rates in this test.[6] As a prospective cohort, it is not yet apparent which patients will develop VH by the time of onset of dementia, only those who have already done so (a minority of the MCI-LB group); while this clinical symptom was modelled as a time-varying predictor, it is not clear at this stage if an increased pareidolia rate in MCI may precede or predict the eventual clinical manifestation of VH.

5 | CONCLUSIONS

Probable MCI-LB had a higher rate of pareidolia responses in the visual noise pareidolia task than MCI-AD, who did not clearly differ from healthy controls. The relationship between hallucinations and pareidolia responses was not as clear as in dementia, with comparisons limited by low rates of hallucinations in MCI. Due to considerable inter-individual variation in task performance, the noise pareidolia test did not accurately classify MCI-LB or VH.

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CONFLICTS OF INTEREST

None.

DATA AVAILABILITY STATEMENT

Data supporting these analyses are available to researchers upon reasonable request through the Medical Research Council Dementias Platform UK (study 'SUPErB')

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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