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IDENTIFYING THE COGNITIVE UNDERPINNINGS OF VOICE-HEARING BY COMPARING NEVER, PAST AND CURRENT VOICE-HEARERS

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Abstract

Objective: The current study aimed to compare specific cognitive profiles corresponding to AVH status, and elucidate which pattern of cognitive deficits may predict voice-hearing status.

Methods: Clinical participants with schizophrenia spectrum disorders were partitioned into: i) Current voice-hearers (*n*=46), ii) Past voice-hearers (*n*=37), and iii) Never voice-hearers (*n*=40), and compared with 319 non-clinical controls. Cognitive assessment employed the MATRICS Consensus Cognitive Battery (MCCB), supplemented by the Delis-Kaplan Executive Function System (DKEFS) Colour-Word Interference Test (Stroop) as a robust measure of executive function.

Results: On the Visual Learning domain, current and past voice-hearers had significantly poorer performance relative to never voice-hearers, who in turn had significantly poorer performance than non-clinical controls. Current and never voice-hearers had significantly poorer performance on the *Social Cognition* domain relative to non-clinical controls. Current voice-hearers also had significantly poorer performance on the *Inhibition* domain relative to non-clinical controls. Binary logistic regression revealed that *Visual Learning* was the only significant cognitive predictor of AVH presence.

Conclusion: Visual learning, and potentially inhibition, may be viable therapeutic targets when addressing cognitive mechanisms associated with AVHs. Future research should focus on investigating additional cognitive mechanisms, employing diverse voice-hearing populations, and embarking on related longitudinal studies.

Keywords: Auditory verbal hallucinations, cognition, visual learning, inhibition, schizophrenia, psychosis

Significant outcomes	○ Visual learning and inhibition may be the two key cognitive domains distinguishing whether schizophrenia spectrum patients experience auditory

	hallucinations or not. ○ However, only visual learning was a cognitive predictor of auditory hallucination presence. ○ These two cognitive constructs may serve as viable therapeutic targets for treatment of voice-hearing, especially if further research can demonstrate that these mechanisms are amenable to change over time, possibly in line with illness severity.
Limitations	○ We were unable to consider additional cognitive and non-cognitive mechanisms (beyond the eight key domains selected), which limited the predictive value of our model. ○ The cross-sectional nature of our study precluded tracking of changes in cognitive variables over time, which would have been an added significant contribution to existing literature.

Data availability: Relevant data will be made available for sharing upon request to the authors.**1. Introduction**

The experience of 'hearing voices' is a hallmark feature of psychosis, and is associated with high levels of emotional distress, poor quality of life, and even suicide (1). These auditory verbal hallucinations (AVHs) refer to the perception of verbal utterances in the absence of corresponding external stimuli. Up to 38% of psychosis patients reported current AVHs, and almost 80% will encounter these experiences over their lifetime (2), yet these symptoms frequently fail to respond to existing treatments. A significant proportion of mechanistic research has thus focused on identifying possible cognitive underpinnings of voices. This mirrors similar work in other psychosis symptoms, such as delusions (3, 4) and formal thought disorder (5, 6). Yet these psychosis symptoms rarely occur in isolation; AVHs and related delusional content often feed into one another, or the absence of AVHs may conversely signify increased cognitive and/or negative symptoms. In fact, psychotic illnesses are often accompanied by broad cognitive dysfunction affecting most domains (7, 8). To develop targeted interventions, it is imperative to not only

identify cognitive factors associated with AVHs, but also uncover patterns of cognitive deficits that may significantly predict voice-hearing status.

1.1 Cognitive deficits implicated in AVHs

Psychosis, and in particular schizophrenia, is associated with a range of severe cognitive deficits, several of which have been directly implicated in AVHs (9). Based on existing etiological theories, two major models have evolved, namely *inner speech* versus *memory*. The *inner speech* model proposes that voices arise from misattribution of self-generated inner speech to a non-self source (10). Based on the 'alien voice' task, 'hallucinators' were significantly more prone to misattribution errors, reflecting problems dissociating self- versus other-generated speech (11). This model is however modality-specific (i.e. auditory), and inherently reliant on language processes and alleged semantic deficits. Alternatively, the *memory* model posits that voices result from faulty intrusions from memory (12). When working memory is compromised, we are unable to sustain a unified sense of self and internal-external representations become fragmented. Supporting this, working memory deficits in the verbal domain have been shown to be a significant predictor of AVH presence (13) and severity (14) in psychosis. While emphasising diverse cognitive processes, these models are not mutually exclusive, and likely depend on a patient's underlying AVH subtype (15). There are also shared cognitive mechanisms between these two theories; the most prominent of these will now be briefly discussed.

Inhibition is an executive function related to the capacity to block out irrelevant information, enabling adaptive control of one's thoughts and behaviours (16). Consequently, a direct link between impaired inhibition of irrelevant stimuli coupled with deficits identifying their origin or context, may lead to voices (9). Poor inhibitory control, reflected in the inability to curb unwanted thoughts or memories (12) or irrelevant behaviours (17), has already been linked to AVH severity. *Working memory*, involving the short-term online storage and manipulation of information, has also been implicated in AVHs. It is plausible that impaired working memory may be partly liable for faulty auditory intrusions being misinterpreted as AVH experiences. A*ttentional processes*, which refer to how we actively select, focus and manage information in our environment, are also a feature of both models. Significantly impaired attention in the auditory verbal domain was noted in frequent versus non-frequent AVH psychosis groups, even three months following an acute episode, possibly indicating a stable cognitive vulnerability to voices (18). Deficits in attention can thus contribute to misattribution of inner speech as well as incongruous

encoding of details in memory. Finally, *verbal learning*, or the consolidation of new information in the semantic realm, is often implicated simply due to the modality-specific nature of AVHs.

1.2 Trait versus state-like cognitive deficits

The following section on trait- versus state-like cognitive deficits in psychosis is not meant to be exhaustive in nature (interested readers are asked to refer to systematic reviews and/or meta-analytic studies cited). Our intention is to position the current study within the broader context of prevailing cognitive research in psychosis, acknowledging inherent complexities therein. Studies have shown that targeted remediation of cognitive deficits has demonstrated efficacy (19-21) in schizophrenia spectrum disorders, and may in turn, convey direct therapeutic benefits to patients with AVHs (though substantial deficits may remain despite moderate improvements). Yet to date, there has been scant research examining specific cognitive profiles associated with voice symptoms. An unresolved issue in the study of the cognitive underpinnings of AVHs is the link between exacerbation of specific cognitive deficits and presence of AVHs. A key distinction thus needs to be made between generalised cognitive deficits in those with psychosis, and cognitive impairments that are specifically present and/or exacerbated amongst persons who hear voices. Therefore, it is important to compare the cognitive profiles of voice-hearers versus non-voice-hearers to elicit patterns of cognitive dysfunction explicitly linked to AVHs. This type of comparison has yet to be completed comprehensively, with such distinctions more likely to manifest in theory than actual scientific practice at present.

Furthermore, there is some evidence to suggest that deficits in cognition may be relatively stable and trait-like in nature, that is, unlikely to fluctuate significantly with illness severity (e.g. 22). In particular, the emergence of stable cognitive trajectories was interpreted as indicative of feasibility for use as specific endophenotypes to predict illness outcomes (23). There is converse evidence citing improvement across most (but not all) cognitive domains in schizophrenia, albeit possibly attributable to external factors, such as practice effects (24). Other research has also implied that state-based cognitive changes according to symptom severity or other circumstantial influences (e.g. stress) are conceivable (25). To this end, an inclusive analysis of the cognitive domain of inhibition in line with the Research Domain Criteria (RDoc) framework revealed that inhibitory control may be seen as somewhat malleable, varying within and amongst individuals, according to life stage as well as illness status. Much less work has been performed across other cognitive domains. However, these disparate conclusions may be somewhat reconciled by considering the specificities of cognition as well as symptom dimensions under consideration. In other

words, it is possible that certain cognitive domains are more amenable to change over time, especially when a particular class or subclass of symptoms (e.g. hallucinations versus AVHs), rather than global illness (i.e. psychosis), is taken into account. Yet on the whole, additional research needs to be carried out before a definitive viewpoint may be reached, though the complex nature of differences across cognitive domains likely signifies there may not be a singular answer.

1.3 Aims of the study

Preliminary research has revealed that executive dysfunction (26) and poor working memory (13, 14, 27) (as well as attentional anomalies and verbal deficits possibly to a lesser extent) are significantly linked to AVHs. Yet few other studies have directly compared the cognitive performance of psychosis patients in line with prevailing AVH status. If posited links between cognitive anomalies and AVH manifestation are true, the patterns and degree of cognitive impairment may differ between patients with current and past voice-hearing experiences, at least for certain cognitive domains. The current study therefore had two main aims: i) to evaluate comparative cognitive profiles of individuals with schizophrenia spectrum disorders based on AVH status (i.e. current, past or never), and ii) to explore which cognitive variables significantly predicted the presence (versus absence) of AVHs. It was proposed that within the clinical groups, current voice-hearers would exhibit significantly poorer performance on specific cognitive domains involving i) attention/vigilance, ii) working memory, iii) verbal learning and iv) inhibition, relative to non-voice-hearers. Whether past voice-hearers would demonstrate a similar pattern of cognitive deficits relative to current voice-hearers, or intermediate performance between current and never voice-hearers may vary according to the cognitive domain under study, depending on which aspects of cognition are more trait- versus state-like. As this part of the analysis was exploratory in nature, we tentatively anticipated that current voice-hearers would exhibit significantly poorer performance on working memory and inhibition relative to past voice-hearers; these two cognitive domains have shown the most robust empirical evidence to date. A secondary research question pertained to whether the observed cognitive deficits would be predictive of the experience of voice symptoms.

2. Methods

2.1 Participants and procedure

Data from 123 patients with schizophrenia spectrum disorders (i.e. schizophrenia or schizoaffective disorder) and 319 non-clinical controls were obtained from the Cognitive and Genetic Explanations of Mental Illness (CAGEMIS) bio-databank. Clinical participants were divided into three groups, including those who: i) were currently experiencing AVHs (*n*=46), ii) had experienced past AVHs only (*n*=37), or iii) had never heard voices (*n*=40). Eligibility criteria for all participants were: i) aged 18 to 65 years, ii) fluent speaker of the English language, iii) an estimated premorbid IQ>70, iv) no known history of significant neurological disorders or serious brain injury, v) no current substance abuse/dependence within the last two months, and vi) no electroconvulsive therapy (ECT) within the last six months. Additional inclusion criteria for non-clinical controls were: vi) no past or current diagnosis of a mental health disorder, and vii) no known first-degree biological relative with a psychotic disorder.

Participants were recruited from metropolitan outpatient or community-based mental health clinics, flyers placed at public venues, or online research participation forums. The study was approved by the human research ethics committee at the Alfred Hospital, Melbourne, Australia. Procedures conformed to the Declaration of Helsinki (28), and participants provided informed consent for analysis of their stored data.

2.2 Materials

A standard demographic-clinical record was used to obtain personal and mental health information, including self-report measures, such as the Launay-Slade Hallucination Scale (LSHS) (29). Clinician-administered instruments were the MINI International Neuropsychiatric Interview (MINI) (30) to verify clinical diagnoses, Wechsler Test of Adult Reading (WTAR) (31) as an estimation of premorbid IQ, and Positive and Negative Syndrome Scale (PANSS) (32), Montgomery Äsberg Depression Rating Scale (MADRS) (33) and Young Mania Rating Scale (YMRS) (34) to assess respective psychosis, depressive and mania severities. Partitioning of clinical participants into *current*, *past* and *never* voice-hearers was achieved using a researcher-administered, forced-choice question: “Sometimes people say that they can hear noises or voices inside their head that others can’t hear. Have you ever experienced this?”, with “Yes, currently”, “Yes, in the past” and “No” as response options. In this case, current voice-hearers were designated as experiencing an AVH episode within the last 14 days.

Cognitive assessment employed the MATRICS Consensus Cognitive Battery (MCCB) (35), where raw MCCB test scores were converted to uncorrected domain *t*-scores (50±10) derived from a normative, non-

clinical sample (36). This was supplemented by the Delis-Kaplan Executive Function System (DKEFS) Colour-Word Interference Test (Stroop) (37) as a robust measure of executive function. Eight cognitive domains were sampled, namely: *Speed of Processing*, *Attention/Vigilance*, *Working Memory*, *Verbal Learning*, *Visual Learning*, *Reasoning and Problem-Solving*, *Social Cognition*, and *Inhibition* (i.e. Stroop). Due to our augmented cognitive battery administered within an Australian sample, normative *z*-scores were calculated using our own non-clinical control performance as baseline (in lieu of MCCB published norms). This allowed creation of an eighth domain (i.e. Inhibition) with corresponding *z*-scores for direct group-wise comparisons within our specified cognitive battery.

2.3 Analyses

Quantitative data were analysed in *SPSS v25.0*. Descriptive data were presented as means and standard deviations, or percentages (as was most meaningful). Preliminary analyses comprised group-wise comparisons of demographic-clinical information, followed by one-way analyses of variance (ANOVAs) for each cognitive domain (based on *z*-scores). When Levene's test was significant, Brown-Forsthye's *F* was reported (38). Post-hoc comparisons used Tukey's Honestly Significant Difference (HSD) test. Effect sizes were η^2: .01=small effect, .06=medium effect, and .14=large effect, or Cramer's U: .01=small effect, .30=medium effect, and .50=large effect (39). Binary logistic regression was conducted to identify which cognitive variables were significant predictors of AVHs. The dependent variable was defined as presence of AVHs (coded *No*=0 and *Yes*=1); current and past voice-hearers were collapsed into a single group (*n*=83). Cognitive domains found to be significantly different amongst the clinical groups were entered into the regression model as possible predictors. A ratio of at least 10 cases per covariate was used for conservative model-building (40); this resulted in a maximum of four covariates for our model (based on *n*=40 for the *No* group).

3. Results

3.1 Demographic-clinical information

Group-wise comparisons on demographic and clinical variables are presented in Table 1. For demographics, there were no significant group differences in age and sex, but clinical groups reported significantly fewer years of education, reduced employment and marital status, as well as lower

premorbid IQ relative to the non-clinical group. In terms of clinical variables, current and past voice-hearers were significantly more prone to hallucinatory experiences and depressive symptoms than never voice-hearers, who in turn were significantly more prone than non-clinical controls. Furthermore, clinical groups experienced significantly elevated mania relative to the non-clinical group. Within clinical groups, there were no significant differences in illness duration or psychosis symptoms. On a subscale level, current voice-hearers reported significantly increased positive (but not negative or general) psychosis severity relative to past and never voice-hearers. When the specific effect of hallucinations (i.e. PANSS *Item P3*) was removed, this was no longer significant. Current voice-hearers were prescribed significantly larger doses of antipsychotics (in *chlorpromazine* equivalents) than past and never voice-hearers (see Table A in Supplementary material for a breakdown of medication use by class and group).

[Insert Table 1 here]

3.2 Comparative cognitive profiles

Cognitive performance of current, past and never voice-hearers on our specified cognitive battery was evaluated. Means and standard deviations as well as group-wise comparisons are shown in Table 1. The three clinical groups performed significantly poorer on *Speed of Processing*, *Attention/Vigilance*, *Working Memory*, *Verbal Learning*, and *Reasoning and Problem-Solving* domains relative to non-clinical controls. On the *Visual Learning* domain, current and past voice-hearers performed significantly poorer relative to never voice-hearers, who in turn performed significantly poorer than non-clinical controls. Current and never voice-hearers performed significantly poorer on the *Social Cognition* domain relative to non-clinical controls (with past voice-hearers exhibiting no significant differences with any other group), and current voice-hearers performed significantly poorer on the *Inhibition* domain relative to non-clinical controls (with past and never voice-hearers exhibiting no significant differences with any other group). For illustrative purposes, corresponding cognitive performance (standardised with respect to non-clinical controls) is displayed in Figure 1. In sum, current and past voice-hearers were markedly impaired on *Visual Learning*, with current voice-hearers also experiencing difficulties on *Inhibition*.

[Insert Figure 1 here]

3.3 Which cognitive variables significantly predict voices?

Based on significant differences in cognitive performance amongst clinical groups identified in preceding ANOVAs, *Visual Learning*, *Social Cognition* and *Inhibition* were simultaneously entered into a binary logistic regression to predict AVH presence. The overall model was significant, $\chi^2(3)=10.0$, $p=.019$, indicating it was able to distinguish between clinical participants who did and did not hear voices. As a whole, the model explained between 9.7% (Cox & Snell) and 13.6% (Nagelkerke) of variance, and accurately predicted AVH presence 68.4% of the time (only a modest increment from 67.3% in the null model). Table 2 shows that only *Visual Learning* was a significant cognitive predictor of AVH presence; voice-hearers were 1.6 times more likely to have difficulties consolidating new information in the visual domain. In contrast, *Social Cognition* and *Inhibition* were not significant predictors.

[Insert Table 2 here]

4. Discussion

The current study aimed to compare cognitive profiles in current, past and never voice-hearers relative to the non-clinical group, and identify significant cognitive predictors of AVH presence. On demographics, participants were well-matched across age and sex, with clinical participants also well-matched across education, employment status and premorbid intellectual functioning. In contrast, current and never voice-hearers were significantly less likely to be partnered relative to non-clinical controls. Clinical groups were well-matched on illness duration as well as mania and psychosis severities. Current voice-hearers scored significantly higher when global positive psychosis symptoms were examined, but as expected, this was no longer significant when the specific impact of hallucinations (i.e. PANSS *Item P3*) was removed. This signified that our clinical groups had comparable positive, negative and general psychosis symptomatology, irrespective of AVH status. Current and past voice-hearers scored significantly higher on hallucination proneness and depressive ratings relative to never voice-hearers, who in turn scored significantly higher than non-clinical controls. An elevated propensity for unusual perceptual experiences in persons with a history of AVHs is to be expected (possibly signifying co-occurrence of hallucinations in other sensory modalities). Current voice-hearers were prescribed significantly higher doses of antipsychotics relative to past and never voice-hearers, possibly reflecting a tendency for increased psychopharmacology directed at AVHs.

As expected, clinical groups in general performed significantly poorer than the non-clinical group across most cognitive domains. There were two exceptions; no differences were evident between past voice-hearers and non-clinical controls for social cognition as well as past and never voice-hearers and

non-clinical controls for inhibition. Given inherent complexities within social cognition, a clear interpretation of this finding is tricky. In particular, the Mayer-Salovey-Caruso Emotional Intelligence Test (MSCEIT) from the MATRICS explicitly assesses the perception of emotion and problem-solving aspects of social information processing (41), but may neglect other pertinent components, such as attributional bias or theory of mind (42). This, coupled with smaller participant numbers encountered during our subgroup analyses may have reduced the sensitivity of our social cognition measure. To reiterate, past voice-hearers showed intermediate performance in social cognition; not only did they did not differ from non-clinical controls, no differences were found with current and never voice-hearers as well.

In contrast, there was little support for our main hypothesis. Though current voice-hearers were significantly impaired on inhibition (relative to non-clinical controls only), this was not significant with respect to past and never voice-hearers. Current voice-hearers were likewise not significantly more impaired in attention/vigilance, working memory or verbal learning relative to past and never voice-hearers. This differs somewhat from limited previous literature (13, 14, 26), but most existing studies did not delineate comparative cognitive profiles of current, past and never voice-hearers (focusing on diagnostic groups instead). Our findings may be further explained by considering that some cognitive deficits could perhaps be more trait-based, meaning these deficits once 'acquired' remain static and do not improve, even if AVHs dissipate. Conversely, other cognitive indicators (e.g. inhibition) could be more state-based, fluctuating in line with hallucination and/or other symptom severity. Inter-individual differences would naturally be expected as well.

Notably, our study found that current and past voice-hearers were significantly impaired on visual learning relative to never voice-hearers (instead of verbal learning, as expected). A possible explanation could relate to the complexity of respective tasks used. In the Brief Visuospatial Memory Test (BVMT; indexing visual learning), six geometric figures are learnt and reproduced concurrently. This contrasts against the Hopkins Verbal Learning Test (HVLT; indexing verbal learning), where a list of 12 words clustered into three taxonomic categories is studied. In fact, the use of these categories likely provides learning support by introducing added structure to the task. Put simply, the HVLT merely requires maintenance of information on a phonological loop (i.e. tapping into working memory), whereas the BVMT additionally entails visual organisation (i.e. executive function-based). So despite both tasks purporting to assess learning, the HVLT may lack sensitivity in detecting subtle learning deficits in the verbal domain that the BVMT is correspondingly able to in the visual domain. If this is the case, a verbal learning task with more equivalent difficulty could be the Rey Auditory Verbal Learning Test (RAVLT; with its longer word list and lack of semantic clustering). This interpretation also accords with our findings

suggesting more significant executive level deficits (and less language-based biases) associated with AVHs. Another possible confounding factor may be antipsychotic medication use, which could exert an undue influence on the visual system, though the evidence supporting this possibility is mixed, with studies finding either significant (43) or minimal (44) impact on eye movements. Therefore, more research is needed to clarify this.

Furthermore, only visual (but not auditory) learning was uncovered as a significant cognitive predictor of AVH presence, with (current and past) voice-hearers being 1.6 times more likely to experience difficulties consolidating new information in the visual domain. Though these findings do not necessarily align with limited previous work, they provide emerging evidence of the role of cross-sensory cognitive deficits possibly contributing to AVHs; that is, deficits in the visual domain as a significant factor in predicting voice symptoms. This is in line with prevailing criticisms involving the multitude of language-based theories currently dominating AVH research (45). In fact, such a bias has culminated in: i) an overemphasis on verbal difficulties in AVHs to the exclusion of other perceptual modalities, and concomitant neglect of broader cognitive dysfunction less dependent on sensory input, as well as ii) the subsequent lack of overarching cognitive models able to account for multimodal hallucinations (i.e. those in the non-auditory realm). Within existing AVH frameworks, our major finding involving significant visual learning deficits also does not clearly fit with the inner speech or memory models. Likewise, this signifies that a broader range of possible underlying cognitive mechanisms (beyond auditory or language-based processes) must be considered to advance an inclusive model of AVHs.

From a therapeutic perspective, a major consideration to have emerged from the current study is that despite the alleged stability of cognition broadly across psychotic illnesses(23), meaningful shifts in line with voice-hearing status are not impossible, especially when individual cognitive facets are analysed. This effect may be compounded when scrutinised from a specific symptom dimension (i.e. AVHs in this case), rather than clinical diagnosis (e.g. schizophrenia), perspective. This fits with calls for greater utility in categorisations used in research settings (46). The pattern of data shown in Figure 1 suggest that some cognitive functions may be more malleable than others. Possibly ‘restored’ inhibition in past voice hearers is a positive indicator this cognitive domain is amenable to change, especially in individuals with a history of AVHs who no longer have these experiences. However before we can delve into possible therapeutic applications, longitudinal research is needed to definitively corroborate the likely direction of causality (i.e. whether past voice-hearing status may be attributed to improved inhibitory control).

4.1 Limitations and strengths

The study had several limitations. Our specified cognitive battery was not exhaustive. There are other viable cognitive mechanisms (e.g. source monitoring deficits) that potentially underlie AVHs, which were not taken into account (9). Similarly, we did not actively consider the role of non-cognitive contributions (potentially accounting for the modest incremental predictive value of our model), such as those involving demographic, clinical or emotional factors. Though comprehensive demographic and clinical participant information was collected, this was primarily employed for matching purposes. Of these variables, the potential influence of age on cognitive performance is of particular importance (22, 25). As we were able to statistically exclude such age effects, we opted to retain our original set of group-wise analyses (see Table B in Supplementary material for additional analyses of covariance). In addition, comorbid psychosis symptoms involving delusions or thought disorder may have exerted a confounding effect. This was beyond the scope of our current study, but also seems less likely as our clinical groups were well-matched in positive, negative and general psychosis symptoms. Given our current state of understanding, a focused study on select cognitive contributors is probably most appropriate, with amalgamation of other non-cognitive factors to take place at a later stage. By the same token, our study was cross-sectional in nature, and did not permit tracking of potential changes in cognitive variables over time, in line with AVH severity. Another key methodological limitation pertained to our definition of *current* voice-hearers as those who had experienced an AVH episode within the past 14 days. Whilst we concur this timeframe may seem somewhat arbitrary, especially given the often sporadic nature of AVHs, until an overriding consensus is reached in relation to the precise definition and measurement of AVH status, a specified cut-off will need to be imposed to advance related research in the field.

Nevertheless, this study was also associated with numerous strengths. Our participants were mostly well-matched across key demographic and clinical variables, with robust participant numbers overall (n=442) as well as within various subgroups under further analyses. Furthermore, we utilised well-validated clinical and cognitive instruments to collect the requisite information, and employed a conservative statistical approach (especially with regards to our logistic binary regression). Lastly, this served as one of the first known studies explicitly delineating comparative cognitive profiles of current, past and never voice-hearers, representing a novel angle to tackle the long-standing conundrum of AVHs in psychosis. This approach is aligned with the RDoC stance of targeting individual mechanistic processes underlying shared psychiatric symptoms (47).

4.2 Directions for future research

There are three key avenues for future research. First, more 'sensitive' cognitive tasks should be employed to tease apart which specific cognitive facets are actually liable, in this case, distinguishing between visual learning versus planning and organisational deficits. Another example would relate to the existing mazes task employed, which despite purportedly assessing reasoning and problem-solving skills, may be surmounted by adequate speed of processing (i.e. quickly and exhaustively going down all dead ends till a solution is reached), as this strategy is not penalised under the current scoring system. To combat this, a more stringent task would be the Porteus maze (48), which does consider these errors in the form of failed attempts. Second, upcoming studies could look towards incorporating added cognitive mechanisms not considered in the current study, or even go beyond this to actively delve into critical non-cognitive factors. Related to this would be expanding the cohorts under study to encompass other voice-hearing groups, involving bipolar disorder and major depressive disorder (49), or even the general population, to explore whether noted cognitive deficits and predictors are disorder-specific, cluster according to certain cognitive patterns, or span the continuum of anomalous perceptual experiences. Embarking on related longitudinal research would also facilitate discovery of whether and if so, which cognitive variables are malleable, and amenable to change in line with varying AVH severity. Such cognitive predictors would serve as the best therapeutic targets for intervention. Third, an alternate prospect would involve broadening the scope of work beyond AVHs to encompass examination of possible cognitive correlates of multimodal hallucinations (e.g. visual, olfactory, tactile and so on). This would reduce the overreliance on language processes in the study of AVHs, and enable creation of an inclusive, overarching model of hallucinations.

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Conflict of interest

The authors have declared no potential conflicts of interest with respect to the research, authorship, and publication of this article.

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Figure legend

Figure 1. Group performance on specified cognitive domains (standardised with respect to non-clinical control performance in green). SOP=Speed of Processing; ATV=Attention/Vigilance; WM=Working Memory; VER=Verbal Learning; VIS=Visual Learning; RPS=Reasoning and Problem-Solving; SOC=Social Cognition; INH=Inhibition.

Table 1

Group comparisons between current, past and never voice-hearers and non-clinical controls on demographic-clinical information and cognitive performance

	Means ± standard deviations/percentages				Statistics[†]			
	Nev (*n*=40)	Pas (*n*=37)	Cur (*n*=46)	NCs (*n*=319)	F/χ^2	*p*	η^2/U	Group contrasts
Demographic								
Age (years)	41.1±9.6	43.5±10.1	40.3±10.3	38.1±7.7	.101	.960	<.001	-
Sex (% male)	67.5	57.4	47.4	47.2	7.1	.070	.126	-
Education (years)	13.9±3.2	14.1±3.6	13.9±2.7	16.4±2.7	20.2	**<.001**	.140	Nev=Pas=Cur<NCs
Employment (% employed[‡])	25.0	21.3	35.1	59.1	39.3	**<.001**	.297	Nev=Pas=Cur<NCs
Marital status (% partnered[§])	0	13.0	5.3	29.2	27.9	**<.001**	.251	Nev=Cur<NCs
Premorbid IQ (i.e. WTAR)	100.6±11.7	103.9±11.7	98.3±13.1	111.2±9.8	134.7[¶]	**<.001**	.179	Nev=Pas=Cur<NCs
Clinical								
Illness duration (years)	15.5±10.6	17.2±10.4	16.6±9.7	-	.249	.780	.004	-
Hallucination proneness (i.e. LSHS)	13.5±13.2	21.8±11.0	26.9±11.3	8.1±7.3	118.0[¶]	**<.001**	.372	Pas=Cur>Nev>NCs
Depression (i.e. MADRS)	7.4±9.5	11.4±9.9	12.1±10.1	1.8±3.2	117.7[¶]	**<.001**	.320	Pas=Cur>Nev>NCs
Mania (i.e. YMRS)	3.8±4.5	5.0±5.2	6.2±7.0	1.1±1.8	68.6[¶]	**<.001**	.236	Nev=Pas=Cur>NCs
Psychosis (i.e. PANSS)	58.9±16.8	59.0±16.1	66.0±18.0	-	2.4	.091	.039	-
Positive	14.5±5.2	14.8±6.1	18.6±5.4	-	7.5	**.001**	.111	Nev=Pas<Cur
Positive - hallucinations (Item P3)	13.2±5.0	12.6±5.1	14.1±4.8	-	1.0	.365	.017	-
Negative	15.2±6.2	13.2±4.9	14.7±5.9	-	1.2	.314	.019	-
General	29.3±9.0	30.8±9.6	32.8±9.8	-	1.5	.238	.024	-
Antipsychotics (mg *chlorpromazine*≈)	564±415	514±379	952±1242	-	3.7	**.027**	.056	Nev=Pas<Cur

Cognitive								
Speed of Processing	41.4±11.4	39.8±13.3	40.7±10.8	55.4±10.1	56.6	**<.001**	.279	Nev=Pas=Cur<NCs
Attention/Vigilance	42.1±12.6	39.1±13.6	38.8±12.4	47.8±9.2	10.8[¶]	**<.001**	.107	Nev=Pas=Cur<NCs
Working Memory	43.8±11.1	42.8±8.7	40.4±9.9	54.5±27.0	7.9	**<.001**	.052	Nev=Pas=Cur<NCs
Verbal Learning	39.9±10.1	38.8±9.2	37.8±9.2	48.7±9.4	33.1	**<.001**	.186	Nev=Pas=Cur<NCs
Visual Learning	43.9±14.9	36.2±14.2	34.7±12.0	52.5±9.6	36.3[¶]	**<.001**	.279	Pas=Cur<Nev<NCs
Reasoning and Problem-Solving	43.3±9.5	43.0±10.0	42.0±9.8	52.1±10.5	24.0	**<.001**	.143	Nev=Pas=Cur<NCs
Social Cognition	38.7±13.5	41.6±13.6	39.9±12.1	47.1±11.5	9.5	**<.001**	.066	Nev=Cur<NCs
Inhibition (i.e. Stroop)	10.8±2.4	10.7±2.8	9.6±3.0	11.1±2.1	3.8[¶]	**.012**	.036	Cur<NCs

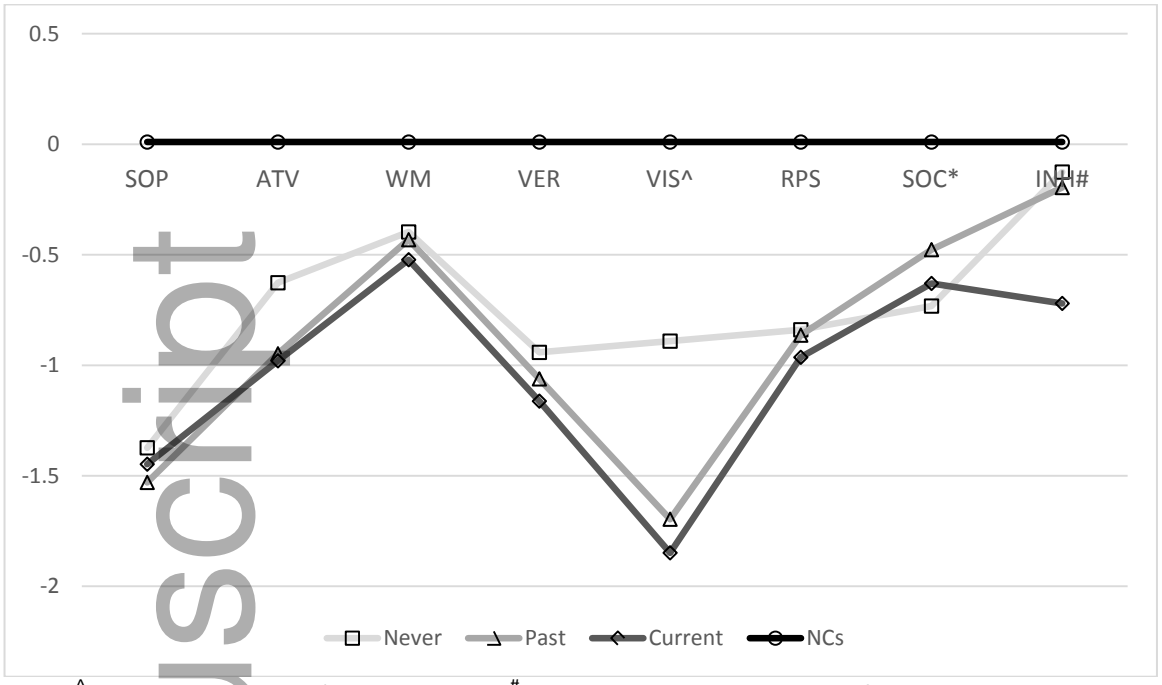
Note. Nev=Never; Pas=Past; Cur=Current; NCs=Non-clinical controls; WTAR=Wechsler Test of Adult Reading (100±15), LSHS=Launey-Slade Hallucination Scale (0-48), MADRS= Montgomery Äsberg Depression Rating Scale (0-60), YMRS=Young Mania Rating Scale (0=60), PANSS=Positive and Negative Syndrome Rating Scale (30-210). For cognition, the first seven domains were derived from the MATRICS Consensus Cognitive Battery (MCCB; 50±10), and the eighth cognitive domain was based on the D-KEFS Stroop test as a measure of inhibition; means and standard deviations presented for descriptive purposes only, normative *z*-scores were calculated and employed in ensuing analyses.

[†] One-way ANOVAs (for continuous variables) with effect size η^2 (.01=small, .06=medium, .14=large), or chi-squared tests for independence (for categorical variables) with effect size Cramer's *V* (.01=small, .30=medium, .50=large)[36]. [‡] Employed includes full- or part-time work. [§] Partnered refers to married or *de facto* status. [¶] Brown-Forsythe.

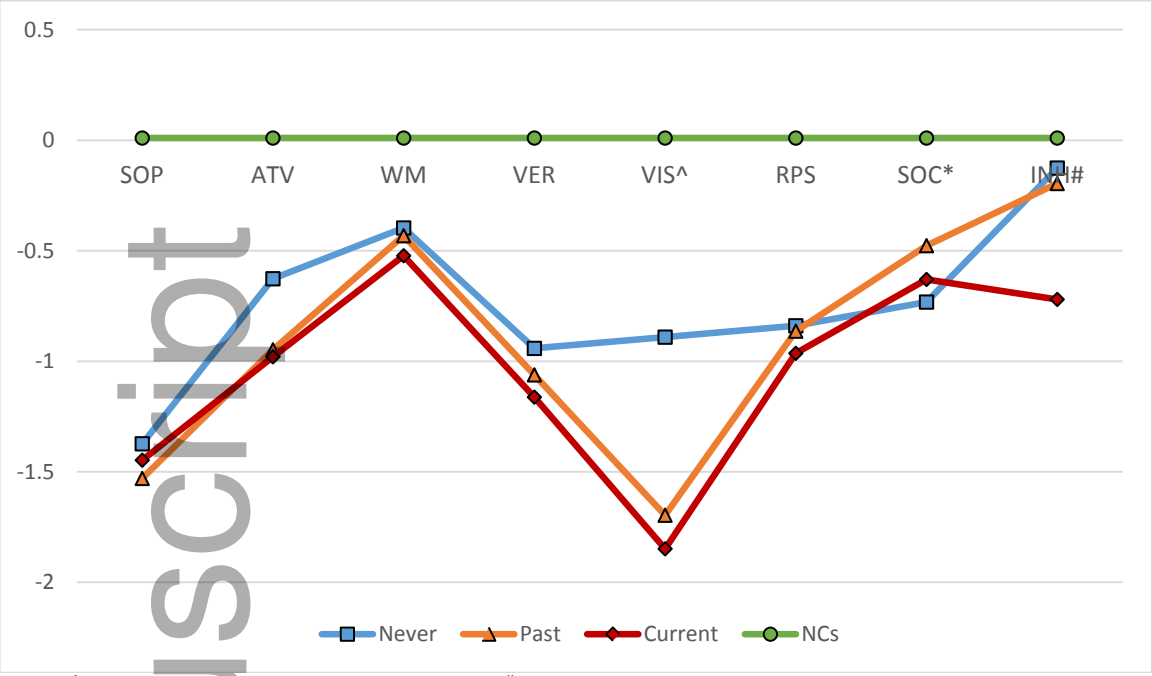
Table 2

Binary logistic regression examining cognitive predictors of AVHs in clinical groups only

Cognitive predictor	**B**	**Wald**	***p***	**Odds ratio**	**95% confidence interval**
Visual learning	.478	7.36	**.007**	1.61	1.14-2.28
Social cognition	.309	2.04	.154	1.36	.891-2.08
Inhibition (i.e. Stroop)	.023	.014	.904	1.02	.706-1.48



Note. ^Pas=Cur<Nev<NCs, *Nev=Cur<NCs, #Cur<NCs; Nev=Pas=Cur<NCs for all other cognitive domains.



Note. ^Pas=Cur<Nev<NCs, *Nev=Cur<NCs, #Cur<NCs; Nev=Pas=Cur<NCs for all other cognitive domains.