

Auditory Verbal Hallucinations in Persons With and Without a Need for Care

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Auditory verbal hallucinations (AVH) are complex experiences that occur in the context of various clinical disorders. AVH also occur in individuals from the general population who have no identifiable psychiatric or neurological diagnoses. This article reviews research on AVH in nonclinical individuals and provides a cross-disciplinary view of the clinical relevance of these experiences in defining the risk of mental illness and need for care. Prevalence rates of AVH vary according to measurement tool and indicate a continuum of experience in the general population. Cross-sectional comparisons of individuals with AVH with and without need for care reveal similarities in phenomenology and some underlying mechanisms but also highlight key differences in emotional valence of AVH, appraisals, and behavioral response. Longitudinal studies suggest that AVH are an antecedent of clinical disorders when combined with negative emotional states, specific cognitive difficulties and poor coping, plus family history of psychosis, and environmental exposures such as childhood adversity. However, their predictive value for specific psychiatric disorders is not entirely clear. The theoretical and clinical implications of the reviewed findings are discussed, together with directions for future research.

Key words: nonclinical/need for care/psychosis/prevalence

Introduction

Auditory hallucinatory phenomena occur on a spectrum ranging from auditory imagery and intrusive and vivid

thoughts to fully developed hallucinations of hearing sounds and voices. Although traditionally associated with psychiatric and neurological diagnoses, hallucinations may also be present in healthy individuals without need for care. It has been observed that individuals with AVH vary widely in their need for care, and clinical status may change over a person's lifetime. Understanding the factors that are relevant in leading to or protecting from need for care can inform clinical interventions. This article brings together research findings on auditory verbal hallucinations (AVH) in the general population and considers the clinical relevance of these experiences. We cover the different methodological approaches that have been adopted to elucidate the factors related to the process of "transition" to a need for care, including longitudinal epidemiological studies, as well as comparison of AVH present in persons with and without a need for care. In this review, we combine the various, often isolated, research streams on multiple aspects of AVH into a cross-disciplinary overview, which documents areas of emerging consensus as well as highlighting contentious and underresearched domains.

The article was initially prepared for the Second Meeting of the International Consortium on Hallucination Research (Durham, UK, September 2013). Beforehand, the authors created a list of topics considered important and/or neglected in the area of AVH in persons with and without a need for care. Then, the authors worked in small groups to expand on these areas, by analyzing

the literature and drawing on their own expertise and research findings, in order to extract the key components in understanding the continuum of AVH experience and risk for clinical disorder.

What Is the Prevalence Rate of AVH in the General Population?

Only a few studies have specifically examined the prevalence of AVH in the general population. The reported prevalence varies widely: in a historical overview of 17 studies from the late 19th to early 21st century,¹ the rates of AVH ranged from 0.6% to 84% (median: 13.2%). Linscott and van Os² retrieved 56 reports containing data on rates of psychotic symptoms in adult community samples and report a median lifetime prevalence rate of 4.1% for hallucinations (all hallucination types grouped together). In the first cross-national (52 countries) study,³ an age- and gender-adjusted estimate of 5.8% for hallucinations (all types grouped together) was reported, but with highly varying prevalence rates across countries (from 0.8% in Vietnam to 31.4% in Nepal). The rates appear higher in children and adolescents: Kelleher et al,⁴ in their meta-analysis, found a prevalence of 14.8% in children and adolescents (age range 9–18 years) specifically for AVH. Moreover, there were no clear differences between rates for children versus adolescents (13.8% and 15.7%, respectively) (also see Jardri et al⁵).

A limit with basing prevalence rates on meta-analyses is the methodological heterogeneity in how AVH are assessed, the timeframe used, and characteristics of the population, resulting in large variations in prevalence rates (cf. Beaven et al¹). Also, important nuances are lost, such as the nature and frequency of the AVH. For example, in Johns et al,⁶ 4.2% of the general population surveyed answered affirmatively to a general hallucination item (“Over the past year, have there been times when you heard or saw things that other people could not”), whereas only 0.7% endorsed a more specific AVH item (“Did you at any time hear voices saying quite a few words or sentences when there was no one around that might account for it?”).

Is There a Continuum of Hallucinations and Psychosis?

A dimensional view posits that (1) AVH and other psychotic experiences lie on a continuum with normal experience⁷ and (2) psychosis exists in the population as a continuous phenotype.⁷ Such a continuum model is helpful for understanding AVH in terms of normal cognitive processes and facilitating research into etiological factors and clinical trajectories. Two types of continua can be distinguished, both within and across individuals⁹: (1) A continuum of experience, whereby different experiences (daydreams, intrusive and vivid thoughts) lie on a common continuum with AVH; (2) a continuum of risk, in which people differ in (a) their proneness to experience

AVH and (b) their risk of developing problematic AVH with need for care. This section considers the continuum of AVH experience across individuals (phenomenological continuity), including the continuum of risk for psychosis (structural continuity), and reviews evidence for and against putative continua. Although there is robust evidence for a continuum of psychotic experiences, with a distribution in the general population, there is less evidence that this represents a single underlying continuum of risk for psychosis.^{10,11}

Evidence for a Continuum

Phenomenological continuity is indicated by studies showing that more people experience AVH and other psychotic experiences than those individuals who receive psychiatric diagnoses,¹² with a range of reported hallucination prevalence rates in nonclinical samples. Further, in these population samples, hallucinations are correlated with delusions, just as they are in psychotic disorders.¹³

Evidence for structural continuity (a single group in the population with quantitative variation in phenotype expression) comes from similar associations between key risk factors and both psychotic experiences and psychotic disorder, suggesting etiological continuity between them.¹⁴ These risk factors include younger age, ethnic minority status, lower education, alcohol and drug use, stressful or traumatic events, urbanicity, and family history of psychotic disorders.^{6,15} Other evidence for etiological continuity comes from direct comparisons of individuals with AVH with and without need for care, which reveal partly similar neurocognitive processes and brain regions underlying AVH in both. This suggests common cognitive mechanisms across the continuum of AVH experiences irrespective of clinical status, but with some cognitive difficulties increasing in severity along the continuum of risk for AVH with a need for care.

A fully continuous relationship between psychotic symptoms and disorder can be distinguished from a quasi-continuous/continuum-threshold model.^{13,16} The latter is more consistent with the observed skewed distribution of AVH in the population, qualitative differences in these experiences along the continuum, and the contribution of various risk factors in making “transitions” from non-clinical to clinical states. Findings from studies comparing the AVH reported by individuals with and without need for care indicate that in addition to similarities, there are also specific differences in the experience, and possibly the underlying mechanisms, of AVH across the continuum, some of which might contribute to clinical status.

Evidence Against a Single Continuum

Factors of individual difference,¹⁶ general psychological distress,¹⁷ and psychosis proneness¹² could either determine where a person lies on a continuum of AVH or reflect different interacting continua (which give rise to

an apparent single continuum). There is emerging evidence that a latent categorical structure of the population underlies the observed continuum of psychosis experience,² with 1 group who are liable to psychosis and another group who are not. In the former, AVH are associated with other cognitive and emotional difficulties and a greater likelihood of need for care, while in the second group, AVH have reduced morbidity and possibly different etiology.¹⁸ This could partly explain why 2 people with the same level of AVH may differ in their clinical outcome. So, although AVH and other psychotic experiences seem to be continuous and distributed across the general population, the risk for developing psychosis might actually be discontinuous rather than truly continuous in the population. Further research is needed to understand these different continua and any underlying factors that could potentially serve as biomarkers.

What Are the Similarities and Differences in the AVH Reported by Individuals With and Without Need for Care? (see figure 1)

Phenomenology

AVH in individuals with and without need for care are broadly similar as perceptual phenomena and in terms of topographical features such as localization (internal or external), loudness, and number of voices.^{19,20} Regardless of need for care, hallucinators tend to personify their voices (ie, attribute their voices to a real person or entity)¹⁹ and seem to share similar underlying brain activity.²¹ Differences lie largely in the frequency and duration with which voices are experienced and age of onset, with the nonclinical group starting to have AVH at a younger

age, often in childhood.¹⁹ The most significant differentiating factor, however, is the degree of negative voice content, with patients reporting a preponderance of negative voices, while AVH in individuals without need for care are mostly neutral or pleasant in content.^{19,20,22} This suggests that negative content is crucial in determining increased distress and need for care.^{23,24} However, many patients also report some positive voice content,²⁵ and it is important to examine the balance of negative and positive voices a person hears.

Cognition

Regardless of need for care, individuals with AVH have difficulty on tasks measuring cognitive control functions, such as controlling the direction of attention in the face of distracting information and the active suppression of intrusions. A meta-analysis²⁶ of 9 studies on source monitoring in individuals with AVH without need for care found a significant, moderate-to-large effect, which did not differ from findings in individuals with AVH with need for care. However, given the relatively small number of studies, further replications are desirable before concluding with certainty that individuals with AVH have a specific, similar-sized source monitoring difficulty independent of their need for care. Due to the range of dysfunctional control components (such as intrusive cognitions, source monitoring, and inhibitory control) in cognitive models of AVH,²⁷ studies of cognitive control functions dominate the literature on cognitive correlates of AVH. Future studies should concentrate on detailing the pattern of affected and intact subcomponents of executive functioning in persons with AVH (cf. Waters et al²⁸).



Fig. 1. Principal differences and similarities between auditory verbal hallucinations experienced by persons with a need for care and those without a need for care.

Memory has also been investigated in hallucinators without need for care. No outstanding deficit appears in control-demanding episodic long-term and short-term tasks nor in binding memories to a specific context (see Badcock et al,²⁹ Chhabra et al,³⁰ and McKague et al³¹). Thus, it appears that lapses in cognitive control in these individuals are not coupled to wider difficulties in memory processing. This represents a potential discontinuity between the cognitive profiles of individuals with AVH with and without need for care²⁷ and could furthermore be a future target for cognitive training interventions.

Given the contribution of low-level sensory and perceptual processing to AVH in individuals with need for care,³² more detailed examination of these influences would be valuable in persons with AVH without need for care. For example, hallucinating individuals without need for care and nonhallucinating controls process various acoustic dimensions of voices similarly, whereas hallucinators with need for care rely less on certain acoustic features.^{33,34} On the other hand, increased tone detection threshold has also been shown in individuals with AVH without need for care,³⁵ suggesting similarity with clinical groups in very basic auditory functions. Furthermore, cognitive dysfunction is a frequent symptom of patients with psychotic disorders, especially those with a diagnosis of schizophrenia. People with AVH without need for care, however, tend to have cognitive functioning within the normal limits.³⁶ It is possible that intact cognitive functioning is a major protective factor for people with AVH who do not develop need for care. More research on this topic is needed.

Neurobiology

Structural neuroimaging in individuals with AVH and a need for care has shown that gray matter loss in superior temporal regions³⁷ as well as the insula³⁸ is associated with hallucination severity. It is not yet known whether the same applies to people with AVH without a need for care. A structural connectivity study has suggested similar alterations in the microstructure of the arcuate fasciculus in hallucinating individuals with and without a need for care³⁹ compared with nonhallucinating controls. Functional neuroimaging suggests that the neural correlates of experiencing AVH are the same in individuals irrespective of need for care. Thus, neuroimaging studies on the “state” (vs trait) of hallucinations in people with and without need for care have not observed significant differences in activation of the brain regions involved.⁴⁰ However, some possible markers for transition to need for care have been found, such as elevated striatal dopamine capacity, which appears to be specific for predicting psychosis but is not associated with the presence of AVH per se.⁴¹ Similarly, Diederer et al⁴² suggest that decreased functional lateralization, a mechanism proposed as important in the development of AVH,⁴³ is specific to psychosis because they found no evidence

for functional lateralization in individuals with AVH without need for care. However, within schizophrenia patients, there appears to be a continuous relationship between the severity of AVH and degree of functional lateralization.^{43,44}

Life Events

Robust associations have been found between traumatic life events and AVH in both those with need for care^{45–47} and without need for care.⁴⁸ Large-scale epidemiological studies of the general population have observed the same association when controlling for a range of confounds.⁴⁹ More recent work has examined whether the type of trauma experienced predicts need for care. Daalman et al⁴⁸ found no differences between 100 psychiatric patients with AVH and 127 individuals with AVH without a need for care in prevalence of specific types of abuse: both groups were more likely to have experienced sexual as well as emotional abuse than were nonhallucinating control participants. Goldstone et al⁵⁰ modeled hallucination proneness among 100 patients with psychotic disorders and 133 students. In the student sample, emotional trauma in childhood, combined with proximal life stressors, was the strongest predictor of proneness to AVH, while sexual abuse was the strongest predictor in the clinical group.

Although there is no clear evidence of trauma type differentiating need for care and non-need for care groups with frequent voices, traumatic stressors may be of lower impact in those without a need for care, and their psychological sequelae may be less persistent. Thus, while sexual and emotional trauma may initiate hallucination onset per se, it may be the psychological impact of the trauma that encourages the development (and maintenance) of clinically significant AVH by negatively influencing beliefs about voices, which in turn predicts the levels of distress and impairment experienced.²² McCarthy-Jones⁵¹ has proposed that 2 specific posttrauma factors may promote the development of negative AVH. The first is the degree of shame and self-blame the person feels in relation to the traumatic event, and the second is the degree of social or emotional isolation following the trauma. McCarthy-Jones⁵¹ argues, following Romme et al,⁵² that it is these emotions and the failure for them to be expressed, which form the basis for the negative content of AVHs. However, this hypothesis remains to be tested. The high rates of trauma exposure in people with AVH both with and without need for care support the need for continued research into relevant developmental events and additive vulnerabilities to understanding the pathways to the distress and disruption that necessitates clinical care.

Appraisals, Coping, and Relationships

The cognitive model of voices proposes that the beliefs people hold about their voices^{53,54} and their social schemata^{55,56} mediate the relationship between the voice

experience and behavioral and affective response. There is accumulating evidence that appraisals about identity,³² intent and power,⁵⁷ and the nature of the relationship with the personified voice^{58–60} are more important determinants of distress and disruption than voice activity per se.

A number of studies have compared voice appraisals, and the relationship between the voice and the individual, in people with AVH with and without a need for care. Individuals without a need for care report higher perceived control over their voices than do people with a need for care.²² They also display less symptomatic coping⁶¹ (ie, going along with the content of voices), engage in fewer safety behaviors in relation to their anomalous experiences,⁶² and score lower on maladaptive response styles in response to experimentally induced anomalous experiences.⁶³ Voice hearers with a need for care are more likely to attribute their voices to real people or agencies, as opposed to spiritual or religious sources,¹⁹ and generally have more “paranoid” appraisals both of their own experiences^{64,65} and of experimentally induced anomalous experiences.⁶³ They are more likely to appraise their voices as malevolent, omnipotent, intrusive, dominant, and coercive and, consequently, are more likely to resist them and keep their distance.^{22,66,67} Individuals with a need for care also display more cognitive biases⁶⁸ and negative metacognitive beliefs about thoughts.²⁰ It is possible, however, that these differences in appraisal are secondary to differences in emotional content, an issue that deserves further attention.

Strengths and Limitations of This Approach

Comparing AVH experienced by groups of people with and without a need for care suggests some factors that may explain why some hallucinators develop a clinical status and others do not. However, if the proposed latent categorical structure of the population is correct in relation to psychosis, then studies comparing hallucinating individuals with and without psychosis/need for care might well be comparing participants drawn (in varying proportions) from 2 qualitatively distinct groups, which will confound their findings. Furthermore, these studies might not be comparing like with like in terms of the phenomenology of the AVH or the severity of other symptoms, such as delusions or cognitive dysfunction, across the 2 groups.⁴¹ Cross-sectional comparisons are limited in answering questions about the continuum of risk for developing AVH with need for care, which are best addressed with epidemiological and longitudinal studies.

What Can Nonclinical Hallucinations Say About Risk for Psychosis and Need for Care?

Clinical Outcomes of Those Who Hallucinate

There are various outcomes of those who experience AVH in early life in terms of continued experience, mental health status, and functioning. The hallucinations may cease or

continue with no negative impact; indeed, the most common outcome of hallucinatory and other psychotic-like experiences in childhood is discontinuation of these experiences^{69,70} (see also Jardri et al⁵). For instance, Bartels-Velthuis et al⁷¹ report that as many as 76% of children who reported hearing voices at 7 and 8 years of age stopped hearing voices by age 12–13. However, for others, AVH persist into adolescence and adulthood and can develop in some people to psychotic disorder or other diagnosable mental health problems. Longitudinal cohort studies have shown that hallucinations and other psychotic symptoms in children and adolescents are associated with an increased risk of later diagnosis of mental illness, but results diverge on whether adolescent AVH specifically increase the future risk of psychotic disorders.^{72–74} It is still not wholly understood why some individuals with AVH develop particular adverse mental health outcomes although a number of specific factors have been identified, which converge with those identified by cross-sectional studies.

Psychological Mechanisms Mediating Transition to Psychosis

The literature suggests 3 key psychological factors that seem to influence risk for developing a psychotic disorder in those with nonclinical AVH: Cognitive biases, negative affect, and coping style. These interdependent processes may synergistically increase psychosis risk by fuelling the impetus for delusion formation and elevating distress associated with hallucinations.^{24,75}

Cognitive Biases. Various idiosyncratic cognitive processes are implicated in transitioning to AVH with need for care, but the mechanisms are sometimes difficult to test directly. These processes include a jumping to conclusions (JTC) reasoning bias, hypervigilance to threat-related stimuli, externalizing and personalizing attributional biases, contextual information integration difficulties, source monitoring errors, and poor Theory of Mind skills.^{76,77} It remains unclear how much these processes contribute to the development of clinical status rather than the occurrence of AVH per se. Top-down decision-making and thinking biases, such as intentionalizing and JTC, seem to be involved in the transition to clinical psychosis,⁶⁸ whereas bottom-up cognitive processes are important for the formation of AVH across the continuum. The influence of top-down cognitive processes on clinical transitions is supported by findings from the longitudinal Netherlands Mental Health Survey and Incidence Study (NEMESIS), in which onset of delusional ideation at 1-year follow-up increased the risk of psychosis at 3-year follow-up in those with hallucinations at baseline.²⁴

Affect. Negative emotional states play a role in both the onset and maintenance of psychotic disorder. The NEMESIS study found that the individuals reporting

hallucinations at baseline who developed depressed mood a year later were at increased risk of developing a psychotic disorder 3 years later.^{78,79} Anxiety levels have also been found to be predictive of higher levels of distress in those experiencing AVH,⁸⁰ which may lead to need for care. Negative affect seems to influence transition to clinical states in various ways: Associated negative thought content can lead to more negative voices^{51,81}; emotional states may exacerbate relevant cognitive biases⁸²; and depressed and anxious mood can reduce effective coping.

Integrating These Psychological Factors. Appraisals of hallucinations are given central importance in cognitive models of psychosis development.⁷⁵ Cognitive biases impact on appraisals of voices, whereby unusual and confusing experiences that seem caused by an external agency are appraised as such. External and personalizing appraisals, particularly those that are threatening and with lower perceived control, are likely to produce feelings of distress and unhelpful reactions of either preoccupation or avoidance, which may ultimately result in need for care. Appraisals of voices influence coping action⁸³ and voices construed as benign have been found to be associated with a greater range of coping strategies.⁸⁴ Conversely, Escher et al⁷⁰ found that adolescents feeling overwhelmed by the experience of voices at baseline used more defensive coping responses and were more likely to develop depression over a 3-year follow-up period. As mentioned, this negative mood may also impact on appraisal process, fuelling the process of development of further psychotic symptoms and disorder. In addition, distal and proximal environmental factors, such as adverse life events, stress, and isolation, have an impact on these psychological processes. For example, Bartels-Velthuis et al⁶⁹ found that exposure to childhood adversity increases the intrusiveness of the hallucinatory experience, together with distress and external locus of control, all of which may increase the risk of secondary delusional ideation.

In summary, there are individual differences in the proneness to experiencing AVH, possibly related to differences in auditory function, cognitive control, self-monitoring, and dissociative tendencies. The association between trauma and AVH suggests that something about the experience of trauma influences the cognitive and emotional processes that give rise to AVH, and there may be more than 1 etiological process.⁸⁵ It is not clear why AVH persist in some individuals or the prognostic significance of this. The onset of AVH tends to be younger in individuals without a need for care, suggesting that persisting AVH are not always clinically relevant. On the other hand, longitudinal studies have found that persistence of AVH into adolescence is associated with negative clinical outcomes.⁷⁶ Studies indicate that, in addition to psychotic disorders, AVH of similar phenomenology are associated with a number of other psychiatric diagnoses, including bipolar, borderline,

and dissociative disorders.⁸⁶ We have yet to elucidate the factors that determine these different clinical trajectories. The biased focus on psychosis and schizophrenia as a risk for individuals reporting AVH draws attention away from other disorders that are associated with AVH.

Discussion

This review raises a number of questions and directions for future research, which are considered below.

Are There Multiple Types of AVH Without Need for Care?

There are differences among nonclinical hallucinators, reflecting either different points on a single continuum of AVH or separate subtypes.^{9,87} Our label “individuals with AVH without need for care” may thus be further divided into (1) “Hallucination-prone” individuals, who experience brief AVH infrequently, usually under specific conditions (eg, sleep deprivation, mourning); there are few other subclinical symptoms, and these AVH do not affect the person’s functioning; (2) “Nonclinical voice hearers,” who experience more frequent AVH of longer duration. These AVH are often associated with other subclinical psychotic and mood symptoms. There may be a family history of psychiatric illness, and the degree of need for care may vary (see below). These 2 groups are usually based on different assessment strategies: The former is determined using hallucination-proneness measures (eg, the Launay-Slade Hallucination Scale, see [supplementary appendix](#)), whereas the latter group is often assessed with interview schedules (as in Sommer et al⁸⁸). This distinction is important when understanding the literature and designing future studies, as findings will be affected the way participants are assessed and grouped in terms of their AVH. For instance, the different results observed in the Daalman et al⁴⁸ and Goldstone et al⁵⁰ studies examining the role of trauma in AVH may be related to the type of participants recruited: Daalman et al⁴⁸ included a nonclinical group who heard frequent voices, whereas Goldstone et al⁵⁰ assessed hallucination-prone students.

What Is the Predictive Value of AVH?

A crucial question is whether experiencing AVH predicts the development of clinical states and/or future need for care. AVH in children mostly cease spontaneously before adolescence, but persistence during and beyond adolescence is associated with greater risk of developing various clinical disorders.⁷⁶ The psychosis proneness-persistence-impairment model¹² attempts to explain this trajectory for psychosis, whereby psychotic experiences that become more numerous and persistent over time (due to an interaction between psychological factors, environmental exposures, and genetic risk) increase the probability of

onset of clinical psychosis. The presence of AVH might give rise to secondary delusional ideation (to explain AVH), resulting in the emergence of a combined “hallucinatory-delusional state,” which may then elicit negative emotions and maladaptive coping, plus other symptoms, leading to functional impairment and a diagnosable psychotic disorder.⁶⁹ The need to explain the often unusual nature of AVH seems a logical necessity for human behavior and might be more likely for AVH that persist rather than transient experiences or when the person experiences AVH as an adolescent or adult rather than as a child. Furthermore, this need for explanation is influenced by cognitive biases,⁷⁵ which may be related to a separate risk for psychosis that interacts with the presence of AVH.

In adult samples, some individuals with AVH “without a need for care” may experience other difficulties for which they do develop a need for care although not requiring specialized psychiatric services. Their AVH might be a reaction to life stress or change, formulated as a symptom of distress or a type of coping mechanism.⁷⁶ The sample of nonclinical voice hearers described by Sommer et al⁸⁸ had additional subclinical symptoms (delusional ideation, schizotypy) and slightly reduced global functioning. In a general population sample (excluding those with clinical psychosis),⁸⁹ it was found that hallucinations in the past year were associated with seeing a family doctor for emotional problems and with counseling/therapy although were not an independent predictor of family doctor attendance after controlling for other help-seeking correlates.

What Are the Clinical Implications?

If transitions to clinical AVH and distress are influenced by cognitive processes, appraisals, and coping styles, then psychological interventions can target these factors (see Thomas et al⁹⁰), both to prevent or delay transition to clinical states and to promote recovery in those with AVH and need for care. Cognitive behavior therapy aims to reduce distress and empower the individual by modifying threatening appraisals and building up a normalizing view of voices.⁹¹ Similarly, acceptance and commitment therapy aims to reduce experiential avoidance of AVH and foster adaptive coping.⁹² Compassion-focused therapies for improving self-esteem and reducing shame associated with trauma can reduce the negative content of AVH.⁹³ Emotion regulation strategies may be helpful if AVH are triggered by intense distress, together with formulating the emotional conflict.⁹⁴ Reasoning training⁹⁵ and metacognitive training⁹⁶ are promising interventions for reducing cognitive biases such as JTC, and cognitive remediation can address various cognitive control and executive functions underlying AVH.⁹⁷

In addition to psychological factors, we can look to the cultural context (see Luhrmann et al⁹⁸) and how accepted AVH are in Western society. Reducing the stigma surrounding AVH would likely impact on how

the person reacts emotionally and behaviorally to them. Furthermore, we should now be intervening to reduce the incidence and impact of childhood adversity, which is a key risk factor for AVH and other psychotic symptoms.⁹⁹

What Methodological Issues Should be Addressed in Future Studies?

Studies of individuals with AVH without need for care often have varying inclusion and exclusion criteria. Including a diagnostic interview would be helpful, together with measures of other subclinical symptoms (eg, anxiety, depression, and delusional ideation), as their presence may have an impact on AVH (see [supplementary appendix](#)). It would also be very helpful to learn more about the type, significance, and content of the voices experienced. More sensitive assessment tools (especially self-report) are needed to identify those with AVH who might be at risk of transitioning to a clinical disorder, and we recommended that studies follow up screening measures with detailed interviews. We need replications of findings in other samples of individuals with AVH without need for care because most of our knowledge about nonclinical, frequent AVH is based on the sample recruited by Iris Sommer’s group. Combining longitudinal and cross-sectional methodologies may also be productive.

Overall, greater methodological rigor is needed to advance our understanding of AVH in persons with and without a need for care. This involves using similar inclusion criteria and assessments of participants, and minimizing confounding variables, in order to improve the comparability of results across different studies. This action point will be taken forward by the Consortium.

Supplementary Material

Supplementary material is available at <http://schizophreniabulletin.oxfordjournals.org>.

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References

1. Beavan V, Read J, Cartwright C. The prevalence of voice-hearers in the general population: a literature review. *J Ment Health*. 2011;20:281–292.
2. Linscott RJ, van Os J. Systematic reviews of categorical versus continuum models in psychosis: evidence for discontinuous subpopulations underlying a psychometric continuum. Implications for DSM-V, DSM-VI, and DSM-VII. *Annu Rev Clin Psychol*. 2010;6:391–419.
3. Nuevo R, Chatterji S, Verdes E, Naidoo N, Arango C, Ayuso-Mateos JL. The continuum of psychotic symptoms in the general population: a cross-national study. *Schizophr Bull*. 2012;38:475–485.
4. Kelleher I, Connor D, Clarke MC, Devlin N, Harley M, Cannon M. Prevalence of psychotic symptoms in childhood and adolescence: a systematic review and meta-analysis of population-based studies. *Psychol Med*. 2012;42:1857–1863.
5. Jardri R, Bartels-Velthuis AA, Debbane M, et al. Hallucinations in children and adolescents: an update. *Schizophr Bull*. 2014;40:S221–S232.
6. Johns LC, Cannon M, Singleton N, et al. Prevalence and correlates of self-reported psychotic symptoms in the British population. *Br J Psychiatry*. 2004;185:298–305.
7. Claridge G. Theoretical background and issues. In: Claridge G, ed. *Schizotypy. Implications for Illness and Health*. Oxford, UK: Oxford University Press; 1997:319.
8. van Os J, Hanssen M, Bijl RV, Ravelli A, Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res*. 2000;45:11–20.
9. Laroí F. How do auditory verbal hallucinations in patients differ from those in non-patients? *Front Hum Neurosci*. 2012;6:1–9.
10. David AS. Why we need more debate on whether psychotic symptoms lie on a continuum with normality. *Psychol Med*. 2010;40:1935–1942.
11. Lawrie SM, Hall J, McIntosh AM, Owens DG, Johnstone EC. The ‘continuum of psychosis’: scientifically unproven and clinically impractical. *Br J Psychiatry*. 2010;197:423–425.
12. van Os J, Linscott RJ, Myin-Germeys I, Delespaul P, Krabbendam L. A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness-persistence-impairment model of psychotic disorder. *Psychol Med*. 2009;39:179–195.
13. Johns LC, van Os J. The continuity of psychotic experiences in the general population. *Clin Psychol Rev*. 2001;21:1125–1141.
14. Linscott RJ, van Os J. An updated and conservative systematic review and meta-analysis of epidemiological evidence on psychotic experiences in children and adults: on the pathway from proneness to persistence to dimensional expression across mental disorders. *Psychol Med*. 2013;1:1–17.
15. Rössler W, Vetter S, Müller M, et al. Risk factors at the low end of the psychosis continuum: much the same as at the upper end? *Psychiatry Res*. 2011;189:77–81.
16. Claridge G. Single indicator of risk for schizophrenia: probable fact or likely myth? *Schizophr Bull*. 1994;20:151–168.
17. Varghese D, Scott J, Welham J, et al. Psychotic-like experiences in major depression and anxiety disorders: a population-based survey in young adults. *Schizophr Bull*. 2011;37:389–393.
18. Kaymaz N, van Os J. Extended psychosis phenotype—yes: single continuum—unlikely. *Psychol Med*. 2010;40:1963–1966.
19. Daalman K, Boks MP, Diederer KM, et al. The same or different? A phenomenological comparison of auditory verbal hallucinations in healthy and psychotic individuals. *J Clin Psychiatry*. 2011;72:320–325.
20. Hill K, Varese F, Jackson M, Linden DE. The relationship between metacognitive beliefs, auditory hallucinations, and hallucination-related distress in clinical and non-clinical voice-hearers. *Br J Clin Psychol*. 2012;51:434–447.
21. Diederer KMJ, Van Lutterveld R, Sommer IEC. Neuroimaging of voice hearing in non-psychotic individuals: a mini review. *Front Hum Neurosci*. 2012;6:1–5.
22. Andrew EM, Gray NS, Snowden RJ. The relationship between trauma and beliefs about hearing voices: a study of psychiatric and non-psychiatric voice hearers. *Psychol Med*. 2008;38:1409–1417.
23. Beavan V, Read J. Hearing voices and listening to what they say: the importance of voice content in understanding and working with distressing voices. *J Nerv Ment Dis*. 2010;198:201–205.
24. Krabbendam L, Myin-Germeys I, Bak M, van Os J. Explaining transitions over the hypothesized psychosis continuum. *Aust N Z J Psychiatry*. 2005;39:180–186.
25. Jenner JA, Rutten S, Beuckens J, Boonstra N, Sytema S. Positive and useful auditory vocal hallucinations: prevalence, characteristics, attributions, and implications for treatment. *Acta Psychiatr Scand*. 2008;118:238–245.
26. Brookwell ML, Bentall RP, Varese F. Externalizing biases and hallucinations in source-monitoring, self-monitoring and signal detection studies: a meta-analytic review. *Psychol Med*. 2013; 43:2465–2475.
27. Badcock JC, Hugdahl K. Cognitive mechanisms of auditory verbal hallucinations in psychotic and non-psychotic groups. *Neurosci Biobehav Rev*. 2012;36:431–438.
28. Waters F, Allen P, Aleman A, et al. Auditory hallucinations in schizophrenia and nonschizophrenia populations: a review and integrated model of cognitive mechanisms. *Schizophr Bull*. 2012;38:683–693.
29. Badcock JC, Chhabra S, Maybery MT, Paulik G. Context binding and hallucination predisposition. *Pers Individ Dif*. 2008;45:822–827.
30. Chhabra S, Badcock JC, Maybery MT, Leung D. Context binding and hallucination predisposition: Evidence of intact intentional and automatic integration of external features. *Pers Individ Dif*. 2011;50:834–839.
31. McKague M, McAnally KI, Skovron M, Bendall S, Jackson HJ. Source monitoring and proneness to auditory-verbal hallucinations: a signal detection analysis. *Cognit Neuropsychiatry*. 2012;17:544–562.
32. Badcock JC, Chhabra S. Voices to reckon with: perceptions of voice identity in clinical and non-clinical voice hearers. *Front Hum Neurosci*. 2013;7:1–9.
33. Chhabra S, Badcock JC, Maybery MT, Leung D. Voice identity discrimination and hallucination-proneness in healthy young adults: a further challenge to the continuum model of psychosis? *Cognit Neuropsychiatry*. 2013:1–14. doi:10.1080/13546805.2013.865512.
34. Chhabra S, Badcock JC, Maybery MT, Leung D. Voice identity discrimination in schizophrenia. *Neuropsychologia*. 2012;50:2730–2735.
35. Kompus K, Falkenberg LE, Bless JJ, et al. The role of the primary auditory cortex in the neural mechanism of auditory verbal hallucinations. *Front Hum Neurosci*. 2013;7:1–13.

36. Daalman K, van Zandvoort M, Bootsman F, Boks M, Kahn R, Sommer I. Auditory verbal hallucinations and cognitive functioning in healthy individuals. *Schizophr Res*. 2011;132:203–207.
37. Modinos G, Costafreda SG, van Tol MJ, McGuire PK, Aleman A, Allen P. Neuroanatomy of auditory verbal hallucinations in schizophrenia: a quantitative meta-analysis of voxel-based morphometry studies. *Cortex*. 2013;49:1046–1055.
38. Palaniyappan L, Balain V, Radua J, Liddle PF. Structural correlates of auditory hallucinations in schizophrenia: a meta-analysis. *Schizophr Res*. 2012;137:169–173.
39. de Weijer AD, Neggers SF, Diederer KM, et al. Aberrations in the arcuate fasciculus are associated with auditory verbal hallucinations in psychotic and in non-psychotic individuals. *Hum Brain Mapp*. 2013;34:626–634.
40. Allen P, Modinos G, Hubl D, et al. Neuroimaging auditory hallucinations in schizophrenia: from neuroanatomy to neurochemistry and beyond. *Schizophr Bull*. 2012;38:695–703.
41. Howes OD, Shotbolt P, Bloomfield M, et al. Dopaminergic function in the psychosis spectrum: an [18F]-DOPA imaging study in healthy individuals with auditory hallucinations. *Schizophr Bull*. 2013;39:807–814.
42. Diederer KM, De Weijer AD, Daalman K, et al. Decreased language lateralization is characteristic of psychosis, not auditory hallucinations. *Brain*. 2010;133:3734–3744.
43. Sommer I, Ramsey N, Kahn R, Aleman A, Bouma A. Handedness, language lateralisation and anatomical asymmetry in schizophrenia: meta-analysis. *Br J Psychiatry*. 2001;178:344–351.
44. Ocklenburg S, Westerhausen R, Hirnstein M, Hugdahl K. Auditory hallucinations and reduced language lateralization in schizophrenia: a meta-analysis of dichotic listening studies. *J Int Neuropsychol Soc*. 2013;19:410–418.
45. Longden E, Madill A, Waterman MG. Dissociation, trauma, and the role of lived experience: toward a new conceptualization of voice hearing. *Psychol Bull*. 2012;138:28–76.
46. Read J, van Os J, Morrison AP, Ross CA. Childhood trauma, psychosis and schizophrenia: a literature review with theoretical and clinical implications. *Acta Psychiatr Scand*. 2005;112:330–350.
47. Sheffield JM, Williams LE, Blackford JU, Heckers S. Childhood sexual abuse increases risk of auditory hallucinations in psychotic disorders. *Compr Psychiatry*. 2013;54:1098–1104.
48. Daalman K, Diederer KM, Derks EM, van Lutterveld R, Kahn RS, Sommer IE. Childhood trauma and auditory verbal hallucinations. *Psychol Med*. 2012;42:2475–2484.
49. Bentall RP, Wickham S, Shevlin M, Varese F. Do specific early-life adversities lead to specific symptoms of psychosis? A study from the 2007 the Adult Psychiatric Morbidity Survey. *Schizophr Bull*. 2012;38:734–740.
50. Goldstone E, Farhall J, Ong B. Modelling the emergence of hallucinations: early acquired vulnerabilities, proximal life stressors and maladaptive psychological processes. *Soc Psychiatry Psychiatr Epidemiol*. 2012;47:1367–1380.
51. McCarthy-Jones S. *Hearing Voices: The Histories, Causes and Meanings of Auditory Verbal Hallucinations*. Cambridge, MA: Cambridge University Press; 2012.
52. Romme M, Escher S, Dillon J, Corstens D, Morris M. Living with voices: 50 stories of recovery. *Tijdschr Psychiatr*. 2009;53:439–440.
53. Chadwick P, Birchwood M. The omnipotence of voices. A cognitive approach to auditory hallucinations. *Br J Psychiatry*. 1994;164:190–201.
54. Morrison AP. A cognitive analysis of the maintenance of auditory hallucinations: are voices to schizophrenia what bodily sensations are to panic? *Behav Cogn Psychother*. 1998;26:289–302.
55. Birchwood M, Gilbert P, Gilbert J, et al. Interpersonal and role-related schema influence the relationship with the dominant 'voice' in schizophrenia: a comparison of three models. *Psychol Med*. 2004;34:1571–1580.
56. Paulik G. The role of social schema in the experience of auditory hallucinations: a systematic review and a proposal for the inclusion of social schema in a cognitive behavioural model of voice hearing. *Clin Psychol Psychother*. 2012;19:459–472.
57. Peters ER, Williams SL, Cooke MA, Kuipers E. It's not what you hear, it's the way you think about it: appraisals as determinants of affect and behaviour in voice hearers. *Psychol Med*. 2012;42:1507–1514.
58. Birchwood M, Meaden A, Trower P, Gilbert P, Plaistow J. The power and omnipotence of voices: subordination and entrapment by voices and significant others. *Psychol Med*. 2000;30:337–344.
59. Hayward M, Berry K, Ashton A. Applying interpersonal theories to the understanding of and therapy for auditory hallucinations: a review of the literature and directions for further research. *Clin Psychol Rev*. 2011;31:1313–1323.
60. Vaughan S, Fowler D. The distress experienced by voice hearers is associated with the perceived relationship between the voice hearer and the voice. *Br J Clin Psychol*. 2004;43:143–153.
61. Bak M, Myin-Germeys I, Hanssen M, et al. When does experience of psychosis result in a need for care? A prospective general population study. *Schizophr Bull*. 2003;29:349–358.
62. Gaynor K, Ward T, Garety P, Peters E. The role of safety-seeking behaviours in maintaining threat appraisals in psychosis. *Behav Res Ther*. 2013;51:75–81.
63. Ward TA, Gaynor KJ, Hunter MD, et al. Appraisals and responses to experimental symptom analogues in clinical and nonclinical individuals with psychotic experiences. *Schizophr Bull*. In press.
64. Brett CM, Peters EP, Johns LC, Tabraham P, Valmaggia LR, McGuire P. Appraisals of Anomalous Experiences Interview (AANEX): a multidimensional measure of psychological responses to anomalies associated with psychosis. *Br J Psychiatry Suppl*. 2007;51:s23–s30.
65. Lovatt A, Mason O, Brett C, Peters E. Psychotic-like experiences, appraisals, and trauma. *J Nerv Ment Dis*. 2010;198:813–819.
66. Lawrence C, Jones J, Cooper M. Hearing voices in a non-psychiatric population. *Behav Cogn Psychother*. 2010;38:363–373.
67. Sorrell E, Hayward M, Meddings S. Interpersonal processes and hearing voices: a study of the association between relating to voices and distress in clinical and non-clinical hearers. *Behav Cogn Psychother*. 2010;38:127–140.
68. Daalman K, Sommer IE, Derks EM, Peters ER. Cognitive biases and auditory verbal hallucinations in healthy and clinical individuals. *Psychol Med*. 2013;43:2339–2347.
69. Bartels-Velthuis AA, van de Willige G, Jenner JA, Wiersma D, van Os J. Auditory hallucinations in childhood: associations with adversity and delusional ideation. *Psychol Med*. 2012;42:583–593.
70. Escher S, Romme M, Buiks A, Delespaul P, van Os J. Formation of delusional ideation in adolescents hearing voices: a prospective study. *Am J Med Genet*. 2002;114:913–920.
71. Bartels-Velthuis AA, van de Willige G, Jenner JA, van Os J, Wiersma D. Course of auditory vocal hallucinations

- in childhood: 5-year follow-up study. *Br J Psychiatry*. 2011;199:296–302.
72. Dhossche D, Ferdinand R, Van der Ende J, Hofstra MB, Verhulst F. Diagnostic outcome of self-reported hallucinations in a community sample of adolescents. *Psychol Med*. 2002;32:619–627.
 73. Fisher HL, Caspi A, Poulton R, et al. Specificity of childhood psychotic symptoms for predicting schizophrenia by 38 years of age: a birth cohort study. *Psychol Med*. 2013;43:2077–2086.
 74. Welham J, Scott J, Williams G, et al. Emotional and behavioural antecedents of young adults who screen positive for non-affective psychosis: a 21-year birth cohort study. *Psychol Med*. 2009;39:625–634.
 75. Garety PA, Kuipers E, Fowler D, Freeman D, Bebbington PE. A cognitive model of the positive symptoms of psychosis. *Psychol Med*. 2001;31:189–195.
 76. de Leede-Smith S, Barkus E. A comprehensive review of auditory verbal hallucinations: lifetime prevalence, correlates and mechanisms in healthy and clinical individuals. *Front Hum Neurosci*. 2013;7:1–25.
 77. Jolley S, Garety P. Cognitive-behavioural interventions. In: Gaebel W, ed. *Schizophrenia: Current Science and Clinical Practice*. Oxford, UK: Wiley-Blackwell; 2011:185–215.
 78. Hanssen M, Bak M, Bijl R, Vollebergh W, van Os J. The incidence and outcome of subclinical psychotic experiences in the general population. *Br J Clin Psychol*. 2005;44:181–191.
 79. Krabbendam L, Myin-Germeys I, Hanssen M, et al. Development of depressed mood predicts onset of psychotic disorder in individuals who report hallucinatory experiences. *Br J Clin Psychol*. 2005;44:113–125.
 80. Hartley S, Haddock G, Barrowclough C. Anxiety and depression and their links with delusions and hallucinations in people with a dual diagnosis of psychosis and substance misuse: a study using data from a randomised controlled trial. *Behav Res Ther*. 2012;50:65–71.
 81. Smith B, Fowler DG, Freeman D, et al. Emotion and psychosis: links between depression, self-esteem, negative schematic beliefs and delusions and hallucinations. *Schizophr Res*. 2006;86:181–188.
 82. Garety PA, Bebbington P, Fowler D, Freeman D, Kuipers E. Implications for neurobiological research of cognitive models of psychosis: a theoretical paper. *Psychol Med*. 2007;37:1377–1391.
 83. Farhall J. Understanding and shaping adaptive coping with voices. In: Larøi F, Aleman A, eds. *Hallucinations: A Guide to Treatment and Management*. Oxford, UK: Oxford University Press; 2010:205–231.
 84. Birchwood M, Chadwick P. The omnipotence of voices: testing the validity of a cognitive model. *Psychol Med*. 1997;27:1345–1353.
 85. Varese F, Barkus E, Bental RP. Dissociation mediates the relationship between childhood trauma and hallucination-proneness. *Psychol Med*. 2012;42:1025–1036.
 86. Larøi F, Sommer IE, Blom JD, et al. The characteristic features of auditory verbal hallucinations in clinical and nonclinical groups: state-of-the-art overview and future directions. *Schizophr Bull*. 2012;38:724–733.
 87. Luhrmann TM. Hallucinations and sensory overrides. *Annu Rev Anthropol*. 2011;40:71–85.
 88. Sommer IE, Daalman K, Rietkerk T, et al. Healthy individuals with auditory verbal hallucinations; who are they? Psychiatric assessments of a selected sample of 103 subjects. *Schizophr Bull*. 2010;36:633–641.
 89. Murphy J, Shevlin M, Houston J, Adamson G. A population based analysis of subclinical psychosis and help-seeking behavior. *Schizophr Bull*. 2012;38:360–367.
 90. Thomas N, Hayward M, Peters ER, et al. Psychological therapies for auditory hallucinations (voices): current status and key directions for future research. *Schizophr Bull*. 2014;40:S202–S212.
 91. Morrison AP, Barratt S. What are the components of CBT for psychosis? A Delphi study. *Schizophr Bull*. 2010;36:136–142.
 92. Morris E, Johns LC, Oliver J. *Acceptance and Commitment Therapy and Mindfulness for Psychosis*. Chichester, UK: Wiley-Blackwell; 2013.
 93. Mayhew SL, Gilbert P. Compassionate mind training with people who hear malevolent voices: a case series report. *Clin Psychol Psychother*. 2008;15:113–138.
 94. Longden E, Corstens D, Escher S, Romme M. Voice hearing in a biographical context: a model for formulating the relationship between voices and life history. *Psychosis*. 2011;4:224–234.
 95. Waller H, Freeman D, Jolley S, Dunn G, Garety P. Targeting reasoning biases in delusions: a pilot study of the Maudsley Review Training Programme for individuals with persistent, high conviction delusions. *J Behav Ther Exp Psychiatry*. 2011;42:414–421.
 96. Moritz S, Woodward TS. Metacognitive training in schizophrenia: from basic research to knowledge translation and intervention. *Curr Opin Psychiatry*. 2007;20:619–625.
 97. Wykes T, Reeder C. *Cognitive Remediation Therapy for Schizophrenia: Theory and Practice*. London, UK: Brunner Routledge; 2005.
 98. Luhrmann TM, Larøi F, Bell V, et al. Culture and hallucinations: overview and future directions. *Schizophr Bull*. 2014;40:S213–S220.
 99. Scott J, Varghese D, McGrath J. As the twig is bent, the tree inclines: adult mental health consequences of childhood adversity. *Arch Gen Psychiatry*. 2010;67:111–112.
 100. Waters F, Woods A, Fernyhough C. Report on the 2nd International Consortium on hallucination research: evolving directions and top-10 “hot spots” in hallucination research. *Schizophr Bull*. 2014;40:24–27.