

Beyond Trauma: A Multiple Pathways Approach to Auditory Hallucinations in Clinical and Nonclinical Populations

Tanya Marie Luhrmann^{*.1}, Ben Alderson-Day², Vaughan Bell³, Josef J. Bless⁴, Philip Corlett⁵, Kenneth Hugdahl^{4,6}, Nev Jones⁷, Frank Larøi^{4,8,9}, Peter Moseley^{2,10}, Ramachandran Padmavati¹¹, Emmanuelle Peters^{12,13}, Albert R. Powers⁴, and Flavie Waters^{14,15}

¹Department of Anthropology, Stanford University, Stanford, CA; ²Department of Psychology, Durham University, Durham, UK; ³Division of Psychiatry, University College London, London, UK; ⁴Department of Biological and Medical Psychology, University of Bergen, Bergen, Norway; ⁵Department of Psychiatry, Connecticut Mental Health Center, Yale University, New Haven, CT; ⁶Department of Psychiatry, Haukeland University Hospital, Bergen, Norway; ⁷Department of Mental Health Law and Policy, Louis de la Parte Florida Mental Health Institute, University of South Florida, Tampa, FL; ⁸Norwegian Center of Excellence for Mental Disorders Research, University of Oslo, Oslo, Norway; ⁹Psychology and Neuroscience of Cognition Research Unit, University of Liège, Liège, Belgium; ¹⁰School of Psychology, University of Central Lancashire, Preston, UK; ¹¹Schizophrenia Research Foundation, Chennai, Tamil Nadu, India; ¹²King's College London, Institute of Psychiatry, Psychology and Neuroscience, Department of Psychology, London, UK; ¹³Psychological Interventions Clinic for Outpatients with Psychosis, South London and Maudsley NHS Foundation Trust (SLaM), London, England, UK; ¹⁴School of Psychological Science, University of Western Australia, Perth, Western Australia, Australia; ¹⁵Clinical Research Centre, Graylands Health Campus, North Metropolitan Health Service, Mental Health, Nedlands, Western Australia, Australia

*To whom correspondence should be addressed; Department of Anthropology, Stanford University, Building 50, Stanford, CA 94305, USA; tel.: 650-723-3421, fax: 650-725-0605, e-mail: luhrmann@stanford.edu

That trauma can play a significant role in the onset and maintenance of voice-hearing is one of the most striking and important developments in the recent study of psychosis. Yet the finding that trauma increases the risk for hallucination and for psychosis is quite different from the claim that trauma is necessary for either to occur. Trauma is often but not always associated with voice-hearing in populations with psychosis; voice-hearing is sometimes associated with willful training and cultivation in nonclinical populations. This article uses ethnographic data among other data to explore the possibility of multiple pathways to voice-hearing for clinical and nonclinical individuals whose voices are not due to known etiological factors such as drugs, sensory deprivation, epilepsy, and so forth. We suggest that trauma sometimes plays a major role in hallucinations, sometimes a minor role, and sometimes no role at all. Our work also finds seemingly distinct phenomenological patterns for voice-hearing, which may reflect the different salience of trauma for those who hear voices.

Key words: hallucination/trauma/psychosis/healthy voice-hearers/spiritual practices

Introduction

The observation that trauma can play a significant role in the onset and maintenance of voice-hearing is one

of the most striking and important developments in the recent study of psychosis. Theoretical models propose that trauma impacts hallucinations on 3 levels.¹ First, trauma may serve as a biological, biopsychosocial, and/or psychological stressor or trigger: those with psychosis commonly report physical assault, sexual trauma, and other forms of victimization before onset.² Second, trauma may influence voice content: what voices say may reflect elements of the original event.³ Third, trauma-related dissociation may create or maintain hallucinations. Some argue early trauma creates auditory hallucinations by leading to the dissociation of self components^{4,5}; others that dissociation mediates the effect of childhood trauma on the hallucination process.⁶ Although some studies have questioned these findings,^{7,8} there is enough evidence to conclude that trauma is a significant risk factor for psychosis and for voice-hearing in particular.^{9,10}

Yet the finding that trauma increases the risk for hallucination and for psychosis is quite different from the claim that trauma is necessary for either to occur. Moreover, trauma is operationalized in a wide range of ways. In the *DSM-IV* diagnosis of posttraumatic stress disorder (PTSD), trauma is conceptualized as the event to which there is a specific psychological reaction (eg, fear, helplessness, and horror); in the *DSM-5*

diagnosis, trauma is limited to a number of specific event types (eg, exposure to actual or threatened death, serious injury, or sexual violence). Studies examining associations between childhood trauma and hallucinations often use a variety of scales that rely on summing-up events experienced in childhood, answered contemporaneously or in retrospect, that may include relatively common stressful experiences such as feeling disliked by a parent (Child Abuse and Trauma Scale; CATS),¹¹ having hurtful or insulting things said to you (Childhood Trauma Questionnaire—Short Form; CTQ-SF),¹² and parental divorce (Adverse Childhood Experience Questionnaire; ACE-Q)¹³ in addition to more extreme classically traumatic experiences of physical or sexual assault. In the trauma and hallucinations literature, trauma is sometimes identified through a rating of the severity of the psychological consequences of trauma regardless of the number or frequency of traumatic experiences or the total number of adverse experiences recorded.¹⁴ It is worth noting that prospective informant-reports and retrospective self-reports of childhood trauma show relatively low levels of agreement¹⁵ (Kappas below 0.31¹⁶).

Furthermore, there is a common if often implicit assumption about the causal pathway between trauma and hallucinations: that the effects underlying the association, whether mediated via cognitive or neurobiological routes, are a result of emotional stress.¹⁷ Yet many traumatic experiences also commonly co-occur with other events that may constitute a parallel risk factor for the development of hallucinations after trauma: for example, concussion and brain injury in intimate partner violence,¹⁸ malnutrition and neglect in childhood trauma,¹⁹ and drug-facilitated sexual abuse.²⁰ The research on social adversity and social disadvantage demonstrates that trauma and psychosis are deeply entangled with a myriad of social and environmental risk factors.^{21,22}

Finally, the experience of trauma is common. In the United States, researchers have found that about 90% of the general population reports at least one significant traumatic event²³; 70% do so globally.²⁴ What counts as trauma may vary in different social worlds,²⁵ but the rate of psychosis (<1%) is substantially lower than the rate of trauma.

The goal of the article is to explore the possibility of multiple pathways to voice-hearing for clinical and non-clinical individuals whose voices are not due to known etiological factors such as drugs, sensory deprivation, epilepsy, and so forth. We do so using multiple sources of data, including ethnography and qualitative observation. Our work also finds seemingly distinct phenomenological patterns for voice-hearing, which may reflect the different salience of trauma for those who hear voices. We suggest that while trauma may play a major role in some hallucinations, and a minor role in many, it may play no role in other hallucinations.

Trauma is Often But Not Always Associated With Voice-hearing in Populations With Psychosis

Trauma can of course never be ruled out, even when individuals do not report it. Still, even in large studies when participants have been asked systematically about trauma, not all who report voices report trauma. One review reports that between 34% and 53% of patients with severe mental illness report childhood sexual or physical abuse, and that 56% of patients admitted with first episode psychosis report childhood sexual abuse²⁶; another reports studies that found a 47% rate of childhood sexual abuse in a schizoaffective population and a 38% rate of sexual abuse in an inpatient psychosis population.²⁷ A retrospective population-based study of 17 337 people found a statistically significant relationship between hallucination and self-reported history of childhood adversity in many forms. Early trauma exposure effectively doubled the risk of hallucination (with odds ratios between 1.2 and 2.5 for different categories of trauma; only 2% of the sample reported hallucinations), but there were individuals who reported childhood abuse (64% reported at least one item on the ACE) but did not report hallucinations, and individuals who reported hallucinations but did not report childhood abuse.²⁸ Other studies have reported a significant minority of individuals with psychosis and voice-hearing who report neither any history of abuse²⁹ nor a clear trigger for the onset of their hallucinations.³⁰

Voice-hearing is Often Associated With Propensity and Practice in Nonclinical Populations

The role of trauma in hallucinations in the general (non-clinical) population is even less clear.³¹ In many culturally diverse settings, unusual sensory experiences are highly valued.³² We know that highly hypnotizable individuals are able to experience hallucinations in response to suggestions,³³ that those who score highly in absorption are more likely to report unusual sensory experiences,^{34,35} and that meditation is capable of eliciting vivid apparitions.³⁶

Those for whom hallucinations are culturally valuable—who expect or even yearn to see spirits or to hear God—often describe some kind of willful “training” or deliberate cultivation that appears to increase the chance of experiencing them. In ethnographic work with charismatic Christians, Luhrmann^{37,38} found that many nonclinical Christian interlocutors (roughly a third) reported at least one experience of hearing God speak audibly. For the most part, these events were rare (people typically reported no more than a handful of such events); brief (rarely more than 4–6 words); and startling, but not distressing (God said things such as, “Slow down”; “I love you”; “I will always be with you”). Compared with people with psychosis, their voices were less commanding, although they shared qualities such as being vivid, substantial, and located in external space. The research found that these events were

associated with mental imagery practice, or unscripted prayer³⁹ and with the trait of “absorption,” as measured by the Tellegen Absorption Scale, which seems to capture whether one can be caught up in sensory experience.⁴⁰ Although some of these individuals did spontaneously speak about trauma, most did not, despite lengthy interviews.

The *Hearing the Voice* project at Durham University⁴¹ and a related project at Yale⁴² work with nonclinical voice-hearers who describes themselves as psychics and spiritualists. As with the nonclinical Christians, these participants often emphasize skill and training in their experience of voices. Those in the Durham group typically spoke of first hearing an unpleasant voice, deciding to treat it as a gift, and then methodically working with the voice until the voice became more positive and controllable. About half, however, always experienced their voices as positive, and all reported a higher degree of control over the voices than usually found in clinical samples. Many work at churches on a regular basis, passing on information from voices usually understood to belong to the dead. Few of these individuals reported trauma in the course of long interviews, despite being asked about their childhoods and about any apparent triggers for their voice-hearing. Those in the Yale group also found their experiences much less distressing than a matched group of patient participants. They reported a degree of volitional control over their experiences, mastered over time, often following initial distress at the initial voice experience, and learned under the tutelage of another voice-hearer. Powers et al⁴² did not explicitly ask about trauma. Yet none of the psychic participants spontaneously offered trauma as an explanation for or harbinger of their experiences, and none met criteria for PTSD.

Again, trauma cannot be ruled out. There is evidence of elevated trauma rates in the Utrecht sample of nonclinical voice-hearers (although it is also true that in that sample, 46% of nonclinical voice-hearers did not report trauma).⁴³ But the role of willful training and cultivation suggests that there may be pathways to hallucination that do not necessarily involve trauma. The presence of positive hallucinations should also alert us to the possibility of other pathways. Rosen et al⁴⁴ have found that negative voice-hearing is a full mediator of a relationship between childhood adversity and distress that may arise from hearing voices. Nonclinical voice-hearers report more positive and more controllable experiences than do their clinical counterparts.⁴⁵⁻⁴⁷ Again, though trauma can never be ruled out, this should incline us to caution in inferring an inherent link between trauma and hallucination.

Trauma is Associated With Different Phenomenologies in Voice-hearing

The most extensive phenomenological studies of voice-hearing to date⁴⁸⁻⁵⁰ have identified subtypes of

voice-hearing among psychotic patients who hallucinate within the schizophreniform diagnoses and have not focused on a trauma etiology. Yet we have some evidence that hallucinations may differ for voice-hearers who do not report trauma. Dorahy et al⁵¹ compared persons with schizophrenia without childhood maltreatment, persons with schizophrenia with childhood maltreatment, and persons with dissociative identity disorder. In this study, childhood maltreatment was assessed by the CTQ,¹² a self-report inventory that asks about emotional, physical, and sexual abuse and physical and emotional neglect. For those with past trauma, their voices were on average louder than normal speech, related to real humans who were influential in the individual’s life, related to memories, and experienced with hallucinations in other sensory modalities. Bless et al⁵² found that 43% of individuals in the general population who reported hearing voices could not identify any specific event (a “trigger,” typically negative) when they heard their voice for the first time. These non-trigger individuals reported less severe and less frequent auditory hallucinations with more neutral emotional content than those reported adverse life events when they heard a voice for the first time. Those who identified a clear trigger more often reported hallucinations in other sensory modalities, poorer general mental health, and were more likely to have been in contact with a health professional about their voices.

Novel Data on Different Phenomenological Presentations of Voice-hearing

Here, we present novel data from an ethnographic study of healthy voice-hearers, which finds that there are different phenomenological presentations of voice-hearing, and different responses to a novel probe, and that these differences are associated with distinctive presentations of trauma.

In Cape Coast, Ghana, “okomfo” are individuals who are understood to talk with the local gods. There is a widely shared cultural model that those who become okomfo are called audibly by the gods; that during this time other people may think that the individual is mad (they also say that people are driven mad if they refuse to answer the call); that during the long and difficult training process, the individual is taught how to hear and see the gods properly and to identify and manage witches and demons. In training, people also repeatedly practice possession, in which the gods take over their bodies and speak through them. After training, candidates are understood to have a positive relationship with a range of gods and spirits with whom they speak on a regular basis.

Despite this shared cultural model, individuals presented different accounts of voice-hearing. In the summer of 2017, Luhrmann³⁷ interviewed 11 okomfo and 7 charismatic Christians who (unusually) said that God or the gods spoke to them audibly at least once a week. (There

were other okomfo who did not hear voices at least once a week.) All were individuals in an ongoing research project on spiritual experience in the area, and most had already been interviewed at length by the local research team, including John Dulin, Vivian Dzokoto, and Eunice Otoo. Data were collected as part of the Mind and Spirit project in a Templeton-funded, Stanford-based comparative, and interdisciplinary project under the direction of TM Luhrmann (PI). Luhrmann conducted a more detailed phenomenological interview. In addition, she played a 45 s audio-track originally created by Pat Deegan to represent the experience of hearing voices. The segment included whispering voices and murmuring voices. It featured a “good” female voice and a “bad” male voice, a man who made derogatory comments such as “you smell” and who gave sharp commands such as “don’t do that.” Luhrmann and her team remade the track with native Fante speakers. At the end of the interview, individuals were asked whether their experience with the gods was similar to or different from this track. All were asked directly about trauma.

Table 1 displays 4 exemplars of patterns of voice-hearing among these nonclinical individuals. Each pattern was represented by several individuals. The ethnographer identified these patterns through the direct responses in interviews and in discussion with other research team members using the iterative methods standard in the discipline.

Pattern I we called “psychosis-like.” The individuals who best represented this pattern clearly assented to hearing audible positive voices (God or the gods) and negative voices (demons) and an array of auditory events: whispering, murmuring, voices, conversing, and commanding. They clearly articulated a period when others thought they were psychiatrically impaired. Individuals gave reasons for this difficult period—losing a job, for instance—but they denied childhood trauma. They were clear that what they heard on the track resembled what they heard gods and demons say. Five of the okomfo exemplified this pattern; another likely did.

Pattern II we called “trauma-related dissociation.” In this pattern, individuals clearly assented to audible positive (God or the gods) voices and to negative voices (demons), but reported a narrower range of auditory events. They less clearly articulated a period when others thought they were psychiatrically impaired. They clearly presented an experience of violence or trauma. These individuals were more ambivalent about whether the track represented their experience of hearing voices. Two of the okomfo and one of the Christians exemplified this pattern.

Pattern III we called “simple trance.” In this pattern, individual voice-hearing is more attenuated, with more emphasis on seeing. The individual did not describe a period when others thought s/he was psychiatrically impaired and the individual denied trauma. In this pattern, the individual

Table 1.

Pattern I: “psychosis-like” presentation

Example: Mary was in her early forties when her business failed. She ran into the bush to follow the call of the gods and was lost for 3 days. Her family took her to a psychiatric hospital but—she reported—the hospital said that nothing was wrong. Now in her late seventies, she repeatedly described the gods’ voices as audible. She had verbal conversations with the gods as well, and other beings would speak with her: demons, cats, and even disembodied neighbors. They would chatter around her, whispering, commenting on what she was doing, commanding her, talking among themselves. She clearly recognized the events on the audio-track and said that what she heard sounded like that.

Pattern II: “trauma-related dissociation” presentation

Example: Ekuwa became an okomfo in her late twenties. She had a violent relationship with her husband, and after a particularly bad fight, she packed her bags and left. She was possessed by a god a month later. She had always had a tumultuous life. She had 4 children by 4 men, and she spoke a lot about fighting. She said that her mother beat her when she was young. When she listened to the track, she said: “this is not the way the gods talk. This is how witches talk.” She heard witches in her dreams, and sometimes they would wake her up and seemingly assault her. Yet while her auditory experience was real, it seemed less prominent and less varied. She seemed to hear whispering only in her dreams.

Pattern III: “simple trance” presentation

Example: Nyakpoo became an okomfo because his mother had been one and he liked what she did. When he was young, he would help out in the shrine and follow her around. His other siblings would not. He was in his teens when he began his training. He never heard the gods speak before he was initiated. He said that the gods never sounded like the audio-track, nor did the witches. He did see and hear the gods, but their degree of audition was never clear to any of his interviewers. He said that no one had ever thought of taking him to a hospital. He reported no trauma. His account was of a vividly imagined world which he clearly enjoyed immensely.

Pattern IV: “incidental” hallucinations

Example: Laura was a Catholic. She prayed for 30–60 min each day, reading her Bible and talking informally with God in her mind, but she did not speak in tongues and she did not describe entering prayer in a vividly trance-like way. She would often wake up and pray in the middle of the night. During this nighttime period she would hear God speak out loud to her, maybe twice a week. She said she had never heard anything like the audio-track.

rejected the track, or gave a description of voice-hearing at odds with the track. Their account of voice-hearing were more novelistic, more rich with imaginative detail, than the accounts of others. There were 3 okomfo like this.

Pattern IV we identified as “incidental hallucination.” In this pattern, individuals appeared to hear voices no more than once a week. They gave no account of other people thinking the individual is psychiatrically impaired. They denied trauma. There was little evidence of elaborated trance. They described no variety of auditory events. In this pattern, people said that the track was unlike their experience. Six Christians gave accounts that fit this pattern.

Within the group as a whole, as the voices were less demanding and overall more positive, the individuals described less trauma. Those who denied trauma unequivocally also denied that the Deegan track resembled the voices they heard from the gods. Those who identified either childhood trauma or a precipitating event tended to say that the voices of the god sounded like the voices on the track. Only those who described past experience with clinical features resembling a psychotic break said that they experienced the full range of auditory events presented on the track. Our authorship group thought that these different categories could be distinguished as well in other data sets—specifically, in the Durham and Yale studies, and also in the Unusual Experiences Enquiry (UNIQUE)⁵³ studies.

Overall, these observations suggest that when people have experienced trauma and the trauma is salient for them, voice-hearing may be more harsh and more auditorily diverse. They also suggest that there may be pathways to hallucination in which trauma plays little role.

Underlying Mechanisms

There are at least 2 types of related mechanisms, psychological/cognitive and neurobiological, which could explain the existence of auditory hallucinations not due to trauma, and the greater distress and auditory diversity of hallucinations associated with trauma salience.

With respect to psychological mechanisms, research on dissociation has distinguished 2 forms, “detachment” and “compartmentalization,” unlikely to be explained by similar cognitive mechanisms, with the former being more likely to be associated with trauma.^{54–57} Detachment-type dissociation involves feelings of depersonalization and/or derealization that is associated clinically with peritraumatic dissociation; as aforementioned, it may be a mediating factor between trauma and hallucinated voices. Compartmentalization-type dissociation is considered to be the basis of the alterations in subjective experience associated with hypnosis.⁵⁸ The basic mechanism in compartmentalization-type dissociation seems to be an individual’s susceptibility to suggestion, and inward attention focus; those at the higher end of hypnotic suggestibility can experience marked hallucinations in response to

suggestions. This susceptibility seems to be a stable life-long trait, normally distributed in the population, and distinct from the effects of persuasion or social conformity.⁵⁹ It seems possible that the voice-hearing of pattern II individuals is mediated by detachment-style dissociation and that of pattern III individuals by the voluntary use of compartmentalization-style dissociation.

This latter mechanisms is consistent with the proposal that, under specific conditions of expectancy or attention, mental processes can influence sensory perception. Theoretical models developed from data on clinical population groups suggest that hallucinations arise from alterations in the balance between bottom-up perceptual and top-down cognitive processing deficits,^{60–62} perhaps influenced by deficits in inhibitory control and cognitive monitoring mechanisms.^{63,64} In the absence of cognitive or neurological deficit (as in the case of spiritual practice), top-down effects may play a central role. There is indeed evidence that top-down influences can produce hallucinations in the absence of any external signals,⁶⁵ and that focused attention has the potential to blur the line between beliefs and experiences.⁶⁶ In support, studies using degraded speech tasks suggest that non-help-seeking voice hearers utilise such top-down influence during speech perception.⁴¹ Indeed, formal computational modeling of participant behavior during perceptual inference (within the Bayesian predictive coding framework⁶⁶) reveals that both clinical and nonclinical voice-hearers are biased toward top-down prior beliefs, and have difficulties updating those priors in light of new evidence⁶⁷

These mechanisms are also consistent with the observation that learning—or training—may alter voice-hearing experience, regardless of pathway. Voice-hearing embedded in an agreeable social world may become more manageable;⁴² distressing voice-hearing may improve in response to specific coaching;⁶⁸ and voice-hearing in religious contexts, as discussed, may become more culturally appropriate over time.

With respect to the neurobiological mechanisms, we suggest that the classic dopaminergic hypothesis for schizophrenia and psychosis-like experience may not apply for the explanation of nonpsychosis hallucinations. Although presynaptic striatum dopaminergic hyperactivation is likely present in psychosis across diagnostic categories,^{69–72} and may be a factor behind trauma-related hallucinations,⁷³ it may not be present in nonclinical voice-hearers in general.⁷⁴ Therefore, it is possible that the trauma-related patterns (I and II) may differ from the non-trauma-related patterns (III and IV) in the degree to which they are dopaminergically mediated.⁷⁵ The incidental murmuring and possibly other blurry speech experience most evident in patterns I and II may arise from a spontaneously hyperactivated cortex, whereas the clear voices in addition may represent intrusive memories that are not adequately suppressed by frontal regions of the brain.⁷⁶ By contrast, some

more incidental voice-hearing may be sleep related⁷⁷ or perhaps also due to the deliberate spiritual practice with inner sensory imagery; hallucination events have been associated with practice in a range of traditions.^{39,78–80}

If dopamine is not involved in patterns III and IV, what other neurobiological process may be at play with the power to disrupt the balance between bottom-up and top-down processes and trauma-related memories? An excitation–inhibition (E/I) imbalance model at the neurochemistry level, following the suggestion by Jardri et al,⁶⁴ proposes that excitatory transmitters, such as glutamate, cause spontaneous hyperactivation of temporal lobe speech areas, which are not inhibited due to hypoactivation of frontal lobe areas caused by aberrant function of inhibitory transmitters, particularly gamma aminobutyric acid (GABA). An E/I imbalance model is indirectly supported by studies showing elevated glutamate levels in trait hallucinators^{81,82} as well as studies in treatment-resistant patients having schizophrenia.⁸³ It is possible that while nontrauma hallucinating individuals show abnormal glutamate levels, which could explain the spontaneous onset of a hallucinatory episode, individuals with a history of trauma in addition show abnormal GABA levels, which prevents them from cognitively inhibiting the voices once they occur, and which would let trauma-related memories flow freely.

An important factor in trauma-related psychosis is stress, and a corresponding dysregulation of the hypothalamus–pituitary–adrenal gland (HPA)-axis. Several studies have shown that HPA-axis hormonal dysregulation in trauma may be a risk factor for transition to psychosis.^{84–86} Although not a common issue in hallucinations research, it is possible that elevated cortisol levels may be a marker for trauma-related hallucinations, not seen in non-trauma individuals.

Conclusion

The discovery of the great importance of trauma for voice-hearing should lead us to consider the possibility of alternate pathways as well. Having established trauma so firmly as a risk factor for voice-hearing, we should now use more careful phenomenological methods to explore voice-hearing for which trauma may be less salient and to consider whether the patterns we describe here might be supported by further research. Ethnographic data—not often considered in psychiatric science—may be helpful here. We should also vigorously explore the ways in which training in many forms may render some of these experiences less caustic.

All research was approved by the authors' Institutional Review Boards.

Funding

T.M.L.'s work was supported by the John Templeton Foundation 55427; B.A.D. and P.M. are supported by

the Wellcome Trust WT 108720; K.H. is supported by the European Research Council (ERC Advanced Grant 693124), Research Council of Norway FRIMEDBIO 21550, and Health Authority of Western Norway (Helse-Vest 912045; V.B. is supported by a Wellcome Trust Seed Award in Science 200589/2/16/7.

Conflict of interest

The authors have declared that there are no conflicts of interest in relation to the subject of this study.

References

1. 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.
2. Mueser KT, Goodman LB, Trumbetta SL, et al. Trauma and posttraumatic stress disorder in severe mental illness. *J Consult Clin Psychol.* 1998;66:493–499.
3. 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.
4. Longden E, Madill A, Waterman MG. Dissociation, trauma, and the role of lived experience: toward a new conceptualization of voice hearing. *Psychol Bull.* 2012; 138:26–76
5. Moskowitz A, Corstens D. Auditory hallucinations: psychotic symptom or dissociative experience?" *J Psychol Trauma.* 2007;6(2/3):35–63.
6. Varese F, Barkus E, Bentall RP. Dissociation mediates the relationship between childhood trauma and hallucination-proneness. *Psychol Med.* 2012;42:1025–1036.
7. Bendall S, Jackson HJ, Hulbert CA, McGorry PD. Childhood trauma and psychotic disorders: a systematic, critical review of the evidence. *Schizophr Bull.* 2008;34:568–579.
8. Morgan C, Fisher H. Environment and schizophrenia: environmental factors in schizophrenia: childhood trauma—a critical review. *Schizophr Bull.* 2007;33:3–10.
9. 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.
10. Varese F, Smeets F, Drukker M, et al. Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective- and cross-sectional cohort studies. *Schizophr Bull.* 2012;38:661–671.
11. Sanders B, Becker-Lausen E. The measurement of psychological maltreatment: early data on the Child Abuse and Trauma Scale. *Child Abuse Negl.* 1995;19:315–323.
12. Bernstein DP, Fink L, Handelsman L, Lovejoy H, Wenzel K, Sapareto E, Gurriero J. Initial reliability and validity of a new measure of child abuse and neglect. *Am J Psychiatry.* 1994;151:1132–1136.
13. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. The Adverse Childhood Experiences (ACE) Study. *Am J Prev Med.* 1998;14:245–258.
14. Anketell C, Dorahy MJ, Shannon M, et al. An exploratory analysis of voice hearing in chronic PTSD: potential associated mechanisms. *J Trauma Dissociation.* 2010;11:93–107.

15. 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.
16. Newbury JB, Arseneault L, Moffitt TE, et al. Measuring childhood maltreatment to predict early-adult psychopathology: comparison of prospective informant-reports and retrospective self-reports. *J Psychiatr Res*. 2018;96:57–64.
17. Misiak B, Kreffl M, Bielawski T, Moustafa AA, Sasiadek MM, Frydecka D. Toward a unified theory of childhood trauma and psychosis: a comprehensive review of epidemiological, clinical, neuropsychological and biological findings. *Neurosci Biobehav Rev*. 2017;75:393–406.
18. Valera E, Kucyi A. Brain injury in women experiencing intimate partner-violence: neural mechanistic evidence of an “invisible” trauma. *Brain Imaging Behav*. 2017;11:1664–1677.
19. Stoltenborgh M, Bakermans-Kranenburg MJ, van Ijzendoorn MH. The neglect of child neglect: a meta-analytic review of the prevalence of neglect. *Soc Psychiatry Psychiatr Epidemiol*. 2013;48:345–355.
20. McCauley JL, Ruggiero KJ, Resnick HS, Kilpatrick DG. Incapacitated, forcible, and drug/alcohol-facilitated rape in relation to binge drinking, marijuana use, and illicit drug use: a national survey. *J Trauma Stress*. 2010;23:132–140.
21. Van Os J, Kenis G, Rutten B. The environment and schizophrenia. *Nature*. 2010; 468:203–212.
22. Veling W, Pot-Kolder R, Counotte J, van Os J, van der Gaag M. Environmental social stress, paranoia and psychosis liability: a virtual reality study. *Schizophr Bull*. 2016;42:1363–1371.
23. Breslau N, Peterson EL, Poisson LM, Schultz LR, Lucia VC. Estimating post-traumatic stress disorder in the community: lifetime perspective and the impact of typical traumatic events. *Psychol Med*. 2004;34:889–898.
24. Kessler RC, Aguilar-Gaxiola S, Alonso J, et al. Trauma and PTSD in the WHO World Mental Health Surveys. *Eur J Psychotraumatol*. 2017;8:1353383.
25. Kohrt BA, Hruschka DJ. Nepali concepts of psychological trauma: the role of idioms of distress, ethnopsychology and ethnophysiology in alleviating suffering and preventing stigma. *Cult Med Psychiatry*. 2010;34:322–352.
26. Morrison AP, Frame L, Larkin W. Relationships between trauma and psychosis: a review and integration. *Br J Clin Psychol*. 2003;42:331–353.
27. Bendall S, Jackson H, Hulbert C. Childhood trauma and psychosis: a review of the evidence and directions for psychological interventions. *Aust Psychol*. 2010;45(4):299–306.
28. Whitfield CL, Dube SR, Felitti VJ, Anda RF. Adverse childhood experiences and hallucinations. *Child Abuse Negl*. 2005;29:797–810.
29. Kingdon DG, Ashcroft K, Bhandari B, et al. Schizophrenia and borderline personality disorder: similarities and differences in the experience of auditory hallucinations, paranoia, and childhood trauma. *J Nerv Ment Dis*. 2010;198:399–403.
30. Honig A, Romme MA, Ensink BJ, Escher SD, Pennings MH, deVries MW. Auditory hallucinations: a comparison between patients and nonpatients. *J Nerv Ment Dis*. 1998;186:646–651.
31. Posey T, Losch M. Auditory hallucinations of hearing voices in 375 normal subjects. *Imagin, Cogn Personal*. 1983;3(2):99–113.
32. Larøi F, Luhrmann TM, Bell V, et al. Culture and hallucinations: overview and future directions. *Schizophr Bull*. 2015;40(suppl. 4):S213–S220.
33. Koivisto M, Kirjanen S, Revonsuo A, Kallio S. A preconscious neural mechanism of hypnotically altered colors: a double case study. *PLoS One*. 2013;8:e70900.
34. Glicksohn J, Barrett T. Absorption and hallucinatory experience. *Appl Cogn Psychol*. 2003;17:833–849.
35. Perona-Garcelán S, García-Montes JM, Ductor-Recuerda MJ, et al. Relationship of metacognition, absorption, and depersonalization in patients with auditory hallucinations. *Br J Clin Psychol*. 2012;51:100–118.
36. Lindahl JR, Kaplan CT, Winget EM, Britton WB. A phenomenology of meditation-induced light experiences: traditional Buddhist and neurobiological perspectives. *Front Psychol*. 2014;4:973.
37. Luhrmann TM. Diversity within the psychotic continuum. *Schizophr Bull*. 2017;43:27–31.
38. Luhrmann TM. *When God Talks Back: Understanding the American Evangelical Relationship with God*. New York: Knopf; 2012.
39. Luhrmann TM, Nusbaum H, Thisted R. “Lord, teach us to pray”: prayer practice affects cognitive processing. *J Cogn Cult*. 2013;13:159–177.
40. Luhrmann TM, Nusbaum H, Thisted R. The absorption hypothesis: learning to hear god in evangelical Christianity. *Am Anthropol*. 2010;112(1):66–78.
41. Alderson-Day B, Lima CF, Evans S, et al. Distinct processing of ambiguous speech in people with non-clinical auditory verbal hallucinations. *Brain*. 2017;140:2475–2489.
42. Powers AR 3rd, Kelley MS, Corlett PR. Varieties of voice-hearing: psychics and the psychosis continuum. *Schizophr Bull*. 2017;43:84–98.
43. Begeman M, Stotijn E, Schutte S, Heringa SM, Sommer IEC. Beyond childhood trauma: stressful events early and later in life in relation to psychotic experiences. Letter to the Editor. *Psychol Med*. 2017;47:2731–2736.
44. Rosen C, McCarthy-Jones S, Jones N, Chase K, Sharma R. Negative voice-content as a full mediator of a relation between childhood adversity and distress ensuing from hearing voices. [published online ahead of print March 23, 2018] *Schizophr Res*. doi: 10.1016/j.schres.2018.03.030.
45. Kråkvik B, Larøi F, Kalhovde AM, et al. Prevalence of auditory verbal hallucinations in a general population: a group comparison study. *Scand J Psychol*. 2015;56:508–515.
46. Woods A, Jones N, Alderson-Day B, Callard F, Fernyhough C. Experiences of hearing voices: analysis of a novel phenomenological survey. *Lancet Psychiatry*. 2015;2:323–331.
47. 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.
48. Nayani TH, David AS. The auditory hallucination: a phenomenological survey. *Psychol Med*. 1996;26:177–189.
49. Copolov DL, Mackinnon A, Trauer T. Correlates of the affective impact of auditory hallucinations in psychotic disorders. *Schizophr Bull*. 2004;30:163–171.
50. McCarthy-Jones S, Trauer T, Mackinnon A, Sims E, Thomas N, Copolov D. A new phenomenological survey of auditory hallucination: evidence for subtypes and implications for theory and practice. *Schizophr Bull*. 2014;40(1):225–235.
51. Dorahy MJ, Shannon C, Seagar L, et al. Auditory hallucinations in dissociative identity disorder and schizophrenia with and without a childhood trauma history: similarities and differences. *J Nerv Ment Dis*. 2009;197:892–898.
52. Bless JJ, Larøi F, Laloyaux J, et al. Do adverse life events at first onset of auditory verbal hallucinations influence subsequent voice characteristics? Results from an epidemiological study. *Psychiatry Res*. 2018;261:232–236.

53. Peters E, Ward T, Jackson M, et al. Clinical, socio-demographic and psychological characteristics in individuals with persistent psychotic experiences with and without a “need for care”. *World Psychiatry*. 2016;15:41–52.
54. Sierra M, Berrios GE. Towards a neuropsychiatry of conversion hysteria. *Cogn Neuropsychiatry*. 1999;4:267–287.
55. Brown RJ. Psychological mechanisms of medically unexplained symptoms: an integrative conceptual model. *Psychol Bull*. 2004;130:793–812.
56. Holmes EA, Brown RJ, Mansell W, et al. Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clin Psychol Rev*. 2005;25:1–23.
57. Humpston CS, Walsh E, Oakley DA, Mehta MA, Bell V, Deeley Q. The relationship between different types of dissociation and psychosis-like experiences in a non-clinical sample. *Conscious Cogn*. 2016;41:83–92.
58. Bell V, Oakley DA, Halligan PW, Deeley Q. Dissociation in hysteria and hypnosis: evidence from cognitive neuroscience. *J Neurol Neurosurg Psychiatry*. 2011;82:332–339.
59. Barnier AJ, McConkey KM. Defining and identifying the highly hypnotisable person. In: Heap M, Oakley DA, eds. *The Highly Hypnotizable Person: Theoretical, Experimental and Clinical Issues*. London, New York: Routledge, 2004:30–60.
60. Bentall RP, Slade PD. Reality testing and auditory hallucinations: a signal detection analysis. *Br J Clin Psychol*. 1985;24(Pt 3):159–169.
61. Collerton D, Perry E, McKeith I. Why people see things that are not there: a novel perception and attention deficit model for recurrent complex visual hallucinations. *Behav Brain Sci*. 2005;28:737–757.
62. Hugdahl K. “Hearing voices”: auditory hallucinations as failure of top-down control of bottom-up perceptual processes. *Scand J Psychol*. 2009;50:553–560.
63. Diederich NJ, Fénelon G, Stebbins G, Goetz CG. Hallucinations in Parkinson disease. *Nat Rev Neurol*. 2009;5:331–342.
64. Jardri R, Hugdahl K, Hughes M, et al. Are hallucinations due to an imbalance between excitatory and inhibitory influences on the brain? *Schizophr Bull*. 2016;42:1124–1134.
65. Engel AK, Fries P, Singer W. Dynamic predictions: oscillations and synchrony in top-down processing. *Nat Rev Neurosci*. 2001;2:704–716.
66. Corlett PR, Frith CD, Fletcher PC. From drugs to deprivation: a Bayesian framework for understanding models of psychosis. *Psychopharmacology (Berl)*. 2009;206:515–530.
67. Powers AR, Mathys C, Corlett PR. Pavlovian conditioning-induced hallucinations result from overweighting of perceptual priors. *Science*. 2017;357:596–600.
68. Craig TKJ, Rus-Calafell M, Ward T, et al. AVATAR therapy for auditory verbal hallucinations in people with psychosis: a single-blind, randomised controlled trial. *Lancet Psychiatry*. 2017; 5:31–40.
69. Howes OD, Bose SK, Turkheimer F, et al. Dopamine synthesis capacity before onset of psychosis: a prospective [18F]-DOPA PET imaging study. *Am J Psychiatry*. 2011;168(12):1311–1317.
70. Waters F, Blom JD, Jardri R, Hugdahl K, Sommer IEC. Auditory hallucinations, not necessarily a hallmark of psychotic disorder. *Psychol Med*. 2018;48:529–536.
71. Egerton A, Chaddock CA, Winton-Brown TT, et al. Presynaptic striatal dopamine dysfunction in people at ultra-high risk for psychosis: findings in a second cohort. *Biol Psychiatry*. 2013;74:106–112.
72. Jauhar S, Nour MM, Veronese M, et al. A test of the transdiagnostic dopamine hypothesis of psychosis using positron emission tomographic imaging in bipolar affective disorder and schizophrenia. *JAMA Psychiatry*. 2017;74:1206–1213.
73. Bailey T, Alvarez-Jimenez M, Garcia-Sanchez AM, Hulbert C, Barlow E, Bendall S. Childhood trauma is associated with severity of hallucinations and delusions in psychotic disorders: a systematic review and meta-analysis. *Schizophr Bull*. 2018;44:1111–1122.
74. 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.
75. Egerton A, Valmaggia LR, Howes OD, et al. Adversity in childhood linked to elevated striatal dopamine function in adulthood. *Schizophr Res*. 2016;176:171–176.
76. Hugdahl K, Craven AR, Nygård M, et al. Glutamate as a mediating transmitter for auditory hallucinations in schizophrenia: a (1)H MRS study. *Schizophr Res*. 2015;161:252–260.
77. Waters F, Blom JD, Dang-Vu TT, et al. What is the link between hallucinations, dreams, and hypnagogic-hypnopompic experiences? *Schizophr Bull*. 2016; 42:1098–1109.
78. Beyer S. *The Cult of Tara: Magic and Ritual in Tibet*. Berkeley, Los Angeles, London: University of California Press; 1978.
79. MacLean KA, Ferrer E, Aichele SR, et al. Intensive meditation training improves perceptual discrimination and sustained attention. *Psychol Sci*. 2010;21:829–839.
80. Noll R. Mental imagery cultivation as a cultural phenomenon, with commentary. *Curr Anthropol*. 1985;26(4):443–461.
81. Hugdahl K, Craven AR, Nygård M, et al. Glutamate as a mediating transmitter for auditory hallucinations in schizophrenia: a (1)H MRS study. *Schizophr Res*. 2015;161:252–260.
82. Čurčić-Blake B, Bais L, Sibeijn-Kuiper A, et al. Glutamate in dorsolateral prefrontal cortex and auditory verbal hallucinations in patients with schizophrenia: a 1H MRS study. *Prog Neuropsychopharmacol Biol Psychiatry*. 2017;78:132–139.
83. Mouchlianitis E, Bloomfield MA, Law V, et al. Treatment-resistant schizophrenia patients show elevated anterior cingulate cortex glutamate compared to treatment-responsive. *Schizophr Bull*. 2016;42:744–752.
84. Ruby E, Rothman K, Corcoran C, Goetz RR, Malaspina D. Influence of early trauma on features of schizophrenia. *Early Interv Psychiatry*. 2017;11:322–333.
85. Lange C, Huber CG, Fröhlich D, Borgwardt S, Lang UE, Walter M. Modulation of HPA axis response to social stress in schizophrenia by childhood trauma. *Psychoneuroendocrinology*. 2017;82:126–132.
86. McIntosh TK, Bush HL, Yeston NS, et al. Beta-endorphin, cortisol and postoperative delirium: a preliminary report. *Psychoneuroendocrinology*. 1985;10:303–313.