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Post-Traumatic Symptomatology and Compulsions as Potential Mediators of the Relation Between Child Sexual Abuse and Auditory Verbal Hallucinations

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Background: Whilst evidence is mounting that childhood sexual abuse (CSA) can be a cause of auditory verbal hallucinations (AVH), it is unclear what factors mediate this relation. Recent evidence suggests that post-traumatic symptomatology may mediate the CSA–AVH relation in clinical populations, although this hypothesis has not yet been tested in the general population. There is also reason to believe that obsessive ideation could mediate the CSA–AVH relation. **Aims:** To test for evidence to falsify the hypotheses that post-traumatic symptomatology, obsessions, compulsions, anxiety and depression mediate the relation between CSA and AVH in a general population sample. **Method:** Indirect effects of CSA on AVH via potential mediators were tested for, using a regression-based approach employing data from the 2007 Adult Psychiatric Morbidity Survey ($n = 5788$). **Results:** After controlling for demographics, IQ and child physical abuse, it was found that CSA, IQ, post-traumatic symptomatology and compulsions predicted lifetime experience of AVH. Mediation analyses found significant indirect effects of CSA on AVH via post-traumatic symptomatology [odds ratio (OR): 1.11; 95% confidence interval (CI):1.00–1.29] and compulsions (OR: 1.10, 95% CI: 1.01–1.28). **Conclusions:** These findings offer further support for the hypothesis that post-traumatic symptomatology is a mediator of the CSA–AVH relation. Although no evidence was found for obsessional thoughts as a mediating variable, a potential mediating role for compulsions is theoretically intriguing. This study's findings reiterate the need to ask about experiences of childhood adversity and post-traumatic symptomology in people with AVH, as well as the likely therapeutic importance of trauma-informed and trauma-based interventions for this population.

Keywords: child abuse, psychosis, rape, schizophrenia, trauma, voice-hearing

Introduction

For over a century there have been proposals that suffering child abuse is associated with an increased probability of later developing the experience of 'hearing voices', formally termed auditory verbal hallucinations (AVH; McCarthy-Jones, 2012). Yet it has only been in the past two decades that large-scale empirical studies have confirmed an association of AVH with child abuse, and in particular child sexual abuse (CSA; Bentall et al., 2012; Daalman et al., 2012; Shevlin et al., 2007; Read et al., 2003; Üçok and Bikmaz, 2007). Evidence is mounting that these associations are causal (Bentall et al., 2012; Kelleher et al., 2013; Kendler et al.,

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2000), although some concerns remain to be addressed (Susser and Widom, 2012), including that this relation could stem from an evocative gene–environment correlation (e.g. Sideli et al., 2012). As AVH can lead to distress, self-harm and suicide (Kjelby et al., 2015), there is an urgent need to better understand their causes to better prevent such consequences.

Little empirical work has been done on what factors may mediate the specific relation between CSA and AVH, despite multiple studies calling for such investigations (e.g. Bentall et al., 2012, 2014). Initial studies that examined mediators of the relations between CSA and psychosis (Bebbington et al., 2011; Marwaha and Bebbington, 2015), and between CSA and hallucinations in general (Sitko et al., 2014), found evidence consistent with depression and anxiety acting as mediators. However, a recent study of people with psychosis by Hardy et al. (2016), which did specifically test for mediators between CSA and AVH, found no evidence that depression mediated the CSA–AVH relation. Instead, evidence was reported consistent with post-traumatic stress disorder (PTSD) symptomatology (avoidance, numbing and hyperarousal) mediating this relation.

Precise estimates of rates of PTSD in CSA survivors are hard to establish, due to methodological and sampling variation between studies and the impact of the nature of the CSA on the probability of PTSD developing. At the low end of estimates, Cutajar et al. (2010) found that 4% of adult survivors of CSA had been formally diagnosed with PTSD, and Maikovich et al. (2009) found that 14% of children who had experienced CSA had clinically significant levels of PTSD symptoms. Other studies have reported higher estimates. Deblinger et al. (1989) found that 21% of psychiatrically hospitalized children who had experienced CSA met diagnostic criteria for PTSD, Widom (1999) reported that 38% of adults who had suffered CSA met lifetime diagnostic criteria for PSTD, and McLeer et al. (1992) found that 10% of children sexually abused by strangers, 42% of children sexually abused by a trusted adult, and 54% of children sexually abused by their fathers met diagnostic criteria for PTSD. Whilst the prevalence of PTSD in CSA survivors remains unclear, what is not in debate is that CSA increases the risk of an individual developing PTSD symptomatology (Chen et al., 2010; Fergusson et al., 2013; Kendall-Tackett et al., 1993).

One of the hallmark symptoms of PTSD, highly prevalent in survivors of CSA, is intrusions (Kuyken and Brewin, 1994; Priebe et al., 2013). The extent of these in CSA survivors was recently documented by Priebe et al. (2013), using an experience sampling methodology. They found that in a 12-hour period, from 08.00 h to 20.00 h, CSA survivors diagnosed with PTSD reported an average of 75 intrusions and 24 flashbacks. As intrusive thoughts are also associated with the presence of AVH (Morrison and Baker, 2000; Jones and Fernyhough, 2006) they are one potential mediator of the relation between CSA and AVH.

Intrusive thoughts are not a homogeneous category. Distinct types have been identified and separated off into named disorders. Intrusive thoughts about personal loss and failure (negative automatic thinking) are viewed as characteristic of depression (Beck, 1967; Clark, 1992). Intrusive thoughts about possible harm and danger are viewed as characteristic of anxiety (Clark, 1992; Clark and Beck, 1989). A third category of intrusive thought that can be distinguished is those which are obsessive (Clark, 1992; Salkovskis, 1985). Obsessive intrusive thoughts do not run parallel to the stream of consciousness, seem to involve socially unacceptable thoughts that indicate a loss of impulse control, and are associated with an exaggerated sense of personal responsibility (Clark, 1992; Salkovskis, 1985).

There is reason to believe that intrusive obsessional thoughts, whose presence are a criterion for obsessive-compulsive disorder (OCD; APA, 2013), may be a mediator of

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the relation between CSA and AVH. The idea that AVH specifically share something in common with obsessions goes back to (at least) 1895 and the French psychiatrist Jules Seglas, who coined the term *hallucination obsédante* (obsessional hallucinations) to refer to a hallucination accompanied by the characteristics of an obsession. Not only is there a marked phenomenological parallel between AVH and thoughts in OCD, with both being highly repetitive (McCarthy-Jones et al., 2012), but there are also preliminary suggestions that drugs with anti-obsessional effects may be able to help AVH (Stephane et al., 2000). Furthermore, patients with OCD have been repeatedly shown to have deficits in intentional cognitive inhibition, the ability to effortfully, consciously suppress cognitions from consciousness (Badcock et al., 2007; Bohne et al., 2005), as have individuals with AVH in both clinical (Waters et al., 2003; Badcock et al., 2005) and non-clinical (Paulik et al., 2007) populations. Indeed, Waters et al. (2003) have argued that a deficit in intentional cognitive inhibition is 'critical' to AVH (p. 276). To this may be added findings from diffusion tensor imaging studies, which have found that the presence of AVH in schizophrenia is associated with reduced structural integrity of the fronto-temporal segment of the inferior occipital-frontal fasciculus (Oestreich et al., 2015). Damage to this tract has been found in other populations to be associated with verbal perseverance (Khan et al., 2014). Not only are AVH and schizophrenia associated with an increased risk of a diagnosis of OCD (Attademo et al., 2015), but so is CSA (Caspi et al., 2008). It is therefore plausible that CSA results in a reduction in inhibitory control, which leads to obsessional ideation, which may in some cases come to manifest as AVH.

It could be objected that obsessive ideation and AVH could be separate manifestations of a reduction in inhibitory control, with no causal relation between the two. However, clinical examples can be found that document the development of AVH from what began as obsessional-compulsive symptomatology. Consider the following example from Poyurovsky (2013):

> '*Since the age of 18 Emma has heard voices which curse her and repeat phrases such as 'yes, correct'. Her first psychiatric problem occurred when she was 12 when she became preoccupied with aggressive and sexual thoughts and fears of saying the wrong thing. As a result she started a range of compulsive checking, counting and repetitive behaviours for up to 2–3 hours a day. At 19 she developed 'obsessive auditory hallucinations' as well as delusions.*'

Less anecdotally, van Dael et al. (2011) have found that levels of obsessive-compulsive symptomatology prospectively predict the later development of psychotic symptoms. It is therefore plausible that obsessive ideation resulting from CSA may come to be experienced as AVH, and hence could act as a mediator between CSA and AVH. Of course, not all AVH have a repetitive nature (McCarthy-Jones et al., 2012), and any such model of the AVH experience would probably only account for a subset of AVH (Jones, 2010).

As a result of this, the first aim of this study was to use data collected by the 2007 Adult Psychiatric Morbidity Survey (APMS; McManus et al., 2009) to test the hypothesis that obsessional thoughts mediate the relation between CSA and AVH in the general population. The specificity of this effect to obsessional thoughts was to be tested for by also examining if there was any evidence that levels of compulsions, depression or anxiety could act as mediators between CSA. The specificity of this effect to CSA was to be tested for by controlling for levels of child physical abuse (CPA), which has previously been found to be associated with AVH in the APMS dataset (Bentall et al., 2012). The second aim of this study was to test the hypothesis that PTSD symptomatology would mediate the relation between CSA and AVH in a general population sample. Although the cross-sectional APMS dataset cannot be used to prove the

existence of a causal, mediating role for the candidate variables, the study nevertheless still had the ability to provide evidence to falsify the hypothesized mediating mechanisms.

Method

Participants

The study used data from the 2007 APMS (McManus et al., 2009). In this survey, 7403 individuals aged over 16 years living in private households completed interview and administered computer-assisted interviews and self-completion questionnaires on topics including demographics, physical health, mental health, social capital and child abuse.

Measures

Child sexual abuse (CSA). Three types of CSA were assessed by the APMS: CSA involving intercourse (CSA_{int}), CSA involving touch but not intercourse (CSA_{touch}), and CSA involving talk but not intercourse or touch (CSA_{talk}). Previous studies of the APMS dataset have found that of these three forms of sexual abuse, only CSA_{int} was associated with AVH (Bentall et al., 2012). This study therefore limited CSA to the binary variable CSA_{int}, which was assessed by the question 'Before the age of 16, did anyone have sexual intercourse with you without your consent?'. This was a binary variable (yes/no). In order to increase the power of the study, those who had experienced CSA_{touch} or CSA_{talk}, in the absence of CSA_{int}, were removed from the study. This left two groups, a CSA_{int} group and a control group who did not report any form of child sexual abuse.

Child physical abuse (CPA). This was assessed using the item 'Before the age of 16, were you ever severely beaten by a parent, step-parent, or carer?'. This was a binary variable (yes/no).

Auditory verbal hallucinations (AVH). Experiences of complex AVHs over the past year were assessed using the Psychosis Screening Questionnaire (Bebbington and Nayani, 1995) item '[Over the past year] 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?'. This was a binary variable (yes/no).

Anxiety. Following the approach of previous studies (Bebbington et al., 2011; Marwaha and Bebbington, 2015), anxiety was defined as the sum of the anxiety and worry subscales of the Revised Clinical Interview Schedule (CIS-R; Lewis et al., 1992). CIS-R (worry) subscale scores (APMS variable name: DVI11) are calculated based on four questions, including 'Been worrying about things other than physical health on four or more days out of past seven days?' and 'Have been worrying too much in view of your circumstances?'. Endorsement of an item is scored 1, and hence total scores on this subscale can range from 0 to 4. CIS-R (anxiety) subscale scores (APMS variable name: DVJ12) are also calculated based on responses to four questions. Endorsement of items such as 'Felt generally anxious/nervous/tense on four or more of past seven days' and 'Anxiety/nervousness/tension has been very unpleasant in past week' are scored one point. Total scores on this subscale can hence range from 0 to 4. Scores on the anxiety measure created by combining the CIS-R (worry) and CIS-R (anxiety) subscales could therefore range from 0 to 8, and this combined measure had satisfactory internal reliability (Cronbach's $\alpha = .80$).

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Depression. Following the approach of previous studies (Bebbington et al., 2011; Marwaha and Bebbington, 2015), depression was defined as the sum of the CIS-R (depression) and CIS-R (depressive ideas) subscales. Responses to four items are used to calculate a depression subscale score (APMS variable name: DVG11). Endorsement of items such as 'Unable to enjoy or take an interest in things as much as usual in past week' and 'Felt sad, miserable or depressed/unable to enjoy or take an interest in things on four days or more in past week' are scored one point. Total scores on this subscale can hence range from 0 to 4. Responses to five items are used to calculate a depressive ideas subscale score (APMS variable name: DVH11). Endorsement of items such as 'Felt guilty when things went wrong in past week' are scored one point. Total scores on this subscale can hence range from 0 to 5. Scores on the depression measure created by combining the CIS-R (depression) and CIS-R (depressive ideas) subscales could therefore range from 0 to 9. This measure had satisfactory internal reliability (Cronbach's $\alpha = .80$).

Obsessional thought. This was assessed using the four-item CIS-R (obsessions) subscale score (APMS variable name: DVN9). Example of items include 'Unpleasant thoughts or ideas kept coming into your mind on four days or more in last week' and 'Tried to stop thinking any of these thoughts in past week'. Endorsement of an item is scored as one point, and total scores on this scale can hence range from 0 to 4. This scale had satisfactory internal reliability (Cronbach's $\alpha = .89$).

Compulsions. This was assessed using the four-item CIS-R (compulsions) subscale score (APMS variable name: DVM9). Examples of these items include 'Found yourself doing things over again (that you had already done) on four days or more in last week' and 'Have tried to stop repeating behaviour/doing these things over again during past week'. Endorsement of an item is scored as one point, and total scores on this scale can hence range from 0 to 4. This scale had satisfactory internal reliability (Cronbach's $\alpha = .85$).

PTSD symptomatology. This was assessed using the total score (APMS variable name: PTSDcom) on the Trauma Screening Questionnaire (Brewin et al., 2002). This is a brief 10-item self-report measure that assesses the presence of re-experiencing and arousal symptoms using items derived from PTSD Symptom Scale-Self Report version (Foa et al., 1993). Examples of items include 'Feeling upset about reminders of the event' and 'Being jumpy or being startled at something unexpected'. Endorsement of an item is scored as one point, and total scores on this scale can hence range from 0 to 10. This scale had satisfactory internal reliability (Cronbach's $\alpha = .82$).

Demographic and other confounds. The following variables were also assessed to control for their potentially confounding effects: age, gender (self-reported male or female), ethnicity (white British, other), highest educational qualification (degree, non-degree higher qualification, A-level, GCSE, other, none), and IQ (assessed through the National Adult Reading Test; Nelson, 1991).

Statistical analyses

Multiple linear regression was used to test for total, direct and indirect relations between CSA and AVH using the PROCESS macro (Hayes, 2013) in SPSS version 22. PROCESS employs maximum likelihood logistic regression for dichotomous dependent variables, and was

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Table 1. Descriptive statistics for sample

Variable	$n = 5788$
Categorical variables (*n*, %)	
Gender (female)	3204 (55.4%)
Ethnicity (white British)	5413 (93.5%)
Highest educational level	
Degree	1036 (17.9%)
Teaching/vocational qualification	417 (7.2%)
A-level	752 (13.0%)
GSCE or equivalent	1500 (25.9%)
Foreign/other	212 (3.7%)
None	1871 (32.3%)
Child sexual abuse (intercourse)	126 (2.2%)
Child physical abuse	207 (3.6%)
Auditory verbal hallucinations	49 (0.8%)
Continuous variables (mean, standard deviation, range)	
Age (years)	51.71 (18.76, 16–95)
IQ	102.61 (15.24, 70–127)
Anxiety	0.86 (1.56, 0–8)
Depression	0.63 (1.49, 0–9)
Obsessions	0.16 (0.66, 0–4)
Compulsions	0.11 (0.56, 0–4)
PTSD	0.50 (1.43, 0–10)

employed using bias corrected, bootstrapped 95% confidence intervals for indirect associations with 1000 bootstrap samples. In the first set of analyses, CSA was entered as the independent variable, AVH as the dependent variable, PTSD, anxiety, obsessions and compulsions as mediating variables (using PROCESS model 4 which uses a parallel mediation model), and age, gender, ethnicity (dummy coded), highest educational qualification (dummy coded), IQ, CPA and depression were added as covariates. Covariates were used in models of both the mediators and the dependent variables. As PROCESS model 4 can only test for four mediating variables at one time, the analyses were then re-run to test if there was any evidence of an indirect effect of depression on AVH. This analysis was performed in the same manner as above, with the only changes being that anxiety was removed as a mediator and included as a covariate, and depression was removed as a covariate and including as a mediator.

Results

A number of participants in the sample had missing data for some of the study variables. As PROCESS cannot work with missing data, these individual were removed from the analysis. In addition, as noted above, participants who had experienced either CSA_{touch} or CSA_{talk}, in the absence of CSA_{int}, were also removed from the sample. This yielded a remaining sample size of 5788. Descriptive statistics are reported in Table 1.

The study variables were a significant predictor of the presence of AVH; χ^2 (16) = 132.11, $p < .001$, –2 Log likelihood = 433.10, Nagelkerke $R^2 = .24$. Details of the final regression

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Table 2. Predictors of presence of auditory verbal hallucinations

Variable	β (standard error, 95% CI)	p
Age	0.01 (0.01, –0.01 to 0.03)	.44
Gender	0.19 (0.33, –0.46 to 0.85)	.56
IQ	–0.03 (0.01, –0.06 to 0.01)	.004
Ethnicity	–0.92 (0.67, –2.22 to 0.39)	.17
Education		
Degree	–0.76 (0.82, –2.37 to 0.85)	.36
Teaching/vocational	1.13 (0.55, 0.06 to 2.19)	.04
A-level	0.07 (0.56, –1.02 to 1.17)	.90
GSCE or equivalent	0.23 (0.43, –0.60 to 1.07)	.59
Foreign/other	–0.18 (1.06, –2.27 to 1.90)	.86
Child physical abuse	0.84 (0.43, –0.01 to 1.69)	.05
Child sexual abuse	1.64 (0.44, 0.77 to 2.51)	<.001
Anxiety	0.15 (0.09, –0.02 to 0.32)	.08
Depression	0.10 (0.09, –0.07 to 0.28)	.23
Obsessions	0.16 (0.12, –0.07 to 0.40)	.18
Compulsions	0.32 (0.13, 0.07 to 0.58)	.01
PTSD	0.19 (0.06, 0.08 to 0.30)	<.001

Effect of gender is for men, relative to a female reference group. Effect of ethnicity is for being non-White British, relative to a White British reference group. Effect of education is relative to a reference group with no qualifications.

Table 3. Direct and indirect effects of child sexual abuse on auditory verbal hallucinations

Variable	Odds ratio (95% confidence interval)
Total effect of CSA	5.81 (2.53 to 13.33)*
Direct effect of CSA	5.15 (2.16 to 12.30)*
Indirect effects of CSA	1.27 (1.06 to 1.55)*
Effect via anxiety	1.04 (0.99 to 1.16)
Effect via PTSD	1.11 (1.00 to 1.29)*
Effect via obsessions	1.00 (0.97 to 1.07)
Effect via compulsions	1.10 (1.01 to 1.28)*

*$p < .05$.

equation for predicting AVH are presented in Table 2; as this table shows, CSA, PTSD symptomatology, compulsions and IQ were significant predictors of AVH.

There was a significant total association between CSA and AVH, consisting of significant direct and indirect effects (Table 3). As Table 3 shows, when the significant indirect effect of CSA on AVH (at $p < .05$) was broken down into its component parts, evidence was only found of a significant indirect effect of CSA on AVH via PTSD symptomatology and compulsions.

It was next tested if there was an indirect effect of CSA on AVH via depression. The original analyses were repeated with the only difference being that depression was now entered as a mediator, not a covariate, and anxiety was entered as a covariate, not a mediator. This analysis

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found no evidence of an indirect effect of CSA on AVH via depression; odds ratio 1.10 (95% confidence interval: 0.93–1.32).

Discussion

This study aimed to test the hypotheses that obsessive thoughts and PTSD symptomatology mediate the relation between CSA and AVH in a general population sample. The hypothesis that obsessive thoughts mediated the relation between CSA and AVH was falsified. There was also no evidence of a main effect of obsessive thoughts on AVH. This was surprising, given the strong theoretical rationale for expecting a relation between obsessive thought and AVH. One possibility is that this was a Type II error, stemming from the measure of obsessive thought employed. The limited sensitivity of the measure, which was only scored on a five-point Likert scale, could have been the reason that no effect was found. However, this argument is mitigated against by the finding of an indirect effect of CSA on AVH via compulsions, a measure that employed the same scoring scale. A second possibility is that the CIS-R measure of obsessions only captures this construct at a high level. It could be helpful for future research to use more sensitive measures that capture specific aspects of obsessive thought. For example, research could examine if AVH are specifically associated with socially unacceptable obsessive thoughts involving an exaggerated sense of personal responsibility (Clark, 1992), which are characteristic of OCD but yet were not specifically inquired about as part of the CIS-R measure of obsessions.

This study failed to falsify the hypothesis that PTSD symptomatology mediated the relation between CSA and AVH. A main effect of PTSD symptomatology on AVH was also found. These findings speak to the argument that AVH should be a named characteristic symptom of PTSD (McCarthy-Jones and Longden, 2015). The evidence for an indirect effect of CSA on AVH via PTSD symptomatology in this general population sample mirrors the finding of such a relation in a clinical sample by Hardy et al. (2016). The failure of this study to find evidence of an indirect effect of CSA on AVH via depression also mirrors the findings of Hardy et al. Further work is now needed, using longitudinal studies, to test if PTSD symptomatology does indeed mediate the relation between CSA and AVH. Such work should also aim to better understand the specific processes involved, such as whether it is intrusions from trauma memory resulting from CSA that form the basis of AVH (Hardy, 2017).

An unexpected finding was that there was an indirect effect of CSA on AVH via compulsions. It is possible that this represents a Type I error, and attempted replication of this finding is needed. Although further speculation on the meaning of this finding is premature, it is nonetheless of theoretical interest. Should the current pattern of findings be replicated, it would need to be explained why compulsions but not obsessions were associated with AVH. An interesting possibility is raised here by a consideration of trichotillomania. This condition involves the compulsive act of hairpulling, yet is not strongly associated with obsessive thoughts (Bohne et al., 2005). Trichotillomania has been found to be associated with abnormal motor inhibitory functioning (Bohne et al., 2008; van Velzen et al., 2014), which is notable given arguments that AVH could be understood as based in inner speech, which can be conceived of as a motor action (Jones and Fernyhough, 2007). This raises the possibility that specifically abnormal *motor* inhibitory functioning could play an important role in AVH. This could be a profitable direction for future study. Of course, another possibility is that compulsions are a consequence, not a cause, of AVH. One way to understand such a relation would be to propose

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that what voices say drives people to undertake compulsive actions. Had the findings of this study come from a clinical population of voice-hearers, in which commands are common (McCarthy-Jones et al., 2012; Nayani and David, 1996), this would be a plausible explanation. However, it appears unlikely that this was the case in this sample of voice-hearing individuals from the general population, as there is some suggestion that commands are less common in non-clinical voice-hearers (Hill et al., 2012).

This study had a number of limitations. First, it relied upon self-reported child abuse history, and such retrospective self-reports are likely to underestimate child abuse (Hardt and Rutter, 2004). Second, despite the parallels of a number of this study's findings to the recent results of Hardy et al. (2016), it is unclear whether the finding relating to compulsions is likely to be generalizable to a clinical population. There is some evidence that compulsions are associated with AVH in people diagnosed with schizophrenia (Guillem et al., 2009). This is also a population with high rates of CSA, with Read et al. (2005) finding that 48% of women with a psychotic disorder, and 28% of men, reported CSA. However, the potential for compulsions to act as a mediator between CSA and AVH in clinical populations remains untested. Third, this study made the assumption that state measures (previous week) of anxiety, depression, obsessions and compulsions were a reflection of trait levels. This may not have been the case. Again this points to the need for prospective study.

Another limitation is that this study did not test for other key potential mediators. As the indirect effects of CSA on AVH through PTSD symptomatology and compulsions were small and could only represent partial mediation, this raises the question as to what other factors may act as mediators. The answer to this could be informed by a three-stage approach that asks: (1) what emotions did the CSA lead to?, (2) how did the individual attempt to regulate these emotions?, and (3) what is their impact on intrusions into consciousness? It has been argued that some post-trauma emotions are likely to be more hallucinogenic than others. In particular, I have argued that shame is likely to be hallucinogenic (McCarthy-Jones, 2017). This is probably in large part due to the emotion regulation strategies that tend to be employed to deal with shame, such as dissociation (McCarthy-Jones, 2017). Indeed, dissociation is one of the best evidenced mediators of the relationship between childhood trauma and hallucinations (Perona-Garcelán et al., 2012; Pearce et al., 2017; Varese et al., 2012). However, continuing with the example of shame, a number of other mediators are possible, such as suppression, rumination and hypervigilance (McCarthy-Jones, 2017). There is hence the need for a large, prospective study that is able to test a complex model of the relation between CSA and AVH. This would assess key post-trauma emotions (e.g. shame, guilt, anger; Andrews et al., 2000), a wide range of coping mechanisms employed to deal with these emotions (e.g. dissociation, suppression, hypervigilance), and the nature of intrusions (e.g. obsessive thoughts, trauma memories, etc.).

Any clinical implications of this study are necessarily tentative. However, as these findings add to the body of evidence that post-traumatic symptomatology may be a mediator of the CSA–AVH relation (Hardy et al., 2016), it reinforces a number of points recently made by Hardy (2017). These are that the presence of trauma and post-traumatic symptomatology should be assessed in psychosis, and that there is a clinical need for trauma-based therapy for psychosis. Whilst there are multiple reasons why people hear voices, and we need multiple models to address this (Jones, 2010), it is clear that trauma is a significant factor in many people's voice-hearing and will often be encountered and need to be addressed in practice.

In summary, this study found evidence consistent with the hypothesis that PTSD symptomatology and compulsions mediate the relation between CSA and AVH, but not depression, anxiety or obsessive thoughts. Further work is now needed to devise and test a comprehensive model of the potential mediating mechanisms between these experiences.

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