

Explaining Auditory Verbal Hallucination in Borderline Personality Disorder: The Role of Childhood Trauma, Anxiety, Depression, and Dissociation

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ABSTRACT

Despite a high proportion of people diagnosed with Borderline Personality Disorder (BPD) experiencing Auditory Verbal Hallucinations (AVH), few studies have investigated factors that might contribute to these distressing experiences.

Aim: The aim of this study was to assess the relationship between trauma, dissociation, mood (i.e., anxiety/depression) and their relative contribution to AVH in a BPD population.

Methods: Thirty adults diagnosed with BPD participated in the study. Participants were categorized into two groups; those who heard voices (BPD-AVH; n = 16) and those who did not hear voices (BPD-No AVH; n = 14). Two theoretical models explored the relationship between trauma, dissociation and AVH, and 1) anxiety; and 2) depression.

Results: The results suggested that model 1 was a better fit, suggesting that the relationship between trauma and AVH is mediated, in sequence, by the experience of anxiety and dissociation. Model 2 was not a good fit.

Conclusion: These findings suggest that therapeutic interventions which address the levels of anxiety and dissociation may assist people with BPD and AVH.

INTRODUCTION

Over the past decade, research has shown that auditory verbal hallucinations (AVH, or hearing voices) experienced by people diagnosed with Borderline Personality Disorder (BPD) are phenomenologically similar to AVH experienced by people diagnosed with a primary psychotic disorder such as schizophrenia [1-3]. In the context of BPD, AVHs are often described as critical, commanding, threatening, terrorizing, or attacking [2], causing substantial distress [4,5], as well as increased rates of suicidal and self-injurious behaviours [4]. Research in other clinical populations has suggested that AVH co-occur with trauma [7,8], dissociation [9,10], increased stress [11,12], an inability to cope/regulate emotions (Hardy, 2017) and insecure and disorganised attachment [5,13]. A meta-analysis investigating features of psychosis concluded that childhood adversity predicted psychotic experiences including AVH [14]. A strong link between childhood trauma and AVH has also been reported in BPD

[14,15]. The prevalence of childhood trauma in people with BPD who experience AVH exceeds what is reported in schizophrenia with AVH [16,17]. In the context of BPD, the voices may repeat phrases used by past abusers or convey the abuser's attitude to the patient as experienced at the time of the trauma [12].

Some authors have argued that AVH, regardless of psychiatric diagnosis, may be more appropriately understood as dissociative rather than psychotic phenomena [18]. That is, AVH may represent dissociated or disowned components of self (or self-other relationships) arising from trauma, loss, or other interpersonal stressors. Dissociation may be used as a psychological defence mechanism in response to acute trauma, resulting in a disruption to the normal integration of psychobiological functioning [19]. As such, it may serve as a protective mechanism that enables individuals to cognitively detach from events that are too overwhelming for them to process consciously [10]. Despite its defensive function during acute trauma, the use of dissociative defences in the longer term can be harmful as they can lead to severe disconnection from aspects of self, others, and the environment. Dissociative symptoms occur in 66-75% of people diagnosed with BPD [20,21], with several authors proposing that AVH in BPD are dissociative in origin [9,11,17,22,23]. A striking finding in a study by Tschoeke and colleagues [17] was that almost all of their BPD-AVH sample (96%, $n = 23$) also met criteria for a dissociative disorder. Furthermore, in comparison with people diagnosed with schizophrenia, people with BPD with AVH felt more controlled by their voices. In clinical settings, people experiencing AVH tend also to exhibit moderate/high dissociative symptoms [12]. A trauma-related dissociative model has often been used to explain AVH in BPD [24, 25]. There is some controversy regarding whether psychotic symptoms of BPD are best described as dissociative in nature and thus fit within the symptom spectrum of BPD, or alternatively whether they represent a comorbid dissociative disorder [17].

There is also growing evidence to suggest that affective symptoms play a key role in the experience of AVH. Hartley, Barrowclough & Haddock [26] conducted a systematic review of psychotic disorders and concluded that both anxiety and depression are associated with the severity of delusions and

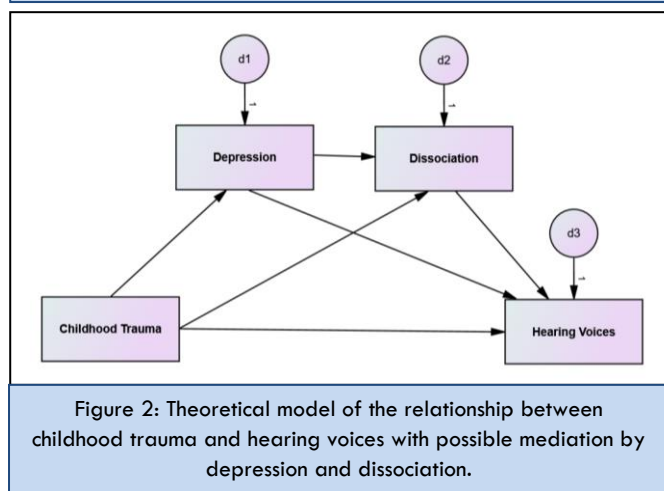
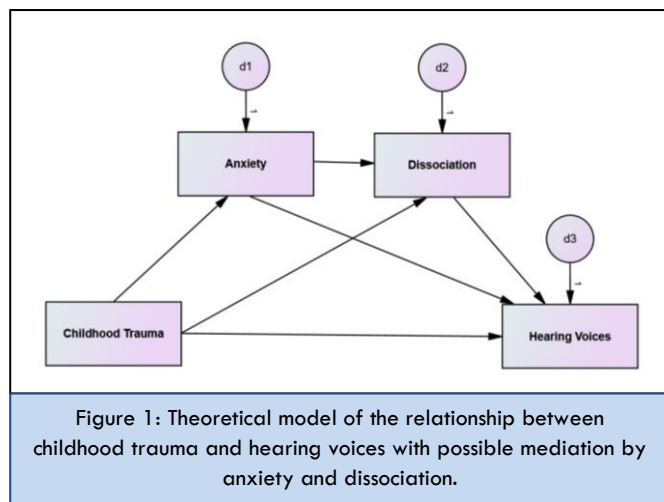
hallucinations, the distress they elicit, and their content. Empirical research by Scott and colleagues [13] found that among voice-hearers with affective psychosis, (i) critical, threatening, and self-harm voice content was predicted by co-occurring anxiety, and (ii) in voice hearers with non-affective psychosis, depression predicted self-harm voice commands. Some literature suggests that negative/critical voice content is related to anxiety and depression in a similar way as self-criticism [27]. However, little is known about whether anxiety and depression play a role in BPD-related AVH. Early work by Chopra & Beatson [28] described a series of clinical interviews with 13 people diagnosed with BPD who experienced AVH. They concluded that the hallucinations appeared to manifest during periods of intense anxiety arising from an inability to cope with stress. More recent studies that have compared people with BPD on the basis of whether they do or do not experience AVH have reported an association of AVH with depression and anxiety $27=4$ [5,15]. However, these findings are not altogether consistent across studies. For example, Benvenuti et al. [29] were unable to establish a difference between psychotic symptoms experienced by BPD patients with and without a co-morbid mood disorder on a lifetime basis.

In sum, there is growing interest in the mechanisms that might help explain the occurrence of AVH, but most research has focussed on AVH in people with primary psychotic disorders, with minimal investigation of AVH in people with BPD. The few studies that have investigated AVH in people with BPD have generally focused on correlations only. The present study seeks to extend the literature by investigating factors that may mediate the occurrence of AVH in BPD.

Aims and hypotheses

Although previous research has established relationships of AVH with childhood trauma, dissociation, and mood (i.e., anxiety/depression), we lack understanding of how these constructs might specifically be associated with/ contribute to the phenomenon of AVH in BPD. Understanding more about processes that might underlie the experience of AVH in BPD could help explain AVH phenomenology and possibly have important implications for the treatment of BPD. The aim of the current study was to assess the relationships between childhood trauma, dissociation and anxiety/depression, and their relative contributions to the aetiology of AVH in BPD population. We

derived two theoretical models to explore the association between childhood trauma and hearing voices. Both were informed by the existing literature and are shown in Figures 1 and 2 below. The difference between the models is that in Model 1, anxiety is the first mediator whereas in Model 2 it is depression. The models are the same in all other respects. Anxiety and depression were examined independently across the two models given the inconsistency of associations reported for these two mood symptoms and AVH in previous BPD literature. Although there is no direct evidence to suggest that anxiety and depression proceed dissociation in people diagnosed with BPD, for the purpose of this study we explored their association to the other variables using this directional model.



Both models are directional, since childhood trauma is known to give rise to anxiety and depression [24], and the literature also indicates that dissociation is a form of an escape from the discomfort associated with trauma, anxiety, and depression [9].

The following hypotheses were proposed for empirical testing:

Model 1

Hypothesis 1: Childhood trauma is positively correlated with AVH.

Hypothesis 2: Anxiety mediates the relationship between childhood trauma and AVH.

Hypothesis 3: Dissociation mediates the relationship between childhood trauma and AVH.

Hypothesis 4: Anxiety and dissociation in combination mediate the relationship between childhood trauma and AVH.

Model 2

Hypothesis 5: Depression mediates the relationship between childhood trauma and AVH.

Hypothesis 6: Dissociation mediates the relationship between childhood trauma and AVH.

Hypothesis 7: Depression and dissociation in combination mediate the relationship between childhood trauma and AVH.

METHOD

Participants

Thirty outpatient adults diagnosed with BPD participated in the study. Participants were categorized into two groups; those who heard voices (BPD-AVH; $n = 16$) and those who did not hear voices (BPD-No AVH; $n = 14$). Eligibility criteria included a) aged 18 to 65 years, b) proficiency in English language, c) estimated premorbid $IQ > 70$ to ensure comprehension of study instructions, d) no known history of significant neurological disorders, e) absence of alcohol or substance use disorder during the previous 12 months, f) no gross hearing impairments, and g) no history of a psychotic disorder (e.g., schizophrenia). BPD participants were recruited from: a) a state-wide specialist personality disorder service in Victoria, Australia, b) national online advertising via the Australian BPD Foundation, and c) advertising on general community online mental health forums.

Materials

Personal and mental health information was collected using a demographic-clinical interview. Three clinician-administered instruments were used. They were (i) the Revised Diagnostic Interview for Borderline (DIB-R; inter-rater reliability $Kappa = .75$; [30]) to ascertain current BPD diagnosis, (ii) the MINI International Neuropsychiatric Interview (MINI; inter-rater reliability $r = .88-1$; [31]) to verify that participants did not meet criteria for a current psychotic disorder; and (iii) the

Wechsler Test of Adult Reading (WTAR; inter-rater reliability $r=.90-r=.94$; Wechsler, 2001) to provide an estimate of premorbid IQ and ensure adequate language abilities. Self-report instruments used in this study were the Beck Anxiety Inventory (BAI; Cronbach $\alpha = .69$; Beck et al., 1988), Beck Depression Inventory (BDI; Cronbach $\alpha=.89$; Beck et al., 1961), the McLean Screening Instrument for Borderline Personality Disorder (MSI-BPD; Cronbach $\alpha = .95$; Zanarini et al., 2003) and the Borderline Evaluation of Severity over Time (BEST, Cronbach $\alpha=.89$; [32]) to assess symptom severity. The Childhood Trauma Questionnaire (CTQ; Cronbach $\alpha=.91$ [33]) and Dissociative Experiences Scale-2 (DES-2; Cronbach $\alpha=.96$; [34]) were used to measure trauma and dissociation, respectively. Phenomenological assessments of hallucinations and delusions (where present) were obtained via an in-depth, semi-structured interview using the Questionnaire for Psychotic Experiences (QPE; Cronbach's $\alpha=.81$; [35]). To maximize consistency, participants were asked to reflect on their experiences of hallucinations and delusions within the previous two weeks.

Procedure

Interviews were conducted either in-person or via video conference and took approximately three hours to complete, with brief breaks as needed. This study was conducted in accordance with the Declaration of Helsinki and multi-site approval was granted by St. Vincent's Hospital, The Alfred Hospital, and Eastern Health Human Research Ethics Committees, Melbourne. Participants provided written, informed consent.

Analyses

An a priori power analysis for a multiple regression with three predictor variables with a small effect size of 0.15 and an error probability of .05 and a power of .8 required a total sample size of 55. Since the sample in the current study was 29 it is acknowledged that the study was underpowered. The mediation model was estimated using the IBM AMOS V27 module available as part of the IBM SPSS V27 suite of features. The model was estimated using Maximum Likelihood Estimation and - in accordance with methodology used by Preacher and Hayes [36] - bootstrapping was employed to calculate the indirect effect of the mediators. Childhood trauma

was measured as a raw score on the Childhood Trauma Questionnaire (CTQ). The maximum score on the questionnaire is 140. The mean score for those who did not hear voices was 55.92, and the mean score for those who heard voices was 62.87. There were no statistically significant differences between the groups in terms of their mean score on childhood trauma $p = .30$. Thus, of itself, trauma was not the real issue, rather the consequences of that trauma and how individuals coped with it is the issue. The reliability (Cronbach's alpha calculated for all materials in this paper; [37] for the CTQ was 0.87 well above the accepted benchmark of 0.70. Similarly, the reliabilities for the BDI and the BAI were satisfactory (anxiety = 0.92, depression = 0.96). The reliability for the DES was 0.81.

RESULTS

Data cleaning

The initial sample comprised 30 cases. An inspection of the Normal Q-Q Plot for dissociation identified one case as an outlier relative to all other cases (see supplementary material1 for plot diagram). That case was removed, leaving 15 voice hearers and 14 non-voice hearers. Missing values were assessed by conducting a Little's MCAR test [38] on each scale for which there were missing data. None of the p values were less than .05. Therefore, it was concluded that missing data for the scales in question were randomly distributed and <5% for all scales; hence missing values were replaced with the mean for each group.

Table 1: Demographics Percentages by Group.

Indicator	BPD-AVH (n = 14)	BPD-No AVH (n=15)	Group Comparisons. p value
Demographics			
Sex (Female)	100	100	1.00
Birthplace (Australia)	93	87	.35
Ethnicity (European)	87	93	.86
Language (English)	100	87	.06
Education (Secondary or Trade/Vocational)	64	93	.31
Unemployed	43	47	.72
Relationship (Never Married)	43	80	.89
Living (Independently)	64	67	.33

Table 1 above shows the demographics for the participant groups. There were no statistically significant difference between the groups on these demographic variables.

Model estimation

Prior to estimating the models, the variables were assessed for Univariate and Multivariate Normality; Skewness and Kurtosis were found to be within acceptable limits. Furthermore, an analysis was conducted to determine whether other comorbidities may have confounded the results that we have found. An analysis of the correlation between comorbidities as measured by the MINI instrument showed that only psychotic disorders were correlated with voice hearing. However, in terms of accepted bench marks the correlation coefficient (0.35) was deemed not to be material, nor was it statistically significant $p = .06$. Thus, comorbidities were ruled out as an influencer of the results that were found.

Table 2 shows the correlation matrix for the variables included in Model 1.

		Traum a	Anxiet y	Voice/N o voice	Dissocia tion
Trauma	Pearson correlation	1	.44*	.20	.24
Anxiety	Pearson correlation	.44*	1	.20	.49**
Voice/no voice	Pearson correlation	.20	.20	1	.41*
Dissociation	Pearson correlation	.24	.49**	.41*	1

The correlation matrix analysis showed that childhood trauma was not directly associated with voice hearing but was associated with anxiety. In turn, anxiety was associated with dissociation which was also associated with voice hearing.

The statistical significance of the direct effects for Model 1 are shown in Table 3. Table 3 shows that there were three statistically significant associations: childhood trauma and anxiety, anxiety and dissociation and dissociation and hearing voices. On this basis, hypotheses 1 and 4 were supported. Hypotheses 2 and 3 were not supported since not all the paths on the relevant pathways related to the hypotheses were not statistically significant. Specifically, in relation to hypothesis 2, the path from anxiety to hearing voices was not statistically

significant. For hypothesis 3, the pathway from childhood trauma to dissociation was not statistically significant. To test goodness of fit, the non-statistically significant paths were removed from the Model. This resulted in a Chi-Square statistic that was not statistically significant $X^2(3) = 0.45, p = .93$. Figure 3 show the standardized parameter estimates (alternatively referred to as the standardized beta coefficients) for Model 1.

Relationship	B	S.E.	t	p	95% CI Lower	95% CI Upper
Childhood Trauma ---> Anxiety	0.33	0.14	2.35	.03	0.06	0.60
Childhood Trauma ---> Dissociation	0.02	0.11	0.18	.86	-0.20	0.24
Anxiety ---> Dissociation	0.36	0.13	2.77	.01	0.11	0.61
Anxiety ---> Hearing Voices	-0.00	0.01	-0.25	.81	-0.02	0.02
Dissociation ---> Hearing Voices	0.02	0.01	2.10	.04	0.00	0.04
Childhood Trauma ---> Hearing Voices	0.00	0.01	0.40	.69	-0.02	0.02

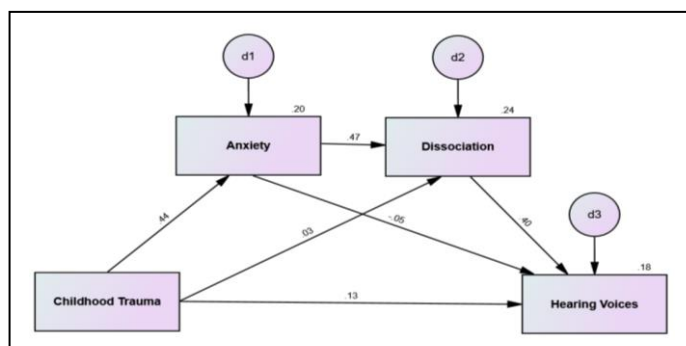


Figure 3: Standardised parameter estimates for Model 1.

The notable feature in Figure 3 was that the greatest weighting was associated with the pathway from childhood trauma to anxiety to dissociation to hearing voices. The R-square coefficients (shown at the top right of the figure boxes) suggest large effect sizes.

Table 4 shows the Standardized Direct, Indirect and Total Effects for Model 1. The total standardized effect of childhood trauma on voice hearing was 0.20. The proportion of the total effect, represented by the pathway that incorporates childhood trauma, anxiety, dissociation, and hearing voices, was 0.35 (a moderate effect size). The direct effect of childhood trauma on hearing voices was not statistically

significant. This was confirmed using a simple regression analysis in which childhood trauma was used as an estimator of hearing voices ($B = 0.01, t(27) = 1.07, p = .28$).

Path	Effect
Direct Effect: Childhood Trauma ---> Hearing Voices	0.13
Indirect Effects	
Childhood Trauma ---> Anxiety ---> Hearing Voices	-0.02
Childhood Trauma ---> Anxiety ---> Dissociation ---> Hearing Voices	0.08
Childhood Trauma ---> Dissociation ---> Hearing Voices	0.01
Total Indirect Effects	0.07
Total Effect	0.20
Mediated Effect: Proportion of Total	.35

Table 4 shows that the total standardized effect of childhood trauma on hearing voices was 0.20. The total indirect effect was 0.07 which was as a proportion .35 of the total standardized effects. Thus, the mediation effect was moderate. Table 5 shows the correlation matrix for Model 2.

		Trauma	Voice/no voice	Dissociation	Depression
Trauma	Pearson correlation	1	.20	.24	.30
Voice/no voice	Pearson correlation	.20	1	.41*	-.36
Dissociation	Pearson correlation	.24	.41*	1	-.02
Depression	Pearson correlation	.30	-.36	-.02	1

$n = 29, * p < .05, ** p < .01$

The correlation matrix for Model 2 suggests that there was no statistically significant direct association between childhood trauma and depression and depression and voice hearing. Thus hypothesis 5 was not supported. There was no statistically significant association between childhood trauma and dissociation and dissociation and hearing voices. Thus hypothesis 6 was not supported. There was one statistically significant direct effect which was between dissociation and voice hearing, similar to what was demonstrated in Model 1. The notable features of the correlation matrix were that depression is negatively correlated to hearing voices, opposite to what was hypothesised. Similarly, depression is negatively

correlated to Dissociation, also unexpected. As with Model 1, dissociation was positively correlated with hearing voices. There were no statistically significant indirect paths from childhood trauma to hearing voices evident within Model 2. Table 6 shows the unstandardized parameter estimates for Model 2. The only statistically significant result was the relationship between depression and hearing voices. Thus, there was no support for our Hypotheses 5, 6, 7 and 8 (the sign for the relationship depression and dissociation was negative as was the sign for the relationship between depression and hearing voices).

Relationship	B	S.E.	t	p	95% CI Lower	95% CI Upper
Childhood Trauma ---> Depression	0.27	0.17	1.59	.13	-0.06	0.60
Childhood Trauma ---> Dissociation	0.14	0.11	1.27	.22	-0.08	0.36
Depression ---> Dissociation	-0.06	0.11	-0.55	.59	-0.28	0.16
Depression ---> Hearing Voices	0.01	0.01	2.60	.02	-0.03	0.01
Dissociation ---> Hearing Voices	0.02	0.01	2.00	.06	0.00	0.04
Childhood Trauma ---> Hearing Voices	0.01	0.01	1.00	.33	-0.01	0.03

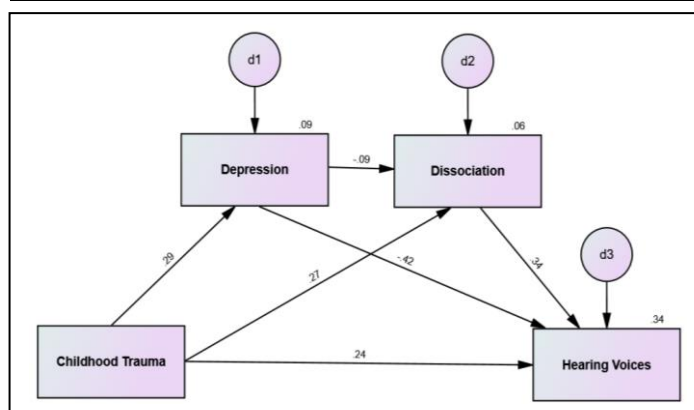


Figure 4: Standardised parameter estimates for Model 2.

The notable feature in Figure 4 is that the greatest weighting is associated with the pathway from childhood trauma to hearing voices. The R-square coefficients (shown at the top right of the figure boxes) suggest a small effect size for the pathway from childhood trauma to depression and the pathways from childhood trauma to dissociation. There was a medium effect size for the pathways from childhood trauma to hearing voices.

The standardized direct, indirect and total effects for Model 2 are provided in the supplementary material. In brief, none of the mediation pathways were statistically significant.

DISCUSSION

This study examined the relationship between childhood trauma and voice-hearing in people diagnosed with BPD, assessing possible contributions from co-occurring mood (anxiety and depression) and dissociation. This was done to investigate whether these features may play a role in the experience of AVH in people diagnosed with BPD. Two theoretical models were proposed to explore whether mood disorder might mediate the relationship proposed between trauma, dissociation, and AVH [12,26]. Model 1 investigated the contribution of anxiety while Model 2 examined the contribution of depression. The results of the present study revealed that Model 1 was a better fit, suggesting that the relationship between Childhood Trauma and AVH is mediated by co-occurring anxiety and dissociation.

Previous studies have reported a direct relationship between childhood trauma and AVH in people experiencing psychotic disorders [7,8]. In contrast, we found no direct relationship between these two features in a cohort of people diagnosed with BPD. Previous BPD studies have reported an association between AVH and (i) trauma [12,16], (ii) Dissociation [9,10,12], (iii) anxiety (Beatson, 1984; [4,5], and (iv) depression [5]. However, none of these earlier studies assessed these factors in a single model to examine their contribution to the experience of AVH in people diagnosed with BPD. It is unclear why the direct relationship between trauma and AVH was not significant in this sample, it is possible that this may have been the result of a small sample size in comparison to other literature.

None of our hypotheses in relation to Model 2 were supported. Furthermore, the signs for the coefficients of the relationship between depression and hearing voices, and depression and dissociation although were negative rather than positive. A possible explanation for the pattern of anxiety and depression findings reported here include peritraumatic responses, such as the emotional and physiological distress activated directly following a threatening situation, such as trauma [39]. This anxious state can arise repeatedly over the course of a day, creating heightened awareness and a sense of immediacy in

terms of dealing with the emotion. Individuals getting stuck in heightened fight/flight/freeze responses and frequently reliving the trauma. Thus, dissociative experiences may arise in response to these experiences of fear and anxiety rather than to depressed mood. While this is purely based on conjecture, it provides a basis for further research and debate. Future research focusing on peritraumatic responses may ascertain whether acute stressful experiences associated with anxiety do indeed trigger AVH experiences. This study focused on anxiety experienced over longer periods of time (e.g., one month, as measured by the BAI) as opposed to the more immediate stress responses, which would be needed to advance our understanding with regards to these relationships.

The statistically significant associations among features explored in Model 1 (i.e., childhood trauma, anxiety, dissociation, and AVH) are consistent with other AVH proneness models applied in studies of AVH in psychosis (Hardy, 2017) [7,10]. The findings from the present study accord with a broad literature showing that AVH are commonly associated with a history of childhood trauma. Maladaptive attempts to cope with this trauma and/or negative emotions experienced following the traumatic event(s) (such as anxiety, fear, low mood, low self-worth, loneliness etc.) become manifest (Hardy, 2017) [7,10,12,22]. Manifestations may include dissociated or disowned aspects of past traumatic experiences, ostensibly as a way of psychologically and emotionally surviving ongoing adversity.

Our findings mark an important step towards understanding how childhood trauma, anxiety, and dissociation may contribute to the experience of AVH in people with BPD. This has clinical implications for our understanding of AVH when they occur in the context of BPD. Firstly, it highlights the importance of assessing for past trauma, mood disorder and dissociative experiences when conceptualizing AVH and formulating appropriate and personalized treatments. Secondly, although trauma-focused treatment programs appear to help voice hearers [40], psychotherapeutic approaches could expand to include the reduction of dissociation as a treatment goal, perhaps in the context of managing anxiety associated with traumatic childhood experiences. In fact, a recent publication by Longden et al. [18] presented a manualised approach to treating AVH, namely, the Talking with Voices trial, which

indicated the feasibility of integrating a dissociation model of voice-hearing within a psychological intervention for people with psychosis. This treatment model could be adapted and trialled for people diagnosed with BPD and may also accumulate evidence to help answer the question of whether psychotic symptoms in BPD are best described as dissociative in nature and thus fit within the symptom spectrum of BPD, or alternatively whether they represent a comorbid dissociative disorder.

There are several limitations to this study including its cross-sectional design and small participant number. The small sample size is likely to have reduced the power of the study to identify significant interactions among the features thought to contribute to the expression of AVH in people with BPD. The cross-sectional design precluded the inference of causation despite the statistically significant correlations identified in this study. Further work is clearly required to establish whether these intriguing correlations with anxiety and dissociation may indeed help to explain the underlying relationship between childhood trauma and voice hearing. Further studies using longitudinal designs and larger sample sizes would optimize advancement of this research.

This study provides a rationale for further investigation of mechanisms underlying the phenomenon of AVH in people with BPD. The results of this study provide support for a model that has the potential to inform diagnostic formulation and therapeutic interventions that will assist with conceptualizing and treating distressing and unwanted hallucinatory experiences.

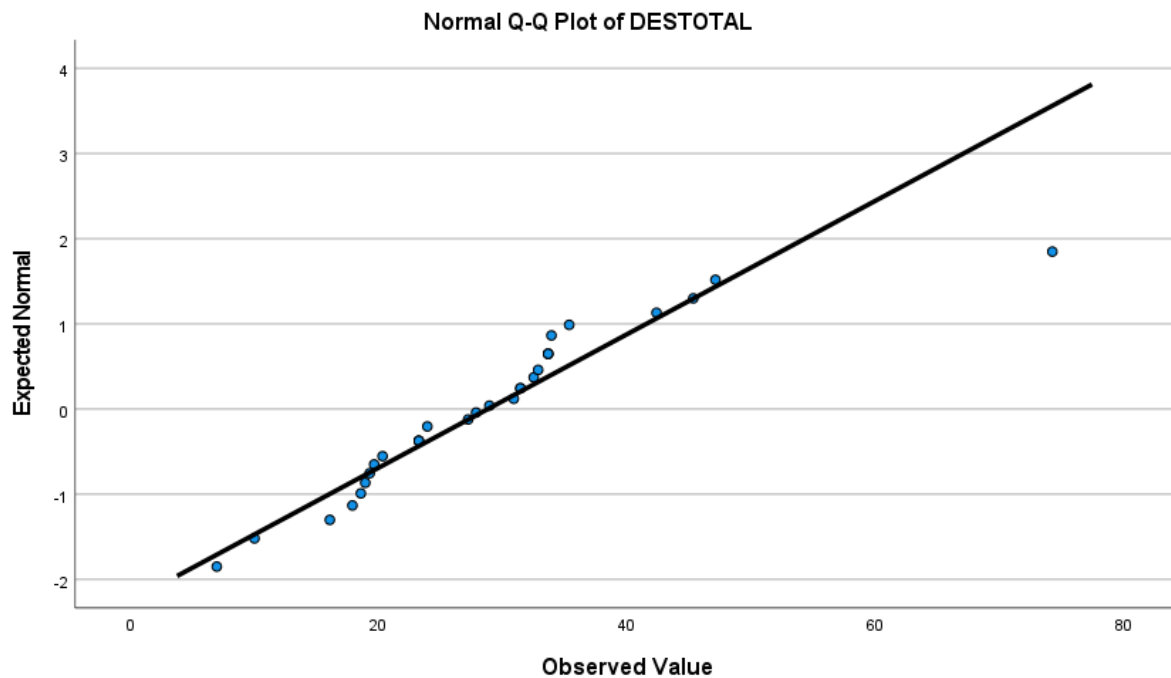
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Supplementary Material 1: Plot diagram of the outlier on the dissociation scale.



Supplementary Material 2: Standardised Direct Indirect Effects and Total Effects: Model 2.

Path	Effect
Direct Effect: Childhood Trauma ---> Hearing Voices	0.24
Indirect Effects	
Childhood Trauma --->Depression ---> Hearing Voices	-0.12
Childhood Trauma --->Depression---> Dissociation --- > Hearing Voices	-0.01
Childhood Trauma ---> Dissociation ---> Hearing Voices	0.09
Total Indirect Effects	-0.04
Total Effect	0.20
Mediated Effect: Proportion of Total	0.20