Auditory hallucinations and PTSD in ex-POWs

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Abstract

Literature has suggested that auditory hallucinations might be prevalent in the general population and could be linked to the experience of trauma. This prospective study examines the prevalence of auditory hallucinations in trauma survivors and its association with posttraumatic stress disorder (PTSD) symptoms, over time. Former prisoners-of-war (ex-POWs) from the 1973 Yom Kippur War (n=99) with and without PTSD and comparable veterans (n=103) were assessed twice, in 1991 (T1) and 2003 (T2) in regards to auditory hallucinations and PTSD symptoms. Findings indicated that ex-POWs who suffered from PTSD reported higher levels of auditory hallucinations at T2 as well as increased hallucinations over time, compared to ex-POWs without PTSD and combatants who did not endure captivity. The relation between PTSD and auditory hallucinations was unidirectional, so that the PTSD overall score at T1 predicted an increase in auditory hallucinations between T1 and T2, but not vice versa. Assessing the role of PTSD clusters in predicting hallucinations revealed that intrusion symptoms had a unique contribution, compared to avoidance and hyperarousal symptoms. The findings suggest that auditory hallucinations might be a consequence of the posttraumatic reaction among veterans.

Keywords: auditory hallucinations, PTSD, trauma, ex-POWs, war captivity

Introduction

To classify a hallucination is a complex and debated topic, resulting in numerous and inconsistent definitions. David (2004) defines hallucinations as an experience of the senses occurring in a wakeful state without corresponding external stimuli, giving a sense of reality that the person experiencing the hallucination does not feel able to control. There are also different types of hallucinations (e.g., auditory, visual, olfactory, tactile) (Aleman & Larøi, 2008). Historically, hallucinations have been acceptable in specific contexts, particularly if those reporting them were religious figures or artists, or members of particular cultures wherein hallucinations were considered an acceptable phenomena (Pierre, 2010). However, the idea that hallucinations are a positive symptom of psychosis has gained prominence among researchers and clinicians.

Lately, hallucinations as abnormal phenomena has become the subject of debate as studies have found that hallucinations are present in the general, non-clinical population (e.g., Moskowitz & Corstens, 2008). For example, a large-scale telephone survey assessing the non-clinical, general population in the United Kingdom, Germany and Italy found that, of the 13,057 participants, 38.7% reported some kind of hallucinatory experience (Ohayon, 2000). Assessing the prevalence of hallucinations in several USA communities, Tien (1991) found that 4.6% of the 13,622 participants reported hallucinations. Similarly, a comprehensive review of epidemiological studies reported that 5-28% of the general population have experienced auditory hallucinations (de Leede-Smith & Barkus, 2013). Finding such rates in the non-clinical population leads one to question the phenomenon of hallucinations, and whether it should only be considered as a symptom of psychosis.

The present study aims to expand the current understanding regarding hallucinations by assessing the link between hallucinations and trauma. Specifically, the present study will investigate auditory hallucinations (AH) in relation to trauma exposure and posttraumatic

reactions among former prisoners-of-war (ex-POWs) and combatants who did not endure captivity.

War Captivity and Hallucinations

Hallucinations are often observed in individuals who undergo highly stressful, traumatic events. This includes grief (Baethge, 2002), sensory deprivation (Leiderman, Mendelson, Wexler, & Solomon, 1958), solitary confinement (Shalev, 2008), captivity (Ohry, 2003), and following childhood trauma (Perona-Garcelán et al., 2014; Varese et al., 2012). Moreover, in a recent literature review of studies among general adult samples, life events, inclusive of trauma, were associated with a continuum of hearing voices (Beavan, Read, & Cartwright, 2011).

War captivity is a highly traumatic experience (e.g., Herman, 1992). Combat exposes individuals to the threat of death and injury, among other stressors (Nash, 2007). However, for POWs, combat is the first step in a protracted traumatic journey as they continue to be exposed to prolonged traumatic experiences. War captivity often includes harsh physical and psychological abuse, torture, deprivation of basic needs (i.e., food, water, sleep), and ongoing humiliation by captors (Herman, 1992). Surviving such conditions may lead to psychopathology once the POW returns home. Research has indicated that ex-POWs remain at a high-risk for psychological impediments, compared to combatants, even decades after repatriation (Rintamaki, Weaver, Elbaum, Klama, & Miskevics, 2009). One might wonder whether the same pattern is applicable with regard to hallucinations.

While the majority of the literature refers to hallucinations in relation to childhood trauma, there is a dearth in investigations of hallucinations among adults who have undergone traumatic events. Nevertheless, studies that investigated this subject indicated a link between war captivity and hallucinations. As early as 1953, a study of US repatriated POWs reported the use of imagination or fantasy to relieve tension (Strassman, Thaler, & Schein, 1956).

PTSD and Hallucinations

The psychological reaction to trauma, i.e., posttraumatic stress disorder (PTSD), might also be related to AH (Morrison, Frame, & Larkin, 2003). PTSD is the most conspicuous psychopathological outcome of trauma, comprising of four symptom clusters: intrusion (e.g., flashbacks), avoidance (e.g., numbness), hyperarousal (e.g., startle reaction) and negative alterations in cognitions and mood (e.g., negative beliefs) (APA, 2013). Moreover, previous studies have revealed a link between PTSD and hallucinations, with a higher rate of hallucinations among traumatized combatants with PTSD, compared to combatants without PTSD and the general population (e.g., Brewin & Patel, 2010).

A questions arises whether the posttraumatic reaction, rather than trauma exposure itself, is linked to hallucinations. Although a previous study pursued this inquiry, and indicated the central role of PTSD (Brewin & Patel, 2010), it was conducted among combatants and did not include ex-POWs. As war captivity involves multiple and unique stressors, aiming to break down captives' basic defences (Herman, 1992), it might have a more prominent effect on the development of hallucinations. The present study aims to elucidate this question.

An important query worth investigation concerns the change in AH over time. Literature indicates that PTSD tends to fluctuate over time. Hence, one may wonder whether there is a fluctuation in hallucinations as well, and whether different patterns are associated with diverse trauma exposures (i.e., war captivity versus combat) or posttraumatic reaction (i.e., PTSD, or lack thereof). Previous studies wherein trauma exposure, PTSD and hallucinations were assessed were cross-sectional, and thus could not address this issue. Consequently, and in an attempt to address the above lacunae, the current longitudinal study was devised.

The Association between PTSD and Hallucinations

PTSD has been implicated in the genesis of AH (Anketell et al., 2010). Possibly, PTSD could predict hallucinations. Re-experiencing of the traumatic event, which is part of PTSD, might deplete the individuals' resources and increase their vulnerability for faulty reality testing, manifested as hallucinations. Additionally, PTSD could lead to hallucinations as an ego-dystonic defense mechanism, activated in the processing of the traumatic event (Longden, Madill, & Waterman, 2012). Thus, hallucinations emerge as a reaction to one's incapacity to integrate their traumatic experience. Accordingly, hallucinations are themselves the re-experiencing symptom.

Conversely, hallucinations might lead to the development or intensification of PTSD over time. Hallucinations could be experienced as traumatic, shattering the person's basic beliefs regarding the self, the world, and others (Davidson & Strauss, 1992). Therefore, hallucinations may be a precursor of PTSD and precipitate its occurrence.

These possible trajectories were taken into consideration in a review addressing the association between trauma and psychosis (Morrison et al., 2003). However, while a link between past trauma and hallucinations has been indicated, to the best of our knowledge the direction and nature of association has not been conclusive.

The Contribution of PTSD Clusters in Hallucinations

Consideration of PTSD symptom clusters may yield a more nuanced understanding of the relation between posttraumatic reactions and hallucinations. Avoidance symptoms, for instance, often include the avoidance of close relationships, leading to social isolation (APA, 2013). Consequently, there may not be an opportunity to test one's perspective of reality with others, as well as a lack of stimulation, interaction and support. This may result in hallucinations as the traumatized individual turns to his or her internal world (Cresswell, Kuipers, & Power, 1992).

Hyperarousal symptoms, manifested in physiological hyper-activation, are associated with a poor prognosis in persons with mental health struggles (Straube & Öhman, 1990). Moreover, overgeneralization of vigilance may be detrimental to the individual's ability to assess probabilities of threat, and might increase susceptibility for reality testing deficits.

Intrusion symptoms could act as a chronic stressor, worsening mental health (Mueser, Rosenberg, Goodman, & Trumbetta, 2002). Traumatized individuals who re-experience traumatic events as intrusive memories, nightmares or flashbacks may be at an increased vulnerability for flawed reality testing. Furthermore, extreme re-experiencing symptoms may take on a delusional intensity in persons prone to psychotic symptoms (Sautter et al., 1999).

The theoretical model suggested by Morrison (2001) implements a cognitive approach, offering that PTSD intrusion symptoms, in particular, might have a unique role in the development of hallucinations. Positive psychotic symptoms, such as hallucinations, can be conceptualized as intrusions and, moreover, if such an intrusion is interpreted as culturally unacceptable, this may cause elevated distress and disability (Morrison, 2001; Morrison et al., 2003). The nature of the intrusive symptoms' interpretation is affected by a faulty sense of self and social knowledge, which may be a consequence of the traumatic experience (Morrison, 2001). For instance, exposure to torture and abuse during captivity may lead ex-POWs to believe that others cannot be trusted, which would make paranoid interpretations of equivocal events more likely.

Three questions were then addressed in the current study, each relying on measurements taken at two time points:

1) Are there differences between ex-POWs who suffer from PTSD, ex-POWs who do not suffer from PTSD, and control veterans in levels of AH, cross-sectionally and over time?

2) Are the relations between PTSD symptoms (PTSS) and AH bidirectional?

3) Is there a unique contribution of specific PTSD clusters in predicting AH?

Method

Procedure and Participants

The present study is part of a longitudinal study on the psychological implications of war and captivity for combatants and ex-POWs (for details see Solomon, Horesh, Ein-Dor, & Ohry, 2012). The current sample consisted of two groups of Israeli male veterans from the 1973 Yom Kippur War (YKW), and used data collected at two time points: 1991 (T1) and 2003 (T2).

Veterans were contacted by telephone and asked to take part in the study. Questionnaires were administered in their homes or a location of their choice. Before filling out the questionnaires, participants signed an informed consent. This study was approved by the Tel Aviv University Ethics Committee.

Ex-POWs. According to Israel's Ministry of Defense, 240 combatants were captured during the YKW and held in Egypt or Syria. Reports by ex-POWs indicate that most were held in solitary confinement in unhygienic conditions, handcuffed, blindfolded and subjected to deliberate and systematic abuse. Torture included beatings, electric shock, sexual abuse, burns and deprivation of medical treatment as well as food and water. They were also subjected to verbal abuse, mock executions, threats (of death, mutilation, killing loved ones), and demoralizing misinformation. Length of captivity ranged from 1.5 to 8 months. The ex-POW group included 164 participants in 1991 (T1) and 144 in 2003 (T2). Data analyzed here are based on the 99 ex-POWs who participated in both waves of measurement.

Controls. A control group of land force combat veterans from the YKW, sampled from Israeli Defense Forces computerized data banks, were matched to ex-POWs in age, ethnic background, marital status, education and military background. There were 185

participants in 1991 (T1) and 143 in 2003 (T2). The present study included the 103 controls who participated in both waves of measurement.

Examination of the two groups' socio-demographic characteristics during the war revealed that age, ethnic background, marital status, and educational background were similar. The mean age of the subjects during the war was 22.

Background Variables

At the time of the study, ex-POWs and controls did not differ in the socio-demographic variables of age (M=52.91, SD=5.20, for ex-POWs; M=51.88, SD=3.42, for controls), length of marriage (M=28.97, SD=9.11, for ex-POWs; M=28.45, SD=7.29, for controls), years of schooling (M=13.90, SD=3.81, for ex-POWs; M=14.12, SD=3.07, for controls), religiosity (62.89% of ex-POWs and 60.19% of controls were secular), or number of children (M=3.21, SD=1.04, for ex-POWS; M=3.21, SD=1.28, for controls).

Measures

PTSD Inventory (PTSD-I; Solomon et al., 1993). The veterans' PTSS were assessed via the PTSD-I, a well-validated 17-item self-report questionnaire. The items on the PTSD-I correspond to the DSM-IV-TR diagnosis for PTSD (APA, 2000). Respondents rated symptoms experienced in the previous month on a scale ranging from (0) not at all to (4) almost always. The number of positively endorsed symptoms was calculated by the number of items with responses of '3' or '4.' Scores were dichotomized (PTSD, no PTSD) using DSM-IV-TR PTSD criteria (APA, 2000). PTSD was assigned if at least one intrusive, three avoidant, and two arousal symptoms were endorsed. The PTSD-I has proven psychometric properties and convergent validity (e.g., Solomon et al., 1993). In the present study, Cronbach's alphas were .95, .96, at T1 and T2 respectively.

SCL-90-R (Derogatis, 1977) measured AH using item 16. Using a 5-point scale, from (0) not at all to (4) extremely, the respondents rated the degree they "hears voices that others

do not hear" during the preceding two weeks. The score reflects the respondent's level of AH, with higher scores reflecting greater AH. Mean scores of AH were used in the analyses.

Results

Inter-correlations between the main study measures are presented in Table 1.

AH among Ex-POWs and Controls as a Function of PTSD

To examine the associations between war captivity exposure, PTSD and AH, ex-POWs and controls were divided according to their PTSD status at T1 and T2. Two ex-POWs did not have data regarding their PTSD status, so were omitted. Seven controls (6.7%) reported PTSD at T1 or T2, or both. Due to the small number of controls with PTSD, they were omitted, leaving 3 groups: ex-POWs with PTSD (at T1, T2, or both) (n=61, 31.6%), ex-POWs without PTSD (n=36, 18.7%) and controls without PTSD (n=96, 49.7%).

Prevalence of AH at T1 and T2 among the groups is presented in Table 2. Ex-POWs with PTSD reported higher frequencies of AH compared to ex-POWs without PTSD and controls.

Next, we conducted repeated measure ANOVAs to examine the difference between the groups, in relation to changes in AH over time. Time was treated as a within subject repeated factor. The groups were treated as a between subjects factor. The dependent variable was the degree of AH at T1 and T2 (Table 3).

Analysis revealed significant effect for time X group interaction, $F(2,190)=12.88$, $p<.001$, $\eta^2=.12$. To understand the interaction, we conducted simple effects analyses. The analyses revealed that while the effect for grouping was non-significant at T1, $F(2,190)=1.44$, $p=.24$, $\eta^2=.24$, it was significant at T2, $F(2,190)=21.82$, $p<.001$, $\eta^2=.19$, with ex-POWs with PTSD reporting higher levels of AH, compared to ex-POWs without PTSD and controls. The analyses also revealed that while the effect for time was non-significant among controls, $F(1,95)=2.16$, $p=.15$, $\eta^2=.02$, and ex-POWs without PTSD, $F(1,35)=.00$, $p=1.00$, $\eta^2=.00$, it

was significant among ex-POWs with PTSD, $F(1,60)=12.72$, $p<.001$, $\eta^2=.18$, indicating an increase in AH from T1 to T2 only among ex-POWs with PTSD.

Associations between AH and PTSS Over Time

Bidirectional relations between AH and PTSS over time were examined using autoregressive cross-lagged modeling (ARCL; e.g., Anderson, 1960). This analysis provides an indicator of temporal precedence in the absence of an experimental design. ARCL allows for simultaneous assessment, enabling the examination of whether an earlier measurement of PTSS predicts later measures of AH and vice versa.

This model was analyzed with AMOS statistics, Version 22. A model is judged as fitting well if the comparative fit index (CFI), that considers sample sizes, normed-fit index (NFI) and the Tucker-Lewis index (TLI) are greater than .95 and are an adequate fit if these indices are greater than .9 and root mean square error of approximation (RMSEA) is equal or lower than .05. We computed the chi square test, but since it is sensitive to sample size (e.g., Kline, 1998), we used the ratio of chi square to degrees of freedom. Values between 1 and 5 indicated a satisfactory fit between the theoretical model and empirical data, a more severe cutoff of 3 is ideal (Kline, 1998).

Fit indices indicated that the theoretical model, $\chi^2(1)=31.52$, CFI=.78, NFI=.79, TLI=-.27, RMSEA=.40, was not a good representation of the data. The next step was to estimate a simpler and parsimonious model, containing only the significant paths found. Fit indices of the nested model were not compared to the general model, as the degrees of freedom of the models were equal. Fit indices of the simpler model indicated that the model, $\chi^2(1)=.54$, $p=.46$, $\chi^2/df=.54$, CFI=1.00, NFI=.99, TLI=1.00, RMSEA=.00, was an excellent representation of the data. Hence, we proceeded with the more parsimonious simple model and the same effects were found.

Figure 1 displays the standardized coefficients and significant paths for the nested model. The analysis revealed high stability of AH and PTSS. That is, those with high levels of AH or PTSS at T1 tended to have high levels of AH or PTSS at T2. Importantly, the analysis revealed that PTSS at T1 predicted an increase in AH between T1 and T2, beyond the stability of AH, and not the vice versa.

The Contribution of PTSD Clusters in Predicting AH

Linear regression using blocks were conducted to investigate the unique contribution of PTSD clusters in predicting AH. Blocks were selected based on the variables' commonalities as well as chronological order. The first block included study group (ex-POWs vs. controls) as well as AH at T1. The second block included the three PTSD clusters of intrusion, avoidance and hyperarousal at T1. Regression beta coefficients are presented in Table 4.

Analysis revealed that the final model explained 34.0% of the variance of AH at T2, $F(5,196)=20.57$, p<.001. The variables that had significant contribution in predicting AH at T2 were the study group, the AH at T1, and the level of intrusion symptoms at T1. Avoidance and hyperarousal symptoms did not have a significant contribution in predicting AH at T2.

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Discussion

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The present study examined the relationship between war captivity exposure, PTSD and AH. The findings indicated that AH were higher among ex-POWs with PTSD at T2, compared to ex-POWs or combatants without PTSD. Moreover, AH increased over time only in this group. Unidirectional, associations between PTSS and AH over time were found, with PTSS at T1 predicting AH at T2, and not vice versa. Lastly, it was found that PTSD intrusion symptoms had a unique contribution in predicting AH, beyond the effects of hyperarousal and avoidance symptoms, which were non-significant.

Enduring trauma in adulthood can leave lasting posttraumatic symptoms that may continue to impact survivors' lives long after the trauma has ended. The current study's results, are in-line with earlier studies reporting a link between PTSD and hallucinations (Brewin & Patel, 2010; Hamner, Frueh, Ulmer, & Arana, 1999), and demonstrate the importance of considering hallucinations in diagnosing and treating traumatized individuals. The present findings indicated that PTSD might have a detrimental effect on the individual, implicated in high levels of AH.

Notably, ex-POWs with PTSD did not differ in AH levels from the other groups at T1, but only at T2, with an increase in hallucinations over time only present among traumatized ex-POWs. Thus, the findings reveal that hallucinations might be more common among traumatized individuals during specific points over the life span.

One explanation may be that the findings reflect a general trend of deterioration among traumatized individuals as they age (e.g., Gagnon & Hersen, 2000). According to theory, the combination of an increased tendency for retrospection and stressful events during old age (e.g., functional decline, loss of loved ones) could lead to the reactivation of past trauma and a deterioration in mental health (e.g., Gagnon & Hersen, 2000). Similarly, Buffum and Wolfe (1995) stress that the multiple losses characterizing old age could lead to the resurfacing of trauma and increase the vulnerability to psychopathology, which might be revealed in damaged reality testing and increased proneness to hallucinations.

Exploring the relation between hallucinations and PTSS over time revealed a unidirectional association, with PTSS predicting an increase in hallucinations. The present findings underscore the important role of PTSD in the amplification of hallucinations among survivors of trauma in adulthood. A few explanations may be suggested.

First, dissociation, which may be part of the posttraumatic reaction (APA, 2013), could impede an objective evaluation of the external world, facilitating hallucinations (Allen,

Coyne, & Console, 1997). Particularly, dissociative detachment may weaken an individual's connection to the surroundings, causing them to turn inwards, and thereby vulnerable to psychosis-like symptoms, including hallucinations (Allen et al., 1997). Hallucinations and dissociation could have a similar function of enabling an emotional 'escape' from the potentially unbearable psychological pain resulting from trauma (Rudegeair & Farrelly, 2008). Meanwhile, hallucinations could be part of dissociated self-states, or dissociated self-other relationships (Longden et al., 2012), and might reflect efforts to cope with the traumatic pain (Longden, 2013).

It has been suggested that those undergoing extreme trauma, such as POWs, might rely on hallucinations, such as the voices of a spouse, as a way to ease emotional pain and find comfort during the trauma (Stein, Crompton, Ohry, & Solomon, 2016). Since ex-POWs may find themselves isolated due to their experiences (Stein & Tuval-Mashiach, 2015), such self-nurturing could be relevant beyond the traumatic exposure as the individual continues to adopt this method of coping in an attempt to grapple with the past trauma.

In examining the role of PTSD symptom clusters, the current study found that intrusion symptoms had a unique contribution in predicting hallucinations, while other clusters were non-significant. This implies that intrusion symptoms could be a key factor in increasing the trauma survivor's susceptibility to hallucinations. The first explanation views intrusion symptoms as a chronic stressor. According to this view, suffering from intrusive symptoms acts as a burden, hindering one's mental health and reality testing (Mueser et al., 2002). This, might be particularly relevant for intrusions regarding a re-experiencing of the past trauma, such as flashbacks, which reflect a focal damage in reality testing (APA, 2013) and therefore might increase the susceptibility for hallucinations.

The second explanation offers that hallucinations in and of themselves reflect intrusion symptoms, and if such symptoms are perceived as culturally unacceptable, this may

increase the distress experienced by the trauma survivor (Morrison, 2001). As a result, the distress of the trauma is compounded as the individual feels he or she is acting in an undesirable manner, creating a cyclical experience of increasing distress and hallucinations.

The third explanation suggests that intrusion symptoms might foster hallucinations as a part of reliving the traumatic past. Re-experienced traumatic memories often give rise to the original emotions, sensations and responses experienced during the traumatic event (e.g., Van der Kolk, 1994). Additionally, as mentioned, there is evidence regarding the use of hallucinations as a coping strategy during captivity (Stein et al., 2016). Thus, one might suggest that the re-emergence of traumatic memories of captivity, as part of intrusion symptoms, could reactivate the ex-POWs' original defensive response of AH.

Lastly, the fourth explanation offers that intrusion symptoms are linked to hallucinations indirectly, through the implications of common correlates of PTSD, such as substance misuse (Mueser et al., 2002). Individuals who suffer from severe intrusion symptoms might rely on substances as a way to self-medicate (Rheingold, Acierno, & Resnick, 2004). Use of alcohol and drugs may help traumatized individuals block or decrease the intensity of intrusive traumatic symptoms (Rheingold et al., 2004), which, consequently, could increase the risk for psychotic-like symptoms, such as AH.

This preliminary study has several limitations that are important to acknowledge. Auditory hallucinations were measured in a general way, without reporting or assessing the content of the hallucinations. In future, it would be important to further investigate the content of the hallucinations and how they contribute to posttraumatic reactions. Furthermore, future research should assess the interpretation of hallucinations by trauma survivors and whether they experience it as an ego-syntonic or ego-dystonic phenomenon. In addition, it would be beneficial to determine the role of other types of hallucinations, besides auditory, in the context of varying experiences of trauma. Lastly, we did not have information

regarding PTSD and AH before or immediately after exposure to war captivity, but only years after the traumatic event ended. This limited our ability to illuminate the relations between war captivity exposure, PTSD and hallucinations, soon after the trauma exposure, which might shape the present results. Hence, one should be cautious while interpreting the present results and not negate the possibility that both PTSD and hallucinations might be discretely trauma-based.

While this study focused on ex-POW and combatant populations, it further adds to the literature on trauma. Moreover, although preliminary, the present results suggest that trauma exposure, and more importantly the posttraumatic reaction, might very well be a risk for hallucinations in cases of adult trauma, and not only following childhood traumas.The findings may have clinical implications in directing therapists towards the importance of the assessment of hallucinations among traumatized populations, and suggest the need for considering a broader notion of posttraumatic reaction wherein hallucinations are incorporated.

References

Aleman, A., & Larøi, F. (2008). The phenomenology of hallucinations. In A. Aleman & F. Larøi (Eds.), *Hallucinations: The science of idiosyncratic perception* (pp. 25-46). Washington, DC: APA.

Allen, J. G., Coyne, L., & Console, D. A. (1997). Dissociative detachment relates to psychotic symptoms and personality decompensation. *Comprehensive psychiatry, 38*(6), 327-334.

Anderson, T. W. (1960). Some stochastic process models for intelligence test scores. *Mathematical Methods in the Social Sciences*, 205-220.

Anketell, C., Dorahy, M. J., Shannon, M., Elder, R., Hamilton, G., Corry, M., . . . O'Rawe, B. (2010). An exploratory analysis of voice hearing in chronic PTSD: Potential associated mechanisms. *Journal of Trauma & Dissociation, 11*(1), 93-107.

American Psychiatric Association [APA]. (2000). *Diagnostic and statistical manual of mental health disorders (DSM*-IV-TR). Washington, DC: APA.

APA. (2013). *Diagnostic and statistical manual of mental health disorders* (DSM-5). Washington, DC: APA.

Baethge, C. (2002). Grief Hallucinations: True or Pseudo? Serious or Not? *Psychopathology, 35*(5), 296-302.

Beavan, V., Read, J., & Cartwright, C. (2011). The prevalence of voice-hearers in the general population: a literature review. *Journal of Mental Health, 20*(3), 281-292.

Brewin, C. R., & Patel, T. (2010). Auditory pseudohallucinations in United Kingdom war veterans and civilians with posttraumatic stress disorder. *The Journal of Clinical Psychiatry, 71*(4), 419-425.

Buffum, M. D., & Wolfe, N. S. (1995). Posttraumatic stress disorder and the World War II

    Veteran: Elderly patients who were in combat or were prisoners of war may have

    special health care needs that may not be obvious. *Geriatric Nursing, 16*(6), 264-270.

Cresswell, C. M., Kuipers, L., & Power, M. (1992). Social networks and support in long-term

    psychiatric patients. *Psychological Medicine, 22*(4), 1019-1026.

David, A. S. (2004). The cognitive neuropsychiatry of auditory verbal hallucinations: an

    overview. *Cognitive Neuropsychiatry, 9*(1-2), 107-123.

Davidson, L., & Strauss, J. S. (1992). Sense of self in recovery from severe mental illness.

    British Journal of Medical Psychology, 65(2), 131-145.

de Leede-Smith, S., & Barkus, E. (2013). A comprehensive review of auditory verbal

    hallucinations: Lifetime prevalence, correlates and mechanisms in healthy and clinical

    individuals. *Frontiers in Human Neuroscience, 7*, 1-25.

Derogatis, L. R. (1977). *The SCL-90 Manual I: Scoring, administration and procedures for

    the SCL-90*. Baltimore: Johns Hopkins University School of Medicine, Clinical

    Psychometrics Unit.

Gagnon, M., & Hersen, M. (2000). Unresolved childhood sexual abuse and older adults:

    Late-life vulnerabilities. *Journal of Clinical Geropsychology, 6*(3), 187-198.

Hamner, M. B., Frueh, B. C., Ulmer, H. G., & Arana, G. W. (1999). Psychotic features and

    illness severity in combat veterans with chronic posttraumatic stress disorder.

    Biological Psychiatry, 45(7), 846-852.

Herman, J. (1992). *Trauma and recovery*. New York: Basic Books.

Kline, R. B. (1998). Software review: Software programs for structural equation modeling:

    Amos, EQS, and LISREL. *Journal of psychoeducational assessment, 16*, 343-364.

Leiderman, H., Mendelson, J. H., Wexler, D., & Solomon, P. (1958). Sensory deprivation:

    Clinical aspects. *AMA Archives of Internal Medicine, 101*(2), 389-396.

Longden, E. (2013). *Learning from the voices in my head.* Paper presented at the TED

    Conferences, Monterey, CA.

Longden, E., Madill, A., & Waterman, M. G. (2012). Dissociation, trauma, and the role of

    lived experience: toward a new conceptualization of voice hearing. *Psychological*

    bulletin, 138(1), 28-76.

Morrison, A. P. (2001). The interpretation of intrusions in psychosis: an integrative cognitive

    approach to hallucinations and delusions. *Behavioural and Cognitive Psychotherapy,*

    29(3), 257-276.

Morrison, A. P., Frame, L., & Larkin, W. (2003). Relationships between trauma and

    psychosis: a review and integration. *British Journal of Clinical Psychology, 42*(4),

    331-353.

Moskowitz, A., & Corstens, D. (2008). Auditory hallucinations: Psychotic symptom or

    dissociative experience? *Journal of Psychological Trauma, 6*(2-3), 35-63.

Mueser, K. T., Rosenberg, S. D., Goodman, L. A., & Trumbetta, S. L. (2002). Trauma,

    PTSD, and the course of severe mental illness: an interactive model. *Schizophrenia*

    Research, 53(1), 123-143.

Nash, W. P. (2007). The stressors of war. In C. R. Figely & W. P. Nash (Eds.), *Combat stress*

    injury: Theory, research, and management. New York: Routledge.

Ohayon, M. M. (2000). Prevalence of hallucinations and their pathological associations in the

    general population. *Psychiatry Research, 97*(2), 153-164.

Ohry, A. (2003). Extracampine hallucinations. *The Lancet, 361*, 1479.

Perona-Garcelán, S., García-Montes, J. M., Rodríguez-Testal, J. F., López-Jiménez, A. M.,

    Ruiz-Veguilla, M., Ductor-Recuerda, M. J., . . . Pérez-Álvarez, M. (2014).

    Relationship between childhood trauma, mindfulness, and dissociation in subjects

with and without hallucination proneness. *Journal of Trauma & Dissociation, 15*(1), 35-51.

Pierre, J. M. (2010). Hallucinations in nonpsychotic disorders: Toward a differential diagnosis of "Hearing Voices". *Harvard Review of Psychiatry, 18*(1), 22-35.

Rheingold, A. A., Acierno, R., & Resnick, H. S. (2004). Trauma, posttraumatic stress disorder, and health risk behaviors. In P. P. Schnurr & B. L. Green (Eds.), *Trauma and health: Physical health consequences of exposure to extreme stress* (pp. 217-243). Washington, DC: APA.

Rintamaki, L. S., Weaver, F. M., Elbaum, P. L., Klama, E. N., & Miskevics, S. A. (2009). Persistence of traumatic memories in World War II prisoners of war. *Journal of the American Geriatrics Society, 57*(12), 2257-2262.

Rudegeair, T., & Farrelly, S. (2008). Pharmacotherapy in the collaborative treatment of trauma-induced dissociation and psychosis. In A. Moskowitz, I. Schäfer & M. J. Dorahy (Eds.), *Psychosis, trauma and dissociation: Emerging perspectives on severe psychopathology* (pp. 307-318). West Sussex, UK: John Wiley & Sons, Ltd.

Sautter, F. J., Brailey, K., Uddo, M. M., Hamilton, M. F., Beard, M. G., & Borges, A. H. (1999). PTSD and comorbid psychotic disorder: comparison with veterans diagnosed with PTSD or psychotic disorder. *Journal of Traumatic Stress, 12*(1), 73-88.

Shalev, S. (2008). A sourcebook on solitary confinement. In M. C. F. Criminology (Ed.), *London School of Economics and Political Science*. London.

Solomon, Z., Benbenishty, R., Neria, Y., Abramowitz, M., Ginzburg, K., & Ohry, A. (1993). Assessment of PTSD: Validation of the revised PTSD Inventory. *The Israel Journal of Psychiatry and Related Sciences, 30*(2), 110-115.

Solomon, Z., Horesh, D., Ein-Dor, T., & Ohry, A. (2012). Predictors of PTSD trajectories following captivity: A 35-year longitudinal study. *Psychiatry Research, 199*(3), 188-194.

Stein, J. Y., Crompton, L., Ohry, A., & Solomon, Z. (2016). Attachment in detachment: The positive role of caregivers in POWs' dissociative hallucinations. *Journal of Trauma & Dissociation, 17*(2), 186-198.

Stein, J. Y., & Tuval-Mashiach, R. (2015). Loneliness and isolation in life-stories of Israeli veterans of combat and captivity. *Psychological Trauma: Theory, Research, Practice, and Policy, 7*(2), 122-130.

Strassman, H. D., Thaler, M. B., & Schein, E. H. (1956). A prisoner of war syndrome: Apathy as a reaction to severe stress. *American Journal of Psychiatry, 112*(12), 998-1003.

Straube, E., & Öhman, A. (1990). Functional Role of the Different Autonomic Nervous System Activity Patterns Found in Schizophrenia-A New Model. In E. R. Straube & K. Hahlweg (Eds.), *Schizophrenia: Concepts, Vulnerability, and Intervention* (pp. 135-157). Berlin: Springer-Verlag.

Tien, A. Y. (1991). Distribution of hallucinations in the population. *Social psychiatry and psychiatric epidemiology, 26*(6), 287-292.

Van der Kolk, B. A. (1994). The body keeps the score: Memory and the evolving psychobiology of posttraumatic stress. *Harvard Review of Psychiatry, 1*(5), 253-265.

Varese, F., Smeets, F., Drukker, M., Lieverse, R., Lataster, T., Viechtbauer, W., . . . Bentall, R. P. (2012). Childhood adversities increase the risk of psychosis: a meta-analysis of patient-control, prospective-and cross-sectional cohort studies. *Schizophrenia Bulletin, 38*(4), 661-671.

Table 1

Inter-correlations Between Main Study Measures

Measure	1	2	3	4	5	6	7	8	9	10
1. Auditory Hallucinations(T1)	1	.46***	.34***	.17*	.31***	.24**	.23**	.11	.30***	.14*
2. Auditory Hallucinations(T2)		1	.39***	.50***	.43***	.51***	.21**	.44***	.32***	.40***
3. PTSS(T1)			1	.45***	.83***	.46***	.76***	.35***	.86***	.42***
4. PTSS(T2)				1	.42***	.88***	.28***	.91***	.40***	.91***
5. Intrusion(T1)					1	.49***	.47***	.30***	.59***	.35***
6. Intrusion(T2)						1	.24**	.68***	.40***	.74***
7. Avoidance(T1)							1	.27***	.47***	.25***
8. Avoidance(T2)								1	.27***	.74***
9. Hyperarousal(T1)									1	.41***
10. Hyperarousal(T2)										1
M(SD)	.16(.66)	.29(.83)	2.32(3.24)	5.88(5.68)	.77(1.25)	1.51(1.87)	.63(1.19)	2.15(2.40)	.93(1.51)	2.22(2.03)

Note. PTSS=posttraumatic stress symptoms. *=$p<.05$; **=$p<.01$; ***=$p<.001$

Table 2

Prevalence of Auditory Hallucinations Among ex-POWs With PTSD, ex-POWs Without PTSD and Controls

Variable		Ex-POWs with PTSD (*n*=61)		Ex-POWs without PTSD (*n=36*)		Controls without PTSD (*n=96*)	
		n	*%*	*n*	*%*	*n*	*%*
Auditory	0	55	90.2	35	97.2	91	94.8
Hallucinations (T1)	1	2	3.3	2	2.8	2	2.1
	2	1	1.6	0	0	1	1.0
	3	1	1.6	0	0	2	2.1
	4	2	3.3	0	0	0	0
Auditory	0	39	63.9	34	94.4	93	96.9
Hallucinations (T2)	1	9	14.8	2	5.6	3	3.1
	2	5	8.2	0	0	0	0
	3	4	6.6	0	0	0	0
	4	4	6.6	0	0	0	0

Note. Ex-POWs= former prisoners of war, PTSD=posttraumatic stress disorder; 0=not at all; 1=a little bit; 2=moderately; 3=quite a bit; 4=extremely

Table 3

Means and SDs of Auditory Hallucinations Among ex-POWs With PTSD, ex-POWs
Without PTSD and Controls

Variable	Ex-POWs with PTSD (*n*=61)		Ex-POWs without PTSD (*n*=36)		Controls without PTSD (*n*=96)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Auditory Hallucinations(T1)	.25	.85	.06	.33	.10	.49
Auditory Hallucinations(T2)	.77	1.24	.06	.23	.03	.17

Note. Ex-POWs=former prisoners of war, PTSD=posttraumatic stress disorder

Table 4

Regression Beta Coefficients Predicting Auditory Hallucinations at T2 (n=202).

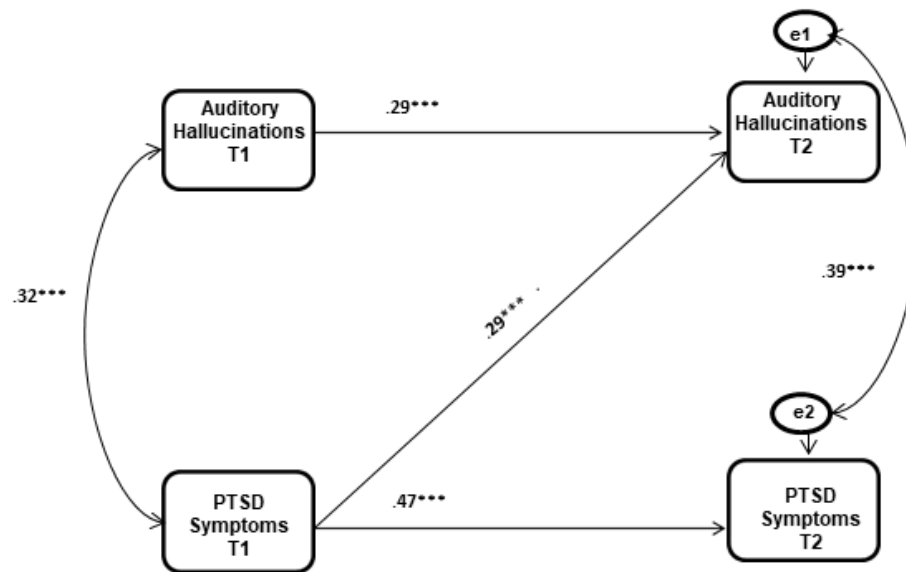| Predicting variables	β	SE	R^2 change	F change (df1, df2)
<u>Step 1</u>			.28	38.31*** (2,199)
Study group	.25***	.10		
Auditory hallucinations(T1)	.44***	.08		
<u>Step 2</u>			.06	6.60*** (3,196)
Study group	.20***	.19		
Auditory hallucinations(T1)	.36***	.08		
Intrusion(T1)	.27***	.05		
Avoidance(T1)	-.05	.05		
Hyperarousal(T1)	.04	.04		

Note. ***p<0.001

Figure 1. Autoregressive cross-lagged model (nested model) assessing relationships between PTSD symptoms and auditory hallucinations. The values represent standardized coefficients. Curved lines represent covariates between constructs..

*** p < 0.001.