



Voices from the storm: A critical review of quantitative studies of auditory verbal hallucinations and childhood sexual abuse

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ABSTRACT

Although an association between hallucinations and sexual abuse has been documented, the relation between specifically auditory verbal hallucinations (AVHs) and childhood sexual abuse (CSA) is less clear. This study reviewed quantitative studies of AVHs and CSA. 36% of psychiatric patients with AVHs, and 22% of non-psychiatric patients with AVHs, reported CSA. At least 16% of the general population with auditory hallucinations also reported CSA. The majority of studies reviewed found that those with AVHs were more likely to be survivors of CSA than individuals without AVHs. 56% of psychiatric patients with CSA reported AVHs, and at least 21% of the general population with CSA reported auditory hallucinations. A majority of studies found survivors of CSA were more likely to report AVHs than individuals without CSA. Ability to impute a causal role for CSA was impaired by such studies' failures to control for potentially confounding variables. Yet, studies of AVH content showed links between the content of voices and the content of CSA in some voice-hearers. It is concluded that although a clear association between CSA and AVHs exists, there is not yet reliable quantitative evidence of a causal relation. Implications for mental health professionals and for future research, are discussed.

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1. Introduction

Recent decades have seen a number of shifts in the paradigm of hallucination research. First, a move has occurred away from studying such experiences within the context of specific psychiatric diagnoses,

partly due to questions being raised as to the reliability and validity of certain diagnoses such as schizophrenia (e.g., Bentall, 2003; Boyle, 2002). This has resulted in the emergence of a complaint-oriented approach to psychopathology (Bentall, 2006; Frith, 1992), with researchers attempting to understand the causal mechanisms underlying the occurrence of hallucinations trans-diagnostically. As a result, a significant literature has been generated on the potential cognitive and neurocognitive mechanisms associated with hallucinations (e.g.,

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Bentall, 1990; Jones, 2010; Seal, Aleman, & McGuire, 2004). The meaning of hallucinations has also been reconsidered, with a movement away from viewing such experiences as meaningful symptoms of a mental illness (Romme & Escher, 1993), and towards a re-conceptualization of them as inherently meaningful events often having their origins in earlier stressful or traumatic events in the individual's life (Honig et al., 1998; Hornstein, 2009; Romme, Escher, Dillon, Corstens, & Morris, 2009; Romme, Honig, Noorthoorn, & Escher, 1992). Such work follows on findings such as that of Romme and Escher (1989) who found 70% of individuals with auditory hallucinations first began to hear voices following a traumatic/emotional event.

One traumatic event that has been found in many studies to be associated with the presence of hallucinations is childhood sexual abuse (CSA). In early studies Ellenson (1985, 1986) found that many female incest survivors experienced hallucinations in a range of modalities including the visual (e.g., shadowy figures, movements in peripheral vision), the auditory (e.g., intruder sounds) and specifically the auditory verbal (e.g., voices giving commands, voices persecuting the voice hearer, or voices helping the voice-hearer). Following this work, an extensive body of work has documented a relation between hallucinations and CSA (Janssen et al., 2004; Read, van Os, Morrison, & Ross, 2005).

A number of studies have further attempted to examine if CSA is associated with hallucinations in specific modalities. Given that intrusive images and auditions, believed to underpin some visual and auditory hallucinations respectively (see Jones, 2010), are underpinned by different cognitive systems (Hagenaars, Brewin, van Minnen, Holmes, & Hoogduin, 2010) it is plausible that different modalities of hallucinations may be preferentially associated with specific causes. Indeed, research (e.g., Read, Agar, Argyle, & Aderhold, 2003) has led to the suggestion that there is a specific relation between CSA and auditory verbal hallucinations (or 'hearing voices'), with Hammersley and Fox (2006) arguing that in studies of psychosis and childhood trauma the link between CSA and auditory hallucinations "is consistently the most reliable finding" (p. 152). Going beyond association, results from quantitative studies have been used to argue that "child abuse is a causal factor for... voices commenting and command hallucinations" (Read et al., 2005, p. 330), yet the causal role of specifically CSA has not been addressed. However, clinical experience, first-person testimonies from individuals who hear voices (e.g., Romme et al., 2009), and case studies (e.g., Heins, Gray, & Tennant, 1990; Bahn & Lee, 2007; Kaufman, Birmaher, Clayton, Retano, & Wongchaowart, 1997; Lysaker, Buck, & Larocco, 2007) are all suggestive of a causal role for CSA in the development of auditory verbal hallucinations (AVHs).

1.1. Theoretical frameworks for an etiological role of CSA in the development of AVHs

A number of theoretical models provide a basis for conceptualizing how CSA may play an etiological role in the development of AVHs. Memory-based models of AVHs propose they result from the unintentional activation of memories (Waters, Badcock, Michie, & Maybery, 2006). Consistent with this model, more severe AVHs (but not visual hallucinations) in people diagnosed with schizophrenia have been found to be associated with worse performance on temporal context memory tasks (Brébion, David, Jones, Ohlsen, & Pilowsky, 2007). This dovetails well with the context memory deficits that have been argued to stem from traumatic events such as CSA (Steel, Fowler, & Holmes, 2005). Steel and colleagues argue that traumatic events are not processed in the normal manner by the hippocampus. During highly traumatic events, information by-passes the hippocampus and is processed via the amygdala, in order to enable a faster release of stress hormones (LeDoux, Iwata, Cicchetti, & Reis, 1988; Steel et al., 2005). Although this allows quicker processing,

it does not allow the hippocampus to perform its normal role of integrating information within a spatial and temporal context (Brewin, 2001; Steel et al., 2005). Thus, potential context memory deficits resulting from CSA form a theoretical framework for understanding how such experiences may result in AVHs. If a review of the CSA and AVHs literature shows that the content of AVHs closely matches that of CSA experiences, then this would help support this memory-based model. However, if more subtle relations between AVHs and CSA are found then this would suggest a need to revise a simplistic memory-based model of AVHs.

In addition to context memory deficits, source-monitoring (Johnson, Hashtroudi, & Lindsay, 1993) provides another mechanism through which CSA may result in AVHs. Bentall (1990) has argued that the ability to discriminate between real, external events and imaginary, self-generated events is a skill (which has been given a number of names; reality-testing, source-monitoring, self-monitoring), in which individuals draw on a number of specific properties of a given cognitive event to make a judgment (not necessarily a conscious one) as to its origin. Bentall proposed that a failure in this skill could result in AVHs, with internal events being misattributed to an external source. While individual studies have found evidence both for and against this proposal (see Aleman & Larøi, 2008 for a review), a recent meta-analysis found deficits in self-monitoring (the ability to recognize self-generated thoughts or actions as one's own) to be more pronounced in people diagnosed with schizophrenia with AVHs than in such individuals without AVHs (Waters, Woodward, Allen, Aleman, & Sommer, *in press*). One specific property of cognitions that has been shown to encourage source-monitoring errors is the amount of cognitive effort associated with their occurrence (Johnson et al., 1993), with spontaneously occurring cognitions (low cognitive effort) being more likely to be misattributed to a non-self source. Relating this to CSA, Hammersley et al. (2003) have noted that CSA is likely to produce intrusive memories, occurring automatically and with low cognitive effort, with such experiences potentially seeding AVHs. Allen, Coyne, and Console (1997) have further argued that trauma-induced dissociation may hamper reality-testing, leaving individuals "vulnerable to the nightmarish inner world" (p. 332).

What is known about the biological impact of CSA on the developing brain also suggests that such experiences may lead to neurological changes that predispose individuals to AVHs. The relatively new discipline of developmental traumatology (Crozier, Van Voorhees, Hooper, & De Bellis, 2011) has begun to trace the neurobiological impact of chronic trauma on the developing child. This considers how measurable aspects of traumatic experiences (such as the type, age of onset, and duration of child maltreatment) and other biopsychosocial factors (e.g., the child's temperament, social support for the child and family) relate to specific neurobiological changes in the brain. While such studies have typically focused on the effects of childhood abuse per se, rather than the differential effects of specific types of abuse (e.g., sexual, physical, emotional), the results of such studies are consistent with abuse causing structural changes that may lead to AVHs. For example, changes in anterior cingulate cortex volume (Cohen et al., 2006; Kitayama, Quinn, & Bremner, 2006), superior temporal gyrus volume (De Bellis et al., 2002; Tomoda et al., 2011), and fractional anisotropy in the arcuate fasciculus¹ (Choi, Jeong, Rohan, Polcari, & Teicher, 2009) have all been found to be associated with childhood abuse. All these areas have been found by both functional and structural neuroimaging (Allen, Larøi, McGuire, & Aleman, 2008) and electrophysiological (Heinks-Maldonado, Mathalon, Houde, Gray, & Ford, 2007) studies to be related to AVHs, and have been interpreted as relating to abnormal functioning in the normal cognitive mechanisms that allow us to recognize internally generated cognitions as self-produced (e.g.,

¹ A measure of the connectivity between speech production areas (e.g., Broca's area) and speech perception areas (e.g., Wernicke's area).

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