THE ROLE OF COGNITIVE DYSFUNCTIONS IN HALLUCINATION FORMATION

Olga Graf, Wolfgang Gaebel, Jürgen Zielasek

Introduction

Hallucinations are one of the most intriguing phenomena in psychopathology of several mental disorders. They are a cardinal feature of schizophrenia and other psychotic disorders and are one of the main diagnostic criteria for the illness (APA, 2013).

In the past two decades the development of neuroimaging techniques allowed researchers to investigate what is happening in the brains of people who experience hallucinations. Nevertheless, the cognitive basis of hallucinatory experience still remains not exhaustively investigated. Many theories concerning the origin of hallucinations implicated specific cognitive dysfunctions as the core abnormality to cause hallucinatory experience (see for Review Daalman et al., 2011). Although dysfunctions of cognitive processes are assumed to be essential in hallucination formation, it is not clear yet which cognitive mechanisms are involved in hallucinatory experiences.

Hallucinatory experience may also occur without a mental disorder (APA, 2013). For instance, some studies reported equal mechanisms in both clinical and non-clinical groups referring to cognitive bias underlies proneness to non-clinical auditory hallucinations and that these experiences are mediated by similar areas of activation to those found in auditory hallucinations and inner speech in patients with schizophrenia (Barkus et al., 2007). Despite this similarity, the study by Daalman and coworkers (2013) showed, using Cognitive Biases Questionnaire for psychosis that healthy persons with proneness to hallucinations show cognitive dysfunctions to a lesser extent compared to persons with mental disorder (Daalman et al., 2013).

Methods

To clarify the role of cognitive processes in hallucination formation, we highlight recent advances in research of the cognitive dysfunctions associated with hallucinations. For this purpose, we conducted a systematic review of the pertinent research literature, including studies with healthy people with the proneness to hallucinations.

Discussion

We presented several cognitive models which describe the cognitive aspects in hallucinations formation. Despite the fact that each of these models contributed to the knowledge about putative cognitive mechanisms underlying hallucinations, neither of them can explain hallucinations fully, covering for example perceptual aspects like loudness or vividness. There is also not enough consistent evidence for one or another model, so that we conclude that there is a need for more detailed and more intensive research on the field of cognitive dysfunctions in order to understand better the phenomenology of hallucinations in schizophrenia and in the general population.

References

Overview of cognitive models explaining hallucinations

Source-monitoring

Reality-monitoring deficit

The reality-monitoring model implies the cognitive process to distinguish internally and externally generated events (Johnson & Raye, 1981). According to this model, hallucinations may result from the erroneous attribution of internally generated information to an external source (Aleman et al., 2003).

Self-monitoring deficit

Inner speech model proposes that auditory verbal hallucinations occur when verbal thoughts are not recognized as being the person’s own but are perceived as someone else’s. This could arise through defective monitoring of inner speech (Firth & Done, 1988). Cognitive self-monitoring can be defined as the ability to identify and distinguish sensations caused by one’s own actions from those that arise from external influences. This model proposes auditory verbal hallucinations to result from self-monitoring deficits leading to inner speech not being recognized as self-produced, and instead being perceived as an external voice (Allen et al., 2007).

Executive functions: poor inhibitory control

Badcock and colleagues (2005) proposed a model of formation of auditory hallucinations, which described a formation of hallucinations through a failure to inhibit irrelevant memories, because of insufficient cognitive inhibition. Poor inhibitory control represents a specific deficit in intentional cognitive inhibition to control unintended and/or intrusive mental activity. Therefore, hallucinations appear to arise from a failure to control previously relevant mental representations, allowing old memories to intrude into current events and become confused with ongoing reality (Badcock, 2010). Several studies confirmed this model with the evidence that auditory hallucination are associated to a failure in intentional forms of cognitive inhibition in the Inhibition of Currently Irrelevant Memories task (Waters et al., 2003), Stroop task (Daalman et al., 2011).

Aberrant perception

Nazzine and colleagues (2012) proposed an expectation-perception model for explaining of auditory hallucination. According to this model normal perception consists of interaction between top-down expectations and bottom-up signals. In the majority of people prior expectations do not lead to hallucinations because the prior probability is modulated by stimulus-driven bottom-up activity. The ascending neural pathways deliver to auditory cortex the sensory input that does not match the prediction and the prior expectation is corrected. Failure of this mechanism may occur when transmission of auditory information consistent with the expectation is enhanced, when the transfer of information that does not match the prediction is suppressed and when the prediction is so broad that random fluctuations of activity in auditory cortex lead to generation of an auditory percept in the absence of external stimuli. These mechanisms might lead to either impaired transmission of prediction error or to its absence. The lack of modulation of expectation by bottom-up signals may underlie the formation of auditory hallucination (Nazzine et al., 2012).

Acknowledgement

The project was supported by Collaborative Research Centre 991 “The Structure of Representations in Language, Cognition, and Science” (B 06 Frame Analysis of Mental Disorders).