COGNITIVE MODELS OF POSITIVE AND NEGATIVE SYMPTOMS OF SCHIZOPHRENIA AND IMPLICATIONS FOR TREATMENT

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SUMMARY
This article reviews cognitive models of positive and negative symptoms of schizophrenia, as well as basic principles of cognitive behavioural therapy (CBT) for hallucinations, delusions and negative symptoms. Cognitive models of schizophrenia posit that individuals’ view of the self is influenced by a combination of genetic vulnerability, early childhood experience and environmental stressors later in life, and that these factors determine how internal and external experiences are interpreted. Cognitive behavioural therapy for schizophrenia is based on the basic principles of CBT and establishes connection between thoughts, emotions and behaviour. Treatment focuses on the meaning the individual attributes to psychotic experience, his or her understanding of it and ways of coping with symptoms, and is intended to reduce the distress caused by psychotic experience and correct thoughts and assumptions that are incompatible with objective evidence. The latest contradictory data on the efficacy of CBT for schizophrenia point to the need to use protocols that are tailored to specific symptoms and subgroups of patients based on the stage of illness, level of neurocognitive impairment and severity of the disorder, and manual-based in order to ensure fidelity of implementation.

Key words: schizophrenia - cognitive behavioural models - implications for treatment

INTRODUCTION
The clinical features of schizophrenia consist of positive and negative symptoms, mood symptoms, disorganisation symptoms and cognitive impairments. There are different underlying pathophysiological mechanisms that are associated with different effects on major domains of patients’ lives and different responses to pharmacological and non-pharmacological interventions. Antipsychotic medication is the first-line treatment for schizophrenia, but medication is not a substitute for therapy and patients should also receive psychological treatment. Cognitive models of positive and negative symptoms of schizophrenia could link genetic vulnerability, early experience and environmental stressors later in life. A CBT based on cognitive models has been developed for schizophrenia and other psychotic disorder.

COGNITIVE MODELS OF POSITIVE SYMPTOMS
A cognitive model of hallucinations proposed by Rector and Beck (2003) connects biological predisposition (genetic factors, neural hyperconnectivity, dopamine and dysfunction of other transmitter systems), vulnerability to stressors and basic negative beliefs about the self and the world. This model posits that, in vulnerable people, during periods of stress and reduced reality testing “hot” cognitions can cross the perceptual threshold and transform into perceptions. Larøi and Woodward (2007) identified two steps before the onset of hallucinations: 1) alienation from inner, self-generated experience and 2) misattribution of these events to external source. The way in which hallucinations are appraised further increases distress (voices appraised as malevolent, powerful, personally acquainted with the individual; the individual disapproves of and rejects the voices) (Mawson et al. 2010). Patients’ distress, hopelessness, and sense of being powerless are maintained by four types of hallucination-related belief: beliefs about the identity, purpose and power (supremacy over patient) of the voices and beliefs about compliance with the voices. These beliefs are supported by the development of quasi-interpersonal relationships between patients and their voices, in which person who is hearing the voices is subordinate and obedient to a powerful other, which means that the validity of hallucinations is not questioned. The perceived ability of voices to carry out their intentions is at the heart of patients’ distress, along with compliant behaviour which prevents disconfirmation of the voices’ ability to realise a threat (Chadwick & Birchwood 1994, 1995). Garcelán’s (2004) model of verbal auditory hallucinations also incorporates personal vulnerability, activation by a high level of distress - which causes faulty e henesthetic feedback - and interpretation of personal experience as alien (e.g. personal voice is interpreted as “Not-Me”). Interaction between a subject and his or her voices leads to a special relationship and the building of an apparently autonomous “Not-Me” identity, and interactions between “Me” and “Not-Me”. Safety behaviour, beliefs about voices and emotional responses maintain the hallucinatory process. An integrated socio-developmental-cognitive model of schizophrenia (Howes & Murray 2014) posits that it is the outcome of genetic vulnerability, early threats to the brain and childhood adversity resulting in dopaminergic excess in the brain. Dopaminergic dysregulation and social adversity make indivi-
duals prone to misinterpreting internal and external stimuli, leading to paranoid delusions and hallucinations. Paranoid delusions are associated with a pessimistic thinking style, low self-esteem, negative emotions and impaired cognition (impaired executive functioning, a tendency to jump to conclusions and inability to understand the mental states of others) (Bental et al. 2009). Freeman et al. (2002) proposed a cognitive model of persecutory delusions: in ambiguous situations vulnerable individuals perceive that something is wrong with the world, rather than with them, due to excessive use of selective attention and a tendency to jump to conclusions. The belief that they are being persecuted by an outside entity limits their distress and preserves their self-respect, serving as a defence mechanism, but this comes at the price of reality-testing. According to Garety et al.’s (2001) cognitive model of psychosis, the disruption of cognitive processes leads, in predisposed persons (genetic factors; childhood adversity) with negative self-schemata, to a maladaptive appraisal of the event, resulting in anomol (unfamiliar, external and potentially threatening) experiences. These experiences and the emotional reactions to them lead to psychosis if they are appraised as externally caused and personally significant. The ABC (Activating events, Beliefs, Consequences) model of emotional disturbances developed by Ellis and Harper (1961) organises emotional experience according to the ABC formula. In the ABC model of hallucinations, the auditory hallucinations are the activating event (A), B represents the person’s beliefs about the voices, and C represents the emotional and behavioural consequences of those beliefs. Patients are taught the BC connection: that their emotional disturbance (C) is a product of irrational thinking (B) about activating events (A). Challenging beliefs about voices and delusions with Socratic questioning and developing alternative, more realistic explanations helps to normalise psychotic experience and enable the patient to accept more rational, logical and pragmatic ways of thinking.

**COGNITIVE MODELS OF NEGATIVE SYMPTOMS**

In contrast to positive symptoms, the negative symptoms of schizophrenia represent a relative absence of feelings, cognition and goal directed behaviour, which has a detrimental effect on psychosocial functioning and quality of life. Negative symptoms are grouped into two main domains: avolition-apathy (amotivation, anhedonia, asociality) and diminished expressiveness (verbal and nonverbal) (Millan et al. 2014, Blanchard & Cohen 2006). Negative symptoms can be primary, i.e. a result of the neurobiology of schizophrenia, or secondary, i.e. due to medication, delusions and voices, depression, social isolation and lack of stimulation (DeQuardo & Tandon 1998, Buchanan et al. 1990). Neurocognitive impairments that manifest in the domains of verbal memory, working memory, attention, social cognition and executive functions (Keeffe & Harvey 2012, Green et al. 2006) are associated with higher levels of negative symptoms and a lower level of social and vocational functioning (Grant & Beck 2009), and may create a vicious circle of failure and reduce the patient’s ability to learn from errors. Negative symptoms are also associated with lower empathetic capacity, a negative global self-concept, lower self-esteem and other dysfunctional beliefs about the self, as well as decreased social cognition (Lincoln et al. 2011). Rector et al. (2005) proposed a cognitive model of negative symptoms, pointing to the protective function of disengagement, which helps the individual to avoid excessive stimulation and preserves limited psychosocial resources. Negative symptoms also serve as a maladaptive strategy, protecting the patient from expected pain and rejection in social situations. Cognitive assessment and beliefs play a role in expression and the persistence of negative symptoms and includes negative social attitudes and attitudes towards self-representation, low expectation of satisfaction and success in socially oriented activities and pessimistic attitude towards the future. As a result, patients minimise social investment, which results in emotional, motivational and behavioural withdrawal. Currently available treatments for schizophrenia do not produce clinically significant improvement in negative symptoms (Fussar-Poli et al. 2015). Because negative symptoms have more influence on outcomes and quality of life than positive symptoms, targeting negative symptoms has the potential to reduce the psychosocial disability associated with schizophrenia.

**IMPLICATIONS FOR TREATMENT**

Cognitive models of psychosis have enabled the introduction of CBT as an adjunctive treatment for schizophrenia and other psychotic disorders. A review of CBT treatment protocols for positive symptoms (Morrison 2017, Quintin et al. 2012, Kingdon & Turkington 2006, Byrne et al. 2003, Smith et al. 2003), indicates that the main components of treatment are as follows: thorough assessment of the patient’s symptoms, gradual establishment of a relationship of trust, psychoeducation and destigmatisation of psychotic experience, Socratic questioning of evidence for the automatic thoughts and inferences, a guided search for alternative, more rational explanations for voices, suspicious and delusional ideas, correction of cognitive distortions and encouraging the adoption of more logical thinking style and rational responses, homework involving gradual exposure to problematic situations, work on the core beliefs that a person is different, evil, damaged unworthy, as well as relapse prevention. The core principle of CBT of hallucinations is to challenge beliefs about the content, identity and power of the voices, with the aim of giving the patient power and control over them and brake down safety behaviour (e.g. obeisance to the voices). The goal is to correct the imbalance of power which maintains
the patient’s obedience and distress and to change the external attribution of the mental experience. CBT for delusional ideas includes collaborative exploration of evidence for and against irrational ideas and the development of alternative, healthy explanations, as well as the planning of behavioural experiments to confirm rational beliefs. Commitment to completing homework between sessions facilitates change, as it is a test of patients’ motivation and an extension of treatment.

Defeatist beliefs (negative beliefs about one’s performance and ability) mediate the relationship between deficit symptoms and psychosocial functioning, (Luther et al. 2016, Grant & Beck 2009), so cognitive behavioural interventions for negative symptoms target the dysfunctional beliefs that maintain negative symptoms (negative beliefs about social affiliation and performance, the perception that one has limited resources, stigma, low expectations of pleasure and success, and negative beliefs activated by positive symptoms) (Perivoliotis & Cather 2009). The goal of CBT interventions for the negative symptoms of schizophrenia is to improve self-efficacy, enhance pleasure, reduce self-stigmatisation and other self-deprecating beliefs, and to gradually activate the patient. It starts with a formal assessment (completion of rating scales and a battery of neurocognitive tests) and observation of the patient’s behaviour, self-monitoring of thoughts, emotions and behaviour, and case formulation. Case formulation provides a picture of the individual’s vicious circle of irrational beliefs, unhealthy emotions and self-deprecating behaviour, all of which help to maintain apathy, avolition, anhedonia, alogia, affective flattening and social withdrawal. Behavioural techniques that are used include activity scheduling with the aim of gradual behavioural activation, undertaking pleasurable activities in everyday life, renewing contact with significant others, practising social skills and problem solving. Automatic negative beliefs and core irrational beliefs about the self, other people and the world are challenged through Socratic questioning.

EFFECTIVENESS OF CBT IN THE TREATMENT OF SCHIZOPHRENIA

Cognitive-behavioural therapy became part of the treatment for schizophrenia in the UK in 2002 (NICE, 2014). Early studies of the efficacy of CBT in the treatment schizophrenia highlighted positive effects on positive and negative symptoms, mood, anxiety and functioning. A meta-analysis of methodologically rigorous studies (Wykes et al. 2008) concluded that it has beneficial effects on positive symptoms. Several recent meta-analyses have, in contrast to the earlier research, found no clear and convincing advance that CBT is better than other non-pharmacological treatments (Jones et al. 2018, Jauhar et al. 2014). A meta-analysis of the effects of CBT on functioning, distress and quality of life (Laws et al. 2018) found a small therapeutic effect, which was not maintained at follow-up. Future studies using treatments tailored to specific subgroups of patients (organised by level of neurocognitive impairment and severity of the disorder) and manual-based protocols to ensure fidelity of implementation, and delivered by the supervised and more experienced therapist, could be used to examine the different effects of various elements of CBT on specific symptoms and outcomes. This might enable development of more effective, personalised forms of CBT treatment becoming available to a greater number of patients with schizophrenia. Treatment protocols also should incorporate elements of the third wave of CBT (mindfulness-based therapy, acceptance and commitment therapy, compassionate mind training and meta-cognitive therapy) which targets the way in which people relate to their thoughts and feelings as well as the content of their thoughts and beliefs.

CONCLUSIONS

Cognitive models of schizophrenia provide a framework for understanding and treating the positive and negative symptoms of schizophrenia. Recent contradictory data on the efficacy of CBT for schizophrenia indicate a need for more personalised, more effective, manual-based treatment protocols that ensure fidelity of implementation and are tailored to specific subgroups of patients.

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