Anhedonia in prolonged schizophrenia spectrum patients with relatively lower vs. higher levels of depression disorders: Associations with deficits in social cognition and metacognition

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**A B S T R A C T**

This study has sought to explore whether there are at least two subtypes of anhedonia in schizophrenia: one closely linked with depression and another that occurs in the absence of depression which is related to a general paucity of internal experience. Participants were 163 adults with schizophrenia who completed assessments of depression, anhedonia, executive functioning, positive and negative symptoms, social cognition and metacognition. A cluster analysis based on participants’ depression and anhedonia symptom scores produced three groups: High Depression/High Anhedonia (n = 52), Low Depression/Low Anhedonia (n = 52), and Low Depression/High Anhedonia (n = 59). An ANCOVA and post hoc comparisons controlling for positive and negative symptoms found that the Low Depression/High Anhedonia group had poorer metacognition and social cognition than other groups. These findings point to the possibility of a subtype of anhedonia in schizophrenia, one occurring in the relative lesser levels of depression, and tied to deficits in the ability to think about oneself and others.

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1. Introduction

Anhedonia generally refers to the diminished capacity to experience pleasure. It has been conceptualized as a multidimensional phenomenon with varying subtypes including physical vs. social and anticipatory vs. consummatory distinctions and also includes a range of cognitive, emotional and interpersonal components (c.f. Buck & Lysaker, 2013a). Anhedonia was considered by early theorists to be a central feature of schizophrenia and a cause of the psychosocial deficits that characterize the disorder (Meehl, 1962). Consistent with these views, empirical studies have suggested that anhedonia is commonly found in schizophrenia, and may be a risk factor for the condition (Buck & Lysaker, 2013b; Gooding, Tallent, & Matts, 2005). Furthermore, evidence suggests that anhedonia is a relatively stable phenomenon (Buck & Lysaker, 2013b) and linked to poorer outcomes over time (Blanchard, Mueser, & Bellack, 1998).
To characterize the place of anhedonia within schizophrenia spectrum disorders, several authors have proposed that anhedonia is a core negative symptom (Kirkpatrick, Fenton, & Carpenter, 2006; Kring & Moran, 2008). Supporting this possibility, research has found that the anhedonia and asociality items of the Scale for the Assessment of Negative Symptoms (SANS) demonstrated a high degree of internal consistency (Andreasen, 1982). Andreasen, Arndt, and Alliger (1995) also found that the anhedonia item of the SANS correlated significantly with scores derived from the avolition and affective flattening items. In tests of the psychometrics of the Clinical Assessment Interview for Negative Symptoms (CAINS), an anhedonia item was found to load on a factor interpreted as experiential as opposed to an expressive negative symptom (Horan, Kring, Gur, Reise & Blanchard, 2011; Kring, Gur, Blanchard, Horan, & Reise, 2013). Llerena et al. (2013) similarly found that the self-report of pleasure was linked with clinician ratings of negative symptoms.

Of note, other research linking anhedonia with negative symptoms has not found significant associations (Fertout, Scoury, Lefebvre, Koudrat, & Loas, 2012; Herbener & Harrow, 2002; Loas, Monestes, Ingelaere, Noisette, & Herbener, 2009). In a recent longitudinal study, Ritsner (2013) also failed to find associations between anhedonia and negative symptoms in individuals with schizophrenia. Buck and Lysaker (2013b) observed that higher levels of anticipatory anhedonia predicted concurrent and prospective depressive symptoms, but not negative symptoms.

One explanation for the lack of a consistent pattern of associations between anhedonia and other negative symptoms is that there are different forms of anhedonia, one form linked with depression and the other form in which anhedonia is not linked depression but with another underlying phenomenon. The possibility that there may be different groups is further supported by a review suggesting that anhedonia may stem from different roots including disturbances in affective regulation and difficulties with the complexity of self-representation (Cohen, Najolia, Brown, & Minor, 2011). With regard to this issue, Cohen and Minor (2010) have proposed that at least two subgroups might exist. They note that, “anhedonia can reflect a secondary negative symptom caused by depression as opposed to an idiopathic disease symptom, so distinguishing between the two might be important” (p. 148). Specifically, it may be that some patients report anhedonia as a result of negative affect (Strauss, 2013) while for others anhedonia could result from the lack of sufficiently stable internal mental representations which integrate memory and affect and allow for reflection about and plans to seek out pleasure (Gold, Waltz, Prentice, Morris, & Heerey, 2008; Strauss & Gold, 2012).

In the current study we sought to pursue these ideas by investigating whether we could detect two groups of patients with anhedonia: one with relatively higher levels of depression and one with lower levels of depression but relatively less complex representations of self and others. We utilized the constructs of metacognition and social cognition to describe the ability to generate and hold complex internal mental representations of self and others. Metacognition refers to a spectrum of activities which involve thinking about thinking, ranging from the consideration of discrete thoughts and feelings to the synthesis of discrete perceptions into an integrated representation of self and others (Lysaker et al., 2013; Semerari et al., 2003). Social cognition refers to processes involved in thinking about social interactions such as theory of mind, emotion processing and attributional style (Pinkham et al., in press). Metacognition and social cognition appear to be significantly impaired in schizophrenia spectrum disorders in both early and later phases of illness when compared to medical and psychiatric controls (Brüne, 2005; Lysaker et al., 2012; Vohs et al., 2014). There is evidence that these constructs are distinct (Lysaker et al., 2013). They diverge theoretically in that metacognition focuses on synthesizing psychological experiences into mental representations that vary by complexity, adaptiveness and flexibility, whereas social cognition is more concerned with the accuracy of perceptions and representations. However, both constructs deal with how cognitive processes are applied to interpersonal experience and some see them as overlapping (Pinkham et al., in press). Studies of this phenomenon in depression are limited but one notable finding suggests first episode depression patients experience significant deficits in social cognition and metacognition relative to healthy controls though these levels of impairments do not appear as severe as those observed in schizophrenia spectrum disorders (Ladegaard, Larsen, Videbech, & Lysaker, 2014).

The possibility that deficits in metacognition and social cognition are uniquely linked with anhedonia is consistent with work suggesting that an integrated representation of oneself in memory and the ability to reflect about that memory are needed for hedonic experience, regardless of one’s affective state (Buck & Lysaker, 2013b; Cohen et al., 2011; Gold et al., 2008). Additional support includes findings linking metacognitive deficits with generally more severe levels of negative symptoms (Hamm et al., 2012; Nicolò et al., 2012; Rabin et al., 2014; McLeod, Gumley, MacBeth, Schwannauer, & Lysaker, in press). Social cognitive deficits might also be a cause of anhedonia in the presence of relatively lesser levels of depression since a lack of an understanding of the emotional responses of others might deprive persons of the opportunity to share pleasure or to have perspectives from which to think about oneself in a manner that might allow for the experience of or anticipation of pleasure. Additionally, social cognition, like metacognition, is widely understood as necessary for persons to think about their own thoughts and feelings, make sense of ongoing experience and pursue meaningful goals (Brüne, Dimaggio, & Lysaker, 2011). Evidence supporting this possibility includes studies linking deficits in social cognition with negative symptoms (Abdel-Hamid et al., 2009; Hofer et al., 2009; Strauss, Jetha, Ross, Duke, & Allen, 2010).

In the current study our primary aim was to examine the relationships among concurrent assessments of anhedonia, depression, metacognition and social cognition. We predicted that cluster analytic techniques would detect three groups: High Depression/High Anhedonia, Low Depression/Low Anhedonia, and Low Depression/High Anhedonia. We proposed that the High anhedonia/Low depression group would have significantly greater deficits in metacognition and social cognition than the other groups. To rule out the possibility that any observed differences were the result of other forms of symptoms or deficits in executive functioning, we included measures of these as potential covariates.
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