EXPERIENTIAL NEGATIVE SYMPTOMS IN YOUNG ADULTS ENDORSING PSYCHOTIC-LIKE EXPERIENCES

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ABSTRACT

While many studies of risk factors for psychosis focus on positive symptoms, such as subthreshold levels of hallucinations and delusions, fewer studies have examined negative symptoms in the early course of the schizophrenia or other psychotic disorders. This relative lack of focus on the role of negative symptoms is problematic, given findings that negative symptoms, such as a loss of motivation and pleasure (MAP), are associated with a more persistent and impairing course of psychosis, and tend to appear earlier in the development of psychotic symptoms. Psychotic disorders, which afflict approximately 3-5% of the population, tend to emerge in late adolescence/early adulthood and are among the most debilitating and costly of mental disorders. The current project explored three areas of negative symptoms in young adults who demonstrated a range of psychotic-like experiences (PLEs). First, a review of the literature pertaining to negative symptoms across the span of psychosis was conducted. Second, we tested whether experiential negative symptoms – specifically MAP deficits – were associated with increases in PLEs, including those that are experienced as distressing (PLEDs). Third, we examined the potential influence of episodic memory performance factors on the relationship between MAP symptoms and PLEs/PLEDs. Collectively, this project highlights the importance of including negative symptoms (i.e., MAP deficits) and/or cognitive performance (i.e., associative/relational learning/memory) outcomes when evaluating people with PLEs/PLEDs to identify those who may be at greater risk for developing a psychotic disorder.

This dissertation is

for my family and friends,

whose support has been unwavering;

For Sterling,

my constant, furry companion,

for—sometimes forcibly—reminding me to play;

And for Branden,

for listening to me fuss and fret,

for helping me through the difficulties and celebrating the earned victories,

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Finally, my most sincere and deepest gratitude goes to the innumerable women before me in science/STEM fields – many of whom continue to go unrecognized – for helping to create the privilege for me and other women to attend graduate school.

"Nevertheless, she persisted."

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CHAPTER 1

EXPANDED LITERATURE REVIEW

Abstract

Our understanding of the role of negative symptoms of psychosis, such as anhedonia and avolition, has been steadily increasing in chronic schizophrenia populations. Yet, little is known about the role of negative symptoms in the early course of schizophrenia. To this point, much of the research on the early stages of schizophrenia has focused on positive (hallucinations and delusions) and disorganized symptoms in identifying and treating people at elevated risk of developing psychosis, particularly in the determination of who may be at clinical high risk (CHR) for the disorder. The relative lack of focus on the role of negative symptoms in risk for psychosis is particularly problematic given that negative symptoms have been associated with worse prognosis, difficulties in social and role functioning, and risk of conversion to disorder in CHR populations. The nature of negative symptoms and their correlates in the course of psychosis are reviewed with a focus on how negative symptoms manifest in the early stages of these disorders.

Introduction

Schizophrenia and related psychotic disorders typically emerge in late adolescence or early adulthood, affect a range of cognitive, perceptual, and emotional functioning, and afflict approximately 5-7% (schizophrenia specifically affecting approximately 1%) of people (Bhugra, 2005; McGrath, Saha, Chant, & Welham, 2008; Saha, Chant, Welham, & McGrath, 2005); these severe mental disorders are among the most debilitating and costly of disorders worldwide (Lopez, Mathers, Ezzati, Jamison, & Murray, 2006). Schizophrenia is the quintessential psychotic disorder, but many disorders (e.g., schizoaffective disorder, major depression with psychotic features, bipolar disorder, post-traumatic stress disorder, and others) can and do involve psychosis. Throughout this review, the terms "schizophrenia" and "psychosis" will be used interchangeably. In the United States alone, schizophrenia is estimated to require more than $60 billion on direct (i.e., treatment) and indirect (e.g., loss of productivity) costs (Solomon & Draine, 1995; Wu et al., 2005), and many people with these disorders require public assistance funds because few are able to maintain full time employment (Anthony & Blanch, 1987; Mueser, Becker, & Wolfe, 2001). Of people who develop schizophrenia, approximately one-third have chronic, severe prognoses (Cannon et al., 2008; Strauss & Carpenter, 1972; Woods et al., 2009) and those people with elevations in negative symptoms have particularly poor prognoses (Lieberman et al., 2005; Velligan & Alphs, 2008); therefore, efforts to understand and treat these symptoms in the course of psychotic disorders is critical.

One way to increase likelihood of good prognosis is through early identification, intervention, and treatment of symptoms related to psychosis (Malla, Norman, &

Voruganti, 1999). While there exist a number of available treatments, the preponderance of treatments target positive symptoms, such as hallucinations and delusions, with fairly limited available treatments on negative symptoms, such as anhedonia and avolition (Cetin, 2015; Elis, Caponigro, & Kring, 2013). The relative lack of focus on the role and treatment of negative symptoms in risk of psychosis is particularly problematic given that in psychosis, negative symptoms have been found to be associated with a poor prognosis, greater cognitive deficits, difficulties in social and role functioning, and elevated risk of conversion to psychosis (Malla et al., 1999). Additionally, some evidence indicates that negative symptoms appear as much as a year earlier than subthreshold positive symptoms in the development of psychotic disorders (Lieberman et al., 2005; Velligan & Alphs, 2008), suggesting that negative symptoms may be a target for early intervention and treatment.

Brief Review of Negative Symptoms of Schizophrenia

At the 2006 consensus development conference on negative symptoms, five negative symptoms were identified: anhedonia, avolition, alogia, affective flattening, and asociality (Kirkpatrick, Fenton, Carpenter, & Marder, 2006). These symptoms are often examined together and have been associated with deficits in cognition (Marder & Fenton, 2004), social, and occupational functioning (Hunter & Barry, 2012; Rabinowitz et al., 2012; Strauss et al., 2013; Wegener et al., 2005), as well as poorer quality of life (Wegener et al., 2005) and suicidal behaviors (Cohen, Test, & Brown, 1990; Soyka, 1994) in people with schizophrenia.

Recent work suggests that the negative symptoms of psychosis can be satisfactorily and parsimoniously described by two broad dimensions: a) symptoms that

reflect diminished verbal and non-verbal expression—*expressive symptoms*; b) symptoms that reflect diminished motivation for engagement in and experience of pleasurable activities—*experiential symptoms*. Expressive symptoms are comprised of *alogia* and *affective flattening*, both of which are prominent in schizophrenia (Cohen, Najolia, Kim, & Dinzeo, 2012; Trémeau et al., 2005, 2010). They are associated with poor functioning, poorer prognosis, and social deficits (Cohen et al., 2012; Fenton & McGlashan, 1991; Herbener, Harrow, & Hill, 2005; Mueser et al., 1996). Experiential symptoms are related to diminishments in motivation and engagement in and experience of pleasurable activities, and encompass *anhedonia*, *avolition*, and *asociality*. These two symptom domains have been suggested as potential targets for treatment (Horan, Kring, Gur, Reise, & Blanchard, 2011).

While several studies point to a link between negative symptoms and adverse outcomes in people diagnosed with psychotic disorders, fewer studies have characterized the nature of negative symptoms over the developmental course of psychosis. Recent longitudinal studies support earlier retrospective self-report studies by suggesting that negative symptoms appear prior to the emergence of positive symptoms (Demmin, Carrión, Auther, McLaughlin, & Cornblatt, 2013), and are associated with a more persistent and impairing course of the disorder than positive symptoms (Lieberman et al., 2005; Velligan & Alphs, 2008). Numerous studies of clinical high risk for psychosis have shown negative symptoms to be associated with more severe functional impairment than other co-occurring symptoms (Comparelli et al., 2014; Fulford et al., 2014; Lee et al., 2014; Rhurmann et al., 2010; Velthorst et al., 2009). Further, negative symptom severity and negative symptoms that persist for at least six months may be the most reliable

predictors of transition to psychosis in at-risk individuals (Nelson et al., 2013; Piskulic et al., 2012; Velthorst et al., 2009).

The present review will briefly discuss available evidence from CHR samples, including current approaches to assessing and measuring negative symptoms, contributions from affective science, and suggestions for future research in negative symptoms.

Clinical High Risk for Psychosis

After much research characterizing symptoms associated with the chronic stages of schizophrenia, the field has recently expanded its focus to include the study of early manifestations of psychotic symptoms such as those experienced by people at CHR psychosis. Such studies may yield critical information for treatment and prevention and, in turn, may lead to improved outcomes for individuals at risk of developing psychosis the economic and societal impacts of psychosis.

Currently the primary method for empirically determining whether a given person may be at CHR for psychosis is by clinical interview. Of the various interviews developed for this purpose, the Structured Interview for Psychosis-Risk Syndromes (SIPS; Miller et al., 2003; Miller et al., 1999) is widely used. If the interviewee meets one or more of the following three SIPS criteria, they are considered to cross the threshold of a psychosis-risk syndrome, which is to say that they are at considered to be at CHR for psychosis. The three psychosis-risk syndromes identified by the SIPS are: (a) brief periods of fully psychotic features (Brief Intermittent Psychotic-Risk Syndrome); (b) recent onset or worsening of attenuated positive psychotic symptoms that meet certain specifications of frequency and severity (Attenuated Positive Symptom Psychosis-Risk

Syndrome); (c) deterioration of functioning within the past year, in addition to either schizotypal personality disorder or genetic risk for psychosis, determined by diagnosis of a psychotic disorder in a first-degree relative (Genetic Risk and Deterioration Psychosis-Risk Syndrome). Of people who are identified as CHR on the SIPS, between 20-40% will go on to develop psychosis, which is substantially higher risk than has been observed in the general population (Cannon et al., 2014; Fusar-Poli, 2012; Mittal et al., 2010; Nelson et al., 2013; Rhurmann et al., 2010; Woods et al., 2009).

While SIPS-determined CHR for psychosis relies almost entirely on positive symptoms or decreased functioning and genetic risk (Yung & McGorry, 1996), findings suggest that negative symptoms and cognitive problems are also associated with increased risk of psychosis (Yung, Phillips, Yuen, & McGorry, 2004). Further, among those at CHR for psychosis, negative symptoms appear to emerge prior to positive symptoms (Lencz, Smith, Auther, Correll, & Cornblatt, 2004; Lyne et al., 2014; Velthorst et al., 2009; Yung et al., 2004), can predict transition to psychosis (Mason et al., 2004), and predict a poorer course for people who develop psychosis (Piskulic et al., 2012). Additionally, inclusion of negative symptoms increases hazard ratio predictions by 10 percent for every 1-point increase in negative symptoms on a subscale of the SIPS (Schlosser et al., 2012), and it is typically the behavioral changes and functional decline associated with negative symptoms, rather than the positive symptoms, that serve as the catalyst for an individual to seek treatment (Lencz et al., 2004; Yung & McGorry, 1996). Yet, surprisingly, negative symptoms are often overlooked in the development of tools measuring and assessing those at CHR and, therefore, when considering transition to a psychotic disorder or when considering treatment planning.

Measurement and Assessment

Interviews

There exist multiple interviews employed in chronic psychosis to measure negative symptoms, including the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszben, & Opler, 1987), the Scale for Assessment of Negative Symptoms (SANS; Andreasen, 1989), and the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962). The SANS exclusively measures negative symptoms, but has not been applied to CHR populations. A number of interview systems developed for use in people at CHR have included measurement of the presence and severity of negative symptoms, but only as part of a longer interview. These include the Comprehensive Assessment for At-Risk Mental States (CAARMS; Yung, Yuen, Phillips, Francey, & McGorry, 2005), the SIPS (Miller et al., 2003), and the Bonn Scale for Assessment of Basic Symptoms (BSABS; Klosterkötter, Hellmich, Steinmeyer, & Schultze-Lutter, 2001). Interviews can provide detailed information not available through other methods of collection, but tend to be time-intensive and require extensive training for reliable administration. Additionally, none of these interview systems were developed to specifically measure negative symptoms, especially as defined in the current, two-factor model, in CHR.

Developed following the 2006 consensus development conference on negative symptoms, two new clinical interviews were established: The Clinical Assessment Interview for Negative Symptoms (CAINS; Horan et al., 2011) and the Brief Negative Symptom Scale (BNSS; Kirkpatrick et al., 2011). Both of these measures cohere to the two-factor model of negative symptoms and have demonstrated good psychometric properties (Horan et al., 2011; Kirkpatrick et al., 2011). Although these interview systems

show significant promise, they were developed and validated in individuals with frank psychosis, which limits applicability to people at CHR. However, one study recently validated the CAINS in people at CHR, with minor age appropriate adaptations (Gur et al., 2015). Findings indicated that the CAINS distinguished those at CHR from healthy controls with moderate to large effect sizes. The authors suggest that their results provide evidence indicating that negative symptoms can be reliably identified in young adults at risk for developing a psychotic disorder. Further, they found associations between negative symptoms, neurocognitive performance, and functioning in line with the literature in those with chronic psychosis.

There have been advances in semi-structured interview systems to specifically ascertain the presence and severity of symptoms in people with CHR and, separately, negative symptoms in people with psychosis. However, only one study has assessed validity of utilizing negative symptom interviews with people at CHR; additional research is needed. With studies suggesting that negative symptoms occur long before the onset of positive symptoms, further work is necessary to develop and finesse negative symptom interviews appropriate for people at CHR.

Self-Report Measures

Self-report questionnaires are a cost effective and quickly administered alternative to clinical interviews to investigate the presence and severity of negative symptoms. Most research studies attempting to understand aspects of negative symptoms, such as anhedonia or avolition, tend to separate and measure only the specific dimensions of negative symptoms, and there are many such self-report questionnaires used across the literature [e.g., Temporal Experience of Pleasure Scale (TEPS; Gard, Germans Gard,

Kring, & John, 2006), General Causality for Orientations Scale – Clinical Populations (GCOS-CP; Cooper, Lavaysse, & Gard, 2015), Intrinsic Motivation Inventory – Schizophrenia Research (IMI-SR; Choi, Mogami, & Medalia, 2010), Community Assessment of Psychic Experiences (CAPE; Konings, Bak, Hanssen, Van Os, & Krabbendam, 2006)]. Assessing each negative symptom construct individually is important for a thorough understanding of the underlying components, but no one self-report measure adequately assesses all constructs within negative symptoms nor do they encapsulate negative symptoms as presented in the two-factor model.

A recent self-report measure was created following development of the CAINS. The Motivation and Pleasure Scale – Self-Report (MAP-SR; Llerena et al., 2013) is a 15-item self-report measure, developed in people with psychosis, with strong psychometric properties. Although this self-report questionnaire shows great promise in measuring the experiential negative symptoms, the authors report purposefully excluding expressive negative symptoms from the MAP-SR as they do not correlate as strongly to functional impairment. Validity of the MAP-SR to people at CHR for psychosis remains unknown, as there have been no published attempts to use it in this population.

Laboratory tasks

Laboratory tasks are employed to control several variables and obtain a more consistent measure of negative symptoms across participants. Reviewed below, there has been much research assessing both expressive and experiential symptoms in patients with psychosis and significantly less research has been conducted in those at CHR.

Expressive Symptoms

Research has shown that people with psychosis who have more severe negative symptoms, have reduced speech production (Cohen et al., 2012). In a 2007 meta-analysis using subjective analysis (i.e., trained assistants) to rate a variety of aspects of diminished vocal expression, the researchers determined large verbal expression deficits in patients with schizophrenia (Hoekert, Kahn, Pijnenborg, & Aleman, 2007). However, a more recent meta-analysis using objective acoustic analysis (i.e., computer software) of diminished vocal expression, which included 13 studies and almost 500 patients, reported more moderate effect sizes than previous studies using behavior-based coding strategies (Cohen, Mitchell, & Elveveg, 2014). Specifically, Cohen and colleagues (2014) found that average length of pause during speech was the most affected in people with schizophrenia, such that those with psychosis paused longer than those without psychosis.

It is widely accepted that people with schizophrenia do not display nonverbal expressions to the same degree that they report experiencing emotion, and that this is true for both men and women diagnosed with the disorder (Kring & Moran, 2008; Mote, Stuart, & Kring, 2014). Though people with schizophrenia display flattened affect, they do display movement in the muscles associated with expressing emotions that is consistent with non-psychiatric controls (Kring, Germans Gard, & Gard, 2011; Kring, Kerr, & Earnst, 1999; Schlenker & Cohen, 1993; Volz, Hamm, Kirsch, & Rey, 2003; Yee et al., 2010).

Data pertaining to expressive symptoms prior to the onset of psychosis are limited, however, follow-back studies assessing youth in the premorbid stage of the

disorder demonstrated symptoms such as flattened affect and social withdrawal (Walker, Grimes, Davis, & Smith, 1993; Watt, Grubb, & Erlenmeyer-Kimling, 1982; Watt, 1979). These studies coded facial expressions in childhood home video recordings (Walker et al., 1993) and qualitative descriptors from school records (Watt, 1979; Watt et al., 1982). In the study using childhood home videos, faces were coded for expressivity. Findings indicated that girls who developed schizophrenia were less likely to express joy between birth and age 16 than their non-schizophrenia siblings, and both boys and girls who went on to develop schizophrenia expressed more negative affect during childhood (Walker et al., 1993). These findings are supported by another study that indicated that people who go on to develop schizophrenia retrospectively self-report flat affect prior to the onset of psychosis (Hafner et al., 2003). Cumulatively, while these findings are suggestive of the early emergence of reduced expression in the course of schizophrenia, results are limited by methodological concerns with potential recall bias, as well as the possibility that environmental factors that may have influenced the child's expressions and/or behavior were not taken into consideration while coding home videos.

Thus far, work conducted to assess deficits in verbal and/or non-verbal expressive symptoms in people at CHR is limited. One such study of people at CHR for psychosis noted varying levels of non-verbal emotional expression over time: expression of emotion was decreased at baseline and significantly diminished at 6-months, but symptoms returned to baseline levels at 12-months (Piskulic et al., 2012). More work will be needed to improve our understanding of the developmental course of diminished verbal and non-verbal expression in CHR.

Experiential Symptoms

Anhedonia, which is the diminished experience of pleasure in something once found pleasurable, has long been considered a key symptom in schizophrenia (Bleuler, 1950; Kraepelin, 1919; Rado, 1953). Anhedonia has been implicated in a number of adverse outcomes in schizophrenia, including increased stress and decreased subjective well-being (Blanchard, Mueser, & Bellack, 1998), poor self-efficacy and low self-esteem (Ritsner, 2014), poorer premorbid functioning (Chapman, Chapman, & Raulin, 1976; Garnet, Glick, & Edell, 1993), low social functioning and less social support (Ritsner, 2014), and increased emotional distress compared to schizophrenia patients without anhedonia (Myin-Germeys, 2001).

Earlier studies of anhedonia in schizophrenia initially distinguished physical from social anhedonia. In chronic schizophrenia and first episode sapmles, data have consistently shown increased reports of both physical and social anhedonia compared with non-schizophrenia samples (Blanchard, Bellack, & Mueser, 1994; Katsanis, Iacono, & Beiser, 1990; Miettunen et al., 2010). Findings in CHR individuals suggest that both physical and social anhedonia are increased relative to controls (Cressman et al., 2015; Horan, Blanchard, Clark, & Green, 2008; Lencz et al., 2004) and social isolation stemming from social anhedonia been identified as a predictor of transition to psychosis (Häfner & an der Heiden, 1999; Yung & McGorry, 1996).

Recent approaches to assessing anhedonia focus on the time-course of anhedonia in psychosis. Pleasure that is being experienced in a given moment has been called *consummatory pleasure*, or in-the-moment pleasure, and the feeling of *liking* (Gard, Gard, Kring, & John, 2006; Gard et al., 2007; Kring et al., 2011; Kring & Barch, 2014;

Kring & Caponigro, 2010; Robinson & Berridge, 2008). Multiple methods, including self-report, naturalistic report, and laboratory tasks, have consistently shown intact consummatory pleasure in chronically symptomatic people with psychosis (e.g., Burbridge & Barch, 2007; Gard et al., 2006; Gard et al., 2007; for review, see Cohen, Minor, & Najolia, 2010).

Until recently, there were no empirical studies assessing the experience of consummatory pleasure in people at risk for psychosis; the belief was that there was no theoretical reason to suggest that at-risk individuals would experience deficits in consummatory pleasure (Barch, 2008). However, recent studies investigating consummatory pleasure in earlier stages of psychosis have conflicting results. One study reported that no differences in consummatory pleasure for first episode of psychosis patients relative to non-psychiatric controls (Mote, Minzenberg, Carter, & Kring, 2014). However, another study using multiple self-report measures, including the Temporal Experience of Pleasure Scale (Gard et al., 2006) and the Behavioral Inhibition Scale / Behavioral Activation Scale (Carver & White, 1994), reported that CHR individuals endorsed decreased consummatory pleasure relative to age-matched non-psychiatric controls and relative to individuals further along the course of psychosis (Schlosser et al., 2014). Combined, these studies suggest that there may be short-term increases in consummatory pleasure deficits before the onset of schizophrenia that potentially remit after transition to psychosis, although more work is clearly needed.

Pleasure that is experienced in contemplation of a future pleasurable stimulus has been called *anticipatory pleasure*, non-current pleasure, and the feeling of *wanting* (Gard et al., 2006; Gard et al., 2007; Kring et al., 2011; Kring & Barch, 2014; Kring &

Caponigro, 2010; Robinson & Berridge, 2008). Diminished anticipatory pleasure in patients with schizophrenia has been consistently shown, using self-report measures, such as the TEPS (Favrod et al., 2010; Gard et al., 2006), physiological and neurological methods (Juckel et al., 2006; Wynn, Horan, Kring, Simons, & Green, 2010), naturalistic report (Gard et al., 2007), and behavioral methods (Trémeau et al., 2010). Anticipatory anhedonia has been linked to deficits in motivational processes and reduced goal-directed behavior, the cycle of which is thought to perpetuate reduced anticipation of pleasure (Cooper, Lavaysse, & Gard, 2015; Davidson, 1998; Gard et al., 2014; Heerey & Gold, 2007).

Until recently, little work had been conducted to understand anticipatory pleasure deficits in CHR, and recent studies offer mixed results. Studies show that individuals with recent onset of a psychosis experience less anticipatory pleasure than either non-psychiatric controls or chronic patients (Mote, et al., 2014; Schlosser et al., 2014). Thus far only one study has been conducted on anticipatory pleasure in CHR individuals. Results of that study indicated that CHR individuals experienced more severe anticipatory pleasure deficits as compared to same-aged peers and to early- and late-course schizophrenia patients, suggesting that anticipatory pleasure deficits may be an early indicator of risk for psychosis (Schlosser et al., 2014).

Avolition, defined as the inability to engage or persist in activity, especially goal-directed behavior has—like anhedonia—long been considered a core feature of schizophrenia (Barch, 2008; Bleuler, 1950; Kraepelin, 1919). Avolition has been shown to be persistently resistant to pharmacological treatment (Kirkpatrick et al., 2006). Research suggests that motivation levels predict functional outcomes in both early and

later stages of schizophrenia (Fervaha, Foussias, Agid, & Remington, 2013; George Foussias & Remington, 2010; Kiang, Christensen, Remington, & Kapur, 2003; Konstantakopoulos et al., 2011). Few studies have investigated motivation deficits in individuals at risk for developing a psychotic disorder.

A lack of motivation is common in schizophrenia (Barch, 2005; Fenton & McGlashan, 1991; Heerey, Robinson, McMahon, & Gold, 2007), and has increasingly become a focus of research. Avolition has been related to both deficits in the integration of information to update and maintain the value of a stimulus (Deci & Ryan, 2000; Deci, Schwartz, Sheinman, & Ryan, 1981; Ryan & Deci, 2000), and taking into account the amount of effort needed in relation to the desired outcome via value computation (Kring & Barch, 2014). Thus, anticipatory anhedonia and avolition are similar yet distinct from one another: whereas both anticipatory anhedonia and avolition involve a diminished ability to accurately predict future consummatory pleasure and reward, avolition encompasses the additional factor of an individual's ability to accurately assess the effort required to obtain a potential, valued reward (Kring & Barch, 2014).

Several studies have highlighted the importance of motivation in schizophrenia, without assessing value or effort computation. These studies find that motivation is negatively correlated with cognition, functioning, and outcome (Foussias, Mann, Zakzanis, van Reekum, & Remington, 2009; Gard, Fisher, Garrett, Genevsky, & Vinogradov, 2009; Nakagami, Hoe, & Brekke, 2010). Recently, the study of motivation has shifted to the earlier developmental stages of psychosis. For instance, Foussias and colleagues found lower levels of motivation to be the single most robust predictor of functional outcome in recent-onset (<5 years) schizophrenia patients, as well as a

mediator for the relationship between cognition and functioning within these patients (Fervaha, Foussias, Agid, & Remington, 2015). Additionally, a study investigating apathy in individuals experiencing their first psychotic episode also demonstrated poorer functioning in those who were the most apathetic (Faerden et al., 2009).

Studies in CHR and schizophrenia populations have also begun to investigate motivation orientation, a construct that encompasses the environmental and personality tendencies that lead to motivation (Breitborde, Kleinlein, & Srihari, 2013; Cooper et al., 2015). Motivation orientations, which come from Causality Orientation Theory (Deci & Ryan, 1985) stemming from a leading macro-theory of motivation known as Self-Determination Theory (Deci & Ryan, 2000; Ryan & Deci, 2000), describe how individuals respond to ambiguous situations; individuals can be more autonomous (intrinsically motivated), controlled (extrinsically motivated), or impersonal (amotivated) in their orientation (Deci & Ryan, 2000). Autonomy oriented / intrinsically motivated individuals are typically motivated by inherent interest and engagement in activities (Deci & Ryan, 2000). Control oriented / extrinsically motivated individuals are more often motivated by external praise and reward (especially monetary), and also away from punishment or criticism (Deci & Ryan, 2000). When individuals lack opportunities for inherent engagement or reward, they may develop a more impersonal/amotivated orientation, and be more disengaged from their environment (Deci & Ryan, 2000). Many studies suggest that individuals across the schizophrenia spectrum have deficits in intrinsic motivation (Fervaha et al., 2013; Gard et al., 2009; Nakagami et al., 2010; Saperstein, Fiszdon, & Bell, 2011). More recent research has indicated that patients

report more amotivation and less intrinsic motivation relative to non-psychiatric controls (Cooper et al., 2015).

Despite evidence linking motivation deficits with schizophrenia populations, there is a dearth of evidence investigating motivation deficits in individuals at CHR, leaving little understanding of whether motivation deficits progress over the course of the disorder. However, one recent study of motivation orientations indicated that individuals experiencing first-episode psychosis reported lower intrinsic motivation and more amotivation than non-psychiatric counterparts (Breitborde et al., 2013). Interestingly, this study also reported that first-episode patients experienced less extrinsic motivation than non-psychiatric individuals, a finding that has not been shown in chronic schizophrenia research. In another study of motivation in CHR individuals, more motivation deficits were found in the CHR populations compared with patients at later stages of schizophrenia, suggesting that there is a spike in motivation deficits before the onset of psychosis (Schlosser et al., 2014). More research will be needed to parse apart the various components of avolition (value computation, effort computation, and motivation orientations) across the psychosis spectrum and especially in those at risk for developing a psychotic disorder.

In sum, research parsing apart and understanding the nuances of negative symptoms is a burgeoning area. Thus far, as discussed above, research shows that negative symptoms are present well before the onset of attenuated or frank psychotic symptoms and continue through to chronic stages of schizophrenia and related psychotic disorders. Specifically, it appears as though chronic, first episode, and CHR patients experience deficits in anticipatory pleasure as well as deficits in motivation and

maintaining emotions. Consummatory pleasure appears to be intact in psychosis, although there are some findings suggesting that those at CHR for psychosis may experience less consummatory pleasure than those in later stages of developing psychosis

Contributions from affective neuroscience

Contributions in understanding negative symptoms in chronic psychosis have been aided by advances in affective science, which bridges research on basic emotion processes with clinical research. A detailed review of this literature is beyond the purview of this paper; what follows is a brief summary of affective science findings as they related to psychosis.

Anhedonia and motivational processes have been studied in both animal and human models (for a review, see Der-Avakian & Markou, 2012). Across models, these processes have been demonstrated to have discrete neurobiological mechanisms. That is, neural circuits related to consummatory anhedonia include the nucleus accumbens, ventral pallidum, and the orbitofrontal cortex, with involvement from mu-opioid, gamma-amniobutyric acid ($GABA_A$), and endocannabinoid receptors. Anticipatory anhedonia involves the anterior cingulate cortex, orbitofrontal cortex, medial prefrontal cortex, basal ganglia, thalamus, and hypothalamus. Motivation is mediated by mesolimbic dopamine pathway (ventral tegmental area to the nucleus accumbens), amygdala mu-opioid receptors, the glutamate pathway from the ventromedial prefrontal cortex to the nucleus accumbens, anterior cingulate cortex, and the lateral hypothalamus. Perhaps most consistently, the ventral striatum has been demonstrated to play a role in motivational processes (Kirschner et al., 2015). Whereas experiential symptoms have been more widely studied, there is little by way of affective neuroscience of expressive symptoms.

Der-Avakian and Markou (2012) were able to determine that although the ventral striatum is involved in aspects of experiential symptoms, it is not involved in diminished expression.

Expressive and experiential deficits have been found to be separate but moderately interrelated clinical symptoms and may, therefore, share some underlying neurobiological mechanisms (Foussias & Remington, 2010). However, a recent article eloquently summarizes that the pathophysiological correlates of distinct negative symptom dimensions remains poorly understood (Kirschner et al., 2015). The literature demonstrating associations between emotion processes—both expressive and experiential—and clinical presentation of schizophrenia is growing, and future research should seek to connect research findings to treatment and intervention. Further, there is little known about the emotion processes in people at CHR for psychosis. By investigating negative symptoms in people at CHR, answers may be found regarding the time course and underlying mechanisms of emotional processes that go awry before the onset of a psychotic disorder, which may provide a target for early intervention and treatment, thus improving the course of psychosis. With time and rigorous studies, affective science research has the potential to greatly contribute to a better understanding of experiential and expressive symptoms.

Limitations and Future Directions

Research assessing negative symptoms and their contribution to other factors in chronic schizophrenia, in first episode of psychosis, and in CHR populations has increased in the past decade. There are, however, many limitations in this work. Integration of findings across different studies is difficult due to the absence of

standardized negative symptom measurement systems (i.e., interviews, self- and informant-reports, laboratory tasks). This becomes especially difficult when attempting to compare the presence/progression of negative symptoms across different stages of psychosis (i.e., non-help-seeking but experiencing attenuated psychotic symptoms, CHR, first episode of psychosis, chronic psychosis). When negative symptoms get combined into a single factor, as many studies have done, application of findings to specific symptomatology is greatly limited. For example, if one study assesses "negative symptoms" by measuring alogia, while another study assesses negative symptoms by measuring anticipatory anhedonia and avolition, it becomes difficult to apply findings to individuals who may experience different constellations of negative symptoms across a continuum of severity and frequency. Further, analyzing summary scores of negative symptoms increases the variance within the experimental group, not only making it difficult to determine the potentially dynamic interplay between different negative symptoms and outcomes, but also making it difficult to detect differences that may exist between groups. Additionally, while negative symptoms have been recently reclassified into a more parsimonious two-factor model (discussed above) (Horan et al., 2011), many researchers continue to assess individual symptoms or report only a total score. Over time, the literature may begin to reflect the definitional shift, but in the meantime, definitional differences across studies will complicate the interpretation and application of research reports.

Methodologically, there are a number of self-report, clinical interview, behavioral, and naturalistic studies used to assess negative symptoms [though there is no consensus on the *best* way to measure them (for review see Blanchard & Cohen, 2006)].

Until assessment tools are agreed upon and/or data indicate that tools can be interpreted in a similar manner, it will remain difficult to determine if an individual who reaches clinical levels on one scale would also reach clinical levels on another scale. Progress has been made to standardize neurocognitive assessment batteries (e.g., the MATRICS Consensus Cognitive Battery; Kern et al., 2011), but no such consensus has been reached yet on how to best measure negative symptoms. A multi-method standardized approach to negative symptom measurement including a clinical interview, such as the CAINS and BNSS, and a self-report measure, such as the MAP-SR, will likely provide data that is more easily interpretable and comparable across and within clinical and research samples, especially those at CHR for psychosis. Additionally, improving the ecological validity of laboratory tasks and/or evaluating ambulatory negative symptoms in people at CHR could dramatically improve the field's understanding of and approach to the development of negative symptoms and their contribution to the development of psychosis.

Currently, the primary diagnostic tools for psychosis and those at CHR for psychosis rely heavily on positive symptom domains (Miller, McGlashan, Rosen, Cadenhead, Cannon, Ventura, Mcfarlane, et al., 2003), which may tend to limit the clinical assessment of negative symptom profiles. A shift in diagnostic emphasis and in treatment planning, to include negative symptom assessment could potentially help support efforts to develop treatment strategies for negative symptoms and potentially for co-occurring symptoms that may be affected by negative symptoms. Research has already shown that when negative symptoms improve, so do a variety of functional outcomes, including independent living skills, social functioning, and role functioning (Velligan et al., 2009). Though pharmaceutical treatments for negative symptoms may be

helpful, another major limitation in researching negative symptoms in people experiencing psychotic symptoms is that the vast majority of research participants receive medications (at least to some degree) for positive symptoms even before the onset of florid psychotic symptoms (reviewd in Cornblatt, Lencz, & Obuchowski, 2002), potentially altering the natural trajectory of symptom development, given findings suggesting that typical antipsychotic, and some atypical antipsychotic, medications can increase negative symptoms (Buckley & Stahl, 2007). Though there are a number of studies in medication-naïve individuals, none of these studies have assessed both negative symptoms and other symptoms, such as cognitive deficits and functional outcomes, thereby limiting the ability to effectively investigate the progression and impact of negative symptoms on other aspects of developing psychosis. Finally, people who are medication-naïve could be an altogether different population than medicated individuals, (if, for instance, medication has not been started due to financial limitations, personal reasons for medication avoidance, inadequate or unavailable medical advice, or less severe symptoms, etc.), thereby potentially limiting applicability of findings.

In addition to the many limitations already mentioned, to our knowledge, no studies have reported any findings about negative symptoms occurring in earlier stages of emerging psychosis (i.e., before clinical help is sought) or in in those experiencing the continuum of psychotic experiences. Given that early and persistent negative symptoms have been shown to represent a vulnerability for the development of schizophrenia (Piskulic et al., 2012), understanding the experience and expression of negative symptoms earlier in the psychosis trajectory of psychosis, including in those experiencing a range of attenuated positive psychotic symptoms, may help differentiate those who may

be at risk for developing psychosis from those who may be at risk for developing other mental health disorders, such as mood disorders. Enhanced understanding of the early stages of psychosis would allow for a more targeted approach to intervention and treatment, which, in turn, may help to quickly get supports in place when an individual develops psychosis and may help delay the onset of psychosis, or even prevent transition altogether.

Given the substantial burden to society, caregivers, and individuals afflicted with schizophrenia and other psychotic disorders, it is imperative that new interventions and treatment for negative symptoms be developed. Understanding negative symptoms during the critical time period before psychosis fully emerges is likely to provide insight into methodological approaches in developing these early interventions and treatments. Further, determining the neurocognitive and functioning profiles in individuals who are endorsing psychotic-like experiences but not seeking help may contribute to understanding deficits and deterioration early in the trajectory of psychosis.

CHAPTER 2

MANUSCRIPT ONE IN JOURNAL ARTICLE FORM

Abstract

Deficits in anticipatory pleasure, intrinsic motivation, and amotivation (jointly, motivation and pleasure—MAP—symptoms) have been consistently shown among chronic, first-episode, and clinical high risk for psychosis populations, but much less attention has been given to non-clinical individuals experiencing psychotic-like experiences (PLEs). Young adults (N = 1,454) were administered the Temporal Experience of Pleasure Scale (TEPS), which measures pleasure, the General Causality Orientation Scale for Clinical Populations (GCOS-CP), which measures motivation, and the Prodromal Questionnaire, which measures PLEs. Analyses examined (a) total PLEs endorsed, and (b) comparisons of groups experiencing PLEs that were endorsed as distressing (distressing PLEs = PLEDs; experiencing more PLEDs = High PLEDs group, a potentially more clinically meaningful group, n = 246; experiencing fewer PLEDs = Low-PLEDs group, n = 720). Results indicated that patterns of MAP deficits in non-clinical young adults mirror findings in psychotic populations, suggesting that MAP deficits appear to occur along the entire continuum of psychotic experiences.

Introduction

Deficits in motivation and pleasure (MAP), which are sometimes characterized as *experiential negative symptoms*, have been demonstrated in psychosis; however, less is known about MAP early in the course of psychosis. Negative symptoms of psychosis, which include MAP deficits, are minimally responsive to treatment (Kirkpatrick et al., 2006), are experienced by approximately 28%-36% of people with schizophrenia (Blanchard, Horan, & Collins, 2005), and persist even when positive symptoms (e.g., hallucinations, delusions) remit (Tamminga, Buchanan, & Gold, 1998). While several studies point to a link between negative symptoms and adverse outcomes in people diagnosed with psychotic disorders, fewer studies have characterized the nature of negative symptoms over the developmental course of psychosis. Nonetheless, deficits associated with negative symptoms, including MAP deficits, include increased stress and decreased subjective well-being (Blanchard et al., 1998), poor self-efficacy and low self-esteem (Ritsner, 2014), poorer premorbid functioning (Chapman et al., 1976; Garnet et al., 1993), low social functioning and less social support (Ritsner, 2014), increased emotional distress (Myin-Germeys, 2001), and substantial functional impairment (Foussias et al., 2011; Foussias & Remington, 2010).

A lack or loss of pleasure, known as anhedonia, has long been considered a key symptom in schizophrenia (Bleuler, 1950; Kraepelin, 1919; Rado, 1953), and is implicated in a number of adverse outcomes (Blanchard et al., 1998; Chapman et al., 1976; Garnet et al., 1993; Myin-Germeys, 2001; Ritsner, 2014). A wealth of literature demonstrates the importance and separate nature of consummatory, or in-the-moment, and anticipatory, or non-current/future, pleasure (for review see Kring & Elis, 2013).

Research in chronic psychosis populations, with schizophrenia being the most quintessential form, have consistently demonstrated anticipatory, but not consummatory, anhedonia (Gard et al., 2006, 2007; Kring & Elis, 2013; Kring et al., 2011; Mote, Stuart, et al., 2014). Less work has been conducted at earlier points in the development of disorder, but studies show that individuals with recent onset of a psychosis also experience less anticipatory pleasure than either non-psychiatric controls or chronic patients (Mote, et al., 2014; Schlosser et al., 2014). Only one study, to date, has investigated anticipatory pleasure in people at clinical high risk (CHR) for psychosis. Results of that study indicated that people at CHR for psychosis experienced more severe anticipatory pleasure deficits—and, surprisingly, more consummatory pleasure deficits—as compared to same-aged controls, suggesting that both consummatory and anticipatory pleasure deficits may be an indicator of risk for psychosis (Schlosser et al., 2014). Recently, work from Cooper and colleagues (Cooper, Kring, & Ellman, In press) have extended these findings to young adults who reported psychotic-like experiences (PLEs). Results of that study demonstrated that as anticipatory, but not consummatory, pleasure decreased, PLEs increased. Further, Cooper and colleagues (In Press) found that decreased anticipatory pleasure was significantly associated with increased odds of experiencing more numerous distressing PLEs, a potentially more clinically relevant group. Thus, deficits in pleasure are a crucial area of impairment in schizophrenia and other psychotic populations that should continue to be investigated at early points on the continuum of psychotic experience as pleasure deficits may confer risk for psychosis.

Motivation dysregulation is also common in schizophrenia, with the lack of motivation—known as *avolition*—having long been characterized as a core feature of

schizophrenia (Bleuler, 1950). Motivation orientations—autonomy, controlled, impersonal/amotivated—from Causality Orientation Theory, define behavioral tendencies that people use in ambiguous situations to interpret their behavior and the environment (Deci & Ryan, 1985). Although they do not completely align, these orientations are often interpreted as intrinsic motivation (autonomy orientation), extrinsic motivation (controlled orientation), and amotivation (impersonal/amotivated orientation). Autonomy oriented / intrinsically motivated people are driven by inherent interest, acting as their own agent, and how activities deepen their experiences. Control oriented / extrinsically motivated people use external praise and reward and/or avoidance of external punishment or criticism to drive their behavior. People who are impersonal/amotivation oriented tend to feel disengaged from their actions and as though their behaviors to not impact their environment. We will be using the terms *intrinsic motivation, extrinsic motivation,* and *amotivation* for the sake of simplicity. Findings suggest that people with schizophrenia or schizoaffective disorder have decreased intrinsic motivation and increased amotivation, but that extrinsic motivation is intact in chronic populations (Choi et al., 2010; Gard et al., 2014; Medalia & Brekke, 2010; Nakagami et al., 2010). Less is known in early psychosis samples, and although investigating motivation in first episode and CHR samples is in its infancy, early studies have demonstrated results that mirror chronic schizophrenia findings. One study assessing motivation in a first episode cohort found that those in their first episode of psychosis demonstrated lower intrinsic motivation and higher amotivation than non-psychiatric controls (Breitborde et al., 2013). A recent study investigating motivational factors in people at CHR indicated that CHR participants were more severely impaired on motivation compared to people early in the course of

psychosis and chronically patients diagnosed with schizophrenia (Schlosser et al., 2014). Cumulatively, these findings suggest that motivation dysregulation appears to be a common experience across different stages of psychosis.

In schizophrenia samples, a wealth of research has reported that decreased intrinsic motivation is associated with impairments in neurocognition, social cognition, occupational functioning, and daily functioning (Fervaha et al., 2015; Fervaha, Foussias, et al., 2013; Gard et al., 2009; Nakagami et al., 2010; Saperstein et al., 2011). Additionally, increased amotivation has been associated with decreased functional outcome and quality of life in schizophrenia populations (Foussias, Mann, Zakzanis, van Reekum, & Remington, 2009; Gard et al., 2009; Nakagami et al., 2010). Although there have been fewer studies in first episode and CHR samples, intrinsic motivation is demonstrated as being significantly associated with functional outcome (Breitborde et al., 2013; Schlosser et al., 2014) and social functioning (Schlosser et al., 2015). Thus, motivation dysregulation appears to be a central component to numerous deficits across the psychosis continuum. Understanding motivation impairment prior to onset of clinically significant symptoms and/or help-seeking is an important next step in devising empirically-driven treatment approaches that people at risk actively engage in prior to transition and in the early stages of psychosis.

As discussed above, studies investigating MAP deficits have almost exclusively focused on people with clinically significant symptoms of psychosis or those at CHR for psychosis. There is an inescapable link between the lack of motivation (avolition) and the lack of pleasure (anhedonia) and it may not be possible to fully disentangle these experiential negative symptoms from one another as avolition and anhedonia may reflect

a common underlying process (Foussias & Remington, 2010). Although pleasure and motivation are often evaluated together and their individual and joint contributions are often assessed on outcomes such as functioning and cognition (Ventura, Hellemann, Thames, Koellner, & Nuechterlein, 2009), there have been few attempts to disentangle the separable contribution of these constructs and investigate potential interactions between motivation and pleasure. While factor structure studies consistently show the interrelations between MAP symptoms (Horan et al., 2011), the relationship of pleasure to motivation in everyday life remains poorly understood (Engel, Fritzsche, & Lincoln, 2013). Furthermore, little is known about MAP deficits in people with psychotic-like experiences (PLEs) that do not meet diagnostic criteria for a psychosis risk syndrome (although see Cooper et al., In press). Psychotic symptoms are comprised of a continuum of perceptual abnormalities, paranoia and suspiciousness, disorganization, and unusual thinking, though only the most frequent and/or debilitating are considered clinically relevant (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). However, constraining psychosis research to symptoms of clinical severity restricts the applicability of findings. Assessing the dimensionality of psychotic symptoms is essential for exploring an extended psychosis phenotype, given that individuals who experience PLEs that do not reach clinical severity are at greater risk for developing a psychotic disorder (van Os & Linscott, 2012). In fact, among those at CHR for psychosis, inclusion of negative symptoms—including MAP symptoms—increases prediction of conversion to a psychotic disorder by 10 percent for every 1-point increase in negative symptoms on a subscale of the Structured Interview for Psychosis Risk Syndromes (SIPS) (Schlosser et al., 2012). This suggests that understanding how MAP deficits are associated with

subthreshold psychotic symptoms may help us with early intervention and prevention strategies.

As such, the present study sought to assess the relationship between MAP symptoms and PLEs in a non-clinical, young adult sample. Specifically, the present study examined the relationships between anticipatory and consummatory pleasure and motivation (Cooper et al., 2015) in those experiencing a range of PLEs, including PLEs that are experienced as distressing as they may be more clinically relevant (Loewy, Bearden, Johnson, Raine, & Cannon, 2005; Loewy, Johnson, & Cannon, 2007). In line with the aforementioned research in chronic schizophrenia samples, we hypothesized that (1) when controlling for other subscales, anticipatory, but not consummatory, pleasure deficits as well as decreased intrinsic motivation and increased amotivation, but not extrinsic motivation, would predict increases in number of PLEs, (2) lower anticipatory pleasure and more impaired motivation (i.e., decreased intrinsic motivation and increased amotivation) would interact to moderate increases in PLEs (3) the same pattern of individual predictors and interactions would predict those who experience more distressing PLEs (PLEDs), and are potentially higher risk of developing psychosis (High PLEDs group), compared to those who experience few PLEDs, and are at presumed lower risk for psychosis (Low PLEDs group) (see "Self-Report Measures" for how groups were determined).

Method

Participants

Participants (N = 1,454) were racially/ethnically and socioeconomically diverse sample of male and female young adults from a non-clinical sample; a subset of the

participants' data were reported in a previous study (Cooper, Kring, Ellman, In press). Participants were recruited via an online subject recruitment website and received college course credit for their participation. The online recruitment website, which was open to all undergraduate students at the university, listed the current study with all other available studies that one could choose to participate in; studies are listed in random order. To participate, an individual would select our study from the list of options, be provided information regarding what the study would entail and where it was located, and then choose a day and time that was conducive to their schedule from the options available. For inclusion, participants were to be between the ages of 18 and 35, which corresponds to the typical age range of individuals developing schizophrenia (*DSM 5*, 2013), and fluent in English. See Table 1 for participant characteristics.

Procedures

Following informed consent, participants were directed to a laboratory computer terminal at which questionnaires were individually, electronically administered (Survey Monkey Inc., Palo Alto, CA). Demographic characteristics were first collected, followed by the administration of additional questionnaires, including the Prodromal Questionnaire (Loewy et al., 2005, 2007), TEPS (Gard et al., 2006), and GCOS-CP (Cooper et al., 2015).

Table 1. Participant demographics and characteristics.

	Total Sample (*N* =1,454)	**Low PLEDs (*n*=720)**	**High PLEDs (*n*=246)**	***p*-value**
Gender: F%(M%)	74.6 (25.4)***	72.9 (27.1)	78.5 (21.5)	.085
Age: M (SD) [Range]	20.15 (2.48) [18-35]	20.20 (2.64) [18-35]	19.84 (2.01) [18-30]	.027*
Race/Ethnicity: N (%)				.880
Caucasian/White	862 (59.3)	445 (61.8)	148 (60.2)	
Af.-American/Black	249 (17.1)	108 (15.1)	40 (16.0)	
Asian/Pac. Islander	193 (13.3)	95 (13.2)	37 (14.9)	
Hispanic/Latino	63 (4.3)	30 (4.1)	9 (3.6)	
Other	87 (6.0)	42 (5.8)	12 (4.6)	
PLEs	9.47 (7.54) [0-44]	5.65 (4.72) [0-30]	20.20 (6.78) [9-44]	<.001***
PLEDs	3.75 (4.76) [0-28]	0.92 (1.05) [0-3]	12.37 (4.60) [8-28]	<.001***
TEPS:				
Anticipatory	47.42 (7.18) [15-60]	48.16 (6.90) [15-60]	45.86 (8.05) [18-60]	<.001***

Consummatory	37.50 (6.44) [8-48]	38.00 (6.47) [8-48]	37.14 (6.01) [12-48]	.070^
GCOS-CP:				
Intrinsic	43.06 (8.58) [8-56]	43.79 (8.74) [8-56]	41.54 (8.02) [11-56]	<.001***
Extrinsic	27.09 (7.36) [8-56]	26.83 (7.72) [8-56]	27.49 (6.84) [8-56]	.234
Amotivation	28.77 (7.77) [8-56]	27.45 (7.57) [8-56]	31.56 (7.64) [15-56]	<.001***

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; TEPS = Temporal Experience of Pleasure Scale; GCOS-CP = General Causality of Orientations Scale for Clinical Populations; *p*-values are the result of either independent sample *t*-tests or $\boldsymbol{X^2}$, as appropriate, comparing High- and Low PLED groups; ^ Trend level significance; * Significant at the <.05 level; ** Significant at the <.01 level; *** Significant at the <.001 level

Self-Report Questionnaires

The Prodromal Questionnaire (PQ) (Loewy et al., 2005, 2007). The PQ is a 92-item, self-report questionnaire that measures subthreshold psychotic symptoms experienced in the past month in four domains: positive, negative, disorganized, and general. The PQ also requires participants to endorse whether symptoms were experienced as distressing. The PQ has been tested against semi-structured interviews commonly used to identify individuals at risk for psychosis and has been found to be both reliable and valid in comparison (Loewy et al., 2005).

We focused on the positive symptom domain, as this domain has been associated with increased risk for psychosis and has been primarily studied in investigations of PLEs in the general population (Cannon et al., 2008; Loewy et al., 2007). In line with previous research, distressing items are an additional focus for the current study, as those who endorse distressing items are at a higher likelihood of developing a psychotic disorder (Hanssen, Bijl, Vollebergh, & Van Os, 2003; Loewy et al., 2007). PLEs were examined continuously (total number of PLEs endorsed), as well as categorically (i.e., High PLEDs = potentially higher risk for psychosis; Low PLEDs = potentially lower risk). Group membership for the categorical variable was determined by endorsement of 8 or more distressing PLEs (High PLEDs, current study n =246) compared to three or fewer distressing PLEs and 8 or fewer total PLEs (Low PLEDs group; the means of PLEs distressing and total PLEs symptoms in our larger undergraduate sample, current study n = 720) (Gibson et al., 2014; Reeves et al., 2014). Endorsing 8 or more positive symptoms has been found to have 90% sensitivity and 49% specificity with individuals identified as

CHR using the SIPS in a clinical population, and in an undergraduate sample 2% met this criterion if 8 symptoms were endorsed as distressing (Loewy et al., 2005, 2007).

Temporal Experience of Pleasure (TEPS) (Gard & Kring, 2009). The TEPS, an 18-item self-report questionnaire, is designed to index trait disposition to experience anticipatory pleasure (i.e., pleasure associated with wanting a given reward or experience) and consummatory pleasure (i.e., pleasure associated with liking a given activity or experience in-the-moment). The TEPS includes the 10-item anticipatory pleasure subscale, and the 8-item consummatory pleasure subscale. This scale is widely used to assess pleasure deficits in individuals with a diagnosis of or at risk for schizophrenia and other psychotic disorders (Cohen & Minor, 2010). The TEPS yields high levels of internal consistency ($\alpha = .79$ overall, $\alpha = .74$ anticipatory, $\alpha = .71$ consummatory) (Gard, Gard, Kring, & John, 2006), is one of the most commonly used self-report measure of hedonic functioning in psychotic disorders, and has recently been used to demonstrate anticipatory pleasure deficits in young adults reporting PLEs (Cooper et al., In press).

General Causality Orientation Scale for Clinical Populations (GCOS-CP) (Cooper et al., 2015)**.** The GCOS-CP is a self-report measure adapted from the General Causality Orientation Scale (Deci et al., 1981) designed to measure motivation orientation, or how individuals respond to ambiguous situations; individuals can be more autonomous (intrinsically motivated), controlled (extrinsically motivated), or impersonal (amotivated) in their orientation (Deci & Ryan, 2000). Autonomy oriented/intrinsically motivated individuals are typically motivated by inherent interest and engagement in activities (Deci & Ryan, 2000). Control oriented/extrinsically motivated individuals are

more often motivated by external praise and reward (especially monetary), and also away from punishment or criticism (Deci & Ryan, 2000). When individuals lack opportunities for inherent engagement or reward, they may develop a more impersonal/amotivated orientation, and be more disengaged from their environment (Deci & Ryan, 2000). In this study, we will be referring to causality orientations as follows: autonomy = intrinsic motivation, control = extrinsic motivation, impersonal = amotivated. Various studies have found deficits in motivation in those suffering from schizophrenia (Gard et al., 2009; Nakagami et al., 2010), as well as those in their first episode of psychosis (Breitborde et al., 2013), and those at CHR for psychosis (Schlosser et al., 2014). The GCOS-CP asks participants to respond to each of the three motivation orientations (autonomy, controlled, impersonal) on 8 vignettes, totaling 24 responses. Each response is indicated on a 1 to 7 Likert scale (i.e., 1 = *Very Unlikely*, 4 = *Moderately Likely*, 7 = *Very Likely*). This measure has acceptable internal consistency in non-clinical (α = .74 autonomy/intrinsic, α = .65 controlled/extrinsic, α = .67 impersonal/amotivated) and clinical (α = .77 autonomy/intrinsic, α = .52 controlled/extrinsic, α = .57 impersonal/amotivated) samples.

Statistical Analytic Plan

Participant ages were checked to ensure that participants from the sample were within the typical age of onset for schizophrenia (18 - 35 years old) (American Psychiatric Association, 2014); all participants reported being within this age range. Data analyses used the total sample of 1,454 participants. Age and gender were examined as potential covariates by conducting bivariate analyses with the main study variables. Linear regressions were conducted to (1) determine the relation between anticipatory and consummatory pleasure, as well as intrinsic motivation, extrinsic motivation, and

amotivation with PLEs, and (2) to assess the possibility of interactions between anticipatory pleasure, and the motivation variables (each in a separate model-intrinsic motivation, amotivation) and PLEs. Significant variables from the first model were subsequently included to improve model fit. We also conducted logistic regressions with (3) pleasure deficits and motivation orientations, as well as (4) the aforementioned interaction terms, as predictors and High PLEDs status as the outcome. Only significant variables from the initial model were subsequently included to improve model fit.

Results

Our sample was nearly representative of the community from which it was drawn, although there were significantly more females than males in the overall sample. Gender (i.e., being female) was found to be related to anticipatory pleasure ($p < .001$), consummatory pleasure ($p < .001$), and intrinsic motivation ($p = .002$). Gender (i.e., being male) was also found to be significantly related to PLEs ($p = .024$), and was approaching trend level for number of distressing PLEs endorsed ($p = .114$), but was not related to PLED status ($p = .453$). Because there was an uneven distribution of gender between the High PLED and Low PLED groups, and because gender was significantly related to several main study variables, we chose to take a conservative approach and include gender as a covariate both for predicting PLEs and PLEDs. Additionally, age was found to be inversely associated to PLEs and PLED status at non-significant levels, and was significantly negatively related to anticipatory pleasure ($p < .001$); therefore, age was controlled in all analyses. Table 1 provides demographic characteristics for our full sample, as well as our High PLEDs ($n = 246$) and Low PLEDs ($n = 720$) groups.

Table 2. Correlation matrix

	1	2	3	4	5	6	7	8	9
1 Age	-	-.124**	-.049	-.059*	-.098**	-.039	.041	.043	-.019
2 Gender		-	-.059*	.042	.218**	.105**	.090**	-.033	-.023
3 PLE			-	.794**	-.102**	-.050	-.080**	.049	.176**
4 PLED				-	-.108**	-.046	-.086**	.050	.200**
5 TEPS: Anticipatory					-	.466**	.275**	.169**	-.027
6 TEPS: Consummatory						-	.274**	.020	-.015
7 GCOS-CP: Intrinsic Motivation							-	.225**	-.090**
8 GCOS-CP: Extrinsic Motivation								-	.258**
9 GCOS-CP: Amotivation									-

PLE = psychotic-like experiences, PLED = distressing psychotic-like experiences, TEPS = Temporal Experience of Pleasure Scale, GCOS-CP = General Causality Orientation Scale for Clinical Populations, * $p < .05$, ** $p < .01$

As shown in Table 3, linear regression results indicated that decreases in anticipatory pleasure and increases in amotivation were significantly associated with increases in PLEs, whereas there were no significant associations between PLEs and consummatory pleasure or extrinsic motivation. Increases in intrinsic motivation were associated with non-significant, trend level increases in PLEs. No significant interactions

Table 3. Regression and logistic regression models examining the relation between anticipatory/consummatory pleasure and motivation orientations with PLEs and High PLEDs.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				<.001***			<.001***
Age	-.059	-.179	-2.268	.023*	.934	.872-1.000	.051^
Gender	-.039	-.668	-1.453	.147	.731	.502-1.064	.102
TEPS Anticipatory	-.095	-.100	-3.176	.002**	.959	.939-.983	.001**
TEPS Consummatory	.012	.010	.332	.740	1.010	.982-1.039	.491
GCOS-CP Intrinsic	-.050	-.044	-1.779	.075^	.979	.960-.998	.028**
GCOS-CP Extrinsic	.035	.036	1.262	.207	1.008	.986-1.030	.500
GCOS-CP Amotivation	.160	.155	5.928	<.001***	1.069	1.047-1.091	<.001***
R^2	.047						
Model 2a				<.001***			<.001***
Age	-.059	-.179	-2.268	.023*	.932	.870-.998	.044*
Gender	-.039	-.668	-1.453	.148	.731	.502-1.064	.102
TEPS Anticipatory	-.095	-.100	-3.165	.002**	.951	.928-.976	.001**
GCOS-CP Intrinsic	-.045	-.040	-1.603	.100^	.978	.959-.997	.024*
GCOS-CP Amotivation	.158	.153	5.803	<.001***	1.067	1.046-1.090	<.001***

Anticipatory X Intrinsic	-.007	-.045	-.275	.783	.937	.824-1.067	.937
R^2	.048						
R^2 change	.001						
Model 2b				<.001***			<.001***
Age	-.059	-.179	-2.264	.024*	.934	.872-1.001	.052^
Gender	-.039	-.672	-1.459	.145	.734	.504+1.070	1.08
TEPS Anticipatory	-.094	-.098	-3.071	.002**	.954	.931-.978	<.001***
GCOS-CP Intrinsic	-.045	-.039	-1.590	.102^	.978	.959-.998	.030*
GCOS-CP Amotivation	.158	.154	5.857	<.001***	1.069	1.047-1.091	<.001***
Anticipatory X Amotivation	-.004	-.031	-.171	.864	.962	.838-1.104	.578
R^2	.047						
R^2 change	.000						

PLEs = psychotic-like experiences; High PLEDs = individuals who endorsed 8 or more distressing PLEs and may be at higher clinical risk for psychosis; TEPS = Temporal Experience of Pleasure Scale; GCOS-CP = General Causality Orientation Scale for Clinical Populations; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the <.05 level; ** Significant at the <.01 level; *** Significant at the <.001 level.

were found in any of the regression models. Similarly, logistic regression results indicated that decreases in anticipatory pleasure, decreases in intrinsic motivation, and increases in amotivation were associated with significantly increased odds of being classified as High PLEDs. No interactions were found in any of the logistic regression models.

All findings were replicated in unadjusted analyses. They are not included herein for brevity.

Discussion

The findings from the present study suggest that patterns of MAP deficits in non-clinical young adults mirror findings in psychotic populations, suggesting that MAP deficits appear to occur in those experiencing the full range of psychotic experiences. The current study extends previous findings from our group indicating anticipatory, but not consummatory, pleasure deficits are evident in people with PLEs, especially those experiencing High PLEDs (Cooper et al., In Press). In the current study, we specifically found that anticipatory pleasure deficits, intrinsic motivation, and amotivation are significantly and independently associated with increases in PLEs in a population of non-clinical young adults. Further, we found the same pattern (i.e., decreases in anticipatory pleasure, increases in amotivation, and decreases in intrinsic motivation) significantly and independently increased the odds of being at potentially higher risk of developing psychosis (High PLEDs group). While the magnitude of our findings are small (i.e., betas and odds ratios), our results are consistent with the larger literature, suggesting that MAP deficits appear to occur along the entire continuum of psychotic experiences. These

findings provide additional evidence for an extended psychosis phenotype and provide potential targets for investigation in early intervention and prevention studies.

As expected, we found that decreases in consummatory pleasure and extrinsic motivation were not associated with PLEs or PLEDs. While these findings extend the literature among people in latter stages of developing psychosis, our consummatory pleasure results are inconsistent with one recent study that found decreased consummatory pleasure among individuals at CHR for psychosis compared to age-matched, non-psychiatric controls and to individuals further along the course of psychosis (Schlosser et al., 2014). It is possible that as subthreshold psychotic symptoms become severe enough to warrant clinical attention, there is an anomalous decrease in consummatory pleasure specific to that developmental period of the emergence of psychosis. It is also possible that comorbid symptoms, such as those related depression, may account for increases in consummatory pleasure deficits during this period, as depression frequently occurs during the prodrome of psychosis (Demmin et al., 2013; Piskulic et al., 2012). Additional work investigating consummatory pleasure deficits in the earliest stages of developing a psychotic disorder is clearly needed. Lastly, although we did not find interactions between anticipatory pleasure, intrinsic motivation, and amotivation, this could be because of the method (self-report questionnaires) used. Future studies should seek to utilize multiple methods—self-report questionnaires, laboratory tasks, clinical interviews, ecological momentary assessment—to more fully investigate potential interactions between motivation and pleasure in emerging psychosis.

Motivation is a complex construct and recent research has considered the joint contributions of hedonic functioning and effort in understanding the motivation/reward

system (Kring & Barch, 2014; Wang et al., 2015). Motivated behavior requires initiation and persistence in goal-directed activity and heavily relies on the experience of pleasure to engage in these processes (Barch, 2008; Barch & Dowd, 2010; Kring & Barch, 2014). Research in schizophrenia samples have shown that reduced anticipatory pleasure and effort (i.e., motivation) remain depressed in people with schizophrenia regardless of the magnitude of the reward (Wang et al., 2015). Additionally, there have been suggestions that the decreases in social functioning (Ritsner, 2014) and daily functioning (Foussias et al., 2011) evident in the prodrome of psychosis may be related to dysfunction in the reward system. Although we did not investigate motivation and pleasure with regard to reward, we did find that some MAP factors (i.e., anticipatory pleasure, intrinsic motivation, amotivation) are important in predicting increased PLEs/PLED status. Further, the degree to which the reward system, including hedonic capacity and motivation, appears to be impaired prior to onset of psychosis has provided predictive validity for CHR populations; that is, increased negative symptoms in people at CHR is associated with poorer functional outcomes and lower rates of recovering from an at-risk psychotic state (Addington et al., 2011; Schlosser et al., 2012). The connection between increases in PLEs/PLEDs and the reward system needs to be further investigated as MAP deficits may disrupt the likelihood of an individual at CHR for psychosis seeking, engaging in, and/or adhering to treatment, which may, in turn, result in poorer prognoses (Medalia & Brekke, 2010; Nakagami, Xie, Hoe, & Brekke, 2008).

This study is particularly notable for its large, diverse sample. Nevertheless, results from this study may not generalize outside of college populations given that the sample consisted of college students, even though the publicly funded university is

comprised of students who are quite diverse from a socioeconomic and ethnic/racial standpoint. Additionally, distinguishing MAP deficits across psychotic spectrum may help researchers target the most helpful point along the psychosis spectrum to intervene with treatments targeted to these symptoms. Finally, findings from this study provide additional evidence for an extended psychosis phenotype. The importance of investigating and assessing psychotic experiences dimensionally is underscored by research supporting the existence of an extended psychosis phenotype, whereby PLEs endorsed by non-clinical individuals have been linked to risk for developing a psychotic disorder, and subclinical and clinical psychosis have been found to have overlapping risk factors (van Os & Linscott, 2012; van Os et al., 2009). Specifically, our data further support an extended psychosis phenotype by providing evidence of previously unexplored shared factors—anticipatory pleasure and intrinsic motivation deficits as well as increased amotivation—between non-clinical young adults who experience increased subthreshold psychotic symptoms and in those with psychotic disorders.

This study is not without limitations. Most notably, the cross-sectional design of this study means we do not know which individuals may transition to psychosis. Second, while we controlled our analyses by gender and by age, and found no significant results, there were significantly fewer men than women in the study and age differed by group, although the latter findings were very slight and may not be meaningful (i.e., not even half a year mean difference between groups). Nonetheless, future research should seek to remediate the gender imbalance when assessing those with PLEs/PLEDs. Additionally, our self-report inventories have limitations. The TEPS is limited in measuring consummatory pleasure because it specifically measures semantic, but not experiential,

emotion knowledge (Gard et al., 2007). Experiential emotion knowledge is important to truly understand pleasure in consummation (Feldman Barrett, Mesquita, Ochsner, & Gross, 2007). The GCOS-CP provides only a momentary glimpse of a person's motivation orientation and limits our understanding of fluctuations of motivation over time or due to other influences (e.g., changes in symptoms, exogenous stressors) (Cooper et al., 2015). Both the TEPS and GCOS-CP are limited by a participant's insight into his/her own behavior. As such, future studies would benefit from using additional measures, including other self-report tools, collateral reports, and through ecological momentary assessment studies, to better capture experiential emotion and motivation. Further, the TEPS focuses on measuring physical, but not social, anhedonia, and people at risk for and with psychotic disorders are known to have deficits in social relationships and likely derive less pleasure in social situations (Gard & Kring, 2009). Understanding both physical and social anticipatory and consummatory pleasure deficits is an important future direction for new studies. Additionally, depression (which shares the symptom of anhedonia with schizophrenia and is also associated with a lack of motivation) and negative symptoms are frequently seen in the prodrome of psychosis (Demmin et al., 2013; Piskulic et al., 2012). Finally, with the array of negative outcomes associated with MAP deficits in psychotic populations, future studies should seek to assess the relation between these deficits and associated negative outcomes (e.g., cognitive problems, functioning) at earlier stages of developing a psychotic disorder, including those with PLEs/PLEDs.

In sum, non-clinical young adults who reported lower levels of anticipatory pleasure, decreased intrinsic motivation, and increased amotivation also reported more

PLEs. Additionally, lower levels of anticipatory pleasure, increased amotivation, and decreased intrinsic motivation increased the odds of being at a potentially higher risk for psychosis (High PLEDs group). Future studies should longitudinally assess MAP deficits to determine hedonic functioning and motivation differences—and their correlates—in people who transition to a psychotic disorder relative to those who do not.

CHAPTER 3

MANUSCRIPT TWO IN JOURNAL MANUSCRIPT FORM

Abstract

Deficits in motivation and pleasure (MAP) are widely noted across the developmental trajectory of clinical psychosis, with recent evidence suggesting that some of these deficits occur in people with psychotic-like experiences (PLEs). Impairment of episodic memory—memory of autobiographical events—is also a common feature of psychosis that has been noted to occur at all stages of psychosis, including clinical high risk, first episode and later stages of the disorders. Negative symptoms, including MAP deficits, have been linked to cognitive deficits, including problems with memory, in people who seek help for psychosis, but less is known about how MAP symptoms and episodic memory may interact in those with PLEs, especially PLEs endorsed as distressing (PLEDs) in non-clinical samples. Young adults (N = 587) were administered the Temporal Experience of Pleasure Scale (TEPS), which measures pleasure; the General Causality Orientation Scale for Clinical Populations (GCOS-CP), which measures motivation; the Relational and Item-Specific Encoding Task (RiSE), which measures aspects of episodic memory; and the Prodromal Questionnaire, which measures PLEs. Analyses examined additive and interactive models of episodic memory (i.e., item recognition and associative recognition based on different encoding strategies) and MAP symptoms (specifically, amotivation and anticipatory pleasure) in their contributions to (a) total PLEs endorsed, and (b) dichotomized PLEDs groups (High PLEDs = experiencing more PLEDs, a potentially more clinically meaningful group, n = 119); Low PLEDs = experiencing fewer PLEDs, n = 334). Findings indicate that associative

recognition using relational encoding acts additively with both increases in amotivation and decreases in anticipatory pleasure to significantly increase odds of being High PLEDs, but not continuous PLEs. Implications of these findings are discussed.

Introduction

Deficits in motivation and pleasure (MAP) have long been noted in psychosis (Bleuler, 1950), and are prominent across the developmental trajectory of schizophrenia, including at the chronic, first episode (FE), and clinical high risk (CHR) stages of the disorder (Kring & Barch, 2014; Llerena et al., 2013; Mote, Minzenberg, et al., 2014; Schlosser et al., 2014; Wang et al., 2015). Findings suggest that, as compared to non-psychiatric controls, individuals with psychosis have lower levels of anticipation of pleasure and intrinsic motivation and higher levels of amotivation (Cooper et al., 2015; Gard et al., 2007). Although less is known about MAP symptoms in people prior to when they first seek help, one recent study identified found that low anticipatory pleasure was associated with higher psychotic-like experiences (PLEs), as well as a greater likelihood of being a potentially higher risk for psychosis (Cooper, Kring, Ellman, In Press).

MAP symptoms are important to investigate because they, along with other negative symptoms, are minimally responsive to treatment (Kirkpatrick et al., 2006), are experienced by approximately 28%-36% of people with schizophrenia (Blanchard et al., 2005), and persist even when positive symptoms (e.g., hallucinations, delusions) remit (Tamminga et al., 1998). Several studies point to a link between negative symptoms (including MAP deficits) and adverse outcomes, such as poorer cognition and memory deficits, in people at CHR for psychosis and in those diagnosed with schizophrenia (Foussias, Siddiqui, Fervaha, Agid, & Remington, 2015; Gard et al., 2009; Nakagami et al., 2008; Schlosser et al., 2015; Strauss et al., 2012). Specifically, findings suggest that MAP symptoms negatively impact neurocognition, social cognition, and functional outcome in people with schizophrenia (Gard et al., 2009) and are associated with poorer

global and social functioning (Fulford et al., 2013; Schlosser et al., 2015). Although there has been some research connecting negative symptoms to cognitive deficits, including memory problems, in the latter stages of disorder, this area of research has only recently begun to touch on the early developmental periods of psychosis, with no studies focusing on those experiencing PLEs prior to seeking help.

Like negative symptoms, memory deficits have also long been identified in psychosis, with episodic memory deficits—problems in the ability to remember past events (Tulving, 2002) —being particularly pronounced in chronic schizophrenia samples (Aleman, Hijman, de Haan, & Kahn, 1999; Heinrichs & Zakzanis, 1998), and also present in FE individuals and CHR for psychosis (Fusar-Poli, 2012; Greenland-White, Ragland, Niendam, Ferrer, & Carter, 2017). Nevertheless, current treatments for psychosis do little to improve memory or other cognitive problems (Goff, Hill, & Barch, 2011), even though episodic memory deficits are known to contribute to poorer daily living skills and are among the strongest predictors of functional outcomes among schizophrenia patients (Ranganath, Minzenberg, & Ragland, 2008). Greater impairment in episodic memory has also been observed in people at CHR who later convert to psychosis relative to those who do not (Fusar-Poli, 2012). In fact, because episodic memory performance is among the strongest predictors of functional outcome and quality of life and has been demonstrated in those who convert to psychosis, it was one of the few variables included in a "risk calculator" developed and validated for individualized prediction of psychosis in people at CHR (Cannon et al., 2016; Carrion et al., 2016; Nuechterlein et al., 2011). Although there are studies demonstrating episodic memory impairment in chronic, FE, and CHR groups, no research to our knowledge, has

investigated episodic memory in people endorsing PLEs or PLEDs in non-clinical samples. With evidence of PLEs experienced by the general population potentially having relevance for psychotic disorder risk (van Os & Linscott, 2012), a better understanding of episodic memory at this early stage of PLEs, could help determine the point at which memory problems begin to emerge in the trajectory of psychosis.

Episodic memory performance consists of a number of cognitive processes, including encoding and retrieval (Tulving, 2002). Encoding, or the learning of information to be stored for later use, of an event involves processing perceptual and conceptual aspects of the event through action representations and by directing attention toward relevant information and away from irrelevant information (Ranganath et al., 2008). How engaged one is during encoding plays a significant role in memory formation (Craik & Lockhart, 1972). Two often used strategies for encoding are by using (1) item-specific strategies and (2) relational strategies (Blumenfeld & Ranganath, 2006; Bower, 1970; Hunt & Einstein, 1981). Item-specific encoding strategies involve thinking about a stimulus through its distinct but superficial features, such as the colors, height, or shape of an object (Hunt & Einstein, 1981). Relational encoding strategies involve focusing on commonalities among stimuli, such as whether all items are the same size or by linking object through a story (Bower, 1970). Retrieval, or the recall of previously learned information, of an event can also include many processes. Retrieval depends upon whether information was adequately encoded, on availability of contextual cues, and on the conditions in which one is attempting retrieve memory of an event (Rugg & Vilberg, 2013; Tulving & Thomson, 1973).

Findings have consistently demonstrated that people with schizophrenia have pronounced impairment in episodic memory, and specifically for associative/relational encoding (how one item relates to another) rather than specific details of an item (Achim & Lepage, 2003; Aleman et al., 1999; Heinrichs & Zakzanis, 1998). Additionally, there is a growing body of evidence for greater deficits in recollection (i.e., recognition of a stimulus in the context of other information associated with the experience) —which is interpreted as reflecting relational memory impairment rather than familiarity—in schizophrenia samples compared to non-psychiatric controls (Eichenbaum, Yonelinas, & Ranganath, 2007). In light of the importance of episodic memory in functioning and in prediction of psychosis, the Cognitive Neuroscience Treatment Research to Improve Cognitive in Schizophrenia (CNTRICS) initiative (Carter & Barch, 2007) and, more recently, the Cognitive Neuroscience Test Reliability and Clinical Applications for Schizophrenia (CNTRACS) consortium (Gold et al., 2012) were developed to, in part, optimize task design and modify existing cognitive neuroscience paradigms to better assess cognitive domains, including episodic memory, in people with schizophrenia. These groups have worked to improve the field's understanding of cognitive and neural underpinnings of how encoding, binding, and retrieval mechanisms, including contributions of the medial temporal and prefrontal regions, relate to memory deficits in people with schizophrenia (Eichenbaum et al., 2007; Ragland et al., 2009). For investigation of episodic memory, CNTRACS researchers modified a paradigm previously used in non-psychiatric controls that assessed episodic memory of verbal stimuli (Blumenfeld & Ranganath, 2007; Murray & Ranganath, 2007). The adapted task for psychosis research was modified to optimize patient tolerability, lessen task switching

demands, and reduce meta-cognitive demands, which are difficult for people with schizophrenia and may result in poorer performance than would otherwise be the case (Gold et al., 2012). Further, these researchers removed the verbal stimuli and replaced it with visual stimuli, which requires less high-order cognition (Heinrichs & Zakzanis, 1998). The task, known as the Relational and Item-Specific Encoding (RiSE) task, has been validated in non-psychiatric controls and schizophrenia groups (Ragland et al., 2012), and has been used with schizophrenia samples behaviorally and in neuroimaging studies (Ragland et al., 2015; Ragland et al., 2012), and in FE and CHR samples (Greenland-White et al., 2017).

Specifically, previous research using the RiSE task has indicated that people with schizophrenia are impaired on both item recognition, regardless of whether a participant uses an item-specific or relational encoding strategy, and associative recognition compared with non-psychiatric controls, but that the magnitude of this impairment is larger for associative recognition (Ragland et al, 2012; 2015). In a more recent study, Greenland-White and colleagues (2017) demonstrated that FE individuals were impaired following both item and relational encoding, but there were more pronounced deficits when using relational encoding strategies. They also found that CHR individuals were impaired only following relational, but not item, encoding. Additionally, both groups performed more poorly than non-psychiatric control participants, but not as poorly as schizophrenia patients.

In recent years, experimentation exploring the interaction of memory and motivation has surged, both in people with and without schizophrenia. This work has established that memory and motivation are highly intertwined (for review, see Braver et

al., 2014). It has been hypothesized that, in people with schizophrenia, abnormalities in cognition—memory, namely—prevent hedonic signals from being translated into motivated behavior (Kring & Elis, 2013). Specifically, people with schizophrenia have difficulties maintaining the intensity of emotional information in memory, which is also associated with decreased motivation (Gard et al., 2011), and much of goal-directed behavior requires the ability to mentally represent stimuli outside of the presence of the stimulus (Burbridge & Barch, 2007; Gold et al., 2012). Motivational salience of information helps prioritize information during memory encoding, which may allow for better retrieval (for review, see Miendlarzewska, Bavelier, & Schwartz, 2016). Further, inadequate encoding and retrieval of information may contribute to lower anticipation of pleasure from future activities, in turn leading to reduced motivation (Strauss, Whearty, Frost, & Carpenter, 2016).

A recent review paper provides evidence that cognitive abilities predict future clinical symptoms of schizophrenia (Lepage, Bodnar, & Bowie, 2014), and memory performance have been noted to be associated with negative symptoms (Hill, Ragland, Gur, & Gur, 2002). Other evidence in people with schizophrenia indicates that poorer episodic memory is associated with higher levels of anhedonia (Strauss & Gold, 2012). Similarly, findings suggests that better episodic memory performance is related to lower negative symptom severity, which in turn is related to better clinical outcomes, such as higher educational, occupational, and social functioning (see review in Lepage, Bodnar, & Bowie, 2014). In FE individuals, episodic memory performance accuracy was associated with negative symptom outcome at one-year follow-up; that is, better memory

performance at baseline predicted less severe negative symptoms at follow-up (Greenland-White et al., 2017).

With demonstrated associations between negative symptoms (e.g., MAP symptoms) and cognition (e.g., episodic memory) in psychosis samples, jointly investigating these constructs in people who are not yet seeking clinical help for PLEs/PLEDs may provide evidence to aid in early identification, intervention, and prevention strategies. Psychotic symptoms are comprised of a continuum of perceptual abnormalities, paranoia and suspiciousness, disorganization, and unusual thinking, though only the most frequent and/or debilitating are considered clinically relevant (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009). However, constraining psychosis research to symptoms of clinical severity restricts the applicability of findings. Assessing the dimensionality of psychotic symptoms is essential for exploring an extended psychosis phenotype, given that individuals who experience numerous PLEs, especially numerous PLEDs, that do not reach clinical severity are at greater risk for developing a psychotic disorder (van Os & Linscott, 2012). It is important to investigate MAP symptoms in addition to relational and item-specific encoding and retrieval in people endorsing PLEs/PLEDs prior to seeking help as the specific pattern of episodic memory impairment seen in later stages of the disorder may serve as a trait marker for vulnerability to psychosis.

The present study, therefore, sought to investigate whether aspects of episodic memory (i.e., item recognition using item-specific or relational encoding, associative recognition using relational encoding) contribute to the relation between specific MAP symptoms (i.e., anticipatory pleasure and amotivation) and PLEs in a non-clinical, young

adult sample. Further, this association was also examined in those experiencing more PLEDs (High PLEDs group) and at potentially higher risk of developing psychosis, compared to those who experience few PLEDs (Low PLEDs group) and are presumed lower risk for psychosis (see "Self-Report Measures" for how groups were determined). We hypothesized that (1) anticipatory pleasure deficits and increased amotivation would be associated with increased number of PLEs as well as higher odds of being in the High PLEDs group; (2) that the addition of associative recognition using relational encoding, but not item recognition regardless of encoding strategy, would independently contributed to PLEs/increased odds of being in the High PLEDs group; (3) due to consistent evidence of both MAP deficits and episodic memory impairments across stages of psychosis, we will seek to determine whether associative recognition using a relational encoding strategy will moderate the relationship between decreased anticipatory pleasure / increased amotivation and increased PLEs / odds of being in the High PLED group. We suspect that those with lower anticipatory pleasure / more amotivation and poorer associative recognition will have increased PLEs / odds of being High PLED.

Method

Participants

Participants (N = 667) were a subset of people who participated in the larger study previously reported (see Chapter 2), and were racially/ethnically and socioeconomically diverse sample of male and female non-clinical young adults. Participants were recruited via an online subject recruitment website and received college course credit for their participation. The online recruitment website, which was open to all undergraduate

students at the university, listed the current study with all other available studies that one could choose to participate in; studies are listed in random order. To participate, an individual would select our study from the list of options, be provided information regarding what the study would entail and where it was located, and then choose a day and time that was conducive to their schedule from the options available. For inclusion, participants were to be between the ages of 18 and 35, which corresponds to the typical age range of individuals developing schizophrenia (*DSM 5*, 2013), and fluent in English. See Table 4 for participant characteristics.

Table 4. Participant demographics and characteristics.

	Total Sample (*N* = 587)	**Low PLEDs (*n* = 334)**	**High PLEDs (*n* = 119)**	***p*-value**
Gender: F% (M%)	78.1 (21.9)	77.5 (22.5)	77.5 (22.5)	.999
Age: M (SD) [Range]	20.03 (2.43) [18-34]	20.07 (2.63) [18-34]	19.79 (1.77) [18-28]	.285
Race/Ethnicity: N (%)				.823
Caucasian/White	359 (59.3)	208 (61.8)	73 (60.2)	
Af.-American/Black	104 (17.1)	51 (15.1)	19 (16.0)	
Asian/Pac. Islander	81 (13.3)	44 (13.2)	18 (14.9)	
Hispanic/Latino	26 (4.3)	14 (4.1)	4 (3.6)	
Other	36 (6.0)	20 (5.8)	6 (4.6)	
PLEs	9.85 (7.82) [0-44]	5.55 (4.69) [0-30]	20.69 (7.13) [10-44]	<.001***
PLEDs	4.19 (5.24) [0-28]	0.85 (1.04) [0-3]	12.83 (4.98) [8-28]	<.001***
TEPS:				
Anticipatory	47.30 (7.22) [24-60]	48.30 (6.17) [30-60]	46.25 (8.39) [24-60]	.005**
Consummatory	37.73 (6.32)) [13-48]	37.94 (5.84) [22-48]	37.97 (5.82) [23-48]	.963
GCOS-CP:				
Intrinsic	43.43 (8.25) [8-56]	44.51 (7.04) [24-56]	43.21 (7.40) [16-56]	.090^
Extrinsic	26.56 (7.90) [8-56]	26.18 (7.07) [10-45]	26.91 (6.42) [8-56]	.283

Amotivated	28.06 (7.82) [8-56]	26.76 (6.84) [8-45]	30.92 (7.72) [17-56]	<.001***
Item: M (SD)				
Item specific encoding	.8772 (.1143)	.8828 (.1111)	.8650 (.1237)	.051^
Relational encoding	.8273 (.1254)	.8273 (.1254)	.8144 (.1268)	.474
Associative: M (SD)				
Relational encoding	.7039 (.2062)	.7226 (.1887)	.6731 (.2121)	.246

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; TEPS = Temporal Experience of Pleasure Scale; GCOS-CP = General Causality of Orientations Scale for Clinical Populations; Item/Associative = recognition accuracy computed using hits minus false alarms on the Relational and Item-Specific Encoding (RiSE) task; *p*-value from independent samples *t*-test, analysis of covariance (ANCOVA) controlling for age/gender, or χ^2, respectively; ^ Trend level significance; * Significant at the <.05 level; ** Significant at the <.01 level; *** Significant at the <.001 level

Procedures

Following informed consent, participants were directed to a laboratory computer terminal. Here, participants were individually, electronically administered the behavioral task (E-Prime 2.0 software, Psychology Software Tools, Pittsburg, PA) and self-report questionnaires (Survey Monkey Inc., Palo Alto, CA). The relational and item-specific encoding and retrieval using the Relational and Item-Specific Encoding (RiSE) task (Ragland et al., 2012), developed via the CNTRACS consortium (Gold et al., 2012), was conducted first, followed by collection of demographic characteristics, and administration of self-report inventories. Self-report inventories included the Prodromal Questionnaire (PQ) (Loewy et al., 2005, 2007), the Temporal Experience of Pleasure Scale (TEPS) (Gard et al., 2006), and General Causality Orientation Scale (GCOS-CP) (Cooper et al., 2015). See Table 1 for participant characteristics on the PQ, TEPS, and GCOS-CP; see Table 2 for performance characteristics on the RiSE task.

Self-Report Measures

The Prodromal Questionnaire (PQ) (Loewy et al., 2005, 2007)**.** The PQ is a 92-item, self-report questionnaire that measures subthreshold psychotic symptoms experienced in the past month in four domains: positive, negative, disorganized, and general. The PQ also requires participants to endorse whether symptoms were experienced as distressing. The PQ has been tested against semi-structured interviews commonly used to identify individuals at risk for psychosis and has been found to be both reliable and valid in comparison (Loewy et al., 2005).

We focused on the positive symptom domain, as this domain has been associated with increased risk for psychosis and has been primarily studied in investigations of PLEs

in the general population (Cannon et al., 2008; Loewy et al., 2007). In line with previous research, distressing items are an additional focus for the current study, as those who endorse distressing items are at a higher likelihood of developing a psychotic disorder (Hanssen et al., 2003; Loewy et al., 2007). PLEs were examined continuously (total number of PLEs endorsed), as well as categorically (i.e., High PLEDs = potentially higher risk for psychosis; Low PLEDs = potentially lower risk). Group membership for the categorical variable was determined by endorsement of 8 or more distressing PLEs (High PLEDs, current study n =119) compared to three or fewer distressing PLEs and 8 or fewer total PLEs (Low PLEDs group; the means of PLEs distressing and total PLEs symptoms in our larger undergraduate sample, current study n = 334) (Gibson et al., 2014; Reeves et al., 2014). Endorsing 8 or more positive symptoms has been found to have 90% sensitivity and 49% specificity with individuals identified as CHR using the SIPS in a clinical population, and in an undergraduate sample 2% met this criterion if 8 symptoms were endorsed as distressing (Loewy et al., 2005, 2007).

Temporal Experience of Pleasure (TEPS) (Gard & Kring, 2009). The TEPS, an 18-item self-report questionnaire, is designed to index trait disposition to experience anticipatory pleasure (i.e., pleasure associated with wanting a given reward or experience) and consummatory pleasure (i.e., pleasure associated with liking a given activity or experience in-the-moment). The TEPS includes the 10-item anticipatory pleasure subscale, and the 8-item consummatory pleasure subscale. This scale is widely used to assess pleasure deficits in individuals with a diagnosis of or at risk for schizophrenia and other psychotic disorders (Cohen & Minor, 2010). The TEPS yields high levels of internal consistency (α = .79 overall, α = .74 anticipatory, α = .71

consummatory) (Gard, Gard, Kring, & John, 2006), is one of the most commonly used self-report measure of hedonic functioning in psychotic disorders, and has recently been used to demonstrate anticipatory pleasure deficits in young adults reporting PLEs (Cooper et al., In press).

General Causality Orientation Scale for Clinical Populations (GCOS-CP) (Cooper et al., 2015)**.** The GCOS-CP is a self-report measure adapted from the General Causality Orientation Scale (Deci et al., 1981) designed to measure motivation orientation, or how individuals respond to ambiguous situations; individuals can be more autonomous (intrinsically motivated), controlled (extrinsically motivated), or impersonal (amotivated) in their orientation (Deci & Ryan, 2000). Autonomy oriented/intrinsically motivated individuals are typically motivated by inherent interest and engagement in activities (Deci & Ryan, 2000). Control oriented/extrinsically motivated individuals are more often motivated by external praise and reward (especially monetary), and also away from punishment or criticism (Deci & Ryan, 2000). When individuals lack opportunities for inherent engagement or reward, they may develop a more impersonal/amotivated orientation, and be more disengaged from their environment (Deci & Ryan, 2000). In this study, we will be referring to causality orientations as follows: autonomy = intrinsic motivation, control = extrinsic motivation, impersonal = amotivated. Various studies have found deficits in motivation in those suffering from schizophrenia(Gard et al., 2009; Nakagami et al., 2010), as well as those in their first episode of psychosis (Breitborde et al., 2013), and those at CHR for psychosis (Schlosser et al., 2014). The GCOS-CP asks participants to respond to each of the three motivation orientations (autonomy, controlled, impersonal) on 8 vignettes, totaling 24 responses. Each response is indicated on a 1 to 7

Likert scale (i.e., 1 = *Very Unlikely*, 4 = *Moderately Likely*, 7 = *Very Likely*). This measure has acceptable internal consistency in non-clinical (α = .74 autonomy/intrinsic, α = .65 controlled/extrinsic, α = .67 impersonal/amotivated) and clinical (α = .77 autonomy/intrinsic, α = .52 controlled/extrinsic, α = .57 impersonal/amotivated) samples.

Behavioral Measure

Relational and Item Specific Encoding Task (RiSE) (Ragland et al., 2012). RiSE tests a person's episodic memory ability to encode and retrieve relational versus item-specific images. This task presents visual stimuli (photographs from a standardized corpus – cvcl.mit.edu/MM/) to participants by alternating encoding tasks (i.e., relational, item-specific) between blocks. This task was developed for use in schizophrenia populations, and findings suggest that patients have less impairment when attending to item-features during encoding compared to relational or abstract features (Ragland et al., 2012; 2015).

Participants underwent two incidental encoding tasks. Item-specific encoding consisted of 36 visual stimuli presented for 2 seconds, each with a 1 second inter-stimulus interval (ISI). Participants determined whether each object was "living" by pressing one of two buttons, labeled "yes" and "no". Relational encoding consisted of 18 object pairs presented for 4 seconds, with a 1 second ISI. Participants determined whether one of the items could fit inside of the other by pressing one of two buttons, again labeled "yes" and "no". In line with Ragland et al. (2012), encoding conditions were alternated in pseudorandom block design, with 3 item blocks (12 trials each) and 3 relational blocks (6 trials each). Before each block, instructions were presented on screen to reorient the participant to the encoding decision (i.e., "living?" or "inside?") they would need to

make. This one-word decision-instruction remained on screen for the duration of each encoding block.

Following encoding, retrieval tasks, which were self-paced, were conducted. Item recognition consisted of the 72 stimuli studied across item-specific and relational conditions with 72 stimuli unstudied. Participants were asked to indicate whether each item was "old" (using their left-hand to respond by pressing the corresponding button) or "new" (using their right-hand to respond by pressing the corresponding button). Associative recognition included the 18-object pairs studied during relational encoding and 18 unstudied object pairs consisting of studied items not originally paired together. Participants were asked to indicate whether each pair had been presented "together" by pressing one of two buttons, labeled "yes" and "no". Item recognition always preceded associative recognition.

Accuracy was computed for both item recognition and associative recognition by subtracting the proportion of false alarms (i.e., new items incorrectly identified as old items) from the proportion of hits (i.e., old items correctly identified as old items) in each condition. Item recognition was parsed into item-specific encoded items and relational encoded items. Associative recognition solely used relational encoded items. In line with previous research, we suspect that associative recognition will be more difficult than item recognition as associative recognition relies upon recollection of an aspect of the encoding to discriminate studied from rearranged pairs whereas item recognition relies upon familiarity of an object (Ragland et al. 2012).

Statistical Analytic Plan

Participant's reported ages were checked to ensure that participants from the sample were within the typical age of onset for schizophrenia (18 - 35 years old) (American Psychiatric Association, 2014); all participants reported being within this age range. Age and gender were examined as potential covariates by determining whether they were significantly related to the main study variables. Participants who performed below chance on a RiSE recognition task (less than 50% hit accuracy; $n = 80$) were removed from analyses. These 80 individuals also provided inconsistent responding (e.g., patterned and/or flat-lined responses) on other self-report questionnaires. Data analyses, therefore, included a total sample of 587 participants.

First, multiple linear/logistic regression models were conducted to determine whether MAP symptoms were associated with PLEs and/or increased odds of being in the High PLEDs group. Second, episodic memory variables (encoding/retrieval strategy) were added to the models to determine whether encoding/retrieval strategy independently contributed to PLEs/increased odds of being in the High PLEDs group. Third, moderated multiple linear regressions were conducted to determine whether episodic memory (s, item recognition using item-specific encoding, item recognition using relational encoding, associative recognition using relational encoding) moderated the relation between MAP symptoms (i.e., anticipatory pleasure, amotivation) and PLEs. Additionally, moderated multiple logistic regressions were conducted to determine whether episodic memory (i.e., item recognition using item-specific encoding, item recognition using relational encoding, associative recognition using relational encoding) moderated the relation between MAP symptoms (i.e., anticipatory pleasure, amotivation)

and being at a higher risk of developing psychosis (i.e., High PLEDs group). A significant interaction in any of the models was indicative of the moderation model better fitting the data than an additive model, whereas if there were no significant interactions, then the additive model was considered to best fit the data.

Results

Our sample was relatively representative of the community from which it was drawn, although there were more women than men in our overall sample. There were significant differences between males and females on anticipatory pleasure (p <.001), consummatory pleasure (p = .031), extrinsic motivation (p < .001), and amotivation (p = .003) scores. Gender was also found to be significantly related to PLEs (p = .043), but not PLED status. Because there was an uneven distribution of gender within the High PLED and Low PLED groups, and because gender was significantly related to PLEs, we chose to take a conservative approach and include gender as a covariate in all our analyses Additionally, age was found to be associated with PLEs (at a non-significant level; p = .080), and was significantly related to anticipatory pleasure (p = .001). Again, continuing our conservative approach, we opted to retain age as a covariate in subsequent analyses. An analysis of covariance (ANCOVA) indicated that those who are High PLED do not differ in retrieval from people who are Low PLED, regardless of encoding strategy. Table 4 provides participant characteristics for our full sample, as well as our High PLED (n = 120) and Low PLED (n = 377) groups.

Table 5. Correlation matrix

	1	2	3	4	5	6	7	8	9	10	11
1 Age	-	-.131**	-.072	-.074^	-.134**	-.052	-.013	.082*	.025	-.094*	-.034
2 Gender		-	-.083*	-.011	.189**	.089*	.063	-.151**	-.099*	.158***	.000
3 PLE			-	.835***	-.093*	.004	-.079*	.083*	.207**	-.109**	.158***
4 PLED				-	-.106**	.009	-.106**	.073	.230**	-.137***	.168***
5 TEPS: Anticipatory					-	.021	.213***	.129**	-.098*	.737***	.407***
6 TEPS: Consummatory						-	..210***	-.032	-.088*	.352**	.094*
7 GCOS-CP: Intrinsic Motivation							-	.113**	-.248***	.810***	-.121***
8 GCOS-CP: Extrinsic Motivation								-	.207***	.151***	.256***
9 GCOS-CP: Amotivation									-	-.231***	.860***
10 Anticipatory X Intrinsic										-	.153***

11 Anticipatory X Amotivation	-

PLE = psychotic-like experiences, PLED = distressing psychotic-like experiences, TEPS = Temporal Experience of Pleasure Scale, GCOS-CP = General Causality Orientation Scale for Clinical Populations, ^ p = trend, * $p < .05$, ** $p < .01$, *** $p < .001$

First, anticipatory pleasure deficits were not significantly associated with increased PLEs. The model did not change when adding (a) item recognition using item-specific encoding strategy, (b) item recognition using relational encoding strategy, or (c) associative recognition using relational encoding accuracies to the model. Additionally, there were no significant interactions of anticipatory pleasure deficits and episodic memory variables. See Tables 6-8.

Table 6. Multiple linear and logistic regressions assessing the relation between anticipatory pleasure deficits and PLEs/High PLEDs group with Item Recognition: Item-specific Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				.007**			.021*
Age	-.096	-.310	-2.294	.022*	.944	.858-1.038	.232
Gender	-.082	-1.560	-1.930	.054^	1.19	.701-2.005	.526
TEPS Anticipatory	-.080	-.091	-1.899	.058^	.954	.925-.984	.003**
R^2	.021						
Model 2				.015*			.022*
Age	-.097	-.314	-2.319	.021*	.941	.856-1.035	.213
Gender	-.082	-1.565	-1.935	.053^	1.18	.694-1.994	.545
TEPS Anticipatory	-.080	-.091	-1.903	.058^	.954	.924-.984	.003**
IR Item-specific	-.024	-1.723	-.594	.553	.299	.050-1.774	.184
R^2	.021						
R^2 change	.000						
Model 3				.029*			.042*
Age	-.098	-.315	-2.327	.020*	.941	.856-1.036	.214
Gender	-.081	-1.550	-1.913	.056^	1.17	.692-1.990	.552

TEPS Anticipatory	-.081	-.092	-1.919	.055^	.954	.924-.984	.003**
IR Item-specific	-.024	-1.719	-.592	.554	.293	.049-1.762	.180
IR Item-Specific X Anticipatory	.015	.127	.369	.712	.983	.794-1.216	.873
R^2	.021						
R^2 change	.000						
R^2 change	.000						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; IR = Item Recognition on the Relational and Item-Specific Encoding Task; TEPS Anticipatory = Temporal Experience of Pleasure Scale, Anticipatory Anhedonia; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Table 7. Multiple linear and logistic regressions assessing the relation between anticipatory pleasure deficits and PLEs/High PLEDs group with Item Recognition: Relational Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				.007**			.021*
Age	-.096	-.310	-2.294	.022*	.944	.858-1.038	.232
Gender	-.082	-1.560	-1.930	.054^	1.19	.701-2.005	.526
TEPS Anticipatory	-.080	-.091	-1.899	.058^	.954	.925-.984	.003**
R^2	.021						
Model 2				.016*			.019*
Age	-.096	-.311	-2.298	.022*	.946	.860-1.040	.252
Gender	-.081	-1.554	.809	.055^	1.19	.700-2.009	.527
TEPS Anticipatory	-.080	-.090	1.883	.060^	.955	.925-.985	.004**
IR Relational	-.016	-1.027	-.387	.699	.296	.056-1.556	.150
R^2	.021						
R^2 change	.000						
Model 3				.019*			.031*
Age	-.096	-.310	-2.295	.022*	.947	.861-1.042	.263
Gender	-.078	-1.495	-18.46	.065^	1.20	.706-2.038	.501

TEPS Anticipatory	-.083	-.095	-1.967	.050^	.954	.925-.985	.004**
IR Relational	-.007	-.462	-.171	.864	.348	.062-1.969	.233
IR Relational X Anticipatory	.049	.405	1.167	.244	1.08	.866-1.355	.486
R^2	.023						
R^2 change	.002						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; IR = Item Recognition on the Relational and Item-Specific Encoding Task; TEPS Anticipatory = Temporal Experience of Pleasure Scale, Anticipatory Anhedonia; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Table 8. Multiple linear and logistic regressions assessing the relation between anticipatory pleasure deficits and PLEs/High PLEDs group with Associate Recognition: Relational Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				.007**			.021*
Age	-.096	-.310	-2.294	.022*	.944	.858-1.038	.232
Gender	-.082	-1.560	-1.930	.054^	1.19	.701-2.005	.526
TEPS Anticipatory	-.080	-.091	-1.899	.058^	.954	.925-.984	.003**
R^2	.021						
Model 2				.011*			.007**
Age	-.096	-.309	-2.287	.023*	.946	.860-1.041	.256
Gender	-.081	-1.540	-1.904	.057^	1.21	.711-2.053	.485
TEPS Anticipatory	-.080	-.091	-1.894	.059^	.954	.924-.984	.003**
AR Relational	-.040	-1.579	-.981	.327	.329	.114-.948	.039*
R^2	.022						
R^2 change	.001						
Model 3				.022*			.014*
Age	-.096	-.309	-2.282	.023*	.947	.861-1.042	.265
Gender	-.080	-1.519	-1.874	.061^	1.23	.719-2.095	.452

Anticipatory Anhedonia	-.081	-.092	-1.911	.054^	.954	.924-.984	.003**
AR Relational	-.040	-1.560	-.968	.333	.345	.118-1.009	.052^
AR Relational X Anticipatory	.018	.144	.431	.667	1.07	.858-1.340	.540
R^2	.022						
R^2 change	.000						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; AR = Associative Recognition on the Relational and Item-Specific Encoding Task; TEPS Anticipatory = Temporal Experience of Pleasure Scale, Anticipatory Anhedonia; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Second, increased amotivation was found to be significantly associated with increased PLEs. However, no episodic memory variables were significantly additive, nor were there any interactions between amotivation and episodic memory variables. See Tables 9-11.

Table 9. Multiple linear and logistic regressions assessing the relation between amotivation and PLEs/High PLEDs group with Item Recognition: Item-specific Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				<.001***			<.001***
Age	-.089	-.287	-2.161	.031*	.959	.870-1.057	.396
Gender	-.076	-1.443	-1.826	.068^	1.10	.649-1.855	.729
Amotivation	.174	.181	4.240	<.001***	1.09	1.052-1.12	<.001***
R^2	.039						
Model 2				<.001***			<.001***
Age	-.090	-.290	-2.185	.029*	.956	.867-1.054	.367
Gender	-.076	-1.449	-1.832	.067^	1.09	.644-1.847	.746
Amotivation	.174	.181	4.237	<.001***	1.09	1.053-1.12	<.001***
IR Item-specific	-.024	-1.660	-.579	.563	.268	.043-1.682	.160
R^2	.038						
R^2 change	.001						
Model 3				<.001***			<.001***
Age	-.091	-.293	-2.201	.028*	.955	.867-1.053	.359
Gender	-.075	-1.429	-1.805	.072^	1.10	.651-1.872	.714

Amotivation	.174	.181	4.244	<.001***	1.09	1.053-1.12	<.001***
IR Item-specific	-.026	-1.813	-.630	.529	.282	.043-1.854	.188
IR Item-Specific X Amotivation	-.026	-.205	-.625	.532	.892	.693-1.150	.378
R^2	.037						
R^2 change	.001						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; IR = Item Recognition on the Relational and Item-Specific Encoding Task; Amotivation = General Causality Orientation Scale for Clinical Populations, Impersonal/Amotivation; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Table 10. Multiple linear and logistic regressions assessing the relation between amotivation and PLEs/High PLEDs group with Item Recognition: Relational Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				<.001***			<.001***
Age	-.089	-.287	-2.161	.031*	.959	.870-1.057	.396
Gender	-.076	-1.443	-1.826	.068^	1.10	.649-1.855	.729
Amotivation	.174	.181	4.240	<.001***	1.09	1.052-1.12	<.001***
R^2	.039						
Model 2							
Age	-.089	-.288	-2.166	.031*	.961	.872-1.059	.424
Gender	-.075	-1.436	-1.816	.070^	1.11	.653-1.877	.705
Amotivation	.173	.181	4.227	<.001***	1.09	1.052-1.12	<.001***
IR Relational	-.014	-.932	-.356	.722	.251	.045-1.388	.113
R^2	.038						
R^2 change	.001						
Model 3				<.001**			.025*
Age	-.090	-.291	-2.191	.029*	.961	.872-1.060	.427
Gender	-.073	-1.388	-1.852	.080^	1.12	.662-1.910	.664

Amotivation	.172	.179	4.190	<.001***	1.09	1.052-1.12	<.001***
IR Relational	-.013	-.812	-.309	.757	.308	.051-1.867	.200
IR Relational X Amotivation	-.043	-.331	-1.402	.298	.888	.702-1.124	.323
R^2	.038						
R^2 change	.001						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; IR = Item Recognition on the Relational and Item-Specific Encoding Task; Amotivation = General Causality Orientation Scale for Clinical Populations, Impersonal/Amotivation; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Table 11. Multiple linear and logistic regressions assessing the relation between amotivation and PLEs/High PLEDs group with Associate Recognition: Relational Encoding as a moderator.

	PLEs				High PLEDs		
	β	B	*t*-value	*p*-value	OR	95%CI	*p*-value
Model 1				<.001***			<.001***
Age	-.089	-.287	-2.161	.031*	.959	.870-1.057	.396
Gender	-.076	-1.443	-1.826	.068^	1.10	.649-1.855	.729
Amotivation	.174	.181	4.240	<.001***	1.09	1.052-1.12	<.001***
R^2	.039						
Model 2							
Age	-.089	-.286	-2.154	.032*	.961	.872-1.060	.429
Gender	-.074	-1.420	-1.797	.073^	1.13	.662-1.913	.664
Amotivation	.174	.181	4.249	<.001***	1.09	1.055-1.12	<.001***
AR Relational	-.042	-1.650	-1.038	.300	.257	.086-.767	.015*
R^2	.039						
R^2 change	.000						
Model 3				<.001**			<.001***
Age	-.089	-.287	-2.159	-.031*	.961	.872-1.060	.428
Gender	-.074	-1.411	-1.781	.075^	1.12	.660-1.912	.667

Amotivation	.175	.182	4.253	<.001***	1.089	1.055-1.12	<.001***
AR Relational	-.042	-1.651	-1.038	.300	.255	.084-.771	.016*
AR Relational X Amotivation	-.011	-.090	-.282	.778	1.01	.782-1.305	.938
R^2	.038						
R^2 change	.000						

PLEs = psychotic-like experiences; PLEDs = PLEs endorsed as distressing; Low PLEDs = individuals who endorsed 3 or fewer PLEDs; High PLEDs = individuals who endorsed 8 or more PLEDs and may be at higher clinical risk for psychosis; AR = Associative Recognition on the Relational and Item-Specific Encoding Task; Amotivation = General Causality Orientation Scale for Clinical Populations, Impersonal/Amotivation; OR = odds ratio; CI = confidence interval; ^ Trend level significance; * Significant at the $p < .05$ level; ** Significant at the $p < .01$ level, *** Significant at the $p < .001$ level.

Third, greater anticipatory pleasure deficits were significantly associated with higher odds of being in the High PLED group. Item recognition—regardless of whether item-specific encoding or relational encoding was used—did not significantly improve the fit of the model. Associative recognition using a relational encoding strategy did significantly independently contribute to the model; however, there were no significant interactions between anticipatory pleasure deficits and associative recognition using a relational encoding strategy. See Tables 6-8.

Fourth, greater amotivation was significantly associated with odds of being in the High PLED group. Item recognition—regardless of whether item-specific encoding or relational encoding was used—did not significantly improve the fit of the model. Associative recognition using a relational encoding strategy did significantly independently contribute to the model; however, there were no significant interactions between amotivation and associative recognition using a relational encoding strategy. See Tables 9-11.

Discussion

The current findings are the first to demonstrate that poor performance on associative recognition using a relational strategy acts additively with both increases in amotivation and decreases in anticipatory pleasure to significantly increase the odds of being at potentially higher risk for psychosis (High PLEDs). We did not, however, find that episodic memory performance predicts PLEs as a continuous measure. These findings are consistent with previous evidence in schizophrenia, FE, and CHR samples showing greater impairment for retrieval using relational encoding than item encoding strategies (Cannon et al., 2016; Carrion et al., 2016; Greenland-White et al., 2017;

Ragland et al., 2012). Our results are also consistent with previous findings showing that lower anticipatory pleasure and higher amotivation are associated with increased odds of being in the High PLEDs group, and extend these findings by suggesting that episodic memory impairments independently contributes to potential risk for psychosis.

As expected, the present findings suggest that associative recognition following a relational encoding strategy was the only aspect of this episodic memory task that contributed to any model. Associative recognition has been indicated as relying more on recollection than familiarity (Yonelinas, 2001; 2002), and although recollection and familiarity were not specifically computed here, evidence has consistently demonstrated that recollection is impaired in people with schizophrenia, FE individuals, and those at CHR for psychosis, relative to non-psychiatric controls (Greenland-White et al., 2017; Ragland et al., 2012). The present study extends these findings by suggesting that associative recollection following a relational encoding strategy independently contributes to those experiencing distressing PLEs, even when accounting for MAP deficits.

Contrary to our hypotheses, episodic memory factors did not moderate the relationship between anticipatory pleasure deficits or amotivation and increases in PLEs or increased odds of being in the High PLED group. While a number of studies have demonstrated a relationship between motivation and cognition in people with schizophrenia, it remains unclear what the causal relationships are between amotivation, anticipatory pleasure, and episodic memory. Further, it is also unclear how these constructs interact with people at the earliest stages of psychotic symptoms (PLEs/PLEDs) who may or may not transition to a psychotic disorder.

Although previous evidence from our lab (Cooper et al., In press) found a significant a relationship between anticipatory pleasure deficits and PLEs, the present study indicated only a trend-level relationship between the two variables. Although we had a large sample, given that the prevalence of clinically significant PLEs are rare in the population, the base rates of the symptoms we are looking at is small, and because we had fewer participants in the current study than our previous study, it may have resulted in the inability to detect relatively subtle associations that occur across the whole spectrum of PLEs. It could be that High PLED participants drove our original findings; however, future studies are necessary to elucidate these possibilities. Contrary to our hypotheses, significant results were limited to models evaluating the odds of being at potentially higher risk of psychosis (High PLED), relative to those at lower risk (Low PLED). By removing individuals (n = 134) who reported a moderate number of PLEs, we compared a symptomatic group to a non-symptomatic group, which more closely mirrors previous studies comparing a psychiatric group (i.e., schizophrenia, FE, CHR) to a non-psychiatric group. As such, our findings suggest that difficulties with MAP symptoms and episodic memory seem to become more apparent as symptoms become more clinically significant.

Our findings support studies that have found disruptions in brain regions involved in episodic memory, motivation, and hedonic functioning in psychosis samples (Brown et al., 2013; Dowd & Barch, 2010; Locke & Braver, 2010; Maddox & Markman, 2010; Pessoa & Engelmann, 2010; Scoville & Milner, 1957; Squire, Stark, & Clark, 2004; Squire & Zola-morgan, 1991), FE individuals, and those at CHR for psychosis (Abi-Dargham et al., 2002; Brown et al., 2013; Dowd & Barch, 2010; Falkenberg et al., 2015;

Locke & Braver, 2010; Maddox & Markman, 2010; Nenadic et al., 2015; Pessoa & Engelmann, 2010). For instance, findings suggest that PFC and MTL activity was impaired (an indication of decreased functional activation) for emotionally evocative stimuli, but not for non-emotional stimuli, in patients with schizophrenia compared to non-psychiatric controls (Ursu et al., 2011). Ragland and colleagues (2015) conducted a multi-site study assessing brain regions using the RiSE task—which uses non-emotional stimuli—and found that people with schizophrenia demonstrated less activation in the PFC and MTL during encoding and retrieval than did people without schizophrenia. Further, people with schizophrenia had disproportional functional impairment in the dorsolateral PFC and the hippocampus (which is a subregion in the MTL) during relational compared to item-specific encoding and retrieval (Ragland et al., 2015). While the PFC and MTL have been implicated in motivation and reward, a number of other separable but related systems (e.g., mesocorticolimbic dopaminergic pathways) have been associated with these functions. For example, neural and behavioral studies consistently suggest that reduced dopamine release in the striatum in response to reward cues is correlated with increased negative symptoms (for a review on the role of dopamine in schizophrenia, see Maia & Frank, 2017). A different study found that negative symptoms, including motivation, predicted reduced activation in the ventral striatum during reward cues in both medicated and unmedicated people with schizophrenia (Juckel et al., 2006). Our results support the independent contributions of motivation, anticipatory pleasure, and episodic memory to increased risk of being at potentially higher risk for psychosis, which may (in part) reflect disruptions in separable (but related) brain systems in these individuals.

A notable strength of this study is the diverse sample. Nevertheless, results from this study may not generalize outside of college populations given that the sample consisted of college students, even though the publicly funded university is comprised of students who are quite diverse from a socioeconomic and ethnic/racial standpoint. Further, our self-report and behavioral measures have been used in both healthy samples and samples at different stages of psychosis. This allows us to utilize the same measure across our entire sample without concern of its applicability or difficulty level.

There are several limitations to the current study, the most notable being the cross-sectional design of the study. Because of the nature of the design, we are not able to discern directionality, rule out the possibility that PLEs lead to MAP symptoms and/or episodic memory performance, or determine which individuals may transition to psychosis. Although gender did not differ between High PLED and Low PLED groups, there were significantly fewer men than women in the study. Future research should seek to remediate this gender imbalance and attempt to study these constructs in a longitudinal methodological design. Additionally, future studies should extend analyses by using receiver operator characteristics (ROC) (Yonelinas & Parks, 2007), which provide estimates of recollection and familiarity in response to items. ROC analyses would allow for estimates of recognition discriminability as a function of response criterion. A dual-process model (Yonelinas, 2001) can also be applied to determine whether recollection or familiarity was relied upon more during recognition trials. These expanded analyses are important to more fully understand episodic memory impairment in those with PLEs/PLEDs, and to better compare these deficits to people with schizophrenia, FE individuals, and those at CHR for psychosis.

In sum, people who experience increased amotivation and decreased anticipatory pleasure, and who perform more poorly on associative recognition following use of a relational encoding strategy, are at increased odds of being at potentially higher risk for psychosis. Although lower anticipatory pleasure and associative recognition using a relational encoding strategy was not significantly associated with PLEs, the trend-level findings provide sufficient evidence to merit further investigation. Future studies should evaluate MAP symptoms and episodic memory performance longitudinally in people that shift into a formal CHR for psychosis diagnosis relative to people who do not to determine the specific time point at which these deficits come online, the course of deficits over the development of psychosis, and the impact these deficits may have on functional outcomes in a person's life.

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