NEGATIVE AFFECT AND POSITIVE SYMPTOMS OF PSYCHOSIS

Audra Crutchfield, B.A., M.A.

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APPROVED:

Craig S. Neumann, Major Professor
Amy R. Murrell, Committee Member
Kenneth Sewell, Committee Member
Randall Cox, Clinical Psychology Program Director
Vickie Campbell, Chair of the Department of Psychology
Michael Monticino, Dean of the Robert B. Toulouse School of Graduate Studies
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The current study utilized structural equation modeling (SEM) to examine the factor-to-factor relations and temporal associations between disturbances in negative affect (NA) and positive symptoms of psychosis (PP). Data were drawn from a large, public-domain data set (MacArthur Violence Risk Assessment Study). A dimensional approach was used to conceptualize and identify latent variables of NA (depression, anxiety, and guilt) and PP (hallucinations, delusions, and thought disorder) among individuals with a diagnosis of primary psychotic disorder. Results showed that anxiety, guilt, and depressed mood modeled an NA latent variable, and that hallucinations and unusual thought content modeled a PP latent variable. As predicted, results revealed strong, significant cross-sectional (synchronous) associations between NA and PP at each measured time-frame, suggesting that NA and PP occurred concurrently within the sample. Contrary to predictions, no significant cross-lagged effect between NA and PP was identified (10 weeks and 20 weeks respectively).
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CLASSIFICATION OF PSYCHOPATHOLOGY: THE UTILITY OF A
DIMENSIONAL FRAMEWORK

Current nosological models used for conceptualizing psychopathology, such as the categorical model employed by *Diagnostic and statistical manual of mental disorders text revision* (American Psychiatric Association, 2000), are insufficient for a variety of reasons. In a recent article, Widiger and Trull (2007) outline a number of fundamental problems with current diagnostic models. Authors of the DSM-IV-TR acknowledge that a categorical model is most useful when members of a specific diagnostic class are homogenous in clinical presentation, have distinct and clear boundaries from other disorders, and are mutually exclusive from one another (DSM-IV-TR, APA, 2000). However, the frequency with which mental disorders co-occur, termed comorbidity, challenges the assumptions upon which the current classification system is based. In addition, the rules for diagnosing a mental disorder are frequently arbitrary and do not incorporate sub-threshold symptoms that are often found in the general population (Widiger & Trull, 2007). Furthermore, heterogeneous clinical presentation, as is often seen in disorders such as schizophrenia, demonstrates that individuals can present with completely different symptoms, yet receive the same diagnosis (DSM-IV-TR, APA, 2000). Current classifications of mental disorders have been criticized on the basis of poor discriminate validity and some
have suggested that more general dimensions may underlie current categorical distinctions (Brown, Chorpita, & Barlow, 1998).

One way of addressing the problems inherent in the categorical model is to study psychological phenomena, and psychopathology in particular, from a dimensional perspective. The dimensional model circumvents the aforementioned problems by examining psychological phenomena in a quantitative manner that includes areas of overlap, or covariation, among disorders and subclinical manifestations of symptoms (Krueger & Markon, 2006). Krueger (2005) has noted that while the categorical approach also relies on a quantification of symptoms, it fails to adequately address the issue of cut-points, many of which are arbitrary in the current diagnostic system. For example, with categorical approaches, the individual has to meet a minimum number of criteria prior to receiving a diagnostic label. However, many mental disorders share (or have very similar) diagnostic criteria and many individuals experience clinically significant and distressing symptoms without meeting a specific cut-point threshold for diagnosis (Kessler, 2002; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). At the same time, there is some empirical support suggesting that the categorical and dimensional models used together is most powerful, rather than either approach used alone (Van Os et al., 1999).

Krueger, Caspi, Moffitt, and Silva (1998) suggested that research in psychopathology would best be served by studying multiple disorders in conjunction across time. Despite the benefits of longitudinal research designs,
Clarkin and Kendall (1992) have noted a paucity of longitudinal comorbidity research in extant literature. Traditionally, the majority of research studies have focused on comorbidity from a cross-sectional perspective, which limits researchers’ ability to formulate potentially causative relationships between phenomena and to study the temporal stability and longitudinal relations between clinical symptoms and disorders. Dimensional conceptualizations in the context of longitudinal studies provide additional information that can shed light on the temporal relations among symptom clusters and potentially uncover etiological information. Given high comorbidity rates among mental disorders, studies whose samples ostensibly pertain to only one specific disorder may, in actuality, contain cases in which symptoms of multiple disorders are present and therefore could be providing convoluted, if not confounded, results. Consequently, researchers may miss important data that contribute to the etiology and development of psychopathology (Krueger et al., 1998).

Furthermore, there may be core psychopathological deficits that underlie a range of mental disorders, reflected in more fundamental dimensions (e.g., negative emotionality), rather than the DSM disorders representing discrete and core entities themselves. For instance, some disorders, such as depression and schizophrenia, may share personality traits that reduce or increase risk of developing a mood or psychotic episode (Van Os & Jones, 2001). Krueger et al. (1998) have taken this a step further and have provided evidence that only a few underlying latent dimensions (e.g., externalizing, internalizing) likely underlie all
psychopathology. As such, individuals who are identified as high-risk on an underlying dimension are also more likely to meet criteria for multiple disorders within a given dimension. Therefore, these authors purport that a dimensional perspective more adequately explains the positive correlation between co-occurring (or covarying) disorders, particularly as severity of mental disorder increases. Some research studies have found that treatment of one disorder (e.g., anxiety disorder) often results in the amelioration of a comorbid diagnosis that was not a direct target of psychotherapeutic intervention (e.g., depression) (Borkovec, Abel, & Newman, 1995; Brown, Antony, & Barlow, 1995), suggesting that there might be a broader dimensions underlying multiple disorders. Determining what those underlying dimensions are constitutes a complex and ambitious endeavor that is only recently being addressed in the literature (Krueger & Markon, 2006).

Finally, a discussion of categorical and dimensional models of psychopathology classification is incomplete without a discussion of the pragmatic implications for both assessment and treatment (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Dimensional approaches to the assessment and treatment of mental health concerns offers rich data with regard to the quantity, frequency, intensity and severity of problematic symptoms. Practitioners are often using dimensional approaches to measure treatment outcome and improvement. For example, progress in mental health treatment, particularly for those individuals with a severe and persistent mental illness is often gauged on
improvement of various dimensions of functioning, even though the individual continues to meet criteria for a specific disorder. Practitioners are often assessing treatment response based on symptom improvement. However, despite the clear advantages for viewing psychopathology from a dimensional perspective, it should also be recognized that over-reliance on this one aspect of outcome (symptom reduction) may have little utility for assessing other important factors, such as quality of life and overall functioning. Additional dimensions related to more positive attributes (e.g., motivation, persistence, resilience, psychological and cognitive flexibility, or emotional willingness) may also provide a rich source of information and hold promise as additional important prognostic indicators (Hafner & an der Heiden, 2008; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996).
THE DIMENSIONAL MODEL OF PSYCHOTIC DISORDERS

While there is considerable empirical support for viewing many mental disorders in terms of externalizing (e.g., substance use, antisocial personality) or internalizing (e.g., depression, anxiety) dimensions of psychopathology (Krueger & Markon, 2006), researchers have also proposed that dimensional approaches to the conceptualization of psychotic disorders can provide more a more meaningful understanding of the heterogeneity and breadth of psychotic symptoms (Krueger & MacDonald, 2005; McGorry, Bell, Dudgeon, & Jackson, 1998). Studies have demonstrated that dimensional classifications of psychotic disorders have greater predictive validity than categorical models, although it varies with the time frame of symptoms used in the analysis (Peralta, Cuesta, Giraldo, Cardenas, & Gonzales, 2002). Given the heterogeneity of clinical presentation among schizophrenia-spectrum disorders, there has been much debate within the literature regarding which dimensions to study, despite the utility of a dimensional conceptualization.

Early factor analytic studies examining the latent structure of schizophrenia symptoms have drawn sharp criticism due to small sample sizes and the inadequacy of using exploratory procedures. Consequently, many researchers have questioned the stability and accuracy of early findings (Lenzenweger & Dworkin, 1996). Strauss (1974) suggested a three-factor model of schizophrenia phenomenology, consisting of a positive symptom dimension, a
negative symptom dimension, and a dimension of poor interpersonal functioning (as cited in Toomey, Kremen, Simpson, Samson, Seidman, Lyons, et al., 1997). Crow (1980) and Andreasen and Olsen (1982) identified a two-factor model consisting of a negative-positive symptom distinction. Liddle (1987) later added to this conceptualization by suggesting that the positive symptom dimension could be further reduced to include a “reality distortion” factor consisting of hallucinations and delusions, and a “disorganization” factor incorporating tangential and derailed speech and bizarre behavior. Despite replication of the three-factor model in multiple settings, more recent factor analytic work has suggested that more dimensions fit the data better than less complex, simpler models (Lenzenweger & Dworkin, 1996; Smith, Mar, & Turoff, 1998).

Lenzenweger & Dworkin (1996) found support for the multidimensional nature of schizophrenia symptoms by examining a correlation matrix of schizophrenia symptoms among participants in the major schizophrenia twin studies. Using confirmatory factor analysis, they identified four factors consisting of negative symptoms, premorbid social adjustment deficits, and a positive symptoms factor that is further subdivided into reality distortion and disorganization (originally proposed by Liddle, 1987). Smith, Mar, & Turoff (1998) completed a meta-analytic study to examine the structural validity of schizophrenia symptoms. Using a confirmatory factor analytic approach, the authors examined the intercorrelations among schizophrenia symptoms from all previous studies (N=28) found in the literature at the time (total cases approx.
Based on the results, the authors concluded that Liddle’s three-factor model consisting of a psychomotor poverty, disorganization, and reality distortion factor best fit the data. The authors added that they supplemented Liddle’s model by placing anhedonia-asociality with the psychomotor poverty factor and bizarre behavior with the disorganized factor (Smith, Mar, & Turoff, 1998).

Despite these findings, a three-factor model has been deemed insufficient and researchers have questioned the comprehensiveness of symptoms studied (Lenzenweger & Dworkin, 1996; Smith, Mar, Turoff, 1998). In another review, evidence across studies suggested that as many as eight broad dimensions (including a depression dimension) can account for the varied expressions of psychotic disorders and that these eight dimensions could be specified to include even narrower dimensions (Peralta & Cuesta, 2001). At the same time, Peralta and Cuesta (2001) have pointed out the disappointing and sometimes confusing nature of uncovering the dimensional structure of psychotic disorders, schizophrenia in particular. Results of factor analytic studies can differ substantially based on the statistics employed, chronicity of illness, and perhaps most importantly, the measure used to assess symptoms. Nonetheless, it is reasonable to hold that at least symptom dimensions reflecting positive, negative, and disorganized factors partially describe the manifestation of schizophrenia. However, there appears to be emerging evidence that a negative affective dimension may also play an important role in describing additional symptom dimensions that underlie the expression of schizophrenia spectrum disorders.
AFFECTIVE DISTURBANCES IN SCHIZOPHRENIA

Historical Perspectives

Despite discrepant views on the centrality of its role, affective and emotional disturbances have long been recognized as prominent symptoms associated with psychotic disorders. Kraepelin’s early work conceptualized affective disorders and schizophrenia as separate and distinct entities, with schizophrenia representing a degenerative disorder with poor prognosis (as cited in Hafner, Maurer, Trendler, an der Heiden, Schmidt, & Konnecke, 2005). Contrastingly, individuals with affective disorders or affective psychoses were believed to have a better prognosis with regard to functional and clinical outcome. Research has since challenged these assumptions, and the role of negative affect within psychotic spectrum disorders remains controversial (Resnick, Rosenheck & Lehman, 2004).

Bleuler (1911/1950) recognized affective disturbance as a fundamental symptom in schizophrenia and believed positive symptoms, such as hallucinations and delusions, represented “accessory” or secondary symptoms. He also identified a discrepancy between the subjective emotional experience reported by schizophrenia patients and the outward expression of that emotion. For example, Bleuler noted that some schizophrenia patients reported experiencing intense emotions but exhibited diminished emotional expression of those emotions. In contrast, Rado (1953; as cited in Kring, Kerr, Smith, & Neale,
1993) concluded that the diminished outward emotional expression shown by schizophrenia patients was a genuine portrayal of their subjective emotional experience. He posited that anhedonia, which refers to an inability to experience pleasure, explained the patient’s lack of emotional expression. He believed that anhedonia, which he described as “integrative pleasure deficiency,” was a primary characteristic of schizophrenia. However, Rado purported that anhedonia applied only to positive emotions and suggested that negative emotions were experienced more intensely, perhaps as an attempt to overcome an inability, or compromised ability, to experience pleasurable emotions (1969; as cited in Kring, Kerr, Smith, & Neale, 1993).

Building upon Rado’s speculations, Meehl (2001) differentiated between anhedonia of primary and secondary origins. Meehl preferred the word “hypohedonia” to reflect the dimensional nature of hedonic capacity, as opposed to anhedonia, which implies a complete absence of hedonic pleasure. He believed that hedonic capacity rested on a continuum of experience, as opposed to a dichotomy. Meehl (2001) indicated that hypohedonia could arise from primary origins, such as genetic contributions, or could develop from secondary origins, such as negative affect, that occurs throughout the course of illness. Meehl suggested that anhedonic individuals may be more susceptible to negative affect and thus, coined the term “aversive drift” (Meehl, 1962). Aversive drift refers to the schizophrenia patient’s tendency to interpret his world as dangerous, depressing, and stressful (Meehl, 1962).
Consistent with Bleuler’s formulations, empirical evidence has shown that individuals with schizophrenia-spectrum disorders show deficits with regard to both emotion recognition and emotional expression. However, research has demonstrated incongruence between patients’ reports of their emotions and the outward expression of those emotions. Based on their subjective reports, schizophrenia patients appear to experience both negative and positive emotions fully (Kring, Kerr, Smith & Neale, 1993). To illustrate, Kring and Neale (1996) noted that schizophrenia patients did not differ from controls in their self-reported experience of either positive or negative emotion after watching a mood-inducing film, although they did exhibit less facial emotion expressivity and increased response on physiological measures. Other studies have shown that schizophrenia patient groups often report decreased positive affect and increased negative affect regardless of whether or not the patients also suffered negative symptoms (Berenbaum & Fujita, 1994; Blanchard, Mueser, & Bellack, 1998; Kring & Earnst, 1999; Kring & Neale, 1996). Furthermore, even those non-medicated patients with negative symptoms such as flat or blunted affect, may actually be subjectively experiencing strong negative emotions (Kring, Kerr, Smith & Neale, 1993).

Despite these findings, it should be noted that negative symptoms in schizophrenia are rather heterogeneous with regard to their origins. Therefore, a subgroup of schizophrenia patients may actually experience diminished
emotionality (Cohen, Docherty, Nienow, & Dinzeo, 2003). For example, recent conceptualizations of negative symptoms differentiate between primary and secondary negative symptoms (Carpenter, Heinrichs, & Wagman, 1988). More specifically, some negative symptoms are the direct result of long-term neuroleptic use or result from the chronicity of the disorder (secondary), while others may be biological or developmental (primary) in nature. Patients with predominantly negative symptoms, often called “deficit-syndrome” patients, are thought to represent a subset of individuals, rather than representing the patient group as a whole. It should be noted that not all schizophrenia patients exhibit flat affect or associated negative symptoms (Kring & Neale, 1996). Fenton and McGlashan (1994) found that approximately 10% of schizophrenia patients showed deficit symptoms at illness onset and 25% showed those symptoms after five years. Nevertheless, taken as a whole, patient groups report experiences of increased negative affect and decreased positive affect, although a subgroup of patients may have difficulty experiencing both positive and negative emotions fully (Cohen, Docherty, Nienow, & Dinzeo, 2003).

It has been suggested that negative affect might play a role in the development of psychotic episodes. For instance, Bell, Bryson, & Lysaker (1997) have suggested that deficits in emotionality might represent a “diathesis” that impairs schizophrenia patients’ ability to manage their own negative affect, potentially triggering an acute episode or leading to relapse. Similarly, inability to identify negative emotions in others paired with the diminished capacity to cope
effectively with their own negative affect likely results in poor social skills and impaired interpersonal functioning characteristic of schizophrenia-spectrum disorders (Bellack, Mueser, Wade, & Sayers, & Morrison, 1992; Bell, Bryson, & Lysaker, 1997).

*The stress-vulnerability model of psychosis.* The diathesis-stress model has been at the forefront of etiological models of schizophrenia-spectrum disorders for many years (Nuechterlein et al., 1994; Norman & Malla, 1993; Walker & Diforio, 1997). In fact, heightened sensitivity to stress and affectively laden material is often thought to represent vulnerability for psychosis in predisposed individuals (Horan & Blanchard, 2003). According to the vulnerability-stress model, an individual is born with a genetic predisposition to developing psychosis and therefore believed to be “high risk.” However, not all individuals with this predisposition will develop a psychotic disorder (Meehl, 1962). Both biological and environmental stressors interact with this vulnerability and are believed to serve as impetus for the onset of psychotic symptoms (Walker & Diforio, 1997). Environmental stressors include pre, peri, and post-natal stressors, biological insults, stressful life events and even negative affective states themselves (Bell, Bryson, & Lysaker, 1997; Walker & Diforio, 1997).

The hypothalamic-pituitary-adrenal axis (HPA axis) is a primary system involved in an organism's response to stress. Individuals with psychotic disorders, schizophrenia spectrum traits (e.g., Schizotypal Personality Disorder) and at-risk individuals exhibit elevated baseline levels of cortisol (Garner et al.,
It is important to note that research has been mixed with regard to biological indicators of stress reactivity. For example, in laboratory settings in which patients have been exposed to psychosocial stressors, results have been inconclusive. Elman et al. (1998) and Walsh, Spelman, Sharifi, & Thakore (2005) found that stressful events caused increased cortisol release in psychotic patients. However, other studies have found an inverse correlation between stress and cortisol release (Jansen et al., 1998; Jansen, Gispen-de Wied, & Kahn, 2000; Marcelis, Cavalier, Gielen, Delespauly, & van Os, 2004). Therefore, additional research is needed to fully elucidate the role of the HPA-axis and its possible relation to psychosis.

Recent studies have examined the role of stressful life events in the emergence and course of psychotic symptoms and provide evidence for the role of an “affective pathway” in the etiology of schizophrenia (Myin-Germeys & van Os, 2007). For example, evidence suggests that patients with schizophrenia may be more susceptible to stressors such as criticism within the family environment (e.g., Butzlaff & Hooley, 1998). Similarly, research suggests that both psychotic and affective disorders can be triggered by life events (Bebbington, Wilkins, Jones, Forester, Murray, Toone, & Lewis, 1993). Individuals with psychosis, major depressive disorder, and psychosis-prone individuals have also shown elevated rates of the personality trait, neuroticism,
which reflects general mood reactivity and sensitivity to stress (Blanchard, Horan, & Brown, 2001).
NEGATIVE AFFECT IN SCHIZOPHRENIA-SPECTRUM DISORDERS

In recent years, the role of emotion and mood in the manifestation of psychotic symptoms has been given increased attention. In particular, researchers and practitioners are beginning to emphasize the importance of addressing the affective non-psychotic components of psychotic disorders, such as depression and anxiety. Despite methodological differences among studies, it is obvious that affective syndromes frequently occur throughout the course of schizophrenia spectrum disorders, including prodromal, acute, and post-psychotic phases and have direct relevance to functional outcome. For instance, Hafner et al (2005) examined causal associations between schizophrenia, depressive symptoms, and episodes of depression in untreated, first episode patients and found that the lifetime prevalence of depressed mood in schizophrenia patients was 83% at the time of their first admission. In fact, there was considerable overlap among symptoms and functional outcome between patients with schizophrenia and patients with unipolar depression as early as four years prior to the onset of their disorder. More specifically, the groups could only be differentiated once the emergence of psychotic symptoms emerged in the schizophrenia group.

Additional studies support the contention that negative affect may predict poorer functioning in a variety of domains including heightened risk for relapse, more hospitalizations, poorer social functioning, diminished work performance,

There is conjecture and some evidence that point to depressive episodes or high anxiety as representing a diathesis for transition into a full-blown psychotic episode in at-risk individuals or a relapse in individuals with a history of the disorder (Yung & McGorry, 1996; Yung, Phillips, Yuen, & McGorry, 2004). Studies of prodromal patients suggest that anxiety, depression, and irritability immediately precede the onset of psychotic symptoms (Yung & McGorry, 1996). In fact, some researchers suggest that depression in particular plays a central role in psychotic relapse (an der Heiden, Konnecke, Maurer, Ropeter, & Hafner, 2005). In a recent study, Yung et al. (2007) identified a simultaneous reduction in both depression and psychotic like experiences in at-risk individuals after six months and suggested that amelioration of depressive symptoms may lessen the intensity of psychotic-like experiences or prohibit psychotic symptoms from developing altogether.

Differential Diagnosis

Differential diagnosis of affective disturbance in schizophrenia, particularly depression and anxiety, is often a complex endeavor due to multiple etiologies
and phenotypic copies of symptoms of other disorders (as cited in Siris & Bench, 2003). For example, organic factors such as autoimmune disorders (e.g., thyroid dysfunction), cardiovascular disease, and neurological disorders can serve as an impetus to depressive episodes (Siris & Bench, 2003). Similarly, medications, particularly the neuroleptics often used to treat psychosis, can result in syndromes that mimic the symptoms of depression and anxiety, such as drug-induced akinesia (dysphoric mood) and akathisia or an inner restlessness/urge to move (Siris & Bench, 2003). Increases in depression and anxiety symptoms have been associated with traditional neuroleptic medications in acute psychotic episodes, but research is still needed with regard to newer atypical antipsychotics (Goff & Shader, 2003). While the relationship between medication, depression and anxiety in schizophrenia remains unclear, it is likely that the majority of depression and anxiety in patients is not directly related to medication (Barnes, Buckley, & Schultz, 2003).

Symptoms of anxiety and depression are often overlooked or disregarded in treatment settings due to their confusion and overlap with the negative symptom cluster characteristic of a subset of schizophrenia patients (Pallanti, Quercioli, & Holander, 2004). According to the DSM-IV, anhedonia, amotivational syndrome, affective flattening, alogia, and social withdrawal constitute the negative symptom cluster in schizophrenia-spectrum disorders (DSM-IV-TR, APA, 2000). However, anhedonia, amotivation, and social withdrawal are also commonly observed in individuals with depression. At the
same time, there is some evidence to suggest that anhedonia may represent a stable “trait disturbance” with regard to schizophrenia-spectrum disorders, and a transient “state disturbance” in affective disorders (Blanchard, Horan, & Brown, 2001; Katsanis, Iacono, Beiser, & Lacey, 1992).

Experts have suggested that the primary distinguishing feature between negative symptoms and depression is a transient, depressive mood state and painful affect, as opposed to the flat affect characteristic of negative symptoms (Siris & Bench, 2003). However, as reported earlier, there is some evidence to suggest that while a subset of patients present as having negative symptoms, they may actually be experiencing intense emotional states (Kring, Kerr, Smith, & Neale, 1993; Kring & Neale, 1996). Others have suggested that anhedonia, characteristic of both schizophrenia and depression, may underlie a susceptibility to negative affect (Meehl, 2001). In psychotic disorders, anhedonia, is thought be temporally stable trait condition and has been correlated with high levels of neuroticism (Berenbaum & Fujita, 1994). Neuroticism, or a tendency to ruminate and worry, refers to trait levels of negative affect and has also been associated with later development of a psychotic disorder (Berenbaum & Fujita, 1994; Horan, Subotnik, Reise, Ventura, & Nuechterlein, 2005; van Os & Jones, 2001). However, neuroticism has been associated with increased risk for broad domains of psychopathology and therefore may not be specific to development of a psychotic disorder (Watson, Kotav, & Gamez, 2006).
Depression

Numerous scholars have outlined the problems with conceptualizing affective and psychotic disorders as distinct and there is little empirical reason for the historical distinction between these disorders (Freeman & Garety, 2003). As described previously, factor analytic studies have repeatedly identified a depression-related dimension in both schizophrenia and depressive disorders (Lenzenweger & Dworkin, 1996; Liddle, 1987; McGorry, Bell, Dudgeon, & Jackson, 1998; van Os et al, 1999). Furthermore, family and genetic studies have suggested that schizophrenia-spectrum disorders and affective disorders may share some common underlying etiology (e.g., Crow, 1995; Taylor, 1992). In addition, according to the widely accepted diathesis-stress model of schizophrenia, individuals with a genetic predisposition towards psychosis may cross a threshold into an acute episode after experiencing negative life events or negative affective states (Walker & Diforio, 1997; for a review, see Bebbington & Kuipers, 2003). Research studies examining correlates of schizophrenia-spectrum disorders in psychosis-prone individuals have identified depression and social anxiety using cross-sectional approaches (Lewandowski et al., 2006). A significant portion of individuals identified as psychosis-prone by self-report measures have been later found to develop a major affective disorder (Chapman, Chapman, Kwapis, Eckblad, & Zinser, 1994; Kwapis, Miller, Zinser, Chapman, & Chapman, 1997; Verdoux et al., 1999). In addition, psychosis-prone individuals who responded to hallucinatory experiences with depressed and anxious mood
(e.g., attributing a voice to a malevolent source) were later diagnosed with an actual psychotic disorder, as opposed to those individuals who experienced psychotic-like experiences but did not respond with depression or anxiety (Krabbendam & Van Os, 2005; Krabbendam, Myin-Germeys, Bak, & Van Os, 2004).

Depression is a component of negative affect commonly observed in individuals with schizophrenia-spectrum disorders and acute psychosis (Siris & Bench, 2003). Early findings suggested that mood disorders, and unipolar depression in particular, were as high as 6% to 75% in patients with schizophrenia (with a modal value of 25%) (McGlashan & Carpenter, 1979). However, Hafner et al (2005) and Siris (2000) are careful to note that methodological differences in early studies account for this range of variability. For example, depression has been defined as symptom, syndrome, and affect in various studies, which makes it difficult to formulate clear conclusions from the literature, given that many studies do not differentiate whether they are examining a disorder, symptoms, affective state, or syndromes. In addition, differences in stage of schizophrenic illness and differences in prevalence rates (lifetime versus point prevalence) further convolute the results. Discrepancies in the operational definitions of schizophrenia-spectrum and depressive disorders and their respective episodes make any conclusive interpretations difficult to reach.
Furthermore, epidemiological studies have shown that individuals with schizophrenia have a significant probability of developing a major depressive episode in their lifetime, approaching a prevalence rate as high as 80% (as cited in Fenton, 2000). Additionally, the National Comorbidity Study found that “81% of patients with schizophrenia were also diagnosed with a mood disorder some time in their lives; 59% of patients with schizophrenia were also diagnosed with unipolar depression, and 22% had a diagnosis of bipolar disorder at some point in their lives” (Fenton, 2000, p. 35). Depressive episodes have been identified across stages of the illness, including prodromal, acute, and post-psychotic depressive states with some indication that depressive states are more severe in actively psychotic individuals (Lancon, Auquier, Reine, Benard, & Addington, 2001). Interestingly, there is some evidence to suggest that depressive states remit in conjunction with the remission of an acute psychotic episode (Koreen, Siris, Chakos, Alvir, Mayerhoff, & Lieberman, 1993; Birchwood, Iqbal, Chadwick, & Trower, 2000). In one recent study, Sands & Harrow (1999) examined the depressive syndrome in the longitudinal course of schizophrenia with assessments at 4.5 and 7.5-year follow-ups after hospitalization. A substantial number of schizophrenia patients (36%) experienced a full depressive syndrome at both follow-ups. An additional 14% showed depressive symptoms that were less severe. In a later follow-up study with the same patients, the authors found that depression represented an independent symptom dimension that was stable.
across time extending ten years after the initial hospitalization (Marengo, Harrow, Herbener, & Sands, 2000).

In one cross-sectional study, Zisook et al. (1999) assessed frequency and severity of depressive symptoms among middle age and elderly schizophrenia patients. Patients who were currently experiencing a major depressive episode or who had received a previous diagnosis of schizoaffective disorder were excluded from the study. Sixty individuals diagnosed with schizophrenia (30 male, 30 female) and sixty normal comparison control subjects were matched for age and gender. Individuals were diagnosed based on DSM-III criteria for schizophrenia, while normal controls demonstrated no current or previous psychiatric history. Results showed that depression was more severe and more frequent in schizophrenia patients than those mood symptoms experienced in a control group without a history of schizophrenia-spectrum disorder. There were no significant differences for gender and no gender X diagnosis interaction was identified.

The stability of depressive symptoms in schizophrenia over time is less clear. Green, Nuechterlein, Ventura, and Mintz (1990) examined the temporal relationship between depressive and psychotic symptoms in a small sample of schizophrenia patients. Using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962; Overall, 1988), subjects were followed every two weeks for at least one year. Results indicated that onset of depressive symptoms occurred simultaneously with onset of psychotic symptoms during the early phase of
schizophrenia, but not at later stages. The authors reported that they used categorical distinctions of depression and schizophrenia and suggested that future research focus on dimensional classifications that incorporate subclinical levels of depression and psychosis. In contrast, depressive symptoms occurring during the chronic phase are poor prognostic indicators resulting in poorer overall functioning and potential suicidality (as cited in Kempf, Hussein, & Potash, 2005). In another recent longitudinal study, at least one depressive symptom was identified in 30-35% of schizophrenia patients over the course of a twelve-year follow-up (an der Heiden, Konnecke, Maurer, Ropeter, Hafner, 2005). Consistent with previous research, the authors also found that depressive symptoms were particularly pronounced during an acute episode.

In an additional study, Norman and Malla (1994) examined the temporal relationship between depression and anxiety symptoms, which they labeled as “dysphoria,” and positive symptoms of psychosis. Individuals diagnosed with schizophrenia were evaluated monthly across a range of 12 to 24 months. The researchers assessed positive and negative symptoms with the Schedule for Assessment of Negative Symptoms, and Schedule for Assessment of Positive Symptoms (Andreasen 1983; Andreasen, 1984). Depression and anxiety were assessed using the Beck Depression Inventory (BDI; Beck, 1978) and the Self Evaluation Questionnaire, respectively (SEQ, Spielberger, Gorsuch, & Luschene, 1968). In addition to finding strong correlations between depression and anxiety
across time, they also found support of a relationship between negative affect and positive symptoms of psychosis, particularly delusions and hallucinations.

Anxiety

Research has shown that anxiety disorders frequently co-occur with psychotic disorders at a much higher rate than those reported in the general population, particularly panic disorder, obsessive-compulsive disorder, and social anxiety (Pokos & Castle, 2006). In addition, anxiety symptoms and/or actual disorders may precede the onset of psychotic episodes (Strakowski, Keck, McElroy, Lonczak, & West, 1995). Despite anecdotal evidence from clinicians regarding the co-occurrence of anxiety disorders and psychotic disorders, there has been little attention paid to this phenomenon in the literature (Pokos & Castle, 2006). Estimates of comorbidity have ranged from 7 to 40% with regard to social anxiety (e.g., Blanchard, Mueser, & Bellack, 1998; Cassano, 1998; Cosoff and Hafner, 1998). The range of prevalence for obsessive-compulsive symptoms in conjunction with schizophrenia-spectrum disorders was 3.5 to 47.6% (as cited in Pokos and Castle, 2006). As with depression, the range of estimates reported is high likely due to methodological issues such as time frame of illness (Voges & Addington, 2005).

In a recent meta-analysis of the literature to date, the cross-sectional comorbidity rates of anxiety and schizophrenia-spectrum disorders were high for a variety of anxiety symptoms, including those consistent with generalized anxiety, obsessive-compulsive symptoms, and social phobia (see Pokos and
Castle for a review). As with depression, the temporal relationship between anxiety and psychosis has not been extensively researched. However, there has been some evidence to suggest that anxiety disorders occur prior to the development of a schizophrenia spectrum disorder in approximately 50% of patients (as cited in Pokos and Castle, 2006.). Similarly, in a study that examined individuals with first-episode psychosis, results showed that a significant number of patients suffered anxiety disorders, excluding PTSD, prior to their first psychotic break (Strakowski, Keck, McElroy, Lonczak, & West, 1995). In another study, those adolescents who later developed schizophrenia-spectrum disorders reported higher elevations of anxiety symptoms prior to the onset of the disorder (Kugelmass et al., 1995).
NEGATIVE AFFECT AND POSITIVE SYMPTOMS

Despite phenotypic similarities of depression and anxiety with a negative symptom dimension, research actually suggests that depression and anxiety are most often associated with a positive symptom dimension (Emsley, Oosthuizen, Joubert, Roberts, & Stein, 1999; Lewendowski et al., 2006; Lysaker, Bell, Bioty, & Zito, 1995). Furthermore, research has questioned any meaningful distinction of anxiety and depressive symptoms, given their high frequency of co-occurrence (Gorman, 1997) that they both can be represented in terms of the broad dimension of internalizing psychopathology (Krueger & Markon, 2006), and it has been suggested that depression and anxiety symptoms may be grouped together to represent a more inclusive “negative affect” factor (Norman & Malla, 1994). As previously reviewed, the extant literature suggests a relationship between negative affect, and the onset and maintenance of hallucinations and delusions in both patient populations and at-risk populations (Delespaul, deVries, & van Os, 2002; Freeman & Garety, 2003; Paulik, Badcock, Maybery, 2006, Norman & Malla, 1994; Zisook, 1999). Negative affective states have been observed prior to the development of a psychotic illness or as a direct precipitant to a psychotic relapse even in neuroleptic-naïve individuals (Subotnik & Nuechterlein, 1988; Tarrier, Barrowclough, & Bhamra, 1991). As described earlier, Norman and Malla (1994) examined the longitudinal relationship of depressive and anxiety symptoms in schizophrenia outpatients and found that depression and anxiety
were strongly associated with positive symptoms. Expanding on previous research in a later study, researchers found correlations between anxiety and reality distortion. (Norman, Malla, Cortese, & Diaz, 1998). Blanchard, Mueser, and Bellack (1992) also found that negative mood states at baseline (via patient self-report) predicted thought disturbance after six months. Researchers have also found evidence that arousal of negative affect, such as that which occurs after talking about stressful life events, exacerbates disordered language and communication patterns in schizophrenia patients and their nonpsychotic family members (Docherty, Evas, Sledge, Seibyl & Krystal, 1994; Docherty, Sledge, & Wexler, 1994; Docherty, Grosh, & Wexler, 1996; Docherty, Hall, & Gordinier, 1998; Docherty, Rhinewine, Nienow, & Cohen, 2001). For instance, Docherty, Sledge, & Wexler (1994) found evidence that negative affect elicited more speech abnormalities suggestive of formal thought disorder than positive affect. This affective reactivity of language was related to the severity of delusions and hallucinations in particular (Docherty & Hebert, 1997). In a related study, Cohen and Docherty (2004) found that discussing events associated with positive affect lessens speech disorder in those individuals with severe symptoms, replicating an earlier study (Docherty, Evans, Sledge, Seibyl & Krystal, 1994).

Asarnow, Cromwell, & Rennick (1978) have suggested that negative affect reactivity occurs more frequently in individuals with a family history of schizophrenia, rather than patients without a family history and research has shown support for this assertion (Norman & Malla, 2000). For example,
subsequent research has shown support that a familial history of the disorder indicates more speech abnormalities associated with negative affect in both inpatient and outpatient samples and may reflect pathology in physiological inhibitory processes such as those involved in habituation responses and sensorimotor gating (Docherty & Grillon, 1995; Docherty, Grosh & Wexler, 1996). To illustrate, there is some research that suggests those patients who are more susceptible to negative affect may also be more susceptible to stimuli in the environment (Docherty & Grillon, 1995).

There have been several studies examining potential affective risk factors in non-psychotic, yet high-risk individuals. To state differently, individuals identified as at-risk for a psychotic disorder have shown evidence of affective disturbance prior to the onset of a psychotic disorder. For instance, Krabbendam, Myin-Germeys, Bak, & VanOs (2004) and Krabbendam, Myin-Germeys, Hanssen, de Graaf, Vollebergh, Bak, M. (2005) found that depressed mood in individuals in the general population who also reported hallucinatory experiences increased the risk of the onset of a clinical disorder such as psychosis. Similarly, Krabbendam and van Os (2005) conducted a longitudinal study examining the psychotic risk and affective experiences in a large sample of general population subjects. They found that individuals without a history of psychotic disorder but who reported some hallucinatory experiences at baseline were more likely to develop a clinical disorder within three years if they also experienced depressed mood. However, they are careful to note that this finding
was mediated by the presence of delusional ideation and suggest that depressed mood may actually result after delusional experiences develop.

In summary, research demonstrates high rates of comorbidity among disorders and suggests that “pure” samples of diagnostic categories may not be representative of the majority of patients presenting with psychopathology (Krueger et al., 1998). Patients meeting criteria for one disorder in the absence of additional psychopathology are rare in the clinical population, with the majority of patients having multiple disorders or symptoms of psychopathology at a given time (Kessler et al., 1994). Dimensional perspectives provide a unique opportunity to examine meaningful covariation among symptom clusters and may help to elucidate underlying etiological mechanisms.

Cross-sectional associations have been found between negative affect, particularly depression and anxiety, and positive psychotic symptoms and likely influence treatment outcomes such as quality of life and post-hospital adjustment (e.g., van Os et al., 1999). However, there is little longitudinal research on this topic and few studies have utilized a dimensional approach. The current study seeks to add to the psychosis literature by examining the longitudinal relationship of negative affect and positive symptoms of psychosis using a dimensional framework. If a meaningful relationship is found, it could provide evidence for a possible etiological role of negative affect in the onset and/or maintenance of positive symptoms of psychosis.
LATENT VARIABLE MODELING OF PSYCHOPATHOLOGY

Structural equation modeling represents an ideal approach for analysis of longitudinal panel data from a dimensional perspective. Given multiple data points across time, (temporal) causal inferences can be stated more strongly than with cross-sectional approaches (Klem, 2000). It allows researchers to test competing cause-effect models of latent variables in more precise ways than traditional approaches (Farrell, 1994). Structural equation modeling also allows one to test the plausibility of reciprocal relationships among latent variables and to test the consistency of these relationships across time and groups while accounting for variation due to measurement error (Hoyle & Smith, 1994).

Structural equation modeling (SEM) provides a unique and flexible approach for examining the relationship among latent constructs (Hoyle & Smith, 1994) and has utility for use in quasi-experimental and non-experimental research (Bentler, 1980). SEM has particular utility with the use of longitudinal panel data in which the measures are taken on the same participants at two or more time points (Klem, 2000). In the literature, the term SEM is used interchangeably with several other terms including *causal modeling, covariance structure analysis, path analysis,* and *linear structural relations (LISREL).* With SEM, investigators can examine the relations between hypothetical, unobservable constructs called latent variables (e.g., depression, anxiety) as opposed to manifest, or directly observable, constructs (e.g., individual items on
a measurement scale). SEM is a confirmatory approach that permits the researcher to assess how well the model fits the data through statistics that assess goodness-of-fit (Bentler, 1980). Besides the structural component of SEM (i.e., factor-to-factor relations), it also includes a confirmatory or measurement factor analytic model (CFA), which involves the relationships between the measured variables and the latent variables (i.e., the variable-to-factor relations) (Anderson & Gerbing, 1988).

Manifest variables (MVs) serve as indicators of specific factors, or latent variables (LVs), and these LVs are believed to account for the correlations among observed variables (i.e., MVs) (Bentler, 1980). In order to test latent variable models, one must initially specify the relationships between the manifest (observed) indicators and the latent construct, also referred to as the measurement model (Farrell, 1994). More specifically, correlations between observed, or manifest variables are accounted for by the latent variables they represent (Bentler, 1988). Structural models refer to the relationship between latent variables (Farrell, 1994).

An advantage of using latent variable causal models is the researchers’ ability to separate the common variance among MVs from unique and error variance, thereby producing a less biased estimate than MV models (Bentler, 1980). More specifically, MV causal models produce a biased estimate of the correlations among variables because the correlation coefficient’s absolute value is affected by that variable’s measurement error (Bentler, 1980). To state
differently, in MV approaches, covariation could be due to either shared error variance or shared trait variance; MV models do not parcel out sources of measurement variance from overall variance. However, SEM allows one to model covariation that is caused by shared error variance and thus, allows for more precise measurements (Hoyle & Smith, 1994).
STATEMENT OF THE PROBLEM

Cross-sectional examinations of affect disturbance in schizophrenia clearly demonstrate the co-occurrence of negative mood states, such as depression and anxiety, and schizophrenia-spectrum disorders, thus, raises the question of whether affective disorders and psychotic disorders are categorically (i.e., distinct) disorders. However, longitudinal research examining the relationship between negative affect and schizophrenia-spectrum disorders is lacking (Sands & Harrow, 1999; an der Heiden, Konnecke, Maurer, Ropeter, & Hafner, 2005), with some research suggesting a possible fundamental relationship between negative affect and positive psychotic symptoms such as hallucinations, delusions, and aberrant speech patterns (e.g., Docherty & Hebert, 1997). Lastly, further understanding of the role of negative affect in schizophrenia-spectrum disorders and its influence on outcome measures is critical for accurate and ethical treatment planning, as well as development of comprehensive models of its etiology.

The purpose of the proposed study was to examine the longitudinal factor-to-factor relationships between a negative affect dimension and a positive symptom dimension of psychosis in patients with psychotic disorders. For purposes of the present study, a longitudinal cross-lagged panel design was used, which involves the evaluation of two or more variables (e.g., negative affect, positive symptoms of psychosis) at two or more points in time. The cross-
lagged design allows for the investigation of cross-sectional or synchronous associations between latent variables, as well as cross-lagged effects, which involve the long-term prediction of one latent variable by the other.

Hypotheses

1). Variables Anxiety, Guilt, and Depressed Mood on the Brief Psychiatric Rating Scale could be used to model a Negative Affect Factor.

2). Variables assessing Hallucinations, Conceptual Disorganization, and Unusual Thought Content on the BPRS could be used to model a Positive Symptom Factor.

3). Synchronous (cross-sectional) associations between the negative affect and positive symptom factors were expected at each individual time frame.

4). Levels of negative affect and positive symptoms would be predicted by their respective baseline levels and thus were expected to be stable over time (stability effects). In addition, correlations between these factors (both exogenous and endogenous) at each assessment time point were expected to be moderate to strong and thus provide evidence of covariation of these factors.

5). A temporal causal relationship was expected between the negative affect factor and positive symptom factor (cross-lagged effects). Specifically, it is expected that negative affect would predict an increase in positive symptoms at subsequent intervals.

6). The proposed model will have an acceptable goodness of fit for the data.
METHOD

Participants

Patients were drawn from a database of a larger study examining violence among civilly committed patients (MacArthur Violence Risk Assessment Study). The John D. and Catherine T. MacArthur Foundation Research Network on Mental Health and the Law and the National Institute of Mental Health funded the original study. However, for purposes of the present study, a subset of research participant data was used. Data was collected between 1992 and 1995 and included patients who had been hospitalized for 21 days or less at one of several inpatient sites: a university-based specialty hospital (Western Psychiatric Institute and Clinic), a public mental health center (Western Missouri Mental Health Center), a state psychiatric hospital (Worcester State Hospital) and a university based general hospital (University of Massachusetts Medical Center). Patients were included in the data analysis if they received a baseline principal diagnosis consisting of schizophrenia ($n=137$), schizophreniform ($n=2$), schizoaffective ($n=66$), brief reactive psychosis ($n=2$), delusional disorder ($n=6$) or atypical psychosis ($n=32$). At baseline, participants include 245 civilly committed patients between the ages of 18 and 40 years old (mean age = 30.87) who are of European-American ($n=127$), African American descent ($n=112$) or Hispanic descent ($n=6$). The model was run both with and without schizoaffective disordered individuals to control for mood symptoms and to have further confidence that any significant results were not due to this subgroup alone.
Hispanic participants were recruited from the Worcester site only. There were 87 females and 158 males.

Measures

The Brief Psychiatric Rating Scale (BPRS) is a widely used clinician-administered instrument designed to assess treatment change across a broad spectrum of psychopathology symptoms (Overall & Gorham, 1962; Ventura, Green, Shaner, & Liberman, 1993). The initial development of the BPRS included 16 items but was later supplemented by 18-item Anchored Version (BPRS-A) and most recently, a 24-item version (BPRS-E) (Overall & Gorham, 1962; Lukoff, Nuechterlein, & Ventura, 1986). The BPRS includes 18 symptom constructs that are addressed via the patient’s self-report and observer ratings. Scoring is conducted on a 7-point Likert-type scale ranging from 1 (not present) to 7 (severe) and scores on individuals items are summed to provide a total score. However, the BPRS allows for scores at the item or dimensional level, providing an ideal avenue for assessing meaningful covariation among symptoms (Lachar, Espadas, & Bailley, 2004).

The BPRS has excellent inter-rater reliability, and studies have reported a range of median Pearson correlation coefficients and intra-class correlation coefficients ranging from .67 to .88 (as cited in Ventura et al., 1993). On average, self-report ratings were more reliable than those ratings of the patient’s behavior by the observer. Ventura and colleagues (1993) assessed inter-rater reliability of the BPRS based upon individuals with advanced degrees and licensure (MDs,
PhDs, and MSWs) and those having predoctoral degrees but no advanced training or licensure. In order to enhance reliability and validity, the original version of the BPRS was supplemented with an administration manual, interview guide, and supplemental training materials, and establishment of anchor points to assist in more accurate rating (e.g., Lukoff et al., 1986; Ventura et al., 1993). Results indicated that both groups, regardless of licensure or degrees, showed excellent inter-rater reliability upon completion of the training program on 22 of the 24 BPRS items.

Original factor analytic models concluded that the BPRS consisted of four relatively independent dimensions and has found much support in the research literature (Overall, Hollister, and Pichot, 1967). The most common and consistently identified factors include a Positive Symptom Factor, a Negative Symptom Factor, a Psychological Discomfort Factor, and a Resistance Factor. Items tapping unusual thought content, hallucinatory behavior, conceptual disorganization, disorientation and/or grandiosity have been found to load heavily on the Positive Symptoms Factor. A Negative Symptoms factor includes items of motor retardation, blunted affect, and emotional withdrawal. The Psychological Discomfort factor is made up of items tapping depression, anxiety, guilt, and somatization, and the Resistance subscale consisting of hostility, uncooperativeness and excitement. However, it should be noted that these specific items have various factor loadings across studies (as cited in Shafer, 2005).
A recent meta-analysis identified strong support for a four-factor structure of the BPRS (Shafer, 2005) similar to Overall’s original model (Overall, Hollister, & Pichot, 1967). Strong and consistent evidence was identified for an affect-related factor, a positive symptoms factor, and negative symptoms factor. There was also evidence of a resistance factor, although to a lesser degree than other symptoms.

Long and Brekke (1999) conducted a longitudinal examination of the factor structure of the 16-item BPRS and the 18-item BPRS with the purpose of determining invariance across time. The authors used the 4-factor CFA model of Mueser, Curran, & McHugo (1997) to establish factorial invariance for both the 16 and 18 item versions. Results showed that the 16-item version had more configural invariance, suggesting that the factors (Thought Disturbance, Anergia, Affect, and Disorganization) remained stable over the three year time period. Configural invariance refers to the ability of a model to remain consistent across groups or time.

The BPRS has been correlated with several additional measures of psychopathology. For example, the Psychological Discomfort scale, which assesses negative affective traits of depression and anxiety, has shown moderate to high correlations with both the Beck Depression Inventory (r=.64), the Calgary Rating Scale for Depression in Schizophrenia (r=.81), and the Hamilton Rating Scale for Depression (for a review, see Lachar, Espadas, & Bailley, 2004). A Positive Symptoms factor was correlated with the Scale for the
Assessment of Positive Symptoms (r=.66), and the Positive and Negative Syndrome Scale positive symptom scale (r=.92).

Procedure

During hospitalization, patients provided historical and demographic information. Next, research clinicians (having PhDs, MAs, or MSW degrees) interviewed participants using the Structured Clinical Interview for DSM-III to obtain diagnostic information, which was then compared to the patient’s initial chart diagnosis. The match rate between these diagnoses was 85.7%. In those cases of disagreement, a psychiatrist was consulted and made the final diagnosis. After patients were released into the community after hospitalization, they were contacted every ten weeks for follow-up over the course of one year after their discharge date from the hospitals. The majority of individuals were interviewed in person at follow-up (89%) with a subset providing telephone interviews (11%).
RESULTS

The following analyses were conducted with SPSS Version 16.0 for Macintosh. The data were screened for multivariate normality to ensure that “(1) that the univariate distributions are normal, (2) the joint distributions of all variable combinations are normal and (3) all bivariate scatter plots are linear and homoscedastic.” (Kline, 1998, p 48-49). Data were also checked for skew and kurtosis to determine if transformations of the data were needed. Overall, the BPRS variables showed minimal skew and kurtosis given the size of the sample. In addition, the dataset was checked for any missing values and/or outliers. With respect to gender, race, and DSM diagnosis, missing data analyses that incorporated Pearson's Chi square values and t-test values, revealed no statistically significant differences in those individuals with scores on all BPRS items across all three time frames and those missing BPRS data at specific time points. Therefore, all analyses incorporated the entire sample (N=245).

The SEM model was tested using Mplus Software (Muthen & Muthen, 1998-2001). Mplus is a software program specifically designed to address questions posed by structural equation models. SEM was used to assess the omnibus relationship between negative affect, and positive symptoms. A CFA was conducted to specify and test the fit of the BPRS items on their respective factors. Coefficient alphas were calculated to assess internal consistency of the BPRS variable sets. Correlation and covariance matrices among variables were
generated. In addition, correlations between BPRS factors were analyzed to determine if they differ by gender or ethnicity. Upon estimation of the model, a goodness-of-fit test was used to determine how the model fits the data. The time points analyzed were baseline, 1st follow-up (Time 1), which occurred ten weeks after the most recent hospitalization, and 2nd follow up (Time 2) which occurred twenty weeks after most recent hospitalization.

Descriptive Statistics

Tables 1, 2, and 3 provide descriptive statistics for each manifest variable at Baseline, Time 1 (first follow-up conducted after initial ten weeks), and Time 2 (second follow-up conducted after twenty weeks), respectively. Correlations among items within each time frame were also analyzed and are provided in Tables 4, 5, and 6. Tables 7, 8, and 9 represent correlations among BPRS items across time frames. Correlations examining the effects of gender and race were also conducted to determine the associations with each indicator variable but revealed only modest demographic effects on the manifest variables within the model. With respect to gender and item correlations across time, women reported slightly more guilt feelings at Time 2 ($r^2 = .032$) and slightly more Hallucinatory Behavior at Time 2 ($r^2 = .022$). Given that the sample included only six Hispanic participants, race and item correlations were analyzed using only European-American and African American participants. None of the correlations across Race and BPRS items were statistically significant at any time point.
### Table 1

**Descriptive Statistics of Variables at Baseline**

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>Skew</th>
<th>Kurtosis</th>
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<tbody>
<tr>
<td>A</td>
<td>3.12</td>
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<tr>
<td>DM</td>
<td>2.70</td>
<td>1.95</td>
<td>.92</td>
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<tr>
<td>G</td>
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<td>1.58</td>
<td>1.33</td>
</tr>
<tr>
<td>H</td>
<td>3.00</td>
<td>2.05</td>
<td>.46</td>
<td>-1.27</td>
</tr>
<tr>
<td>CD</td>
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<td>1.43</td>
<td>.57</td>
<td>-.51</td>
</tr>
<tr>
<td>UTC</td>
<td>4.17</td>
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<td>-.29</td>
<td>-1.38</td>
</tr>
</tbody>
</table>

*Note:* A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content

### Table 2

**Descriptive Statistics of Variables at Time 1 Follow-up (10 weeks after baseline)**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>Skew</th>
<th>Kurtosis</th>
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<td>H</td>
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<tr>
<td>UTC</td>
<td>2.35</td>
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<td>-.61</td>
</tr>
</tbody>
</table>

*Note:* A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content
Table 3

*Descriptive Statistics of Variables at Time 2 Follow up (20 weeks after baseline)*

<table>
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<th>Variable</th>
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<th>SD</th>
<th>Skew</th>
<th>Kurtosis</th>
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<td>UTC</td>
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</table>

*Note:* A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content

Table 4

*Correlations across BPRS Items at Baseline*

<table>
<thead>
<tr>
<th>Items</th>
<th>A</th>
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<th>G</th>
<th>H</th>
<th>CD</th>
<th>UTC</th>
</tr>
</thead>
<tbody>
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<td>.408**</td>
<td>.219**</td>
<td>-.104</td>
<td>.121</td>
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<tr>
<td>DM</td>
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<td>.369**</td>
<td>.305**</td>
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<td>.036</td>
<td></td>
</tr>
<tr>
<td>G</td>
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<td>.200**</td>
<td>-.115</td>
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<td>H</td>
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<td></td>
<td></td>
<td>-.081</td>
<td>.243**</td>
<td></td>
</tr>
<tr>
<td>CD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.273**</td>
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<td></td>
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</tr>
</tbody>
</table>

*Note:* **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content*
Table 5

*Correlations across BPRS Items at Time 1 (10 weeks after baseline)*

<table>
<thead>
<tr>
<th>Items</th>
<th>A</th>
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</tbody>
</table>

Note: **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content

Table 6

*Correlations across BPRS Items at Time 2 (20 weeks after baseline)*

<table>
<thead>
<tr>
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</table>

Note: **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content
### Table 7

**Correlations across BPRS Items at Baseline and Time 1 (10 weeks after baseline)**

<table>
<thead>
<tr>
<th>Items</th>
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**Note:** **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content; x axis refers to Baseline, y axis refers to Time 1

### Table 8

**Correlations across BPRS Items at Baseline and Time 2 (20 weeks after baseline)**

<table>
<thead>
<tr>
<th>Items</th>
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</table>

**Note:** **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content; x axis refers to Baseline, y axis refers to Time 2
Table 9

*Correlations across BPRS Items at Time 1 and Time 2 (10, 20 weeks after baseline)*

<table>
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<th>Items</th>
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<td>.588**</td>
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Note: **p < .01, *p < .05, A=Anxiety, DM=Depressive Mood, G=Guilt, H=Hallucinations, CD=Conceptual Disorganization, UTC=Unusual Thought Content; x axis refers to Time 1 and y axis refers to Time 2
STRUCTURAL EQUATION MODELING RESULTS

Confirmatory factor analysis and structural equation modeling procedures were conducted with Mplus Software (Muthen & Muthen, 2001) using the full information maximum likelihood estimation procedures (i.e., using all pairwise data available). To determine model fit, comparative fit index (CFI: Bentler, 1990), root mean square of approximation (RMSEA), standardized root mean square residual (SRMR) and Bayesian information criterion (BIC) statistics were employed per recommendations proposed by Hu & Bentler (1999). More specifically, the chi-square test is insufficient to determine model fit and experts have recommended the use of additional fit indices such as CFI, RMSEA, and SRMR. When compared to other fit indices, one strength of the CFI is its ability to handle problems with sampling variability (Hu & Bentler, 1999 check). Traditionally, a CFI value of .90 or higher has been considered adequate (Hoyle, 1995). However, more recent research has suggested CFI values close to .95 or larger may be preferred for identifying excellent model fit (Hu & Bentler, 1999). Also, a RMSEA value of less than or equal to .06 are acceptable indices of excellent model fit. SRMR values range between 0 and 1; however, a good fitting model results in a value <.08 (Hu & Bentler, 1999). The information-theoretic fit criterion used in current analyses was the Bayesian Information Criterion (BIC). BIC is widely used for latent variable modeling (e.g., Krueger, Markon, Patrick, & Iacono, 2005) and can be used for comparisons between
latent variable models. Minimizing BIC corresponds to maximizing model parsimony and thus models with smaller BIC values are preferred over those with larger BIC values.

The Measurement Model

Confirmatory factor analysis (CFA) was conducted to test the fit of the measurement model. Each manifest variable was specified to load on its respective factor, and the latent variables were set to freely correlate. The CFA results revealed an adequate, though somewhat sub-optimal, model fit to the data (CFI = .90, RMSEA = .06, SRMR = .07). Analyses revealed strong factor loadings of manifest indicator variables on their respective latent constructs, with all factor loadings significant within a range of $p < .05-.001$. With one exception, BPRS items loaded on their respective latent factors as predicted. However, BPRS item Cognitive Disorganization did not load onto a positive psychotic symptom factor as was initially predicted, showing non-significant loadings at Baseline and T2. As can be seen in Tables 4-6, this BPRS variable displayed an erratic pattern of associations with the two other psychotic symptom manifest variables. Thus, this variable was dropped from the model to provide a more uniform psychotic symptom latent variable. The results for this slightly modified model resulted in excellent model fit (CFI = .96, RMSEA = .04, SRMR = .04).

The Structural Model

Next, a structural equation model was specified by regressing the Negative Affect latent variable (LV) and Positive Psychotic Symptoms LV at later
time frames onto earlier LVs (Time 2 onto Time 1, and Time 3 onto Time 2). Also, the loadings for respective BPRS manifest variables were constrained to be equal (e.g., Hallucinations at baseline, T1, & T2 all set to be estimated as same value). In this way, the latent variables are specified to be structurally equivalent across time and therefore it is reasonable to assume (with good model fit) measurement invariance of the respective latent variables over time (i.e., the same construct is being assessed at each time point). The SEM results suggested good fit to the data (CFI = .94, RMSEA = .05, SRMR = .05). In addition, the invariant model was compared with the same model but without the imposition of constrained factor loadings. A smaller BIC value for the invariant model (BIC = 12023) suggested that it was the preferred model, compared to the non-invariance model (BIC = 12041).

As predicted, analyses revealed strong, statistically significant cross-sectional (synchronous) associations between the Negative Affect LV and the Positive Psychotic Symptoms LV at each measured time frame, suggesting that Negative Affect symptoms and Positive Psychotic symptoms occurred concurrently within the psychotic disorders sample (see Figure 1). In addition, the moderate (residual) correlations between the Negative Affect and Positive Psychotic Symptom factors (Time 2 & Time 3 correlations respectively) remained stable even after accounting for baseline and previous effects of these latent variables.
To ensure that the strong co-occurrence of negative affect and psychosis symptoms was not due primarily to presence of the schizoaffective diagnostic group within the total sample, the model was run again while excluding those individuals with schizoaffective disorder. There was no change in the modeling results when the schizoaffective diagnostic group was removed from the analysis. Therefore, the strong relationship between affective and psychotic symptoms is not simply due to those individuals having a diagnosis of schizoaffective disorder within this sample (i.e., those individuals who meet criteria for both a major mood and psychotic disorder).

Contrary to expectation, there was no significant cross-lagged effect between negative affect and positive psychotic symptoms. There was a cross-lagged trend between Negative Affect at Time 2 and Psychotic Symptoms at Time 3; however, this relationship did not meet conventional levels of statistical significance.
Figure 1. Structural equation model.
DISCUSSION

There is a strong research base within the literature noting the co-occurrence of depressive and anxiety symptoms among individuals with psychotic disorders. Results of the present study provide further support for this phenomenon and suggest that the relations amongst negative affective and psychotic symptom domains remain stable over time. Negative affectivity can stem from biological, psychological, or environmental sources including stress hormones, personality traits, or psychosocial stressors (Walker & Diforio, 1997). Regardless of the source, negative affectivity may play a role in the etiology and/or expression of psychosis and may affect illness onset, relapse, or response to treatment (Myin-Germeys & van Os, 2007; Norman & Malla, 1994).

Most of the current literature examining the relationship between negative affect and psychosis has been cross-sectional, and few longitudinal studies have been conducted. The present study sought to examine the relationship between negative affect (as defined by BPRS items of Depressed Mood, Anxiety, and Guilt) and positive symptoms of psychosis (as defined by BPRS items of Hallucinations, Unusual Thought Content, and Cognitive Disorganization) utilizing a longitudinal approach. Structural equation modeling was used to examine the relationship between the latent variables of negative affect and positive symptoms in psychosis. Specifically, it was predicted that negative affectivity and positive psychotic symptoms would exhibit a strong cross-sectional relationship,
and this relationship would remain stable over time. Furthermore, it was speculated that a cross-lagged and directional relationship would demonstrate that negative affectivity leads to an increase in positive symptoms of psychosis.

With regard to the measurement model, the results provided support for specific hypotheses on how manifest variables would load onto each latent factor. The only exception to this was the BPRS item of Cognitive Disorganization. Given an erratic factor-loading pattern of the Cognitive Disorganization item, it was dropped from the final model for the purpose of providing a more homogeneous and consistent positive symptom factor. There are two possibilities as to why this particular item did not demonstrate the expected factor loading. Had analyses included only individuals with a schizophrenia spectrum diagnosis (as opposed to psychotic disorders more generally); this particular indicator variable may have had a stronger loading on the positive symptom latent variable. While methodological issues (e.g., specific sample characteristics) could account for this finding, it is more likely that cognitive disorganization corresponds to an altogether separate symptom dimension when contrasted with a positive symptom dimension. As described earlier, factor analytic studies of schizophrenia spectrum disorders have yielded results suggestive of several underlying dimensions, including positive symptoms, negative symptoms, disorganization/thought disorder, premorbid social adjustment, and affective dimensions (Lenzenweger & Dworkin, 1996).

More specifically, it is plausible that cognitive disorganization is more
representative of a thought disorder dimension rather than a positive symptom
dimension (e.g., Mueser, Curran, & McHugo, 1997). This argument is further
buttressed by evidence suggesting that cognitive disorganization and associated
thought disorder symptoms are more often associated with a negative symptom
dimension rather than a positive one (Green, 1998).

Consistent with expectations, results of this study demonstrated both
synchronous and stability effects for the negative affect and positive psychotic
symptom factors. A moderately strong co-occurring relationship between the
negative affect and positive symptom of psychosis factors was identified and this
relationship remained stable across the span of approximately six months (with
measurements taken at baseline, 10 weeks, and 20 weeks). Additionally, this
relationship remained even after accounting for baseline levels of the respective
factors, allowing for additional confidence in the interpretation of the strong,
synchronous co-occurrence of these specific latent variables. Moreover, the
current SEM results held even after excluding patients with schizoaffective
disorder from the total sample. As such the results from the present study
provide further evidence that negative affect is a commonly observed aspect of
the clinical picture in psychotic disorders (broadly defined) and this relationship
remained robust and temporally stable across monthly intervals.

Contrary to hypothesis #5, a cross-lagged, longitudinal relationship
between the negative affect and positive symptom factors was not identified. The
results provided little evidence of a statistically significant predictive relationship
between negative affect and exacerbation of positive symptoms. Therefore, based on the present results, there is no evidence to suggest that negative affect plays a direct (temporal) causal role in the development or exacerbation of positive symptoms in a chronic mentally ill population. It is important to note that results do not determine whether negative affect played a causal role in the development of positive symptoms in the earlier stages of psychosis; however, negative affect does not appear to play such a role once the disorder has become chronic.

The aforementioned findings hold important treatment implications for working with individuals with psychotic disorders, particularly with regard to psychosocial interventions. Most often, mental health providers employ antipsychotic medications as a first line treatment to target overt psychotic symptoms such as hallucinations and delusions, placing less importance on psychosocial treatments to address co-occurring depressive and anxiety symptoms. However, many psychotic individuals only experience limited relief from positive symptoms, with a subset of individuals experiencing continued residual positive psychotic symptoms and continued negative affective symptoms such as depression and anxiety (Gould, Mueser, Bolton, Mays, & Goff, 2001; Lehman, Steinwachs et al., 1998; Siris, 2000). While positive symptoms are certainly distressing and need to be managed, there is little evidence to suggest that targeting positive symptoms in isolation leads to any significant improvement of daily functioning, such as completing Activities of Daily Living, enhancing
quality of life more generally, or influencing the ability of an individual with serious mental illness to participate in supported employment programs and achieve vocational goals (Bond et al, 2001; Velligan, Mahurin, Diamond, Hazleton, Eckert, Miller, 1997). Appropriate intervention with negative affective symptoms and neurocognitive limitations may represent more useful treatment targets to improve psychosocial functioning, rather than just targeting positive symptoms alone. For example, given the strong relationship between negative affect and positive symptoms of psychosis, adequate treatment of depression and anxiety might improve overall quality of life, vocational and social skills, and activities of daily living. Conversely, rehospitalization rates and suicide risk may diminish if negative affective symptoms are appropriately assessed and managed.

With regard to treating specific affect-related symptoms of psychosis, the decision between pharmacologic interventions and implementation of psychosocial treatments is less clear and more controversial; nevertheless, medications are commonly relied upon as a first line of treatment in hopes of reducing emotional reactivity (Bebbington & Kuipers, 2003; Spaulding, Sullivan, & Poland, 2003). When evaluating treatment interventions for affect-related symptoms in psychosis, it is important to evaluate potential sources of the symptom when possible. Given the heterogeneity of clinical presentation within psychotic disorders, it is likely that the illness is determined by a complex interplay of polygenetic biological, neurodevelopmental and environmental risk factors. Additionally, depressive and anxiety symptoms observed in individuals
with psychosis likely stem from different sources. For example, depression and anxiety in psychotic disorders may play an etiological role, may result directly from having the illness itself (e.g., post-psychotic depression, anxiety associated with paranoia and delusions), or may result from environmental contexts such as family discord, high expressed emotion (High EE), or homelessness.

In addition to the risk factors that may precipitate the onset of mental illness, society often marginalizes and discriminates against individuals exhibiting psychotic behavior and consequently, these individuals are left to cope with situational stressors that healthier individuals do not have to address. For example, if an individual with a psychotic disorder is experiencing depression, anxiety and homelessness, or returning to a highly critical home environment after hospitalization, it is important to assess the impact of these contextual factors on symptoms and subsequently intervene when possible. In the previous example, it would be important for treatment providers to address the patient’s lack of housing by identifying appropriate homes or transitional facilities or to implement family interventions that that enable the family to decrease problematic displays of anger and guilt and learn skills to facilitate more effective communication. In this way, one can eliminate environmental contributors to depression and anxiety while also offering other interventions to treat depression and anxiety such as pharmacology, cognitive-behavioral therapy, or psychoeducation (Spaulding, Sullivan, & Poland, 2003).
Depression and anxiety symptoms in psychotic disorders and their effective treatment holds promise for enhancing patients’ quality of life and may directly impact one’s ability to cope with symptoms. For example, Smith et al (2006) found that those psychotic patients with more severe depressed mood and poor self-esteem were more likely to experience their auditory hallucinations and persecutory delusions as having higher negative content, described their symptoms as more severe and distressing, and felt their symptoms were harder to control. If depression and anxiety can be treated within the course of treatment for psychosis, patients may experience an improvement in social and cognitive functioning and improved quality of life, despite continued presence of psychotic symptoms.

Future studies should examine negative affect within the context of earlier phases of psychotic disorders. Additionally, completing models should be examined and, to the extent possible, ruled out. Of particular importance is the emphasis of examining negative affect within phenotypic at-risk individuals as well as those with a genetic liability towards a psychotic disorder; there may be some variation in affective and cognitive symptoms between those with a family history and those without (Asarnow, Cromwell, & Rennick, 1978; Docherty & Grillon, 1995; Docherty, Grosh, & Wexler, 1996; Norman & Malla, 2000).

Limitations and Suggestions for Future Research

Several methodological limitations of the present study should be considered and offer a useful framework for guiding future research. For
example, one limitation involves the severity and chronicity of symptoms and functional impairment of the sample population. The severity of psychopathology of the clinical sample may have precluded the ability to identify a potential relationship between target variables. Individuals within the present sample represent severely impaired, chronic cases of serious mental illness, many of whom had been involved in treatment of some kind (e.g., pharmacotherapy) for many years. Length and type of medications or additional treatment effects could have mitigated or otherwise impacted symptoms in such a way that a relationship could not readily be identified. Furthermore, the present study did not control for intellectual capacity, cognitive performance, or substance abuse, all of which could potentially affect affective and psychotic symptoms. Given the relatively limited sample size, particularly across time, the possibility of estimating additional model parameters was not realistic.

Additionally, individuals were included in the sample if they had a general diagnosis of a psychotic-spectrum disorder. Individuals with a diagnosis of Major Depression with Psychotic Features or Bipolar Disorder were excluded from the sample due to the predominant mood symptoms. While analyses included individuals with a diagnosis of schizoaffective disorder, the model was analyzed both with and without schizoaffective clients in order to control for mood symptoms in this diagnostic subgroup. The relationship amongst symptoms occurred even when the schizoaffective disorder subgroup was excluded, allowing additional confidence in the present findings. Furthermore, given the
heterogeneity of psychotic disorder diagnosis in the current sample, the impairment and severity of symptoms in the present population may be reflective of a generalized dimension (e.g., neuroticism) of psychopathology or vulnerability. A generalized, underlying dimension of severe psychopathology may have clouded the relationship between negative affect and positive symptoms of psychosis, or changed the relationship amongst symptoms in some manner. However, the two symptom factors in the present study only showed moderately sized correlations, which casts some doubt on the possibility of such an underlying generalized factor accounting for the associations between the negative affective and psychotic symptom factors.

On a related note, the present study utilized a dimensional framework for categorizing psychotic disorders and did not differentiate between specific types of psychoses. When examining symptom correlates, extant literature suggests a dimensional approach may be beneficial and even preferable to traditional categorical models (Krueger & MacDonald, 2005; Peralta, Cuesta, Giraldo, Cardenas, & Gonzales, 2002). However, it remains possible that negative affect only predicts positive symptoms of psychosis in those individual with a specific psychotic disorder, such as schizophrenia. Examining negative affect within specific psychotic disorders may uncover a potential relationship that is overlooked when looking at general clusters of disorders. Conversely, given the heterogeneity of psychotic symptoms and strong diagnostic comorbidity with other psychiatric problems, identification of general, more fundamental
dimensions of psychopathology (e.g., negative affect, positive symptoms) may provide important etiological information that can be more effectively used in treatment development and implementation. Knowledge of the etiology of specific symptoms could potentially aid clinicians and researchers to develop/implement interventions for earlier periods of treatment, therefore arresting the development of full-blown psychotic disorders.

The current study could have failed to find direct or indirect effects due to the specific assessment time frame utilized in this study (10 and 20 weeks follow-ups). In future studies, researchers could potentially vary the time frames for assessment with more or less frequent observations of symptom ratings. As described earlier, numerous studies have established a link between depressive symptoms and anxiety prior to developing psychosis in at-risk individuals. Future studies involving medication-naïve, prodromal individuals, or individuals whose psychotic episodes are earlier within the psychotic trajectory may offer additional insight into the relationship between negative affect and positive symptoms of psychosis. Researchers should continue to incorporate prospective longitudinal designs with both clinical and at-risk populations.

While both self-report and clinician-rated measures of symptoms provide useful information, the present study relied on one clinician-rated measure to assess the presence and change among symptoms across time. The BPRS is a well-established and frequently used measure of symptom change within psychiatric populations (Overall & Gorham, 1962; Lukoff, Nuechterlein, &
Ventura, 1986). It is a clinician-administered and clinician-rated (as opposed to self-report) which provides particular benefit when working with individuals who may not be able to accurately report their symptoms due to complications associated with a lack of insight. However, the value of self-report measures should not be underemphasized and studies suggest that self-report measures are valuable tools for information gathering from psychotic patients (Bell, Fiszdon, Richardson, Lysaker, & Bryson, 2006). In future studies, researchers may find it useful to incorporate multiple self-report, narrow-band measures of depression, anxiety, and positive symptom clusters into their research designs. For instance, measures specifically designed to identify depression or anxiety symptoms within schizophrenia (such as the Calgary Depression Scale for Schizophrenia) or psychotic disorders may lend themselves to identifying a causal role of (self-reported) negative affect in positive symptoms. Additionally, researchers may find it useful to incorporate both self-report and clinician-rated measures to gather more comprehensive information about symptoms, their stability over time, and any potential method effects in the assessment of symptoms.

The present study did not examine competing models or mediating/moderating variables. For example, one should consider the possibility that experiencing psychotic phenomena, such as hallucinations and delusions, may play a causal role in the development of depression and anxiety in individuals with psychotic disorders. It is plausible that the experience of positive
psychotic symptoms is so distressing that it may lead one to become depressed and anxious, as in the case of post-psychotic depression. Furthermore, additional variables related to social cognition, such as level of insight or attributional style may play a role in the development or maintenance of negative affect within psychotic-spectrum disorders. In contrast to earlier, more dynamic views, anosognosia, or lack of insight, is now believed to be an inherent neurocognitive deficit of schizophrenia and is seen in a subset of schizophrenia patients. (Amador & Paul-Odouard, 2000). Individuals with schizophrenia who have more insight into their illness often experience more depression due to their awareness that they are mentally ill and the repercussions of having such a debilitating illness (Iqbal, Birchwood, Chadwick, & Trower, 2000). It has been suggested that psychotic individuals with high levels of insight have a better prognosis, although they may be at risk for increased feelings of hopelessness and suicidal ideation (Birchwood, Iqbal, Chadwick, & Trower, 2000). Therefore, level of insight may be an important mediating or moderating variable in the relationship between negative affect and psychotic symptoms.

With regard to potential mediating and moderating variables, there has been a recent interest in attributional style and how it relates to psychotic phenomena, particularly the development and maintenance of delusions. Emerging cognitive conceptualizations of psychosis are useful for understanding the relationship between depression, anxiety, and positive psychotic symptoms (Chadwick & Birchwood, 1994; Garety, Kuipers, Fowler, Freeman & Bebbington,
2001 Morrison, 2001). In general, the cognitive model as applied to psychosis, assumes that it is the interpretation of psychotic experiences, rather than the experiences themselves that is problematic (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001). While studies are few in number and have offered mixed results, there has been a recent interest in exploring the usefulness of cognitive approaches to treat psychosis. Meta-analyses of studies using cognitive therapy to treat residual psychotic symptoms has been shown to be moderately effective in the treatment of positive symptoms such as hallucinations and delusional thought, although moderating variables and dismantling studies are lacking (Gould, Mueser, Bolton, Mays, & Goff, 2001; Morrison 2008; Steel 2007; Zimmerman, Favrod, Trieu, Pomini, 2005). Identification of a relationship between negative attributional style and negative affect in schizophrenia suggests that cognitive therapy of schizophrenia may hold promise as an intervention for depression in schizophrenia as well. Conversely, the fact that cognitive behavioral approaches are showing moderate treatment effectiveness suggests that the exploration of cognitive processes underlying psychosis is a useful endeavor for future research.

One final, yet important limitation of the present study involves lack of assessment of neurocognitive deficits and their relationship to symptom clusters involving positive psychosis and negative affect. Individuals with psychotic disorders, and schizophrenia-spectrum disorders in particular, are known to exhibit significant neurocognitive deficits involving aspects of attention, working
memory, executive functioning, perception and language. In addition, many individuals who are psychosis-prone or at-risk for development of psychosis show similar, yet attenuated, neurocognitive deficits similar to those seen in schizophrenia (e.g., Chapman, Chapman, Kwapił, Eckblad, & Zinser, 1994; Gooding, Kwapił, & Tallent, 1999) Many of these neurocognitive domains, particularly language and selective attention, have been shown to worsen due to negative affect (Asarnow, Cromwell, & Rennick, 1978; Docherty, Evans, Sledge, Seibyl, & Krystal, Freeman & Garety, 2003; Mohanty et al 2008). Future research should examine the interplay between negative affect and positive symptoms of psychosis while taking into account performance on neurocognitive measures and the presence and severity of negative symptoms.

In conclusion, research on the exact role that negative affect may play in the development and maintenance of psychotic symptoms remains an open area of investigation, particularly those symptoms that lie along a positive symptom dimension. While the role of negative affect in the etiology and course of psychosis remains unclear, the impact of these symptoms on social and interpersonal relationships, suicide risk, hospitalization rates, and overall quality of life and psychosocial functioning is of paramount importance. Fortunately, a more comprehensive and integrative model of psychosis is beginning to take its place in mainstream psychology and psychiatry. No longer solely focused on reduction of overt positive symptoms, researchers and treatment providers are beginning to question the role of depression and anxiety in the etiology and
maintenance of psychotic symptoms. Additionally, the field is beginning to recognize the potential impact of specific symptoms on psychosocial functioning for individuals with schizophrenia-spectrum and associated psychotic disorders. The advent of newer, evidence-based psychosocial interventions and the burgeoning study and use of psychological approaches in treating individuals with psychosis and their families has shown promise. Despite these advances, further research is needed to fully understand the relationship between negative affect and psychosis, both etiologically and its functional impact.
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