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Aspects of cognitive vulnerability as predictive of general and specific themes of delusional ideation in individuals at risk for psychosis

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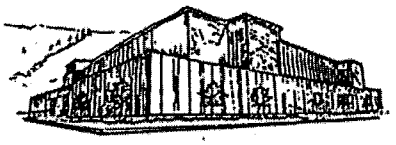

Aspects of Cognitive Vulnerability as Predictive of General and Specific Themes of Delusional Ideation in Individuals at Risk for Psychosis

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Given the debilitating nature of schizophrenia, as well as recent research supporting the efficacy of cognitive therapy to treat schizophrenia and, more specifically, delusional ideation, it is important to hone in on aspects of cognitive vulnerability that may be related to the development of this disorder. Despite advances in understanding cognitive vulnerability processes in schizophrenia, studies investigating these processes in individuals at risk for the disorder have been very limited. The purpose of this study was to examine specific types of cognitive vulnerability (attributional style and negative self-schema) in individuals at risk for psychosis in order to identify how overall dysfunctional thinking, as well as its particular facets, predict specific types of delusions in individuals at risk for psychosis. It was hypothesized that the existence of negative self-schema and type of attributional style, in combination with different risk factors, would predict delusional ideation.

Results suggested that potentially At-Risk individuals exceeded Control Group members on measures of dysfunctional attitudes and delusional ideation, but that there were no significant differences in attributional styles between the two groups. It appears that the combination of risk factors (specifically genetic risk and general psychological symptoms), negative self-schema, and a "blaming others" for negative events attributional style best predicts overall delusions, as well as specifically persecutory and grandiose ideation. These findings are discussed in terms of gaining important knowledge about risk factors for schizophrenia, cognitive predictors of delusions, and prevention of full-blown psychosis.

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Introduction

Overview of Schizophrenia and its Etiology

Schizophrenia, affecting approximately one percent of the population, has been described as one of the most debilitating psychological disorders. Hallmark symptoms include "positive symptoms" such as delusions, hallucinations, disorganized speech and/or behavior, and "negative symptoms," including flattened affect, anhedonia, and avolition (APA, 2000). As the disorder is difficult to treat effectively, individuals with schizophrenia are often heavily reliant on anti-psychotic medication. In addition, this population has a very high suicide rate and frequently suffers from comorbid disorders, such as substance abuse/dependence, depression, and anxiety (APA, 2000).

Due to a variety of factors, patients often alternate between acute psychotic phases and phases of improvement or recovery (Herz, 1990), and relapse to psychosis has been found to be quite characteristic of this disorder. Some research supports the finding that there is an equal distribution among patients who recover completely from the disorder or have a very long remission (33%), those who generally recover from the positive symptoms of the disorder but who are left with residual symptoms in the form of flat affect, social withdrawal, and other negative symptoms (33%), and those who relapse back into psychosis (33%); the so-called "1/3 – 1/3 – 1/3" rule (e.g. Doering, et. al., 1998). Other research has found a similar proportion of individuals with schizophrenia who recover completely (25%), but with a small percentage (10%) who suffer a severely chronic course and remain permanently hospitalized (e.g., Herz, 1990), also known as

"high redundancy" schizophrenia (Cromwell, 1972). The remainder of patients (between 50 and 75%) alternate between acute psychotic phases and phases of improvement or recovery (Herz, 1990), termed "low redundancy" schizophrenia by Cromwell (1972). The high and low redundancy classifications, in addition to pertaining to acuteness, chronicity, and prognosis, roughly correspond to positive and negative symptom subtypes of schizophrenia. Others have found that even with adherence to an antipsychotic medication regimen, 20 – 48% of patients relapse (e.g. Heinrichs, Cohen, & Carpenter, 1985).

As a result of the often acute and incapacitating nature of schizophrenia, a great deal of research has been conducted on predictors of onset and relapse in this population. Studies have shown that certain demographic characteristics, such as gender (being male), age (under 40), and marital status (being single), play a significant role in relapse (Doering, et. al., 1998) as does alcohol and drug use (Cuffel & Chase, 1994).

Despite the psychosocial factors that play a significant role in onset and relapse, much attention has been given to the biological etiology of schizophrenia, as it appears to have a large genetic component (Gottesman, 1991; Kendler & Diehl, 1993); however, it is widely acknowledged that there are likely several genes dynamically involved to produce the disorder (Green, 2001; Levinson, et al., 1998). Twin and adoption studies have found an overall concordance rate for schizophrenia in the range of 27-77% in monozygotic twins (MZ) and approximately 9% in dizygotic twins (DZ; Gottesman & Shields, 1972). It is notable that these types of studies have been sometimes criticized for methodological complications that preclude a clear determination of degree of genetic

versus environmental influence. For instance, many researchers who have studied monozygotic twins reared apart posit that these individuals do not share any environmental commonalities whatsoever. However, other researchers suggest that Pregnancy Birth Complications (PBCs), regarded as environmental (yet biological) risk factors for the development of schizophrenia play an important role prenatally and should not be ignored (Davis & Phelps, 1995).

While the examination of special environmental factors such as PBCs may complicate conclusions drawn from traditional genetics studies, many still consider the MZ-DZ twin model when assessing genetic influence and degree of risk for the development of this disorder. It has been suggested that as degree of genetic closeness grows to a relative with schizophrenia, so does the proband's risk for schizophrenia. That is, first-degree relatives (siblings, parents, and offspring) of individuals of schizophrenia have a much higher chance to develop the disorder themselves (9%, 6%, and 13%, respectively) than do second or third degree relatives (Gottesman, 1991). Thus, continued research on PBCs and genetic aspects of twins concordant and discordant for schizophrenia, as well as other relatives, can provide valuable information to understand this complex disorder.

While the biological heritability of schizophrenia cannot be denied, the disorder is most often conceptualized from a diathesis-stress perspective, which highlights the interaction of a genetic or other biological disposition for a disorder that creates a vulnerability, and events from the environment, which may "trigger" the predisposition and lead to the onset of the disorder (Mirsky & Duncan, 1986; Monroe & Simons, 1991;

Nuechterlein,1987). For an individual with schizophrenia, stressors from the environment include not only negative life events, but events that are considered pleasant as well, such as a social gathering, or some type of achievement or accomplishment (Herz, 1990). Zubin and Spring's (1977) stress-vulnerability model states that an individual can repel this stress if the stressful event falls below his/her tolerance threshold, which is defined by the level of genetic vulnerability to schizophrenia that the individual possesses; however, if the stressful situation is above the tolerance threshold, then a psychotic episode may develop. Nicholson and Neufeld (1992) have extended this model and developed a "dynamic vulnerability perspective" which hypothesizes that an individual's ability to cope with stress is influenced by genetic vulnerability, levels of stressors, as well as by his/her symptoms. They suggest that not only can stressors create an increase in symptoms, but that the existence of symptoms can also increase stress in an individual.

In addition to general environmental events and stressors, certain family factors have been found to be predictive of onset of schizophrenia as well as relapse to a schizophrenic episode (Herz, 1990; Nicholson, 1998; Wynne & Singer, 1963a, 1963b). It has been suggested that parental Communication Deviance (e.g. lack of commitment to ideas and percepts, language anomalies, disruptive speech, unclear or idiosyncratic communication of themes or ideas, and closure problems) may directly affect the offspring's cognitive development and reality testing and potentially lead to subsequent thought disorder (Lukoff, Snyder, Ventura, & Neuchterlein, 1984).

One of the most significant interpersonal predictors of relapse to a psychotic

episode is a relative's level of "Expressed Emotion," generally defined in terms of an influential family member's behavior and feeling expressions toward the patient with schizophrenia. Family members who are considered to exhibit "high EE" tend to be critical, hostile, and emotionally overinvolved toward the patients and tend not to offer positive remarks or warmth (Brown, Birley, & Wing, 1972; Hooley, 1985; Vaughn & Leff, 1976). Studies have shown that patients who have frequent contact with high EE family members are two times more likely to relapse within a year than those who have more limited contact with high EE family members, or return to a low EE environment post-hospitalization (e.g. Brown, Birley, & Wing, 1972; Leff & Vaughn, 1981;Vaughn & Leff, 1976). Relatedly, the construct of "Affective Style" (AS), described as the manner in which parents and patients with schizophrenia (as well as with other disorders) interact during an emotionally charged discussion, has also been found to be a significant predictor of relapse (Doane, Falloon, Goldstein, & Mintz, 1985; Doane, Goldstein, Miklowitz, & Falloon, 1986). The AS system categorizes verbal behavior during the parent-child exchange into classes such as guilt induction, intrusiveness, and benign and personal criticism and support. Research suggesting that EE and AS behaviors only partially overlap reinforces the notion that family interaction patterns and their role in the etiology and exacerbation of schizophrenia (and other disorders) is very complex (Doane & Becker, 1993).

Research focusing on both the biological and interpersonal correlates of risk to psychosis has proven fruitful in increasing knowledge about this disorder. Recently, researchers have given more attention to cognitive factors, such as metacognitive

processes and levels of sociotropy and autonomy (Morrison, et al., 2002), that put someone at risk for the development of schizophrenia and relapse to psychosis. These factors, described later, are the focus of the present study. Given the debilitating effects of schizophrenia, investigation into new areas of risk prediction is important.

Rationale and Strategies for Assessing Risk for Schizophrenia

The literature suggests that psychotic symptoms, in particular delusions, can be conceptualized as severe expressions of beliefs and traits that exist at lower intensities in "normal," or "sub-clinical" populations (Claridge, 1972, 1987; Edell, 1995). Lower-level delusional ideation may therefore manifest itself in individuals who do not have a diagnosable psychiatric condition, but who may show signs of psychopathology or psychotic thought processes (Peters, Joseph, & Garety, 1999). Given the existence of this low-level symptomatology and the research that suggests that such ideation may develop into more severe psychosis in the future (Chapman, Chapman, Kwapil, Eckblad, & Zinser, 1994; Peters et al., 1999) it is important to examine the beliefs and perceptual experiences of such people, in order to gain a greater understanding of the thought processes of more disturbed individuals, as well as the longitudinal processes that can lead to breakdown. This allows gains in knowledge about the development of psychosis, as well as the environmental risk and protective factors related to such disturbances (Chapman & Chapman, 1985). As research suggests that a longer Duration of Untreated Psychosis (DUP) results in a poorer prognosis (Loebel, Lieberman, Alvir, Mayerhoff, Geisler, & Szymanski, 1992), an examination of individuals with subclinical psychopathological symptoms that can aid in prediction may therefore help with

prevention of full-blown psychosis, or with decreasing its duration and hence lessening the debilitating financial, emotional, and interpersonal consequences of the disorder.

There have been several approaches to the assessment of risk for schizophrenia. Initially, researchers focused on the genetic component and conducted longitudinal studies examining first-degree relatives (e.g. offspring) of individuals with schizophrenia, as well as other family members, in order to monitor their potential development of the disorder via continuous follow-up assessments over a period of several years (Asarnow, 1988). While this research technique has some benefits, it is quite time-consuming and expensive. More importantly, in these targeted individuals, the transition rate to psychosis is generally not large (Asarnow, 1988), as the genetic risk for first-degree relatives of individuals with schizophrenia only ranges from approximately 6 to 13% (Gottesman, 1991), leaving a false positive rate of approximately 85% and thus precluding the attainment of high-yield data about risk.

Another strategy frequently utilized to gain information about correlates of risk is associated with the study of the "prodrome," or the changes in behavior that occur just before the transition to a full psychotic break (E. Bleuler, 1911/1950; Heinrichs, et al., 1985; Herz, 1990). These indicators are often conceptualized as low-level or subthreshold symptoms and have led to a line of research which suggests that some individuals, who may or may not have a genetic predisposition for psychosis, likely experience these symptoms ("schizotypal" symptoms or "schizotypy") well before the development of a psychotic disorder (Chapman & Chapman, 1985; Strauss, 1969). "Hypothetical psychosis-proneness," earlier termed "schizotaxia" by Meehl (1962, 1990, 1993), is

regarded as a vulnerability characteristic for the long-term development of psychosis, and can be measured via endorsement (from a series of paper and pencil measures) of deviantly high levels of psychotic-like experiences such as the identification of thought transmission, passivity experiences, voice experiences and other auditory hallucinations, as well as aberrant beliefs (Chapman & Chapman, 1985)

In the 25-month follow-up study of former college student participants who had previously been identified as hypothetically psychosis prone, Chapman and Chapman 1985) found that only three subjects (out of approximately 200 at-risk) had made the transition to diagnosable psychosis. At the 10-year follow-up mark, Chapman and colleagues (1994) found that participants significantly exceeded control subjects on diagnoses of DSM-III-R psychoses, as well as on psychotic-like experiences, schizotypal symptoms, and reports of having psychotic relatives. While the construct (and measurement) of psychosis proneness does have some long-term predictive validity, the Chapman group found that, in fact, only a small percentage of putatively at-risk individuals break down after 10 years (approximately 14 out of 182 participants), estimated elsewhere (Meehl, 1990; 1993) as approximately 10% of "schizotypes" proceeding to develop clinical schizophrenia. Due to this limited predictive power, the Chapman group developed and validated additional measures to add to their high-risk assessment battery, including more "trait-like" measures of schizotypy.

This line of research reinforces the idea that not everyone who presents with certain types of vulnerability (e.g. genetic predisposition or characteristics of prodromal schizophrenia) will go on to develop a psychotic disorder. It is suggested that they may

not develop any diagnosable psychological problem at all, or may develop another disorder instead, such as bipolar disorder or major depression. In addition, it has been suggested that the construct of the "prodrome" can only truly be evaluated and understood retrospectively (Yung, McGorry, & McFarlane, 1996). Thus, researchers have adopted the term "at-risk mental state" to describe individuals who are at risk for psychosis, but who may not definitely develop a full-blown disorder (Yung, et. al., 1996; Yung et al., 1998).

Using strategies based on those of Chapman and colleagues and others to assess state-like psychotic characteristics, other researchers (e.g., Yung et al., 1998; Morrison, et al., 2002) have identified individuals who are at risk for schizophrenia by examining specific transient and attenuated psychotic symptoms. Transient psychotic symptoms, also known as Brief Limited Intermittent Psychotic Symptoms (BLIPS), are defined by these researchers as including at least one of the following, operationalized by cut-off scores on the Brief Psychiatric Rating Scale (BPRS) within the hallucinations, delusions, and/or Formal Thought Disorder subscales of the measure. All symptoms must also last for less than one week and resolve spontaneously. The concept of attenuated symptoms relies on the notion of subclinical psychopathology mentioned above and these indicators are defined by the presence of at least one of the following symptoms of DSM-IV schizotypal personality disorder: Ideas of reference, perceptual disturbance, odd beliefs or magical thinking, odd behavior or appearance, or paranoid ideation. These symptoms require slightly lower cut-off scores on the BPRS, have existed for longer than one week,

and occur several times each week. When combined with additional state and trait factors (such as decreased functioning and genetic predisposition for schizophrenia, respectively), this attention to the transient and attenuated psychotic symptoms may increase the chances of targeting individuals who are at risk to develop a psychotic disorder.

More specifically, this "trait plus state" strategy employs combining "trait" factors, such as a family history of schizophrenia in a first degree relative or a diagnosis of schizotypal personality disorder in the index subject, with a variety of mental state characteristics indicative of risk, including elevated levels of general psychopathology, specific psychotic-like symptoms, or a decline to some degree in overall social or occupational functioning. In a study targeting an at-risk group and examining their rate of transition to psychosis through monthly follow-up assessments, Yung and colleagues (1998) developed and utilized both a state-only and the state-trait risk criteria and collected a very heterogeneous sample of individuals, some of whom were at imminent risk for the development of a psychotic disorder. Participants who had: a) a first degree relative with a history of any psychotic disorder or b) a diagnosis of schizotypal personality disorder and c) "any change in mental state or functioning which results in a loss of 30 points or more on the Global Assessment of Functioning (GAF) scale for at least one month" (p. 16) and were d) between the ages of 16 and 30 (high risk age range) were included in the Yung et al. study.

Morrison and colleagues (2002), in an investigation of the efficacy of cognitive therapy as prevention of the transition to psychosis in high-risk subjects, utilized similar

criteria for inclusion in the study. In his study, participants who 1) possessed transient psychotic symptoms (BLIPS) or attenuated symptoms (measured using Yung and colleagues' criteria) and 2) had a family history of a first-degree relative with any psychotic disorder or a diagnosis themselves of Schizotypal Personality disorder and 3) had an at-risk mental state as defined by a drop in GAF of 30 points or more and/or an elevated score on a measure of general psychopathology (the General Health Questionnaire, GHQ, Goldberg & Hillier, 1979) and 4) were between the ages of 16 and 36 were admitted to the study and randomly assigned to one of two intervention conditions.

Yung and colleagues found that approximately 40% of their participants made the transition to psychosis after six months. In the Morrison and colleagues study, approximately 22% of participants became psychotic within six months. These findings suggest that utilizing a structured, comprehensive strategy for targeting at-risk individuals is beneficial and reduces the false positive rate that has previously occurred in these types of studies that rely on fewer indicators. Indeed, there is a good deal of predictive power in the "trait plus state" approach to identifying at-risk individuals; this is promising as researchers and clinicians work to determine other vulnerability factors (such as dysfunctional cognitions) and to develop effective methods of prevention.

Overview of the Cognitive Model of Psychopathology

While only recently being applied systematically to the study of schizophrenia, cognitive models have been widely accepted as valid conceptualizations of several

psychological disorders, particularly the anxiety disorders and depression (Beck, 1976; Beck, Rush, Shaw, & Emery, 1979; J. Beck, 1995). In general, cognitive theories of psychopathology highlight the existence and consequences of maladaptive cognitive styles. These styles may be further defined as manners of interpreting and understanding events or interactions that lead to a distortion or misinterpretation of reality (Freeman & Reinecke, 1995). Such thinking patterns in turn may precipitate or exacerbate psychological symptoms (Beck, 1976; Beck, et al., 1979). Dysfunctional thinking styles, as they relate to the development of depression in particular, have been conceptualized as latent diatheses (separate from the genetic and biological diatheses previously discussed), which in the presence of environmental stressors make an individual more vulnerable to developing the disorder (Abramson, Alloy, & Metalsky, 1988; Ingram, Miranda, & Segal, 1998). The specific mechanisms through which this cognitive dysfunction develops and sustains itself are varied and have been widely researched and examined in psychotherapeutic interventions over the years. As much of cognitive theory was initially developed to conceptualize clinical depression, the discussion of these mechanisms will focus on that disorder.

The Cognitive Triad and Schemata

The Cognitive Triad, first proposed by Aaron Beck several decades ago (Beck, 1976), is used in the model as a way to describe more specifically the negative thoughts or maladaptive cognitions that a person with depression likely possesses. It has been suggested that individuals with depression hold these dysfunctional thoughts in relation

to one (or all) of the three important areas of the triad – the self, the world, and the future, and that the problems of virtually all patients can be linked to maladaptive beliefs in one of the three portions of this "Triad," (Freeman, & Reinecke, 1995).

Schemata, originally conceptualized in the fields of cognition and cognitive-developmental psychology, are generally defined as abstract mental structures that aid in organizing information in one's environment. According to Beck and colleagues, schemata may be described as "cognitive structures of the mind," (J. Beck, 1995, p. 166): Stable, underlying beliefs and assumptions concerning the elements in the Triad. Schemata provide meaning and understanding for one's past and current experiences as cognitive (as well as emotional) representations of events. These representations of the self, the world, and the future are maintained and solidified through the process of "assimilation" (Piaget, 1928). It has been suggested that individuals are more prone to assimilate their experiences to their existing schemata, rather than "accommodate" the belief to fit new events which are discrepant with the schema (Kovacs & Beck, 1978). Developing in infancy as the child interacts with others and with his/her environment, these beliefs are consolidated throughout childhood, and they often represent "internalizations of ongoing or repetitious parental behavior" (Freeman, & Reinecke, 1995, p. 190). Later in life, the set of beliefs becomes repeatedly activated by events that appear similar to those formative early experiences.

Generally speaking, people maintain basically positive schemas about themselves, the world, and their capabilities (e.g., "I am worthwhile," "I can function on a daily basis," "People are generally good," "The world is basically a safe place," etc.).

Although these self-schemas may be unrealistically positive ("positive illusions"), it has been suggested that overly, even inaccurately, positive beliefs about the self are often more adaptive than accurate beliefs (Taylor & Brown, 1988). While it is not theorized to be the case that a single traumatic or negative life event produces a maladaptive schema (Freeman, & Reinecke, 1995), repeated damaging interactions with others (e.g., family members) are stored in memory and may aid in the encoding of beliefs about people (i.e., "Others will let you down") and oneself (i.e., "I am difficult to love"), which become templates for how a person evaluates events in his/her adult life.

It has been suggested that such negative schemas are present in individuals who suffer from psychological disorders, that they underlie many symptoms, and play an etiological role. In the case of clinical depression, for example, negative self-referent schemas are present and often include themes of personal inadequacy, self-blame, and negative assumptions and expectations about oneself. Such maladaptive self-schemas may be broadly categorized as beliefs associated with helplessness ("I am powerless; I am defective") and with unlovability ("I am unlikable; I am bound to be alone") (Beck, cited in J. Beck, 1995). It is suggested that some individuals with depression may have negative self-schemas that fall into one category or the other, while others may have beliefs that are based on both helplessness and unlovability (J. Beck, 1995). Schemas are believed to serve as the source for the manifestation of the repetitious maladaptive thoughts, interpersonal interactions, and behaviors frequently seen in individuals with psychological disorders.

Automatic Thoughts and Cognitive Distortions

Automatic thoughts, streams of uncontrolled thinking that are often in our immediate awareness, are produced by stimuli (external events or thoughts about events), are fairly transitory, and are typically related to a specific set of emotions or behaviors (Beck, Epstein, & Harrison, 1983a). In general, people engage in reality testing (evaluating and correcting thoughts or perceptions after examination of information from the environment) as they respond to automatic thoughts or cognitions, especially if the thoughts are negative. However, distressed individuals may not have the skills to conduct such frequent "reality checks" and may therefore have consistently maladaptive or distorted cognitions, which greatly influence their affect and actions.

There are a multitude of examples of cognitive distortions to which individuals with depression fall prey. While an exhaustive list is beyond the scope of this paper, some illustrations of these dysfunctional thinking styles include: "dichotomous thinking" ("If I don't get an A, I am a failure"), "overgeneralization" ("I can never get a break in this world"), "should statements" ("I should do whatever my spouse tells me to"), "selective abstraction" ("Nothing else matters except the negative details of this situation"), and "emotional reasoning" ("Because I feel stupid right now, I must be stupid") (Beck, cited in J. Beck, 1995).

In keeping with the diathesis-stress model of the development of psychological disorders, it has been suggested that cognitive distortions are created when a "stressful external event... activates an individual's unrealistic schemata" (Beck, Epstein, & Harrison, 1983a, p. 2). In this case, a person's longstanding negative schema is regarded

as a diathesis or vulnerability factor (unrelated to biological/genetic diatheses), and as it interacts with an environmental stressor, a host of distorted cognitions are produced, all of which become more negative and difficult to control than they would were it not for the onset of the stressful life event. As previously noted, these now-activated schemata and negative automatic thoughts may greatly influence the person's emotional state and behaviors, and will create depressive symptoms, for example, in individuals who are at risk.

Attributional Style and Helplessness

In addition to the cognitive structures (e.g., maladaptive schemata, cognitive distortions) that may make an individual prone to develop a psychological disorder like depression, attention is often given to one's cognitive *style* as a vulnerability marker; the former relates to specific perceptions about self while the latter appears to be more associated with the general manner in which one perceives causality of events in the world. The concept of learned helplessness (Seligman, 1972) suggests that, after a series of learning trials without reinforcement, one develops the expectation of negative outcomes that are non-contingent upon the response (Abramson, Seligman, & Teasdale, 1978). Relatedly, theory and research have suggested that the specific manner in which one interprets events – regarding the causes, stability, and universality of events -- is related to the affective and behavioral response to the event (Abramson, Metalsky, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978). According to this model of attributional style, there are three dimensions involved in the emergence of helpless

cognitions – internal/external ("caused by me" or "caused by another"), stable/unstable ("permanent damage" or "temporary damage"), and global/specific ("always" or "just in this situation").

As is the case with schemata, the development of a particular attributional style may theoretically be traced back to childhood and is likely related to three important interpersonal realms in which a child is traditionally involved: Family and parent-child relationships, peer interactions, and teacher-child relationships (Haines, Metalsky, Cardamone, & Joiner, 1999). It is suggested that negative interactions in any or all of these realms act as a risk factor for a child to develop a depressive attributional style in the future.

Many studies have examined specific aspects of helplessness and attributional style as related to depression. Helplessness has been found to be a cognitive style highly connected to vulnerability to the disorder (Ingram, Miranda, & Segal, 1998). In addition, studies have demonstrated that depressed individuals tend to make more internal, stable, and global assessments about negative events in life, and more external, unstable, and specific attributions about positive events (Abramson, Seligman, & Teasdale, 1978).

Thus, cognitive theory provides a model of the development of psychological disorders, particularly depression, complementary to the diathesis-stress paradigm, which suggests that one has a latent cognitive vulnerability (apart from any biological vulnerability) in the form of negative schemas and a maladaptive cognitive style. When an environmental stressor occurs, these specific cognitive responses are triggered, thus activating a host of negative automatic thoughts or cognitive distortions and a

depressogenic attributional style. It is important to note, however, that although this cognitive style is conceptualized as a generally stable vulnerability factor, it is not necessarily irreversible and permanent (Ingram, Miranda, & Segal, 1998).

More recently, proponents of the cognitive model have turned to schizophrenia as a disorder to conceptualize within the realm of overall disturbed thinking (Kingdon & Turkington, 1994). Historically, Bleuler (1911) focused much of his theoretical work on the pattern of loosened associations of thought that he observed in patients with schizophrenia. Similarly, the view of schizophrenia as a disorder of thinking was held by Kraepelin (1919/1971), who coined the term "dementia praecox" (the original name for schizophrenia), suggesting that individuals with this set of disturbed thinking symptoms were entering an early phase of dementia.

The cognitive model views schizophrenia's disordered thinking as consisting of several components: Disturbed content of thinking (delusions), disturbed mechanism of thinking (loosening of associations, overinclusion, concrete thinking, illogicality and irrational reasoning), disturbed expression of thoughts (the linguistic component of formal thought disorder, language disturbance), and disturbed manner in which "events in the real world are thought about or judged" (Kingdon & Turkington, 1994, p. 43), also described as a "lack of common sense" (Cutting & Murphy, 1988).

Once regarded as the disorder that is not able to be effectively treated with cognitive therapy, as this set of interventions was originally targeted for the "neuroses" (Beck, in Kingdon & Turkington, 1994), schizophrenia has now been conceptualized in terms of negative schemata, dysfunctional attributional styles, and cognitive distortions.

Indeed, cognitive and cognitive behavioral therapies have been empirically tested and supported as viable interventions for schizophrenia in general (Kingdon & Turkington, 1994; Kuipers et al, 1997; Rector, Seeman, & Segal, 2003; Sensky et al, 2000; Tarrier, Beckett, Harwood, Baker, Yusupoff, & Ugaarteburu, 1993), and as promising interventions to target specific positive symptoms such as delusions (Chadwick & Lowe, 1994).

The Importance of Studying Delusions

Regardless of past doubts of the applicability of cognitive interventions to symptoms of schizophrenia, delusions actually lend themselves well to cognitive theory and techniques (discussed below) and are, in addition, very important to study. Historically, the study of delusions was given a great deal of attention, as theorists such as Kraepelin (1919/1971), Bleuler, Jaspers (1963), and Schneider spent a great deal of time organizing and classifying categories of this symptom. Bleuler (1911/1950) proposed that delusions were a product of disturbances of affectivity and associations, and could be divided into the categories of "basic delusions" (the main set of beliefs) and "elaborative delusions" (basic beliefs extend to other areas of thinking). Schneider (1959) suggested that a delusion was not a primary disturbance of perception or sensation, but one of symbolic meaning or attribution.

Since the work of these early theorists, however, delusions have not received a great deal of attention in the psychopathology literature, despite their prevalence (Winters & Neale, 1983) and their importance in the definition, diagnosis, and course of several

psychiatric conditions (Harrow, MacDonald, Sands, & Silverstein, 1995; Jorgensen, 1994). Not limited to schizophrenia, delusions occur in a variety of disorders, including unipolar and bipolar affective disorders, delusional disorder, substance use disorders, personality disorders (schizotypal personality disorder and transiently in borderline personality disorder), and organic psychoses (Winters & Neale, 1983). Perhaps because they occur in so many manifestations of psychopathology, they are not given primary diagnostic importance, as many researchers choose to give attention to more general syndromes or "basic processes" instead (Jorgensen, 1994; Oltmanns & Maher, 1988, p. xi). It is quite valuable, however, to study specific symptoms of disorders as well, in that frequently, a patient's symptoms can often be more reliably identified and more meaningfully related to an individual's past experiences and social background than can a syndromal diagnosis or more microscopic psychological processes. In addition, when symptoms are not studied individually, "fascinating and important psychological phenomena are ignored" (Persons, 1986, p. 1253). Other advantages of studying specific symptoms include the avoidance of misclassification of research subjects, the ability to formulate and test hypotheses about relationships between symptoms, as well as relationships between symptoms and causes and their underlying mechanisms (Persons, 1986).

Furthermore, delusions are extremely common in psychotic patients; they are one of the hallmark symptoms of schizophrenia, occurring with much more frequency than formal thought disorder (Winters & Neale, 1983). While theorists such as M. Bleuler (1978a, 1978b) have suggested that delusional ideation in schizophrenia generally

subsides after five years, more recent studies have found that even with antipsychotic medication, delusions persisted over an eight-year period in 75% of psychotic subjects (Jorgensen, 1994), and that, although delusions existed in both patients with bipolar affective disorder and schizophrenia, they were more severe, more frequently occurring, and persisted for a longer period of time in subjects with schizophrenia (Harrow, et al., 1995). Delusions also appear to play a significant role in the onset and relapse process (Herz, 1990; Jorgensen & Jensen, 1994) and may also influence the process of a first psychotic break. They clearly represent a disturbing and socially disruptive symptom that often becomes quickly apparent to others in a patient's environment (Chapman & Chapman, 1988; Harrow, Rattenbury, & Stoll, 1988; Yung, et. al., 1998).

While some theorists have eschewed the existence of connections between a patient's specific areas of distress and their delusional content (e.g., Berrios, 1991), there are many researchers who have suggested that delusions often have a content which can frequently be understood and described in terms of the patient's social, interpersonal, and psychological history, as well as his or her current situation (e.g., Lucas, Sainsbury, & Collins, 1962): "In delusions everything which one wishes and fears may find its level of expression," (E. Bleuler, 1911/1950, p. 117). Research suggests that concerns, ideas, and aberrant beliefs premorbidly held by individuals who had a subsequent psychotic break tend to manifest themselves in the patient's ensuing delusional content (Chapman & Chapman, 1988; Harrow & Prosen, 1978; Harrow, Rattenbury, & Stoll, 1988). Given the prevalence, meaning, and persistence of this symptom, as well as the conceptualization of schizophrenia as a disorder of thinking, it is useful to understand delusional ideation from

the perspective of the cognitive model.

The Cognitive Theory of Delusions

Conventional definitions of delusions highlight the following: They are abnormal beliefs or ideas that are 1) certainly false, 2) held with absolute conviction, not changeable by facts or arguments, 3) not sanctioned by one's culture or religious subgroup, 4) often fantastic, and 5) of great personal significance to the individual (Butler & Braff, 1991; Oltmanns, 1988; Winters & Neale, 1983; APA, 2000). In contrast, the use of cognitive conceptualizations of this symptom involves an explanation with a focus more on the thought processes that are involved rather than simply the specific content (Beck, & Rector, 2002). This is similar to the traditional distinction between "form" and "content" of thought.

According to Alford and Beck (1994, p. 370), delusions "involve severe cognitive dysfunction that leads to negative (harmful) consequences; simply put, delusions are maladaptive cognitive constructions of internal or external phenomena." Contrary to classic psychoanalytic approaches toward delusions, which suggest that delusions can be understood mainly through examination of unconscious drives and conflicts that obtain symbolic expression, the cognitive model posits that patients experience delusional beliefs and the thoughts that lead up to them consciously, within the realm of personal awareness. Also diverging from traditional behaviorist approaches, which focus on the objective realm of verbal behavior in delusions, the cognitive approach offers that delusions are "*less* than the psychoanalysts theorize them to be (they can be directly

understood apart from psychoanalytic theory) and *more* than the 'verbal behavior' conceptualization of some behaviorists" (Alford & Beck, 1994, p. 371). In line with cognitive theory's conceptualization of other psychological disorders, that thoughts originate from and influence emotions in a cyclical manner, this theory suggests that the content of delusions may also be associated with patients' affective experiences (Freeman, Garety, & Kuipers, 2001).

A traditional and widely-accepted approach to understanding the development and maintenance of delusional ideation, the "anomalous perception model" proposed by Maher (1988), posits that delusions develop through an individual's "normal" attempt to make sense of an abnormal sensory experience, such as an hallucination. Maher suggests that the individual does not have impaired reality testing or judgment, but that he/she is naturally disturbed by this perceptual symptom, and "reasonably" searches for a way to find meaning in his/her experience, hence arriving at a delusion that is almost demanded by the abnormal perception. Notably, this theory has been further tested as a cusp catastrophe where the person makes a sudden, near-discontinuous transition to a delusional belief, and findings suggest that individuals with reported paranormal (related to delusions) experiences exhibit two basic states in reaction to these anomalous perceptions. For individuals with a low tolerance for ambiguity, fear tended to exceed one's belief in the event. In the other state, belief in the anomalous event exceeded degree of fear in those who had a higher tolerance for ambiguity (Lange & Houran, 2000).

As empirical evidence for the anomalous perception theory, Maher notes that people with schizophrenia may have an overly broad attention span, which makes it

challenging for them to focus on one set of stimuli, and also that illogical reasoning is not generally found in delusional individuals (Winters & Neale, 1983). While Maher's theory has received some support, other research has found that abnormal perceptions play a role only in some delusions (Bentall, 2001). In addition, studies of hypothetically psychosis-prone individuals revealed that many subjects experienced aberrant beliefs (i.e., low-level delusions) without reporting the existence of any perceptual anomalies, and vice versa (Chapman & Chapman, 1988). Thus, the connection between delusion development and abnormal perceptual experiences is by no means obvious or absolute.

Cognitive Biases in Individuals with Delusions

As an alternative explanation of delusions, Bentall (2001; see also Beck & Rector, 2002) suggests that the documented distorted inferential processes or cognitive biases in people with schizophrenia are at the core of understanding the development of delusions. The focus here is on cognitive distortions rather than perceptual anomalies. Several studies involving patients' performance on a variety of judgment and reasoning tasks lend support to this theory. Research has shown that individuals with delusions are prone to give an abnormal amount of weight to evidence that appears to support their initial hypotheses, and they tend to "jump to conclusions," requiring less information before reaching a judgment than non-deluded people (Bentall, 2001; Garety, Hemsley, & Wessely, 1991). In addition, they appear to have selective biases in information processing. On an emotional Stroop task, patients with persecutory delusions (diagnosed with either paranoid schizophrenia or delusional disorder) selectively processed words with threat and paranoid-related content (Bentall & Kaney, 1989), and on a similar task,

recalled more threatening story themes than control subjects (Kaney & Bentall, 1992). These findings lend support to the idea that individuals with delusions may have a "strong emotional need to reach definite ideas about the world and an inability to tolerate uncertainty" (Bentall, 2001, p. 141) and also exhibit a great deal of similarity in their use of the same types of cognitive distortions (dichotomous thinking, overgeneralization, selective abstraction) employed by individuals with clinical depression.

Relatedly, other evidence for cognitive biases in individuals with delusions is demonstrated through attribution theory. In contrast to the internal attributions that depressed individuals make for negative events and the external attributions made for positive events, studies have shown that individuals with persecutory delusions (some of whom had diagnoses of paranoid schizophrenia and some with diagnoses of delusional disorder) have the opposite pattern; they tend to exhibit abnormally high levels of attributing positive events to themselves and negative events to outside factors (Candido & Romney, 1990; Kaney & Bentall, 1989; Lyon, Kaney, & Bentall, 1994). When examining specific external attributions – attributions that involve "circumstances" ("external-situational attributions") and those that involve "people" ("external-personal attributions"), these deluded individuals tended to blame negative happenings on other people and rarely attributed positive *or* negative events to circumstances, suggesting that they perhaps excessively attribute events and experiences to others, which may in turn contribute to the emergence of thoughts of persecution (Bentall, 2001). While the internal/external dimension was opposite from the attributions of those with depression, both groups tended to exhibit stable and global attributions for negative events. The

abnormal internal/external attributional style demonstrated by deluded individuals is regarded as an "extreme form of what social psychologists have termed the 'self-serving bias'" (Kaney & Bentall, 1992, p. 774).

Other evidence for these types of cognitive biases in schizophrenia comes from Beck and Rector (2002), who suggest that such individuals exhibit an egocentric bias, in that they "see themselves in a drama during which most events are relevant to them... threats are everywhere... delusional patients may attach personal meanings to almost the most mundane events" (p. 457-458). This is related to the classic phenomenology of "paranoia." They continue to explain this egocentricity as a comparison to the elevated levels of self-focused attention experienced by individuals with social anxiety disorder, suggesting that once the "fear network has been activated," even unimportant happenings are regarded as self-referent. The comparison of elements of social anxiety and persecutory delusions, although discussed merely as a theoretical example by Beck and Rector, suggests a potential conceptual link between these two symptoms, which may further elucidate the developmental process of delusions.

Dysfunctional Self-Concept

Although congruent with the overall nature of a delusional belief, which is often an unusual explanation for an upsetting experience, it is perhaps counterintuitive that individuals who feel persecuted by others would formulate a grandiose, "self-serving" attributional style. Potential answers to this conundrum may be found in theories describing persecutory delusions as serving a defensive function against low self-esteem

(Beck & Rector, 2002; Bentall, 2001; Bentall, Kinderman, & Kaney, 1994; Lyon, Kaney, & Bentall, 1994), which are actually quite similar to Kohut's (1972) theories of the development and function of narcissism.

It has been suggested that in "normal" individuals, self-esteem is maintained through the use of a cognitive style that generally attributes negative events to factors outside oneself, thus removing personal responsibility when this is harmful to self-esteem. Therefore, for people with persecutory delusions (or elements of narcissism, as suggested by Kohut) who have a strikingly exaggerated external attribution style, the abnormal beliefs may be an extreme method of saving their low self-esteem at the expense of deeming other people as malicious or evil.

While past researchers have not been able to discover this pattern using traditional, direct measures of self-esteem, others (e.g., Lyon et al., 1994) have found that on a more subtle task of attributional style, deluded (again with diagnoses of either schizophrenia or delusional disorder) subjects exhibited a cognitive style similar to the depressed subjects (internal attributions for negative events; external for positive events), while maintaining their more common self-serving bias on an obvious attribution test. In the Lyon and colleagues study, in addition to the Attribution Style Questionnaire (ASQ; Peterson, Semmel, Von Baeyer, Abramson, & Seligman, 1982), participants were given an implicit attribution task, the Pragmatic Inference Task (PIT; Winters & Neale, 1985). In this task, subjects listen to a series of stories describing themselves participating in successful and unsuccessful activities. They are then asked to determine which of two causes (one external; one internal) was responsible for the positive or negative outcome

of each story, although the stories are designed so that neither attribution is more obviously correct than the other. It is argued that the causes chosen will reflect participants' true self-evaluations.

The similarity between the implicit self-evaluation style in the deluded group and the depressive group in the Lyon and colleagues study supports Zigler & Glick's (1988) hypothesis that delusions of persecution may reflect a "masked" form of depression. (Of course, delusions – mood-congruent ones – can be a feature of major "psychotic" depression). Further, it has been suggested that developing a delusion may help a person reduce the self-ideal discrepancy that he/she experiences, which often develops as a result of negative life events or everyday interactions with others (Bentall, et al., 1994). Diathesis-stress formulations of other disorders, such as depression, theorize that the dysfunctional self-concept (a.k.a., negative self-schema) becomes activated during times of stress or in response to criticism from others. This emergence of the dysfunctional self-concept is hypothesized to take place in the formation of schizophrenia symptoms, particularly delusions (Martin & Penn, 2002). As the individual perceives disapproval, negative self-schemas may become activated. To reduce the impact of the emergence of such negative views of self, persecutory delusions may emerge as a defense against threatened self-esteem and reproach from others.

Beck and Rector (2002) construct a comprehensive theoretical framework in which to understand the interaction of cognitive biases and dysfunctional self-concepts as precipitants to delusion formation. In keeping with the work of Bentall and colleagues, they suggest that delusions are compensations for underlying deficits in self-esteem, as

well as feelings and beliefs of "loneliness, unworthiness, evil, incompetence, or powerlessness" (p. 461). With regard to the formation of grandiose delusions, they suggest that the delusion begins as a fantasy or a daydream that helps promote one's self-concept, and that eventually, people become unable to differentiate between the fantasy and reality; they begin to adopt the identity of the figure that they previously emulated. However, although one would expect that adopting the identity of a powerful and respected figure such as the president, celebrity, or religious deity would offer the person feelings of power and joy, dialogue with individuals with grandiose delusions suggests that the underlying feelings of isolation and pain are still very much evident. Beck and Rector suggest a linear pathway to the development of a grandiose delusion; an example of this is presented in Table 1, describing a patient who had the delusion that she was the poet laureate.

Explanations of proximal precipitants of the formation of a persecutory delusion involve a fear of being evaluated, endangered, or hurt in some manner, often at the hands of a group from whom the individual anticipates retaliation. In order to exemplify the process more clearly, Beck and Rector provide a case example of a patient who reported drug dealing to the police and then saw an article about policemen involved in drug rings. The pathway to the subsequent persecutory delusion is presented in Table 2. It follows in the theory that one may develop combined grandiose and persecutory delusions, as individuals who compensate for deficiencies in positive self-concept would be disheartened to realize that others in their day to day lives do not treat them with the respect and awe that would be appropriate for someone of their talent or stature. It is

likely that they in turn will try to find reasons for the lack of respect from others, and may come to the conclusion that others are jealous, and therefore plotting to destroy them. An example of such a combined delusion would be the belief that one is Jesus and about to be crucified.

Thus, it is suggested that delusions may be the result the interplay of an "epistemological impulsiveness" (frequent "jumping to conclusions," perhaps due to a strong need for certainty; Bentall, 2001, p. 142), selective biases of information processing, an externalizing attribution style for negative events, and an egocentricity about the cause and relevance of environmental events. These cognitive biases and the use of cognitive distortions, likely made worse during the experience of negative emotion such as depression or anxiety (Garety, Kuipers, Fowler, Freeman, & Bebbington, 2001), influence normal processes of belief formation. Coupled with a previously existing dysfunctional self-concept or negative self-schema and the onslaught of environmental stress, criticism, or the fear of hurt from others, attempts are likely made to neutralize the activation of negative beliefs about self by creating compensatory delusions that take on the characteristics of excessive grandiosity or paranoia.

While these theories of delusion formation may appear quite complex and detailed, relatively straightforward interventions based in cognitive therapy with roots in this theory have been found to be effective in reducing delusional ideation in individuals with schizophrenia. Borrowing cognitive therapy techniques from the treatment of other disorders such as depression and anxiety, specific belief modification interventions for delusions include psychoeducation (educating the patients about the symptoms and

course of schizophrenia and/or delusions), collaborative empiricism (a therapist-client "teamwork" perspective regarding the questioning and testing of beliefs and behaviors), planned reality testing (experimentation used to help the client discover new ways to cope with negative thoughts and modify beliefs), normalizing (de-stigmatizing symptoms by examining them through rational argument), and verbal challenge in the form of evidence examination (encouraging the client to view a delusion as only one possible way to interpret events; Alford & Beck, 1994; Chadwick & Lowe, 1994). These techniques have been effective in reducing the conviction of delusions, as well as the subjective distress that inevitably accompanies them.

Cognitive Treatment and Cognitive Vulnerability of Individuals at Risk

Given the complex and persistent nature of delusions and the emotional suffering that they often cause, as well as the overall debilitating effects of schizophrenia, it is worthwhile to attempt to find ways to prevent the onslaught of delusions and therefore, presumably florid psychosis in individuals who may be at risk to develop the disorder. The recent emergence of efficacy data on cognitive interventions for schizophrenia shows potential (Kingdon & Turkington, 1994; Kuipers et al, 1997; Rector, Seeman, & Segal, 2003; Sensky et al, 2000; Tarrier, Beckett, Harwood, Baker, Yusupoff, & Ugaarteburu, 1993) and may contribute to a model for use of these techniques in an at-risk population. As the goal of at-risk intervention studies is primary prevention of schizophrenia, or at a minimum, delay of the disorder's onset, the use of psychological interventions has been suggested for this population (Yung, et al., 1998; Morrison, et al., 1998). Unfortunately,

there appears to be only one completed study of a cognitive therapy intervention with individuals at risk for schizophrenia, but results look promising. In a series of case studies of a homogenous group of clients at risk (as defined by the Yung et al., 1998 criteria), French, Morrison, Walford, Knight, & Bentall (2003) utilized various cognitive therapy techniques such as selective attention strategies, belief modification, and the manipulation of safety behaviors within a problem-solving context. They found that two of the three subjects responded well to the interventions, and that their transient and attenuated psychotic symptoms diminished.

Morrison and colleagues (2002) are currently in the midst of conducting a much needed randomized controlled trial of cognitive therapy for individuals at high risk (using the Yung et al., 1998 criteria) for schizophrenia. While preliminary results of treatment efficacy are not yet available, these researchers have attempted to tap into areas of cognitive vulnerability in these subjects, a strategy that has not previously been employed with high-risk individuals. This study used the Meta-Cognitions Questionnaire (MCQ; Cartwright-Hatton, & Wells, 1997), a measure that examines beliefs about internal cognitive events ("meta-cognitions" generally defined as "cognitions about cognitions") and has been shown to correlate with measures of psychotic-like symptoms in non-patients (Morrison & Wells, 2000). The questionnaire measures negative beliefs about the controllability of thoughts ("I cannot ignore my worrying thoughts"), levels of cognitive confidence ("I have a poor memory"), general negative beliefs about thoughts such as superstition and punishment ("If I did not control a worrying thought, and then it happened, it would be my fault"), and levels of cognitive self-consciousness ("I pay close

attention to the way my mind works"). The researchers found that the at-risk sample had significantly higher scores on these subscales than the control group. Morrison has posited that negative meta-cognitions play an important role in the development of positive symptoms like delusions, and the high scores on this measure were interpreted as support for this theory.

In addition, these researchers administered the Sociotropy-Autonomy Scale (Beck, Epstein, & Harrison, 1983b). Related to Blatt and colleagues' (2001) constructs of "anaclitic" (concern with interpersonal relationships) and "introjective" (concern with securing a positive sense of self) dimensions of depression, Beck et al. (1983a) suggest that the traits of sociotropy (or social dependence) and autonomy (need for independent goal attainment) influence one's perception of and interaction with the world, and may act as predisposing factors in the development and maintenance of depressive symptoms. According to Morrison and colleagues, this measure was included in their study as an assessment of dysfunctional self-schemata. On the two subscales, Sociotropy, measured as levels of fear of rejection and criticism, and Autonomy, measured as levels of self-demands of accomplishment and control, subjects reported how closely statements regarding these two areas described them. It was found that the at-risk sample scored significantly higher than the control group on the Sociotropy subscale, but not on the Autonomy subscale. The finding of higher scores on Sociotropy in the at-risk group is an important one, and is consistent with previously described theories of delusion formation (Beck & Rector, 2002; Bentall, 2001) which suggest that delusions may function as defenses against threatened self-esteem, as such elements of dysfunctional self-concept

are often triggered as a result of criticism from or negative interactions with others.

Preliminary findings in the schizophrenia treatment literature suggest the feasibility of reducing or preventing delusion formation using cognitive techniques, and the at-risk literature has demonstrated that researchers can measure specific forms of cognitive vulnerability. Given these two promising yet limited research advances, a clearer, more detailed assessment of aspects of cognitive vulnerability in individuals at risk for psychosis is warranted, with an eye toward defining specific foci for intervention.

The Present Study

The purposes of the present study were multifaceted. As prevention of schizophrenia is clearly an important and possibly feasible goal, this research built on the work of Yung and colleagues (1998) as a continuation of the use of "trait plus state" factors in order to measure variables related to risk for psychosis. Additionally, as previously described, components of cognitive vulnerability have been discovered in individuals with schizophrenia, and in order to prevent breakdown, it is therefore necessary to examine more closely these subsets of dysfunctional cognitions in individuals at risk. Although Morrison and colleagues (2002) pioneered this particular line of research, their investigation into the details of cognitive vulnerabilities was limited, as the MCQ does not quite seem to tap into negative self-schemas or dysfunctional attitudes; and, the Sociotropy-Autonomy scale is narrow in scope, as it only examines to two specific facets of self-schemas. There appear to be additional areas of cognitive vulnerability to study that have not yet been pursued in the literature. While

several researchers have implicated abnormal attributional style (the self-serving bias) in the development of schizophrenia, there have been no studies to date looking at this pattern in individuals at risk, and as previously mentioned, only one (Morrison et al. 2002) has investigated negative self-schemata.

This research examined more specific facets of cognitive vulnerability, such as attributional style, cognitive distortion, and negative self-schemata in individuals at risk for psychosis. This study departed from Yung and colleagues (1998) and Morrison and colleagues (2002) in that it was not a prospective investigation examining rate of transition to full-blown psychosis; instead low-level delusional ideation was used as the dependent variable. Utilizing presence and type of delusions as the dependent variable seemed appropriate, given the prevalence of this symptom, as well as its phenomenological importance and significance as a cognitive process. Both general cognitive vulnerability and specific components were examined in their ability to predict low-level delusional ideation in general, as well as more specific types, in particular delusions of persecution and delusions of grandeur. The study examined specific types of cognitive vulnerability in individuals at risk for schizophrenia (as well as in a control group, for comparison purposes) in order to identify how dysfunctional thinking patterns/negative self-schemata and attributional style predicted specific delusion types. With thorough investigation of these potentially related areas, this research adds to the literature on cognitive processes, on risk to psychosis, and on delusional ideation. It is hoped that knowledge about specific cognitive vulnerabilities in individuals at risk will aid clinicians in developing particular interventions to target and modify these

dysfunctional, unhealthy beliefs *before* they lead to full-blown delusions, thus preventing the pervasive nature of this symptom and its relationship to overall deterioration in functioning.

Based on a review of the literature and the rationale for the present study, the following hypothesis were tested:

1. Individuals who are categorized as "at-risk" (using a modified version of the Yung et al. criteria) have significantly higher levels of overall cognitive vulnerability (e.g., more dysfunctional attitudes, more pronounced negative self-schemas) than do those in the control group.
2. At-risk individuals exhibit an attributional style similar to that found in the literature with schizophrenia patients (external attributions for negative events and internal attributions for positive events), and this attributional style differs significantly from the attributional style of the control group.
3. At-risk individuals have significantly higher levels of delusional ideation than those in the control group. The exhibited ideation can be divided into persecutory and grandiose delusion components.
4. Participants' levels of overall cognitive vulnerability possessed, as measured by the IPSAQ and the DAS (see Measures in Methods section) are significantly positively correlated with the degree of delusional ideation exhibited in both groups.
5. Specific risk factors (e.g. genetic risk, high score on general psychopathology

measure, age in early 20s, etc.) are related to level of delusional ideation, and certain combinations of these aforementioned risk factors are significantly more predictive of delusional ideation than other combinations.

6. There is an interaction between the level of risk for psychosis -- as determined by a "risk score" (discussed below) -- and level of cognitive vulnerability/ specific attributional style in predicting delusional ideation. Inclusion of the interaction of the two accounts for a significantly larger proportion of the variance in ideation than does either variable alone.

Method

Participants

Participants for this study comprised two distinct groups: The At-Risk Group and the Control Group. Characteristics of subjects and recruitment strategies are described below. Results from a power analysis, conducted with the Sample Power (Borenstein, Rothstein, & Cohen, 1997) computer program to determine the appropriate *n* for each group, revealed that a sample size of 50 per group was necessary to have adequate power (.70) to detect group differences. The total *n* for this study was 133 (61 in the At-Risk Group and 72 in the Control Group). Gender and age matching procedures are described below.

At-Risk Group

Inclusion criteria for the At-Risk Group were based on those outlined in Yung et al. (1998) and Morrison et al. (2002) and modified slightly for the sake of feasibility and

practicality in this research. Subjects in this group who reported having a first-degree relative (sibling, parent, or child), second degree relative (aunt/uncle, grandparent), or third degree relative (cousin, great-aunt/uncle, great-grandparent) with a history of any DSM-IV diagnosis of any psychotic disorder (e.g., Schizophrenia, Schizoaffective disorder, Delusional disorder, Schizophreniform disorder, Schizotypal personality disorder, and Psychosis NOS) were selected for participation in the study. As it was not feasible to confirm reported family member diagnosis with an assessment or chart review, participants were included in the study based on their report alone. Furthermore, as it was not possible to confirm specific family member symptoms and whether or not those symptoms truly were indicative of the six targeted Psychotic Spectrum Disorders, endorsed symptoms were not used in isolation to determine whether an individual qualified as having a psychotic relative. Thus, subjects were included in the At-Risk Group only if they endorsed being certain that their family member was diagnosed with one of the six listed psychotic disorders (in addition to other criteria described below); if they *in addition* happened to report the existence of relatives' specific psychotic symptoms (as was most often the case), that was also acceptable for entrance into the At-Risk Group. Thus, inclusion criteria for the present study's At-Risk Group was in some ways narrower than the Yung et al. (1998) criteria (e.g., BLIPS not included) and in some ways wider (e.g., participants could have either 1st, 2nd, or 3rd degree relatives).

At-Risk participants were between the ages of 18 and 36 years old, which is an age-range targeted in other at-risk studies (e.g., Morrison, et al, 2002). In addition, many At-Risk Group subjects also had an elevated score on the study's measure of general

psychopathology, the Outcome Questionnaire (OQ-45.2; Lambert & Burlingame, 1996); in fact, approximately 52% of At-Risk subjects had OQ scores in the clinical (raw score greater than or equal to 63) or subclinical range (raw score between 57 and 62). Data from this subset of subjects were included in a series of level of risk-to-psychosis analyses described below in the Results section. Of the 61 At-Risk subjects included in this study, 27.9% (n = 17) reported having a first degree relative with a psychotic disorder, 55.7% (n = 34) reported having a second degree relative, and 16.4% (n = 10) reported having a third degree relative.

Control Group

The Control Group was composed entirely of undergraduates from the University of Montana Introductory Psychology subject pool. These subjects also participated in the initial course "screening" and were given the same survey as those who were in the At-Risk group (survey details described in Procedure section below). The Control Group subjects were randomly chosen from a subgroup of screening subjects who reported not having any family history of any DSM-IV psychotic disorder, nor suspecting any psychotic symptoms in their family members, and who were between the ages of 18 and 36 years old. In addition, after completing the questionnaires, potential Control Group members whose Outcome Questionnaire scores exceeded the clinical cutoff score delineated in the manual (raw scores greater than or equal to 63) were excluded from the analyses (approximately 5 potential Control Group subjects were excluded for this reason).

Subjects were matched for gender and age across groups. To achieve this, all

participating screening questionnaires were initially sorted into two categories: those from subjects who endorsed having a relative with a psychotic disorder, and those who did not. Then, screening measures were sorted by gender and age range within each group. For each At-Risk Group member to be included in the study, a Control Group member of the same gender and age range was randomly chosen as well. Results indicate no significant gender differences, X^2 (1, n = 133) =. 20, p = .66; age differences, t (131) = .93, p = .36; or ethnicity differences, X^2 (1, n = 133) =. 11, p = .74 between groups. There was however, a significant between-group difference in marital status (ever-married vs. never-married), X^2 (1, n = 133) = 3.84, p = .05 as At-Risk Group subjects were more often married and Control Group subjects were more often single. Demographic data for all study participants are shown in Table 3.

Measures

Cognitive Vulnerability Measures

Attributional style was assessed using The Internal, Personal, and Situational Attributions Questionnaire (IPSAQ; Kinderman & Bentall, 1996). This measure was designed in response to criticism of low internal consistency in the commonly-used Attributional Style Questionnaire (ASQ; Peterson, Semmel, Von Baeyer, Abramson, Metalsky, & Seligman, 1982); it has been suggested that the internality subscale produces the least reliable scores on the ASQ (as well as on the related EASQ; Metalsky, Halberstadt, & Abramson, 1987), which is a problem given that for research with disorders other than depression (e.g., psychosis), the internality scale is generally the one

of interest (Kinderman & Bentall, 1996).

The IPSAQ has 32 items and measures attributions for both positive and negative social situations via a fill-in response and then a categorization of the causality of each particular event using a multiple-choice format. An example scenario is: "A friend betrayed the trust you had in her. What caused your friend to betray your trust?" (Subject writes in brief response). "Is this a.) something about you?, b.) something about the other person or other people?, or c.) something about the situation (circumstances or chance)?" Six subscales are calculated to reflect the different types of attributional styles (e.g., internal attributions for positive events, external attributions toward people for positive events, external attributions toward situations for positive events, internal for negative events, external toward people for negative events, and external toward situations for negative events). As the purpose of the present study was to evaluate the attributional style demonstrated in an at-risk group and draw theoretical comparisons to individuals with psychosis, only the two subscales relevant to that particular "delusional" attributional style were used in the analyses: internal attributions for positive events and external attributions toward people for negative events. The authors of the scale suggest constructing " bias" scores from these subscales in order to reflect common cognitive biases described in the literature, "Externalizing Bias (EB)" and "Personalizing Bias (PB)" (Kinderman & Bentall, 1996). These scores were calculated in this study; however, they were not used in the analyses as the guidelines for correct interpretation that was provided by the authors were unclear. Thus, only the "internal positive" subscale and the "external personal negative" subscale were used in the analyses.

Of note is that the developers of the scale, in a follow-up article (Kinderman, Kaney, Morley, & Bentall, under review), highlighted the importance of scoring the IPSAQ items based solely on the subjects' own causality ratings, and not modifying them to reflect the ratings of an independent observer. For example, a deluded subject may perceive the cause of a particular event as "caused by me," whereas an independent observer would clearly interpret the response as "caused by circumstances." In their article, the authors note that while there may not be discrepancies between the deluded subjects' *descriptions* of the actual causes of the events and those of the independent rater, but that the discrepancies apparently emerge in the subjects "attributions about their attributions" (Kinderman et al., under review, p. 2). Thus, it is not the deluded subjects' specific indicated cause of the event (e.g. Q: "A friend gave you a ride home;" A: "because the bus broke down") that is per se abnormal or significantly different from non-deluded subjects' responses, but it is their perception of who or what causes the reason for the event taking place that is noteworthy.

In the validation study using an undergraduate sample, internal consistency was acceptable (ranging from .61 and .76 for the six subscales) and improved as compared to the internality subscales of the original ASQ (reported by Kinderman and Bentall as ranging from .39 and .52). In this study, the *alpha* coefficient for the overall scale items was .81; for the internal positive subscale and the external personal negative subscale, *alpha* coefficients were .74 and .80, respectively. As regards the measure's validity, both clinical and analogue studies have found that IPSAQ scores were significantly associated with the ASQ's internality subscales, and were also highly correlated with levels of

paranoid ideation (Kinderman & Bentall, 1997).

Negative self-schemas were assessed using the Dysfunctional Attitudes Scale (DAS; Weissman, & Beck, 1978), a 40-item self-report questionnaire on a seven-point Likert scale ranging from "totally agree" to "totally disagree" that measures one's expectations about desired outcomes ("My life is wasted unless I am a success"), concern with the evaluations of others ("I cannot be happy unless most people I know admire me"), and attachment of importance to certain goals ("It is difficult to be happy unless one is good looking, intelligent, rich, and creative"). It is suggested that this questionnaire taps negative self-schemas as well as elements of cognitive distortions. A total score is obtained by summing the value corresponding to the choice endorsed for each item, and there are no subscales. Reliability in the validation sample of patients and non-patients was adequate, with *alpha* coefficients ranging from .89 to .92 (Weissman, 1979, cited in Hammen & Krantz, 1985). In this study's sample, the *alpha* coefficient was .92. Dobson and Breiter's 1981 validation study (as cited in Beck, Epstein, & Harrison, 1983) found that the DAS was correlated $r = .36$ with the original Beck Depression Inventory (Beck, Ward, Mendelson, Mock, and Erbaugh, 1961) and $r = .43$ with the Automatic Thoughts Questionnaire (Hollon & Kendall, 1980). Beck's initial validation study, based on a sample of undergraduates, found comparable correlations between the DAS and measures of cognitive distortion commonly used at that time (Beck, Epstein, & Harrison, 1983).

Delusions Assessment

Delusional ideation was assessed using a modified version of the Peters et al.

Delusions Inventory (Gottlieb, Schuldberg, Peters, & Caruso, unpublished manuscript). The original version (PDI; Peters, Joseph, & Garety, 1999) is a 40 item self-report questionnaire that measures delusions in the normal or "subclinical" population. Examinees are presented with a question, such as, "Do you ever feel as if there are forces around you which affect you in strange ways?" Subjects must initially mark "yes" or "no" for each item. If the answer is "yes," subjects are then asked to indicate: (1) the level of distress their belief causes, (2) their level of preoccupation with the belief, and (3) their level of conviction that the belief is true, all corresponding to five-point Likert scales (with anchors of "not at all distressing," "hardly ever think about it," "don't believe it's true" to "very distressing," "think about it all the time," "believe it's absolutely true" on the three scales). The original version includes subscale scores that are calculated by adding the Likert ratings of each subscale for the items; however, subscales were not used in the present study. Instead, an overall score was calculated by adding the subscale scores, as well as the initial yes-no item, to come up with a composite delusional ideation score.

Cronbach's α for data scored with the present method from a previous study with an undergraduate student sample was calculated as .90 (Gottlieb, 2001, unpublished manuscript). Criterion validity with the original PDI was established with the norming sample, as a deluded inpatient psychiatric sample scored significantly higher on the PDI than a non-psychiatric normative sample (Peters, Joseph, & Garety, 1999). In addition, concurrent validity has been demonstrated within the normative sample, with scores on the PDI correlated with scores on other delusional ideation measures (the Magical

Ideation Scale, Eckblad and Chapman, 1983; the Schizotypal Personality Scale, Claridge & Broks, 1984; and the Delusions Symptom-State Inventory; DSSI, Foulds & Bedford, 1975). In the revision study (Gottlieb et al.), a significant correlation between the shortened PDI total score and the DSSI total score was found ($r = .19, p = .002$, between the new PDI Grandiosity subscale and the DSSI Grandiosity subscale ($r = .26, p < .001$, and between the new PDI Persecutory subscale and the DSSI Persecutory subscale ($r = .14, p = .02$).

Due to inconsistencies in results from previous factor analyses and the lack of clear categories of delusional ideation, a revised version of this measure was created (Gottlieb, et al., unpublished manuscript) in order be in keeping with the most common categories of ideation (persecutory and grandiose) and to create a more parsimonious assessment (details of this modification are presented elsewhere; see Gottlieb et al., unpublished manuscript). The shortened measure has a slightly different scoring system (which involves recalibrating the Likert items to range from 0-4 instead of 1-5 and collapsing the subscale totals for each item and the initial yes-no item into one composite item score) and contains eight items which factor into two subscales (Grandiosity and Persecution) when used with the undergraduate sample from the revision study. As preliminary analyses of this measure with that sample demonstrated adequate psychometric properties (Gottlieb, Schuldberg, & Caruso, unpublished data), the present study utilized both the original 40-item measure (for breadth) as well as the newly shortened and revised measure (factor analysis with the present study's sample described

below). *Alpha* coefficients for the original measure (with the overall score), the shortened version (total score), and the Grandiose and Persecutory subscales in this present study were .89, .67, .56, and .72, respectively.

In order to examine the factor structure of the shortened 8-item version of the PDI with this At-Risk sample and to ascertain whether these eight items factored into the common delusional themes categories of Persecutory and Grandiose, confirmatory factor analysis (CFA) was conducted using the AMOS computer package (Arbuckle, 1997). The items and the scoring of the original PDI were modified, retaining 8 of the original 40 items, categorized into the two modified subscales. Details of the procedure of the selection of the items are presented elsewhere (Gottlieb, et al., unpublished manuscript). Both orthogonal (uncorrelated factors) and oblique (correlated factors) models for the two-factor shortened measure were tested. While CFA results from the original Gottlieb et al. revision study found that the oblique model was a significantly better fit than the orthogonal model, in the present study there was no statistically significant difference between the two X^2 $(1, N=61) = 0.51, p > .10$. According to the chi-square fit test, neither the oblique, X^2 $(19, N=61) = 36.63, p = .01$ nor the orthogonal, X^2 $(20, N=61) = 37.13, p = .01$, were a particularly good fit for the data; however, each was significantly better-fitting than the independence model, oblique: $X^2 \Delta (9, N=61) = 88.96, p < .001$, orthogonal: $X^2 \Delta (8, N=61) = 88.45, p < .001$. In addition to the chi-square tests, two other fit indices, "the goodness of fit index (GFI)" and the "adjusted goodness of fit index (AGFI)" were used to compare each model to the independence model. The "root mean square error of approximation (RMSEA)" was also calculated in order to determine the

discrepancy between the observed covariances in the data and those predicted in the model. Specifically, the GFI compares each model to the independence model and ranges from 0 to 1, with higher values (especially over.90 or .95) indicating a good fit. RMSEA values of equal to or less than .05 are considered indicative of good fit; .08 is considered adequate. See Table 5 for summary fit statistics and Figure 1 for the two-factor oblique model.

Despite not meeting the absolute strictest criteria for "good fit" on the basis of these fit indices, which may be due to a fairly small *n* in this At-Risk Group, the internal consistency coefficient for the Persecutory subscale is more is adequate (.72). The Grandiosity subscale's *alpha* coefficient is slightly lower than expected (.56) and therefore results utilizing this subscale as a criterion variable (see Hypothesis 6) should be considered exploratory and interpreted with caution.

Assessment of General Psychopathology

The Outcome Questionnaire (OQ-45.2; Lambert & Burlingame, 1996), a 45-item self-report measure, was used to assess levels of psychological symptoms in study subjects. Although designed to measure client progress in therapy, this measure is also used in research studies, as it is relatively brief and easy to complete. The OQ-45.2 assesses a range of common symptoms that occur within a wide variety of psychological conditions and also provides information about a person's interpersonal relationships, current stress and distress, and performance in social roles.

The OQ is composed of a total score and three subscales: Symptom Distress (SD), Interpersonal Relations (IR), and Social Roles (SR). The developers of the scale

suggest utilizing a cut-off score of greater than or equal to a raw score of 63 (the scale has a possible score range of 0 - 180) to distinguish between clinical and non-clinical groups, and this cut-off score was used as exclusionary criteria for the Control Group. Analyses from normative samples of undergraduates and patients demonstrate test-retest reliability coefficients of .84 for the OQ Total, and coefficients ranging from .78 to .82 for the OQ subscales. The internal consistency *alpha* for the OQ Total was .93 for both students and patients, and the OQ subscales coefficients ranged from .70 to .92 across both sample groups. Concurrent validity for this same sample was demonstrated as Pearson correlations between the OQ and several other well-established symptom measures (e.g., SCL-90, Derogatis, 1977; BDI, Beck et al., 1961; State-Trait Anxiety Inventory, Spielberger, 1983; Social Adjustment Scale, Weissman & Bothwell, 1976; etc.) were all significant beyond the $p < .01$ level. In the present study, the *alpha* coefficients for the OQ-45 were as follows: .93 for the total score, .93 for Symptom Distress, .79 for Interpersonal Relations, and .61 for Social Roles.

Procedure

The participants who completed this study (within both the At-Risk and Control Groups) were recruited from the University of Montana undergraduate Introductory Psychology course subject pool. Approximately 1000 potential subjects (about 550 from Fall Semester course and 462 from Spring Semester course) were assessed with a paper and pencil measure during a course "screening day." This screening tool was developed for use specifically to screen participants for this particular study and included questions

addressing potential subjects' knowledge about the mental health of their family members (e.g., specific diagnoses of specific biological relatives, characteristics/traits/symptoms of specific relatives, etc) as well as their willingness to participate in a future study for course credit or monetary compensation ($10) if their course credit requirements had already been filled at the time of their future recruitment.

Follow-up phone calls were made to subjects who met inclusion criteria (as previously described) by one undergraduate research assistant who followed a script and who was blind to subjects' group membership (e.g. At-Risk or Control Group). These subjects were invited to attend one session, where they completed the questionnaires described in the Measures section. Undergraduate research assistants, who were in charge of the data collection sessions, also were blind to the participants' group membership. All subjects consented to participation and completed their questionnaire packets in individual rooms to ensure privacy. After completion of the questionnaires, participants received written debriefing information and a phone list of mental health referral services in the area. All subjects received course credit for their participation. Those who were no longer in need of course credit received $10.

Unfortunately, although discussed in the planning stages of this research, an essential question involving the participants' own history of psychosis was inadvertently omitted from the measures administered. As this information is important to the conceptualization of the subjects as either "at-risk" or "not at risk" for psychosis, there was an attempt to recontact the 133 participants to obtain consent to gather this important mental health history information. An email was sent to 105 subjects asking for

permission to telephone them to ask them this question and 26 were sent a written letter by mail (when an email address was not available) with the same request. Two subjects were not recontacted because they had not provided an email address or regular mailing address and making contact via telephone was not permitted by the IRB. Of those 130 sent the email or regular mail request, 48 replied and consented to the phone call (37%). Forty-three of those 48 consenting subjects were reached by phone (90%) and all of them denied any history of psychosis.

While the response rate/successful recontact rate of these subjects was fairly low (about 1/3 of the entire sample), all of those who were recontacted denied a history of psychosis. Given the base rate of psychosis in the general population (approximately 1%; APA, 2000), the chance that Control Group subjects in this study would report a history of past psychosis is approximately 1 in 100 (n=72). In the At-Risk Group, the chance is a bit higher (Gottesman, 1991); however, the statistical likelihood of individuals in this young age range having already had a previous psychotic episode is rather small. Thus, while lack of full response (and therefore denial of psychosis history) from participants precludes the certain determination of the pureness of the data, it may be assumed with reasonable confidence that the At-Risk Group is representative of a "true" risk group and the Control Group is comprised of individuals without risk to psychosis as defined by this research.

An attempt was made to recruit At-Risk participants from the University Counseling Center (serving University students) and the Clinical Psychology Center (serving the Missoula community). Therapists at these locations were given a description

of the study as well as copies of a recruitment letter to distribute to their clients. Interested clients were then directed to call the study's voicemail number and leave a confidential message with contact information in order to receive a follow-up phone call to confirm inclusion criteria (participant's age, diagnosis of biological relative) and set an appointment to complete the questionnaires. Flyers describing the study were also posted at both of these locations, as well as in the main lobby of the Curry Health Center (see Appendix 2). One potential University Counseling center referral subject called the voicemail; however, she did not meet the inclusion criteria for the study, as she reported that she did not believe that her biological relative formally received a diagnosis of one of the DSM-IV psychotic disorders. After the data collection portion of the study closed, two individuals referred from the Clinical Psychology Center called the voicemail. One of these subjects is in the process of completing the questionnaires; the other has not yet made an appointment to participate in the study. Due to the late date of these referrals, the data from these two individuals were not included in the present analyses. Thus, all 61 At-Risk participants (and, as previously mentioned, all 72 Control Group members) who completed the study were recruited from the Introductory Psychology Course pool.

Results

Reliability analyses and descriptive data from all questionnaires administered are displayed in Table 4. As predicted, members of the At-Risk Group demonstrated significantly higher levels of dysfunctional attitudes and more pronounced negative self-schemas as measured by the DAS than did members of the Control Group (Hypothesis

1), t (131) = 3.48, p = .001 with a medium effect size (d = .58). The At-Risk Group, as predicted (Hypothesis 3) also had significantly higher levels of overall delusional ideation (PDI) than did the Control Group, t (131) = 3.33, p = .001, with a medium effect size (d = .56). In addition, there were between-group differences for persecutory ideation: t (131) = 2.51, p = .01 and grandiose ideation: t (131) = 2.29, p = .02, also with medium effect sizes (d = .43 and d = .39, respectively).

A 2 (Group) by 2 (Attributional Bias) mixed factorial ANOVA analysis revealed a main effect for positive event biases: across groups, individuals made significantly more internal attributions than external attributions for positive events, F (1, 131) = 94.92, $p < .0001$. There was no main effect for Group in attributional style for positive events, F (1, 131) = 1.62, p = .21, and no Group by attributional style interaction for positive events, F (1, 131) = 1.14, p = .29.

An additional 2 by 2 mixed factorial ANOVA demonstrated that there was no significant main effect for negative event biases, F (1, 131) = .01, p = .94, as individuals made a comparable amount of internal attributions as external attributions regarding the cause of negative events. There was also no main effect for Group: participants in the Control Group and the At-Risk Group did not significantly differ on attributional style for negative events, F (1, 131) = .23, p = .63, and there was no Group by attributional style interaction for negative events, F (1, 131) = .26, p = .61. These findings suggest that At-Risk individuals do not demonstrate significantly different attributional biases from the Control Group, for positive or for negative events.

As regards the relationship between negative self-schema, attributional style, and

delusional ideation (Hypothesis 4), significant positive Pearson Product-Moment correlations emerged between dysfunctional attitudes/negative self-schema (DAS scores) and delusional ideation ($r = .38, p < .0001$), but not between the predicted attributional style scores (internal attributions for positive events: $r = .04, p = .61$, and external attributions toward people for negative events: $r = -.04, p = .67$) and delusions. When these analyses were conducted for the At-Risk Group alone, stronger correlations were found between dysfunctional attitudes and delusions ($r = .49, p < .0001$), but again no significant relationships were observed between attributional style variables and overall delusional ideation (internal positive: $r = .20, p = .12$; external negative toward people: $r = -.04, p = .76$).

When correlating the newly created PDI subscales (Grandiose and Persecutory) with the IPSAQ subscales and the DAS in the full study sample, no significant relationships emerged between either PDI subscale and these cognitive variables. However, within the At-Risk Group, there were significant associations between Persecutory delusions and the IPSAQ internal attributions for positive events subscale ($r = .26, p = .04$) and between Persecutory delusions and the DAS score ($r = .34, p < .01$). There were no correlations between the Grandiose subscale and the cognitive variables. In both the total sample and the At-Risk Group analyses, there were no significant relationships between dysfunctional attitudes/negative self-schema and attributional style. See Tables 6a and 6b for Pearson correlations among relevant study variables.

To analyze the specific risk factors most predictive of delusional ideation in the At-Risk Group, a series of hierarchical regression equations were conducted with the

various risk factors (degree of genetic risk, OQ-45 score, age) used alone and in combination as predictor variables and PDI score used as the criterion variable. Total score on OQ-45 was entered first into the equation and was found to be significantly related to delusional ideation ($R^2 = .40, p < .0001$). Degree of genetic risk (reporting either a 1st, 2nd or 3rd degree relative with a psychotic disorder, coded as 3: highest degree of genetic risk, 2: medium degree of genetic risk, and 1: lowest degree of genetic risk, respectively) was entered second and was found to contribute significantly to the variance in PDI scores over and above OQ score alone ($R^2 \Delta = .06, p = .02$). Age was entered third into the equation and did not add any explained variance to the criterion ($R^2 \Delta = .00$, $p = .68$), nor did the interaction of degree of genetic risk and OQ score, which was entered fifth into the equation ($R^2 \Delta = .00, p = .65$), or the genetic risk by OQ score by age interaction, entered sixth ($R^2 \Delta = .00$, $p = .55$). See Table 7 for results of hierarchical regression analyses.

It appeared that OQ score was the strongest predictor of PDI scores, followed by degree of genetic risk, and the two variables together additively contributed to approximately 45% of the variance in delusions. From the results of this hierarchical regression equation, a "risk score" for each At-Risk subject was calculated using different combinations of OQ scores (e.g. greater than or equal to the 63-point cutoff = greater risk; less than or equal to the 63-point cutoff =less risk) and degree of genetic risk (e.g., 1st degree, 2nd degree, 3rd degree), such that having an OQ score equal to or exceeding 63 in combination with a 1st degree relative with psychosis equaled the "highest" level of risk with a "risk score" of six, and having an OQ score below 63 in combination with a

3rd degree relative equaled the "lowest" level of risk with a risk score of one. See Table 8 for more detailed calculations of the "risk score."

To test the hypothesis that the combination of certain risk factors and heightened cognitive vulnerability (conceptualized as dysfunctional attitudes/negative self-schema and dysfunctional attributional style) best predicted both general and specific types of delusional ideation, multiple regression analyses were calculated for the At-Risk Group. As a way to hone in on specific vulnerability variables in this group, and to create a more stringent test for risk, these analyses were conducted with data from the At-Risk Group only. Subjects' risk scores were used as predictors in conjunction with DAS scores and IPSAQ scores to predict PDI scores (used as the criterion variable) (Hypothesis 6). From the regression analysis utilizing risk score and DAS score as predictors, it was found that the two variables together predicted 24% of the variance in PDI scores ($p = .001$), and that there was no significant interaction among risk and DAS score ($R^2 \Delta = .00, p = .79$).

To examine the relationship between risk and attributional style in predicting delusions, subjects' risk scores and the relevant IPSAQ subscales (internal attributions for positive events, external attributions toward people for negative events) were entered into separate regression equations. When the risk score and internal attributions for positive events were entered together as predictors, they did not contribute significantly to the variance in total PDI scores over and above the risk score's contribution alone, which was 7.5% ($R^2 \Delta = .03, p = .18$), nor did the interaction of the two variables ($R^2 \Delta = .00$, $p = .80$).

When external attributions toward people for negative events and the risk score

were entered together, they did not contribute significantly to the variance in PDI over and above the risk score alone (R^2 Δ = .00, p =.77). Although there was no main effect for this IPSAQ subscale, when the interaction of the two variables was entered into the equation, the contributed variance in PDI scores jumped from 7.7% to 16% (R^2 Δ = .08, p = .02) which suggests that this type of attributional style has a moderating effect on the relationship between risk and delusional ideation. See Table 9 for detailed results of these regression equations.

In order to understand the relationship between risk for psychosis, cognitive vulnerability, and *specific* types of delusions, additional multiple regression equations with these same predictor variables and different criterion variables were conducted. The criterion variables (Persecutory delusions and Grandiose delusions) were the two subscales of the shortened 8-item PDI calculated on the basis of the confirmatory factor analysis and were entered separately as downstream variables for each analysis. In the present analyses, these delusion categories were utilized as unit weights, not as weights based on factor loadings. While the risk score alone was not significantly correlated with Persecutory delusions (r = .23, p = .07), the DAS in combination with the risk score predicted a significant amount of variance in Persecutory delusions (R^2 = .12, p = .04), with no interaction effect (R^2 Δ = .03, p = .20). There were no significant main effects and no interaction effect for the DAS and risk score in predicting Grandiose delusions.

When risk score and IPSAQ subscales regression equations were calculated to predict the two types of delusions, it was found that there was a trend toward significance for the combination of the internal attributions for positive events subscale and the risk

score ($R^2 = .11, p = .058$) in predicting Persecutory delusions, but that external attributions toward people for negative events (neither alone nor in conjunction with risk) did not contribute to this type of delusion. Examining Grandiose delusions, there was no significant predictive relationship between risk and internal attributions for positive events ($R^2 = .00, p = .51$). Although there were no main effects for risk or external attributions toward people for negative events alone or in combination, there was a highly significant interaction effect for these two variables in predicting Grandiose delusions, as the multiplicative interaction term predicted about 23% of the variance in Grandiosity ($R^2 \Delta = .22, p = .000$); this is suggestive of a "pure moderation" effect, as defined by Baron and Kenny (1986). See Table 10 for more detailed results of these multiple regression equations.

Subgroup analyses for the basic main analyses were conducted to examine gender differences, and whether there might be gender effects on the relationships among dysfunctional attitudes, attributional style, and delusional ideation variables. The pattern of correlations among the DAS, IPSAQ subscales, and the PDI remained essentially the same when data from males and females were analyzed separately within the total sample and within the At-Risk data alone, in that significant correlations between delusions and dysfunctional attitudes (DAS scores) of the same average magnitude emerged with both genders. Of note is that in the At-Risk all male group data, the correlation between these variables remained particularly strong ($r = .52, p = .02$), despite the small subgroup sample size of 19. See Tables 11a and 11b for gender-separated correlations of study variables.

Two-way ANOVA analyses revealed no significant gender (male vs. female) by group (At-Risk vs. Control) differences in DAS scores, F (1, 129) = 1.83, p = .31 or in overall PDI scores, F (1, 129) = 1.31, p = .26. Given that there were no significant differences between males and females on these variables or differential patterns of correlations, regression equations analyzing specific risk factors predictive of delusional ideation were not pursued.

Discussion

The results from this study regarding correlates of risk to psychosis are in keeping with similar lines of research illustrating the existence of elements of cognitive vulnerability (e.g. dysfunctional self-concept/negative self-schema) in individuals with schizophrenia and other psychotic disorders. They are also in keeping with the emerging literature on targeting at-risk individuals via a state-trait recruitment methodology.

Targeting At-Risk Individuals Via the "State-Trait" Methodology

As Yung and colleagues (1998) and Morrison and colleagues (2002) have determined that it is feasible to target members of a legitimately at-risk group and predict their transition to psychosis, findings from the present research suggest that it is also feasible to utilize "trait plus state" techniques (in this case age, and most robustly degree of genetic risk and general diffuse psychological symptoms) to predict the existence of low-level delusional ideation. In fact, when targeting these at-risk individuals and comparing them to young adults with similar demographic characteristics who do not

have these risk factors, there was a very strong difference between these two groups in number of delusions possessed. Not only does this finding support the notion that low-level delusional ideation may be more common than once thought (Peters, et al., 1996), it also presents the possibility that these low-level delusions may foreshadow development of more fine-tuned, potentially disturbing, and clinically-relevant beliefs in these young adults who have an elevated genetic predisposition for future psychosis. Furthermore, even with the modified "trait plus state" methodology utilized in the present study, important group differences are uncovered.

Interestingly, the strongest predictor of delusions (which served as the "outcome variable" for the purposes of this study) was general psychopathology. While the existence of diffuse symptomatology has been used as at-risk criteria in some studies (e.g., Yung, et al., 1998; Morrison, et al., 2002), it was surprising to find that this variable was a larger predictor than was degree of genetic risk, which, in the literature (particularly medical research) tends to emerge as the sine qua non of vulnerability factors for the development of schizophrenia (Gottesman, 1991).

Two arguments can be made against regarding general psychopathology (OQ score) as a legitimate predictor of delusions. First, as low-level delusional ideation was used as the downstream variable in this study, as opposed to full transition to psychosis, it can be argued that having subclinical abnormal beliefs is not really indicative of anything close to having full-blown psychosis, and that using diffuse symptoms to predict such beliefs is not very meaningful. However, as this was a "risk" study and not a "transition to psychosis" study, was aimed at more developmental, "upstream" analyses of correlates

of propensity for future psychosis, and utilized a sample of young adults who are on the cusp of being in the most at-risk age range for schizophrenia, it appears appropriate (and even more logical) to use low-level delusions as an outcome variable in place of some another more "diagnostic" criterion variable. Further, the presence or absence of delusions is often used as the defining factor in determining whether someone has made the transition to full psychosis and/or has a diagnosable psychotic condition (Larsen, McGlashan, & Moe, 1996) and is therefore an important criterion variable of interest, even though delusions did not mark this transition in the present sample.

Secondly, it may be argued that using diffuse psychopathology to predict delusional ideation is a circular analysis – predicting symptoms with symptoms. Several researchers involved in targeting individuals at-risk for psychosis conceptualize "state" vulnerability as, " [including] nonspecific symptoms such as anxiety; depressed mood; reduced drive, energy, and concentration; sleep disturbances; and behavioral changes such as deterioration in role functioning..." (McGorry, Yung, & Phillips, 2002). This suggests that these symptoms may be representative of several disorders and are not limited to any of the psychotic spectrum disorders, but should be used in conjunction with other traitlike indicators. The measure used in this study (the OQ) has 45 questions, none of which are specifically related to abnormal beliefs or delusions. Furthermore, the OQ does not just address basic symptoms, but also has several items that assess interpersonal interactions and more general social behaviors, which compose two of the three OQ subscales (Interpersonal Relations and Social Roles); it is very close to the conceptualization of the "at-risk mental state" defined by other researchers (e.g.,

McGorry & Singh, 1995; Yung, et al., 1996; Yung et al., 1998). Thus, it appears that general psychopathology, measured as a combination of diffuse symptoms, dysfunctional interpersonal relationships, and instances of abnormal social behavior, can be conceptualized as a legitimate and useful predictor of delusional ideation. Therefore, utilizing the existence of general psychopathology to assess risk to psychosis not only makes conceptual sense, but when it is used in conjunction with additional "trait" characteristics (e.g., age and genetic risk), has demonstrated effectiveness in accurately aiding in the assessment of the transition to full psychosis. The combination of close genetic relatedness and this psychosocial measure of diffuse pathology as strong predictors in the current study further supports the diathesis-stress model of the development of psychological disorders, and in this case, of delusional beliefs that may be a component of burgeoning full-blown psychosis.

Results from the present research point to the feasibility of creating a "risk score" to quantify somewhat accurately and evaluate an individual's degree of vulnerability to psychosis (or in this case, presence of general and specific facets of delusional ideation). Admittedly, the method used to calculate the risk score in this study was somewhat rudimentary. The dichotomization of scores from the general psychopathology questionnaire (using a cut-off score to determine "symptomatic" or "not symptomatic"), and the assumption that genetic risk increases via equal linear intervals depending on degree of genetic family member closeness, together are likely to have detracted somewhat from the richness of the data and its interpretability. Future "at-risk" research should attempt to fine-tune and modify the computation and accuracy of such a risk

score, as the development of a "quick and dirty" way to assess vulnerability to psychosis could be of great advantage in pinpointing individuals and providing early intervention. Nevertheless, the present study's attempt to quantify risk does have some utility in predicting who will exhibit delusions and who will not, especially within this rather small sample, and can be regarded as a good first endeavor.

Aspects of Cognitive Vulnerability in the At-Risk Sample

As predicted, individuals in the At-Risk Group had more pronounced aspects of disordered self-concept as compared to their Control Group counterparts. Although some recent studies have examined negative self-schema and dysfunctional attitudes in individuals with psychosis (e.g., Alford & Beck, 1994; Freeman, Garety, & Kuipers, 2001) and have found this maladaptive cognitive style to be related to (and even theorized as a pathway to) delusions (Beck and Rector, 2002; Bentall, 2001; Bentall, Kinderman, & Kaney, 1994; Martin & Penn, 2002), there has been only one study to examine overall disordered self-concept in individuals at risk for psychosis (Morrison et al., 2002). Thus, the finding that negative cognitions occur to a greater degree in individuals who have a genetic predisposition for psychosis than in those who do not is a potentially important one. This suggests that the cognitive distortions about self, others, and the world that may be the cause of delusions likely emerge early on in one's development into adulthood; they seem to become fairly solidified by the time one reaches the stage of young adulthood, which also happens to be the apex of the age range for initial psychotic breakdown.

The interpretations of the findings in this study related to attributional style are somewhat more complex. As previously mentioned, research has suggested that while depressed individuals tend to attribute negative events to themselves and positive events to circumstances or to outside influences, individuals with delusions have the opposite pattern: they are more likely to believe that positive events are caused by their own doing and that negative events are caused by the actions of others (Candido & Romney, 1990; Kaney & Bentall, 1989; Lyon, Kaney, & Bentall, 1994). Overall, in the present research, the particular attributional style discussed in the literature as existing in individuals with full-blown psychosis was not found to be related to overall delusional ideation or to negative self-schema within the At-Risk Group. Contrary to prediction, the At-Risk Group did not attribute the causes of positive events to themselves more often than the Control Group, nor did they attribute negative events to others more often. In fact, both groups demonstrated a bias toward attributing the causes of positive events to themselves, the "self-serving bias" (Kaney & Bentall, 1992). While this is a somewhat surprising finding, it is of note the aforementioned theories about the attributional style of people with delusions developed from studies examining these biases in individuals who had diagnosable psychotic conditions, and the present study is the only one to date that directly measured attributional style in putatively *at-risk* individuals. Therefore, it is likely that the patterns of attributional biases are different between these two groups. It is possible that for many young adults at risk, while more diffuse disordered self-concept is related to delusions, a solidified abnormal bias about the cause of events, which has been said to serve as protection against low self-esteem (Beck & Rector, 2002; Bentall, 2001;

Bentall, Kinderman, & Kaney, 1994; Lyon, Kaney, & Bentall, 1994) has not yet developed. It may be the case that a true "style" (e.g. a self-serving bias) emerges later in one's developmental trajectory and/or in one's psychotic symptom trajectory in those who go on to develop diagnosable psychosis.

Although in general there was no significant indication that At-Risk Group individuals displayed an attributional style similar to patients with psychosis, it was the case that a particular attributional style, in conjunction with other risk factors, played a role in the prediction of general and specific delusions, as discussed below.

A Basic Model of Risk for Psychosis

In creating a model to understand better the relationship between genetic and psychosocial risk for psychosis and cognitive vulnerability in the prediction of delusions, it appears that it is generally some combination of risk and disordered thoughts about oneself and the causes of life events that actually forecast the existence of aberrant, distressing beliefs. Interestingly, it is the existence of a particular attributional style (believing that negative events in one's life are caused by the acts of "malevolent others") that has an exacerbating detrimental effect on being at risk for psychosis and may create a greater amount of delusional beliefs. If an individual has a genetic predisposition to psychosis and exhibits diffuse maladaptive psychological symptoms, it is the presence of this persecutory "style" of thinking about life events that really interacts with the genetic and symptom risk factors to create distressing beliefs that likely influence one's behavior. Thus, it is not simply genetic factors, or even the combination of genetic and psychosocial risk, that truly predicts having such potentially upsetting beliefs; it is in fact

a particular view of causation about positive and negative events in the world, in conjunction with risk, that plays a substantial role in the determination of which "at-risk" individuals develop potentially disturbing delusions.

In relation to the psychosis literature on common types of delusion themes (persecutory and grandiose) in individuals with full-blown ideation (Appelbaum, Clark Robbins, & Roth, 1999; Beck & Rector, 2002; Junginger, Barker, & Coe, 1992; Lucas, Sainsbury, & Collins, 1962; Sinha & Chaturvedi, 1989), results from this study suggest that even in subclinical individuals who may be at risk for psychosis, the themes of delusions may mirror those in patients with diagnosable psychotic conditions. This finding not only confirms the existence of elements of psychotic symptoms in at-risk individuals, but also lends support to the idea that delusion types do not manifest themselves in an arbitrary manner, as some would hypothesize (Berrios, 1991), but in a way that can actually be understood in terms of more specific concerns, fears, and ideas about oneself, one's world, and one's future (Beck & Rector, 2002; Chapman & Chapman, 1988; Harrow & Prosen, 1978; Harrow, Rattenbury, & Stoll, 1988). Findings from the present research support this idea of the individual "meaningfulness" of particular delusions, as it was found that the additive combination of general dysfunctional beliefs about oneself and other risk factors contribute greatly to the participants' degree of paranoid or persecutory delusions. In addition, grandiose delusions were most prominent in those at-risk individuals who specifically blamed others for negative events in their lives. While this finding may seem somewhat counterintuitive, in that it may be expected that those with the tendency to excessively

blame others for bad happenings would have persecutory delusions and not grandiose ones, it is possible that this blaming of others serves to bolster the individual's self-confidence. If one can make others the scapegoats, it follows that one can let oneself "off the hook;" this may create an inflated sense of worth or ability that may grow in magnitude into a delusional belief. This "compensatory" view of delusions is consistent with the work of Beck and Rector (2002), Bentall and colleagues (1994), and Martin and Penn (2002).

Limitations of the Present Research

While this study sheds light on some of the important, previously unresearched correlates of vulnerability to psychosis, it has some limitations. Obviously, an unfortunate circumstance during the data collection process was the omission of the question involving the participants' own history of psychosis. Coupled with the low subject response-to-be-recontacted rate, this reduces to some degree the "pureness" of both the At-Risk and Control groups. As a result, it is theoretically possible that some members of both groups have had a prior psychotic episode. However, as described previously, the statistical likelihood of individuals in this age range having already had a previous psychotic episode is quite small. Undoubtedly, future researchers assessing risk should be certain to attend to participants' own past psychiatric history in order to distinguish more clearly among at risk, remitted, and currently ill groups.

Another potential limitation of the present research is that the entire sample was composed only of undergraduate introductory psychology students, which can be

regarded as an "analogue" sample, not a "clinical" sample, and therefore perhaps not a legitimately "at risk" sample. Despite the homogeneity of this sample, this group should still be regarded as representative of a true "vulnerable" sample for several reasons. First, these subjects are certainly at elevated genetic risk, as all of them were clear in their endorsement of having a first, second, or third degree biological relative with a diagnosed psychotic disorder. Their age range (broadly, between 18 and 36, and specifically, with an average age of 21) puts them squarely in the documented high-risk zone for onset of an initial psychotic break (APA, 2000). These two criteria (genetics and age) are two of the three used in past research that prospectively followed similar individuals, approximately 22-40% of whom had diagnosable psychotic symptoms six months after being targeted (Morrison, et al., 2002; Yung, et al., 1998). A third criterion for at-risk participants used in these past risk studies was the existence of diffuse psychological symptoms not necessarily related to psychosis. In the present study, a high percentage of subjects in the At-Risk Group had general psychopathology symptom levels that fell into the clinical or subclinical range (Lambert & Burlingame, 1996), and many had mildly elevated scores on the OQ. With such high levels of endorsed symptoms in so many of the At-Risk Group participants, it is likely that many of them were fairly distressed, and as a result, it is also fairly likely that many of them had already sought professional help for their distress and/or had some current involvement in mental health services. Thus, these individuals may be at more imminent risk for psychosis than is assumed. While a more heterogeneous participant recruitment strategy would have been ideal, the validity of the present study's At-Risk Group as a true at-risk group should not be dismissed.

Early Intervention Implications and Future Directions

With regard to clinical intervention, results from this study point to the need and the potential utility in creating a comprehensive program that both targets and provides mental health services to at-risk individuals. The literature suggests that there is a recent shift in focus regarding treatment of psychosis, as more and more researchers (and clinicians) are aiming to attend to the prevention of psychosis before it becomes treatment-refractory.

This emphasis is reflected in the recent development of the International Prodromal Research Network (IPRN; Cornblatt, et al., 2003), the CBT randomized controlled trial with at-risk individuals conducted by Morrison and colleagues (2002), and the work McGorry, Yung, Phillips, and others in Australia, who have developed a research and treatment program for young-adult prepsychotic individuals. Their clinic provides interventions aimed at reducing patients' symptoms and preventing the worsening of these symptoms into acute psychosis through the use of Cognitive-Behavioral therapy principles. They utilize other collaborative techniques as well (such as the therapist providing case management services to help with housing, benefits, work placement) in order to help patients reduce the impact of stressors that can exacerbate symptoms and "push" one into full-blown psychosis (McGorry, Yung, & Phillips, 2003).

While prevention and early intervention of schizophrenia have received a great deal of attention recently, there is not much data to date regarding what particular types of psychotherapeutic interventions are most useful to prevent the transition to psychosis. It is generally agreed upon that Cognitive and Cognitive-Behavioral interventions make

intuitive sense (French, Morrison, Walford, Knight, & Bentall, 2003; McGorry, Yung, & Phillips, 2003; Morrison, et al., 2002), given the affective disturbances and distorted thinking that has been found to be present in these at-risk individuals. (Not to be confused with elements of Cognitive or Cognitive-Behavioral psychotherapy aimed at changing maladaptive cognitions, another type of intervention, called Cognitive Remediation, deals with the "retraining" of low-level memory, attention, and information processing deficits in patient populations; Green, 2001). However, specific CT or CBT interventions have not been officially targeted and tested with these at-risk groups in a true experimental fashion.

Results from this study help to elucidate specific vulnerability factors that put someone at risk to develop low-level delusional ideation. The findings indicate that a combination of genetic risk *and* psychosocial/diffuse general psychopathology are most predictive of delusions and therefore call for mental health workers and other health care providers to monitor more closely individuals who demonstrate these particular characteristics. The finding that disordered self-concept and a specific attributional style, in conjunction with these risk factors, predicts a high degree of potentially distressing delusions, may be a start in isolating the particular facets of cognitive vulnerability factors that afflict at-risk individuals. Results from this research could be expanded upon and used to create more specific targeted interventions based in CT and CBT in order to modify negative thoughts about self and maladaptive biases in the attributions of causes of life events. A reduction in negative self-schema and dysfunctional attributional style

could in turn reduce levels of delusional ideation, or prevent these cognitions from increasing to the point where full-blown delusions (and/or other psychotic symptoms) develop, and more serious, continued treatment becomes necessary.

Given both the limitations of this study and the important knowledge gained, future research should attempt to replicate these findings utilizing larger, more diverse samples. These samples would ideally include heterogeneous groups of individuals within the appropriate age range from varied socioeconomic, ethnic, and geographic groups. A greater mixture of individuals recruited from different sources (e.g. university clinics and counseling centers, community mental health centers, private practitioner referrals) could potentially provide clearer, more useful, more generalizable, and more definitive data about genetic, psychosocial, and cognitive predictors of risk for psychosis. Such data could lead to more specific and efficacious psychotherapeutic interventions for this population.

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Table 1

Cognitive pathway to the formation of a grandiose delusion (taken from Beck and Rector, 2002)

1. *Underlying negative self-image.* Socially awkward, alone, and disliked (although intellectually capable).
2. *Extended fantasy/daydream.* "Books were my only friends: -- fantasizes about becoming a famous writer, receiving great praise and international recognition.
3. *Critical incident.* Mounting stress with school; difficulties completing school assignments; subsequent failure and shame.
4. *Activities of fantasy world.* Patient begins to see self as the poet laureate.
5. *Egocentric bias.* Spends much of her time jotting down ideas "to be later published" and sits in public library throughout the day.
6. *Confirmatory bias.* Discounts/ignores information that challenges the belief.
7. *"Connecting the dots."* Patient begins to live as if she is the poet laureate, with the expectation of special recognition and privileges.

Table 2

Cognitive pathway to the formation of a persecutory delusion (taken from Beck and Rector, 2002)

1. *Fear of retaliation.* For reporting the drug dealers.
2. *Elaboration of conspiracy theory.* After the reports of the police involvement in the drug dealing.
3. *Egocentric bias.* Selective focus on police to test his theory. The patient had a low threshold for danger signals, which gradually accumulated and further energized his persecutory focus.
4. *Confirmatory bias.* Patient assimilated examples of the police following him and ignored or discounted incidents that did not fit.
5. *Enhancement of similarity of "persecutors."* As the patient developed a mental representation of the "conspirators," an increasing number of individuals seemed to fit this image.
6. *Capitalizing on coincidence.* The patient saw the same individual initially when shopping at a grocery store and later when attending a reception at his church. This coincidence confirmed that he was being followed.
7. *"Connecting the dots."* Putting together all this evidence, the patient became convinced that he was being observed by the police who presumably intended to harass and harm him.

Table 3

Demographic data for At-Risk and Control Group subjects, n = 133

Demographic Variables	At-Risk Group	Control Group
n	61	72
Mean age	21.31 (3.79)	20.74 (3.36)
Males	19 (31.1%)	25 (34.7%)
Females	42 (68.9%)	47 (65.3%)
Never Married/Single	50 (82.0%)	67 (93.1%)
Ever Married	11 (18.0%)	5 (6.9%)
Caucasian	55 (91.7%)	63 (88.7%)
Native American/Indian	1 (1.7%)	2 (2.8%)
African American/Black	0	1 (1.4%)
Hispanic/Latino	0	1 (1.4%)
Other	4 (6.7%)	4 (5.6%)

Table 4

Descriptive statistics and alpha reliability coefficients for study measures and relevant subscales

Measure	Mean	SD	α
Outcome Questionnaire (OQ-45.2)	50.06	21.05	.93
Dysfunctional Attitudes Scale (DAS)	111.67	31.77	.92
Internal, Personal, Situational Attributions Questionnaire (IPSAQ) Internal attributions for positive events	8.05	2.83	.74
IPSAQ External personal attributions for negative events	5.95	2.79	.80
Peters et al. Delusions Inventory original scale (PDI)	62.08	47.83	.89
PDI new 8-item shortened scale	17.80	13.59	.67
PDI new Grandiose subscale	10.50	9.35	.56
PDI new Persecutory subscale	7.31	7.87	.72

Table 5

Summary fit statistics for competing 2-factor models of the modified PDI, n = 61

Model	X^2	df	$X^{2/df}$	GFI	AGFI	RMSEA	90% CI
Independence	125.578	28	4.49	0.65	0.54	0.24	0.20 – 0.29
Orthogonal	37.13	20	1.86	0.86	0.75	0.12	0.06 – 0.18
Oblique	36.63	19	1.93	0.86	0.74	0.12	0.06 – 0.18

Note: $X^{2/df} = X^2$ to df ratio; GFI = goodness of fit index; AGFI = adjusted goodness of fit index; RMSEA = root mean square error of approximation.

Figure 1

Two-factor oblique model of shortened Peters et al. Delusions Inventory with newly-developed Persecutory and Grandiose subscales. n = 61.

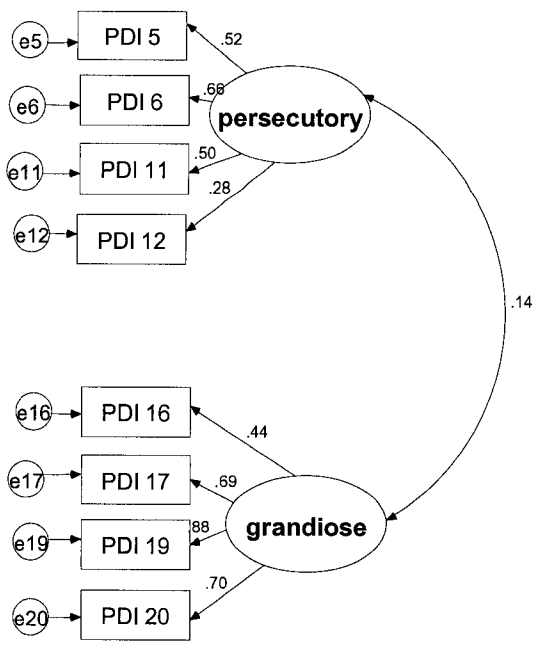

Table 6a.

Correlations among cognitive vulnerability and delusions measures for the total study sample, n = 133

Measure	DAS	Internal Positive	External Negative	PDI total	Grandiose	Persecutory
DAS	--					
Internal Positive	.12	--				
External Negative	-.05	.10	--			
PDI total	.39**	.04	-.04	--		
Grandiose	.11	.02	-.12	.68**	--	
Persecutory	.30**	.09	-.06	.71**	.24**	--

Note: $**p < .01$. DAS = Dysfunctional Attitudes Scale; Internal Positive = IPSAQ internal attributions for positive events subscale; External Negative = IPSAQ external attributions for negative events towards people subscale; PDI total = Peters et al. Delusions Inventory original scale total score; Grandiose = shortened PDI grandiose subscale; Persecutory = shortened PDI persecutory subscale.

Table 6b

Correlations among cognitive vulnerability and delusions measures within the At-Risk sample, n = 61

Measure	DAS	Internal Positive	External Negative	PDI total	Grandiose	Persecutory
DAS	--					
Internal Positive	.20	--				
External Negative	-.14	.09	--			
PDI total	.49**	.20	-.04	--		
Grandiose	.10	.09	-.13	.63**	--	
Persecutory	.34**	.26*	-.07	.70**		--

Note: $*p < .05$. $**p < .01$. DAS = Dysfunctional Attitudes Scale; Internal Positive = IPSAQ internal attributions for positive events subscale; External Negative = IPSAQ external attributions for negative events towards people subscale; PDI total = Peters et al. Delusions Inventory original scale total score; Grandiose = shortened PDI grandiose subscale; Persecutory = shortened PDI persecutory subscale.

Table 7

Hierarchical regression analysis summary for determining risk variables that best predict overall delusional ideation, n = 61.

Variables/Model	R^2	ΔR^2	β	p
1. OQ score	.396	.396	.63	.0001
2. OQ score	.451	.055	.63	.02
Genetic Risk			-.24	
3. OQ score	.453	.002	.64	.68
Genetic Risk			-.22	
Age			-.04	
4. OQ score X Genetic Risk Interaction	.455	.002	-.05	.65
5. OQ score X Genetic Risk X Age Interaction	.458	.004	.07	.55

Note: OQ = Outcome Questionnaire (general psychopathology measure); Genetic Risk = degree of risk based on family member genetic closeness (1st, 2nd, or 3rd degree); Age = between 18 and 36.

Table 8

Criteria used to determine "risk score" for use in multiple regression analyses (Hypothesis 6), calculated using data from hierarchical regression analysis (Hypothesis 5, Table 7)

OQ Score	Genetic Risk (1^{st}, 2^{nd}, or 3^{rd} degree relative)	Risk Score Assigned
≥63	1^{st} degree relative	6
≥63	2^{nd} degree relative	5
≥63	3^{rd} degree relative	4
< 63	1^{st} degree relative	3
< 63	2^{nd} degree relative	2
< 63	3^{rd} degree relative	1

Note: OQ Score determined by Lambert & Burlingame (1996) OQ manual clinical cutoff score; Genetic Risk: 1^{st} degree = parent, child, sibling, 2^{nd} degree = aunt/uncle, grandparent, 3^{rd} degree = cousin, great-aunt/uncle, great-grandparent.

Table 9

Multiple regression analysis summary for risk score and cognitive variables predicting general delusional ideation, n = 61.

Table 9a

Analysis for risk score and level of dysfunctional attitudes (negative self-schema) in predicting delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.075	.075	.28	.03
2. DAS Total Score	.243	.167	.50	.001
3. Risk Score X DAS Total Score Interaction	.244	.001	-.04	.79

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); DAS Total Score = Dysfunctional Attitudes Scale.

Table 9b

Analysis for risk score and attributional style (internal attributions for positive events) in predicting delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.075	.075	.28	.03
2. IPSAQ internal positive attributions	.104	.029	.17	.18
3. Risk Score X IPSAQ internal positive attributions Interaction	.105	.001	-.03	.80

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ internal positive attributions = Internal, Personal, & Situational Attribution Questionnaire internal attributions for positive events subscale.

Table 9c

Analysis for risk score and attributional style (external attributions towards people for negative events) in predicting delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.075	.075	.28	.03
2. IPSAQ external negative attributions	.077	.001	-.04	.77
3. Risk Score X IPSAQ external attributions Interaction	.160	.084	-.31	.02

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ external negative attributions = Internal, Personal, & Situational Attribution Questionnaire external attributions towards people for negative events subscale.

Table 10

Multiple regression analysis summary for risk score and cognitive variables predicting specific types of delusional ideation (persecutory and grandiose), n = 61.

Table 10a

Analysis for risk score and level of dysfunctional attitudes (negative self-schema) in predicting persecutory delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.053	.053	.23	.07
2. DAS Total Score	.119	.065	.31	.04
3. Risk Score X DAS Total Score Interaction	.144	.025	.18	.20

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); DAS Total Score = Dysfunctional Attitudes Scale.

Table 10b

Analysis for risk score and level of dysfunctional attitudes (negative self-schema) in predicting grandiose delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.000	.000	.00	.99
2. DAS Total Score	.014	.014	.14	.37
3. Risk Score X DAS Total Score Interaction	.059	.045	-.25	.10

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); DAS Total Score = Dysfunctional Attitudes Scale.

Table 10c

Analysis for risk score and attributional style (internal attributions for positive events) in predicting persecutory delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.053	.053	.23	.07
2. IPSAQ internal positive attributions	.111	.058	.24	.06
3. Risk Score X IPSAQ internal positive attributions Interaction	.111	.000	-.02	.90

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ internal positive attributions = Internal, Personal, & Situational Attribution Questionnaire internal attributions for positive events subscale.

Table 10d

Analysis for risk score and attributional style (internal positive attributions for positive events) in predicting grandiose delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.000	.000	.00	.99
2. IPSAQ internal positive attributions	.007	.007	.08	.51
3. Risk Score X IPSAQ internal positive attributions Interaction	.041	.033	-.19	.16

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ internal positive attributions = Internal, Personal, & Situational Attribution Questionnaire internal attributions for positive events subscale.

Table 10e

Analysis for risk score and attributional style (external attributions towards people for negative events) in predicting persecutory delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.053	.053	.23	.07
2. IPSAQ external negative attributions	.058	.005	-.07	.59
3. Risk Score X IPSAQ external attributions Interaction	.059	.001	-.04	.80

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ external negative attributions = Internal, Personal, & Situational Attribution Questionnaire external attributions towards people for negative events subscale.

Table 10f

Analysis for risk score and attributional style (external attributions towards people for negative events) in predicting grandiose delusions.

Variables/Model	R^2	ΔR^2	β	p
1. Risk Score	.000	.000	.000	.99
2. IPSAQ external negative attributions	.016	.016	-.13	.36
3. Risk Score X IPSAQ external attributions Interaction	.233	.217	-.50	.000

Note: Risk Score = weighted combination of OQ score and genetic risk (see Table 8); IPSAQ external negative attributions = Internal, Personal, & Situational Attribution Questionnaire external attributions towards people for negative events subscale.

Table 11a

Correlations among cognitive vulnerability and delusions measures for males in the total study sample, n = 44

Measure	DAS	Internal Positive	External Negative	PDI total	Grandiose	Persecutory
DAS	--					
Internal Positive	.22	--				
External Negative	-.11	-.02	--			
PDI total	.47**	.07	-.13	--		
Grandiose	.24	-.07	-.17	.64**	--	
Persecutory	.30	.26	-.13	.69**	.25**	--

Note: $^{**}p < .01$. DAS = Dysfunctional Attitudes Scale; Internal Positive = IPSAQ internal attributions for positive events subscale; External Negative = IPSAQ external attributions for negative events towards people subscale; PDI total = Peters et al. Delusions Inventory original scale total score; Grandiose = shortened PDI grandiose subscale; Persecutory = shortened PDI persecutory subscale.

Table 11b

Correlations among cognitive vulnerability and delusions measures for females in the total study sample, n = 89

Measure	DAS	Internal Positive	External Negative	PDI total	Grandiose	Persecutory
DAS	--					
Internal Positive	.06	--				
External Negative	.00	.15	--			
PDI total	.30**	.03	.04	--		
Grandiose	.01	.06	-.07	.70**	--	
Persecutory	.30**	-.02	.01	.72**	.22*	--

Note: $^{**}p < .01$, $^{*}p < .05$. DAS = Dysfunctional Attitudes Scale; Internal Positive = IPSAQ internal attributions for positive events subscale; External Negative = IPSAQ external attributions for negative events towards people subscale; PDI total = Peters et al. Delusions Inventory original scale total score; Grandiose = shortened PDI grandiose subscale; Persecutory = shortened PDI persecutory subscale.

Family Mental Health History Questionnaire

Please carefully read the following questions and answer to the best of your ability. Thank you in advance for your participation. All of these questions apply to a BIOLOGICAL ("blood") relative, NOT to yourself.

*Your age _____

1. Sometimes people behave in a way that seems abnormal or even "psychotic" or "crazy" to others. Sometimes people observe these behaviors in friends or family members, or are told by other people that their friends or family members did or said something extremely bizarre or seemingly abnormal, "psychotic" or "crazy."

Have you ever observed (or been told by another person) that a biological relative of yours fit the above description?

____ yes

____ no

If so, what relation is that person (or people, if more than one) to you? (check all that apply)

Parent ____ Aunt/Uncle ____ Other, please specify _____

Child ____ Grandparent ____

Sibling ____ Niece/Nephew ____

2. To your knowledge, have you ever noticed or been told by another person that a biological relative of yours said or did any of the following: (check all that apply)

____ Mentioned that they felt that others were talking about them or taking special notice of them?

____ Mentioned that they felt that they were receiving special messages from the TV, radio, or newspaper, or from the way things were arranged around them?

____ Mentioned that they felt that they were especially important in some way, or that they had the power to do things that other people couldn't do?

____ Mentioned that they felt that someone (or several people, or an important group) was going out of his/her way to give your relative a hard time, or try to hurt them?

____ Mentioned that they felt that something was wrong with them physically, even though their doctor said nothing was wrong?

____ Mentioned that they felt that something strange was happening to parts of their body?

____ Mentioned that they felt that they had committed a crime or done something terrible for which they should be punished?

____ Mentioned that they felt that other people could read their mind?

____ Mentioned that they felt that someone or something outside themselves was controlling their thoughts or actions against their will?

____ Mentioned that they felt that certain thoughts that were not their own were being put into their head?

____ Mentioned that they felt that thoughts were being taken out of their head?

____ Mentioned that they felt that thoughts were being broadcasted out loud so that other people could actually hear what they were thinking?

____ Mentioned that they heard things sometimes (or often) that other people couldn't hear, such as noises or the voices of people whispering or talking?

____ Mentioned that they had visions or saw things that other people couldn't see (in a way that others regarded as a problem)?

____ Mentioned that they had strange sensations in their body or on their skin?

____ Mentioned that they could smell things that other people couldn't smell?

____ Did you yourself see them appear to be talking to themselves out loud or seem like they were having a conversation or an argument with someone else, although no one else was really there?

____ Did you yourself notice that they appeared to see or hear or smell something that other people couldn't see or hear or smell?

If you checked any items in the previous list:

What relation is that person (or people, if more than one) to you?
(This can be the same person or people that you answered about in question #1) (check all that apply)

Parent ____ Aunt/Uncle ____ Other, please specify _____

Child ____ Grandparent ____

Sibling ____ Niece/Nephew ____

3. Sometimes the above "abnormal" feelings, thoughts and actions take place when people are under the influence of alcohol, drugs, or other prescription medication. To your knowledge, did these things occur at times OTHER than when your relative (or relatives) was drinking, using drugs, or taking medication?

____ yes, they took place when he/she was <u>not</u> under the influence of any substances that could have caused these behaviors.

____ no, they <u>only</u> took place when he/she <u>was</u> under the influence of a substance that could have caused these behaviors.

4. To your knowledge, have <u>any</u> of your biological relatives <u>ever</u> had (or currently have) a psychological diagnosis of any of the following disorders:

Schizophrenia _____ Psychosis, not otherwise specified _____

Schizoaffective Disorder _____ Delusional Disorder _____

Schizotypal Personality Disorder _____ Schizophreniform Disorder _____

*If you checked any items in #4:

What relation is that person (or people, if more than one) to you?
(This can be the same person or people that you answered about in question #1 and/or #2) (check all that apply)

Parent ____ Aunt/Uncle ____ Other, please specify _____

Child ____ Grandparent ____

Sibling ____ Niece/Nephew ____

And one last question...

5. Would you be willing to be contacted in the next few weeks to a month or so in order to come and answer more questions? Please note that all responses at this second meeting will be completely confidential (as they are today) AND your name will NOT appear on any documents. Psyc 100 credits will be offered, or if you already have all of your necessary credits, another form of compensation for your time will be provided.

_____ yes

_____ no

6. If "yes" to the previous question, please GO BACK TO THE BOTTOM OF YOUR CONSENT FORM and provide the requested contact information so we can get in touch with you. Please be sure to complete <u>all</u> of the information if possible. Note that all of the contact information will be known <u>only</u> to the research team for this study, will be kept in a locked cabinet, and will be destroyed after this study ends.

Now please flip back to your consent form and fill in the contact information if you are willing to be recontacted. Thank you!

Again, thank you for your participation today!!!

Men and Women who have a relative with schizophrenia or another psychotic disorder are needed to participate in research

Adults between the ages of 18 and 36 who have a *blood* relative who may have any of the following diagnoses:

- schizophrenia
- schizoaffective disorder
- schizophreniform disorder
- schizotypal personality disorder
- delusional disorder
- psychotic disorder, not otherwise specified

*please note that even if you are unsure about your relative's exact diagnosis, you will likely still qualify for the study.

- **Participation is completely confidential and takes only 30-45 minutes.**
- **Participants will receive $10 in appreciation of their contribution.**

For more information or to participate, please call and leave a message at:

243-6530
Jennifer Gottlieb, M.A.
University of Montana
Department of Psychology

Relative with Psychotic Disorder Rsh Project - $10 243-6530

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