8-2017

Exploring a multifactorial, clinical model of thought disorder : application of a dimensional, transdiagnostic approach.

Mara Ann Hart
University of Louisville

EXPLORING A MULTIFACTORIAL, CLINICAL MODEL OF THOUGHT DISORDER: APPLICATION OF A DIMENSIONAL, TRANSDIAGNOSTIC APPROACH

By

Mara Ann Hart
B.A., Loyola College in Maryland, 2006
M.S., Loyola College in Maryland, 2009

A Dissertation
Submitted to the Faculty of the
College of Arts and Sciences of the University of Louisville
In Partial Fulfillment of the Requirements
For the Degree of

Doctor of Philosophy
in Clinical Psychology

Department of Psychological and Brain Sciences
University of Louisville
Louisville, KY

August 2017

EXPLORING A MULTIFACTORIAL, CLINICAL MODEL OF THOUGHT DISORDER: APPLICATION OF A DIMENSIONAL, TRANSDIAGNOSTIC APPROACH

By

Mara Ann Hart
B.A., Loyola College in Maryland, 2006
M.S., Loyola College in Maryland, 2009

A Dissertation Approved on:

July 29, 2016

by the Following Dissertation Committee

Dissertation Director
Richard Lewine

Benjamin Mast

Janet Woodruff-Borden

Suzanne Meeks

Tracy Eells

I cannot experience your experience. You cannot experience my experience. We are both invisible men.

– R.D. Laing, The Politics of Experience

ACKNOWLEDGEMENTS

I was introduced to thought disorder on my first day of graduate school; so began a five-year journey through which this dissertation evolved. Many people provided help and support along the way. Above all, I am grateful for Rich Lewine, an incredible mentor, researcher, and clinician, who helped me see that grappling with complexity is more important than searching for answers.

I am deeply grateful for Debbie Levy, who shared this remarkable dataset with me, without which, this dissertation would not have been possible. Thank you to Morgan Martin, who helped me code thousands of lines of data… twice, somehow staying interested and enthusiastic throughout the process. And to my lab mates, Rachel and Cat, who were there from the beginning and made this journey a whole lot more fun.

I would also like to thank to mom and grandma, my cheerleaders, who still can't quite figure out what this dissertation is about, but have had unyielding interest and confidence in me nonetheless. And to Brian and Olive who have been limitlessly patient and supportive.

ABSTRACT

EXPLORING A MULTIFACTORIAL, CLINICAL MODEL OF THOUGHT DISORDER: APPLICATION OF A DIMENSIONAL, TRANSDIAGNOSTIC APPROACH

Mara A. Hart

July 29, 2016

Background: Bleuler saw thought disorder as the core defining feature of psychotic phenomena, reflective of the "splitting of the psychic functions" that occurred when, in the process of thinking, one's ideas and feelings disconnect, becoming fragmented and competing functions. Unfortunately, interest in thought disorder as the conceptual core of psychosis was lost with rise of the modern DSM system, paralleling the shift towards a more simplistic, categorical way of defining psychiatric disorders.

Aims: This study examined thought disorder from a dimensional perspective, with the aim of disentangling qualitative heterogeneity and diverse sources of influence. Analyses were based on a large, transdiagnostic sample (n = 322), including individuals diagnosed with schizophrenia, schizoaffective disorder, and bipolar disorder. Structural equation modeling was used to estimate the unique and combined effects of family psychiatric history, age-at-onset, affective state, and sex on two dimensions of thought disorder, namely idiosyncratic thinking and combinatory thinking. We also explored the utility of categorical (i.e. DSM) diagnosis, by estimating the relative proportion of variance it accounted for within the model.

Results: The overall model accounted for 11% of variance in idiosyncratic thinking and 3% of the variance in combinatory thinking. Negative affect was the strongest predictor of idiosyncratic thinking (r = .27), although this effect was significantly more robust in those with a family history of psychosis (r = .37) compared to those without (r = .02). DSM diagnosis was a significant predictor of IV, explaining 7% of unique variance when entered into the full model compared to 9% of the variance when estimated independently, which suggests that the portion of variance explained by diagnosis was largely independent of other predictors in the model.

Discussion: The pattern of associations among family psychiatric history, age-at-onset, and negative affect that predicted idiosyncratic thinking are suggestive of a developmental process. This hypothesis is explored in the context of previous research. The broad implications of this research on the classification and study of psychosis is also discussed.

TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Thought is a complex and uniquely-human phenomenon, through which our understanding of self, world, and reality is constructed. Connected to our capacity for language, thought is critical to communication and plays an important role in how we understand others and make our own experiences known. Said simply, "the use of words reflects [the] organization of the inner world of thought" (Lidz, 1973). Breakdowns in the thought system (e.g., how one perceives, interprets, structures, and responds to information) is intrinsically linked to difficulties with psychological wellbeing and the ability to function adaptively in the world. At its extreme, these disturbances are the core of psychotic experiences. In fact, disturbances in thought have been central to the conceptualization of psychosis since Emil Kraepelin introduced the first formal diagnostic construct, dementia praecox, which would later be reformulated as "schizophrenia" by his successor, Eugen Bleuler.

Kraepelin's application of a disease model to the classification of psychological disturbances had a profound and enduring impact on the conceptualization and study of psychosis. He defined dementia praecox as a dementing disease, characterized by a progressive deterioration of mental functions beginning in early life. He viewed language as an important marker of cognitive processes, describing "derailments" and "incoherence" in patients' speech as reflective of underlying thought disturbances. Kraepelin acknowledged the clinical variability of dementia praecox, noting that the "disease picture appears so varied that upon superficial observation the fundamental

symptoms are not recognizable" (Adams & Sutker, 2000, p. 405). While anxiety, hallucinations, delusions, attentional difficulties, loss of interest and pleasure, and lack of volition were common features of dementia praecox, symptoms alone had little diagnostic significance in his system (Adams & Sutker, 2000). Despite his appreciation for clinical heterogeneity, Kraepelin believed that a precise taxonomy of psychopathology would lead to the identification of discrete disease processes.

Bleuler sought to refine the dementia praecox construct, describing the dimensional nature of psychotic phenomena, which he viewed as existing on a continuum with normal experience. He conceptualized "schizophrenia" in terms of internal psychological processes and, with this, detailed the vast clinical heterogeneity among patients. For Bleuler, the essence of schizophrenia was in the "splitting" of mental functions, in which the psychological force that holds together facets of the psyche—perception, affect, memory, thought, behavior—breaks down in some way. The primary manifestation of splitting was disturbed thinking, which he observed in the speech of patients. He described subtle phenomenological differences in the form of thought disturbances, detailing processes in which "the most important determinant of the associations is lacking… the concept of purpose" (p. 15) and others in which "associations do not become entirely senseless, but they still appear odd, bizarre, distorted" (p. 19). For Bleuler, clinical heterogeneity was critical to understanding the nature of psychosis. Unlike Kraepelin, he believed schizophrenia to have multiple etiologies, involving biological, developmental, and social mechanisms, which underlay differences in clinical picture.

When the first Diagnostic and Statistical Manual was published in 1952, disturbances in thought remained central to the conceptualization of psychosis. The category of "schizophrenic reactions" was defined as "represent[ing] a group of psychotic reactions characterized by fundamental disturbances in reality relationships and concept formations, with affective, behavioral, and intellectual disturbances in varying degrees and mixtures. The disorders are marked by strong tendency to retreat from reality, by emotional disharmony, [and] unpredictable disturbances in stream of thought" (DSM, 1952, p. 26). The conceptualization and classification of disorders in the DSM-I was rooted heavily in the psychodynamic orientation of the time and did not delineate clear boundaries between normal behavior and pathology.

The complex and functional view of "psychoses" was retained with the release of the second edition in 1968, as was the centrality of thought disorder. Schizophrenia was described as a "large category" (p. 33), characterized by "disturbances in thinking [that] are marked by alterations of concept formation which may lead to misinterpretation of reality and sometimes to delusions and hallucinations, which frequently appear psychologically self-protective" (DSM-II, 1968, p. 33).

Release of the DSM-III in 1980 marked a turning point in the conceptualization and classification of psychosis. Responding to growing skepticism regarding the legitimacy of psychiatry as a scientific discipline, the DSM-III aimed to disseminate a common nomenclature and establish reliability of psychiatric diagnosis. The detailed descriptions of "schizophrenic reactions" (DSM, 1952) and "the schizophrenias" (DSM-II, 1962) were replaced by sets of observable, categorical criteria and rigid diagnostic boundaries. In the interest of inter-rater reliability, hallucinations and delusions (i.e.

content of thought), once considered “accessory symptoms” by Bleuler, were redefined as the core characteristics of schizophrenia. Thought disorder (i.e. form of thought) was placed under the heading of “other characteristic symptoms,” defined as “incoherence, derailment (loosening of associations), marked illogicality, or marked poverty of content of speech." The complexity and conceptual significance of thought disturbances was further reduced with the revision of schizophrenia in the DSM-IV, referred to as simply “disorganized speech.”

The transformation of the DSM over the past 60 years reflects a growing effort to standardize psychiatric diagnosis in order to improve professional communication, facilitate research progress, and guide treatment decisions. Unfortunately, with the shift toward categorical classification has come the assumption of mental disorders as discrete disease entities, an overly simplistic view of psychological disturbances that has had unintended consequences for how we understand and study these phenomena. As written by Jaspers (1963), “when we design a diagnostic schema, we can only do so if we forego something at the outset……and in the face of facts we have to draw the line where none exists…. A classification therefore has only provisional value. It is a fiction which will discharge its function if it proves to be the most apt for the time” (p. 605).

Limitations of DSM Classification

When Kraepelin applied a disease-model to the classification of psychopathology, it was with the expectation that defining syndromes at the symptom-level would facilitate discovery of underlying biological causes of mental disorders. Thus, despite his belief that dementia praecox was a disease of the brain, he accepted this as tentative until it could be demonstrated empirically. In fact, Kraepelin was explicit about the evidence

required to establish nosological entities, stating that "similar disease processes will produce identical symptom pictures, identical pathological anatomy, and an identical etiology (1907, p. 117)." Years later, these criteria were revisited by Robins and Guze (1970), who developed a framework for establishing the validity of diagnostic constructs. They proposed that a meaningful diagnostic entity should: (1) represent a cohesive clinical construct; (2) be distinct from other disorders; (3) accurately predict course, prognosis, and treatment response; (4) aggregate in families; (5) have clear and consistent biological markers.

Schizophrenia-spectrum disorders (i.e. schizophrenia, schizoaffective disorder, & schizophreniform) and affective psychoses (i.e. bipolar & major depressive disorders) are associated with a significant degree of clinical heterogeneity, which has long been recognized as an obstacle to research and clinical progress. Since development of DSM-III (APA, 1980), the classification of psychiatric disorders has been based on polythetic criteria, in which each disorder is defined by a list of possible symptoms, but not all are needed for diagnosis. Inherent to this approach is a high degree of clinical heterogeneity within diagnostic categories. Take, for instance, the DSM-IV (2000) construct of schizophrenia, which is defined by the presence of any two (or more) of five core criteria or one of three "special" symptoms. This amounts to twenty-nine distinct symptom profiles all subsumed under the diagnosis of schizophrenia. The clinical picture of bipolar disorder is comparably diverse, such that two patients can have the same diagnosis, but share no common symptoms. Further, because the same symptoms can present across multiple disorders, it is possible for individuals with similar clinical features to hold different diagnoses.

There is also considerable variation within individual diagnostic criteria resulting from the reduction of broad symptom dimensions into dichotomous categories. This is well illustrated by the heterogeneity of hallucinatory experiences, which vary in form (e.g., auditory, visual, gustatory, etc.), tone (e.g., derogatory, violent, reassuring, etc.), and severity across individuals, but are assumed to be equivalent in the DSM system. In addition to the poor specificity of clinical criteria, there is also significant variability in the onset, course, and outcomes of symptoms, which is not accounted for by diagnosis (Wright et al., 2013). A major point of criticism of the DSM system has been the virtual lack of evidence supporting its utility to describe behavior, predict outcomes, or guide treatment decisions (Lillienfeld & Treadway, 2016).

Influenced by Kraepelin's disease perspective, construction of the modern DSM system was based on the assumption that mental disorders are discrete, biologically-determined entities. Despite accumulated evidence contradicting the validity of diagnostic categories, this assumption continues to have a powerful influence on how we understand and study psychological disturbances. Recent advances in neuroimaging and genetics have facilitated investigation into the biological underpinnings of mental disorders, results of which have illustrated a complex and non-specific etiological perspective. While certain neurological patterns have been associated with schizophrenia, effects have been small with significant variability across individuals. Similarly, molecular genetics studies of schizophrenia have implicated a large number of associated genes with very small effects, results which are not indicative of a genetic disorder. This is consistent with findings from heritability studies, which support a model of generalized

risk for psychiatric conditions instead of disorder-specific aggregation within families (DeVylder & Lukens, 2013; Fusar-Poli et al., 2013; Rasic et al. 2013).

Dimension-Focused Approach

Progress in understanding psychotic phenomena is contingent upon the clarity and precision of the constructs that we study. This was an area pioneered by Paul Meehl (1955, 1962), who sought to explore the natural distribution of psychotic phenomena without a priori assumptions about the structure of data. Meehl believed that latent categories of psychopathology do exist, but have to be empirically distinguished from latent continua. Others oppose syndromal classification, arguing the need for alternative approaches to better capture clinical heterogeneity and disentangle complexity at the etiological level.

A large body of conceptual and empirical literature has addressed the heterogeneity problem and introduced alternative systems for classifying and studying psychosis (Andreasen, 2007; Tsuang, Lyons, & Faraone, 1990). One such approach has focused on the study of symptom dimensions, backed by empirical validation, including evidence of symptom-specific heritability (Buchanan & Carpenter, 1994; Strauss, Carpenter, & Bartko, 1974). In recent years, domain- or dimension-based studies have become increasingly prevalent in psychological science (Berenbaum, 2013). This approach has gained wide acceptance in the field of personality research, contributing to an advanced understanding of the structure and etiology of personality pathology (Krueger & Piasecki, 2001).

Dimensional frameworks also have a long history in psychosis research. In 1974, well before release of the DSM-III, Strauss and colleagues proposed the Domains of

Pathology model, which deconstructed six psychotic dimensions: disorders of content of thought and perception, disorders of affect, disorders of personal relationships, disordered speech and thought, disordered motor behaviors, and lack of insight. Reiterations of this approach have followed, including Buchanan and Carpenter's Domains of Psychopathology (1980) and Peralta and Cuesta's Psychopathological Dimensions (2001). More recently, the domain-based approach was endorsed by the National Institute of National Health (NIMH), who introduced their Research Domain Criteria (RDoC) in 2008, initiating a shift away from categorical diagnosis towards an empirically-driven, symptom-based taxonomy. Despite having yet to become the dominant paradigm in psychological science, domain-based research has yielded promising evidence, elucidating clearer links among psychotic experiences, etiological factors, and outcomes (Dutta et al., 2007; Leask, Vermunt, Done, Crow, Blows, & Boks, 2009; Peralta & Cuesta, 2007; Van Os et al., 1999).

Dimensional Measurement: Capturing the Psychosis Continuum

The traditional conceptualization of psychotic experiences as categorical constructs is increasingly refuted (Read, Mosher, & Bentall, 2004). With this, there is growing acceptance that psychotic phenomena exist on a continuum with normal experience, supported by the well-documented prevalence of subtle psychotic experiences in the general population (Johns & VanOs, 2001). The continuous nature of psychotic symptoms is purported to have etiological significance, corresponding with models of complex risk. As support for the continuum model has become more widespread, so has recognition of the inadequacy of categorical measurement approaches (Krueger & Piasecki, 2001; Lenzenweger, 2010). Dimensional assessment of symptoms

has superior validity compared to categorical approaches (e.g., rating scales), as they are better able to capture the natural distribution of psychosis phenotypes (Dutta et al., 2013; Esterberg & Compton, 2009). In terms of methodology, they are more sensitive to subtle forms of pathology that may be overlooked by categorical methods. Given their increased sensitivity to detect differences in psychotic experiences, continuous measures maximize statistical power and enhance the meaningfulness of data. Additionally, continuous data permit the use of sophisticated statistical techniques, which are valuable when modeling multifactorial relationships (Krueger & Piasecki, 2001). Despite conceptual and empirical advantages, continuous measurement is not the norm in psychopathology research. Consequently, availability of psychometrically-established assessment instruments is limited.

Thought Disorder: Conceptualization and Assessment

Thought disorder (TD) is a complex and multidimensional construct, reflecting peculiarities in thinking, language, and communication. Broadly, TD is defined as any disturbance that affects the form of thinking, including the organization, control, processing, or expression of thoughts. Given the breadth of the construct, TD has been defined and classified in a number of different ways. Different perspectives have placed varying emphasis on features related to the contextual appropriateness of ideas, the way in which they are organized, and the language used to express them.

A variety of approaches have been developed to measure TD, including reasoning tasks, clinician rating scales, and self-report measures. Perhaps the most widely-used medium has been the Rorschach inkblot test, which has a rich history in the measurement of disordered thinking and been the basis of several scoring systems. The

Rorschach lends itself well to TD assessment, as it is a relatively standardized technique with an unstructured format that allows examinees to interpret and structure the task freely (Singer & Wynne, 1964). Its projective nature offers a glimpse into how an individual perceives, interprets, and responds to ambiguous stimuli. As described by Singer, Wynne, & Toohey (1978), the Rorschach constitutes "an analogue of those many daily occurrences in which two or more individuals attempt to establish a consensually shared view of an ambiguous reality."

The use of the Rorschach in assessing thinking disturbances can be traced back to Rorschach himself, who observed a characteristic style of responding in psychotic protocols. He described a tendency for these individuals to formulate responses based on absurd, narrow details of the blot (i.e. ignoring typical determinants of form, color, and shading), often ascribing idiosyncratic or personal meaning to perceptual features (Kleiger, 1999). Building upon Rorschach's work, Rapaport sought to develop a more formal system for classifying thought disturbances, which he referred to as "deviant verbalizations." With this, he introduced the concept of "distance," which served as the foundation of his approach. He believed adaptive thinking was a function of the integration of perceptual (e.g., blot features) and associative (e.g., internal ideas, memories, feelings) processes. The relative pull of one process over the other resulted in either a loss or increase of distance, from which thought disturbances arose. A loss of perceptual distance reflected a tendency to see the blot as too real, while an increase of perceptual distance was associated with an overly-symbolic view of percepts. Rapaport's scoring system included 21 categories of disordered thinking, reflecting disturbances in perception, synthesis, interpretation, and expression of responses.

Rapaport's system became the basis for the Delta Index (Watkins & Stauffacher, 1952), the first standardized assessment to exclusively measure disordered thinking. The Delta Index included 15 of Rapaport's scoring categories, each of which was assigned to a four-point level of severity. The Delta Index was innovative, in that it captured the multifaceted and continuous nature of TD. For this reason, the scale was revised decades later by Johnston and Holzman, who developed the Thought Disorder Index based on it.

Thought Disorder Index. The Thought Disorder Index (TDI; Johnston & Holzman, 1979) provides a system for identifying, categorizing, and evaluating the severity of disordered thinking as expressed in language. TDI scoring can be based on any verbal sample, including the Wechsler Adult Intelligence Scale (WAIS), but is most commonly derived from verbatim responses to the Rorschach Inkblot Test (Rorschach, 1921). The Rorschach is believed to elicit greater instances of thought disorder than more structured methods (Johnston & Holzman, 1979), such as interviews (e.g., the Scale for the Assessment of Thought, Language, and Communication) and non-projective tests (e.g., WAIS).

The TDI specifies 23 categories of thought disturbances, most of which are based on Rappaport's original classification (see Appendix A). Because TD is understood as existing on a continuum, each category is assigned to a level of severity, ranging from mild to severe (.25, .50, .75, and 1.00), mirroring the structure of the Delta Index. The .25 level reflects subtle instances of cognitive slippage, which are commonly observed in healthy individuals, particularly in times of anxiety, stress, and fatigue (Johnston & Holzman, 1986). Disturbances at the .50 level "convey[s] an impression of loss of mooring, shaky reality contact, emotional overreaction, and distinct oddness" (Johnston

& Holzman, 1986, p. 490). Significant instability in thinking and perceiving is represented at the .75 level, while responses at the 1.0 level indicate a complete break from reality.

In developing the TDI, Johnston and Holzman (1979) distinguished four qualitative dimensions of TD based on the conceptual relatedness of individual categories. These include:

- **Associative Looseness**, in which responses appear to be driven by internal processes instead of demands of the task;
- **Combinatory Thinking**, in which percepts, ideas, or images are joined in an inappropriate, incongruous or unrealistic manner;
- **Disorganized Responses**, in which a lack of clarity of thought and sense of confusion are displayed;
- **Deviant Verbalizations**, in which word usage is odd, idiosyncratic, or undecipherable

A subsequent factor analysis yielded six discrete factors that partially overlapped with the original, conceptually-derived dimensions (Shenton et al., 1987). These dimensions, named "empiric factors," included: (1) combinatory thinking; (2) idiosyncratic verbalizations; (3) autistic thinking; (4) fluid thinking; (5) absurdity; (6) confusion.

The TDI is a highly sensitive measure of thought disorder and thus is able to detect subtle disturbances in language that may be overlooked using other methods. This is facilitated by the scoring protocol, as ratings are based on written transcriptions of verbal samples, which allows for systematic analysis of thought disturbances, in terms of qualitative form and severity. While this approach has clear advantages as a research tool, it is cumbersome to administer and score, which limits its utility in clinical settings.

Scale for the Assessment of Thought, Language, and Communication.

Alternative approaches to assessing thought disorder have been developed that are not as methodologically rigorous. Of these measures, the Scale for the Assessment of Thought, Language, and Communication (TLC; Andreasen, 1979a; 1979b; 1986) is perhaps the most widely-used clinician-rated assessment of thought disorder in research and clinical practice. Its development was undertaken as part of the broader objective to establish a standard set of thought disorder subtypes for inclusion in the glossary of the DSM-III. Subtypes were identified and defined based strictly on clinical observation, with no assumptions of underlying etiological mechanisms. This atheoretical approach was assumed deliberately in the service of designing an instrument with high inter-rater reliability and clinical utility (Andreasen, 1979a). The original definitions were piloted in a small sample of patients (n = 44) and subsequently revised to improve clarity. The severity of each item is rated on a 4-or 5-point scale, ranging from "absent" to "severe." These anchor points are defined quantitatively (e.g., speech behavior occurs 5 to 10 times during interview) and are item-specific. In addition to the relative severity of each item, thought disorder subtypes are identified as "more pathological" (e.g., poverty of speech, pressure of speech, clanging) or "less pathological" (e.g., circumstantiality, perseveration, blocking; Andreasen, 1986). Thus, items are not equally weighted in determination of global thought disorder severity.

Several studies have examined the factor structure of the TLC, with mixed results. In her early examination of the TLC, Andreasen (1979b) conducted an exploratory factor analysis of 12 TLC items, which yielded a single "verbosity" factor, on which derailment, illogicality, loss of goal, perseveration, incoherence, and pressure of speech loaded

positively and poverty of speech loaded negatively. This suggests that poverty of speech is a comparable indicator of verbosity, although measured inversely. Nonetheless, results were interpreted as evidencing distinct "positive" (florid) and "negative" (diminished) dimensions of thought disorder. In a subsequent factor analysis of the complete TLC, Andreasen and Grove (1986) reported three distinct domains, represented as Fluent Disorganization, Emptiness, and Linguistic Control. A comparable three-factor model was generated from an exploratory factor analysis of eight TLC items (Berenbaum et al., 1985) and later replicated through confirmatory factor analysis (Harvey et al., 1992). Interestingly, however, the inclusion of all 18 TLC items has typically revealed a more complex factor structure, with six to seven distinct dimensions (i.e. Cuesta & Peralta, 1992; Cuesta & Peralta, 1999; Peralta et al., 1992). Taken together, these data illustrate the potentially problematic impact of methodology on conceptual models.

The TLC is a generally reliable measure, with high clinical utility. Rating scales can be advantageous, as they are well-defined, structured, and facilitate communication among treatment providers. Further, TLC ratings can be based on a standard clinical interview and scoring can be completed quickly. Despite these strengths, the TLC has several disadvantages as a research tool. A general disadvantage of rating scales is that they yield categorical data (Lenzenweger, 2010). This is particularly problematic in the study of thought disorder, as categorical measures do not adequately capture the continuous nature of the construct. Further, because assessing the nuances of language is inherently challenging, ratings of thought disorder made in real-time or from memory are susceptible to examiner bias. The TLC does not control for speech output, although fluency has been shown to correlate with severity. Consequently, greater weight is given

to "positive" disturbances (e.g., pressure of speech, looseness, tangentiality, etc.), as they are directly related to increased verbal output. On the other hand, the TLC tends to underrepresent "negative" disturbances (e.g., poverty of speech, poverty of speech content), which are more difficult to assess through observation and comprise only 3 of the 18 items. While the positive/negative distinction appears to be a valuable heuristic for clinical practice, the TLC is not sensitive to different forms of thought disorder.

Diagnosis-Based Study of Thought Disorder.

Thought disorder has been the topic of extensive study over the past several decades. Unfortunately, the majority of this research has been based on broad diagnostic categories, results of which do not clearly inform a dimensional model. Nonetheless, it is important to reexamine this evidence from a dimensional perspective.

For much of the 20th century, TD was widely-accepted as a schizophrenia-specific feature and studied within this context. This perspective shifted in the 1970s, with recognition that thought disturbances were also prevalent in mania, spurring considerable efforts to identify patterns of thought disorder that differentiated diagnostic groups (see Appendix B). The earliest of these studies, which assessed thought disorder using abstract reasoning tasks (e.g., Gorham's Proverbs Test), demonstrated that under-inclusive thinking tended to characterize schizophrenia, while over-inclusive thinking was more typical of mania (Andreasen & Powers, 1974; Breakey & Goodell, 1972). Using this approach, depressed patients have been shown to exhibit deficits characterized by under-inclusive and concrete thinking compared to controls, although to a lesser degree than schizophrenic patients (Braff, Glick, & Griffin, 1983; Braff et al., 1988).

Similar patterns were found in a series of later studies using the TLC, with poverty of speech and speech content (i.e. negative thought disorder) common to schizophrenia and pressured speech, tangentiality, incoherence, illogicality, and loss of goal characterizing mania (i.e. positive thought disorder) (Andreasen, 1979b; Andreasen, 1984; Andreasen & Grove, 1986; Docherty, Schnur, & Harvey, 1988; Harvey, 1984). However, inconsistent evidence has also been reported, with several studies finding greater tangentiality, looseness, and illogicality in schizophrenia compared to mania (Cuesta & Peralta, 1993; Jampala et al., 1989; Ragin & Oltmanns, 1987). Schizoaffective patients were included in only two studies, both of which evidenced thought disorder profiles comparable to manic patients (Andreasen & Grove, 1986; Ragin & Oltmanns, 1987). Examining differences by schizophrenia subtype, Andreasen and Grove (1986) reported a significantly higher prevalence of poverty of speech content in patients with hebephrenic versus paranoid schizophrenia (Andreasen & Grove, 1986).

The presence of negative thought disorder has been evidenced in patients with primary depression, although overall TLC severity tends to be considerably lower than that of schizophrenia and mania (Andreasen, 1979b; Ragin & Oltmanns, 1987; Wilcox et al., 2000). Further, patients diagnosed with psychotic depression tend to exhibit significantly greater alogia, poverty of content, blocking, and perseveration than their non-psychotic counterparts (Wilcox et al., 2000).

A series of studies by Holzman and colleagues examined the form and severity of thought disorder in schizophrenia, mania, and schizoaffective disorder using the TDI (Holzman et al., 1986; Johnston & Holzman, 1979; Shenton et al., 1986; Solovay et al., 1986). Results evidenced disorder-specific patterns of thought disorder, in which

schizophrenia was characterized by “fluid thinking, interpenetrations of one idea by another, unstable verbal referents, and overly concise and contracted communications which give the impression of inner turmoil and confusion (p. 369).” Conversely, manic thought processes were described as “loosely tied together ideas that are excessively and immoderately combined and elaborated” with “a playful, mirthful, and breezy quality to their productions (Holzman et al., 1986, p. 369).” Contrary to the findings of Andreasen and Grove (1986), Johnston and Holzman reported no significant differences in TDI scores between paranoid and non-paranoid patients.

The pattern of thought disorder in schizoaffective patients was less consistent, as significant differences were found between those in manic and depressed states. Schizoaffective – manic patients exhibited a high level of combinatory thinking comparable to manic patients. However, they lacked the characteristic flippancy and humor of the manic patients and resembled the schizophrenic patient in terms of their frequency of idiosyncratic verbalizations, confusion, and autistic thinking. The schizoaffective – depressed patients were similar to the schizophrenic group in terms of frequency of absurd responses and relatively constricted protocol length. However, they had very low levels of thought disorder overall, with TDI scores largely resembling healthy controls. In contrast, their rate of absurd responses was similar to the schizophrenic group.

Despite evidence of differences in qualitative form and severity across disorders, the variability in thought disorder presentation is not fully explained by diagnostic groupings. Heuristically, comparative studies of thought disorder consistently report significant within-group variance (i.e. large standard deviations) in TDI total and factor

scores, across diagnostic and control groups. This point was empirically demonstrated using discriminant-function analysis, which found that TDI total scores correctly classified only 63.0% of manic and schizophrenic patients (Solovay, Shenton, & Holzman, 1986). Qualitative factors were shown to more accurately differentiate groups, as a subset of five empirically-derived categories (irrelevant intrusions, combinatory thinking, fluid thinking, confusion, and idiosyncratic verbalization) correctly classified 76.5% of the sample. However, when re-examined in a subsequent study that also included a schizoaffective subsample, the accuracy rate of the same factors dropped to 57.7% (Shenton, Solovay, & Holzman, 1987).

Course and Chronicity. The course of thought disorder is highly variable across individuals. Diagnosis has been shown to account for a portion of this variance. It is well-evidenced that thought disorder in schizophrenia is more stable and persistent compared to schizoaffective disorder and affective psychoses (Harvey & Earle-Boyer, 1986; Marengo & Harrow, 1988). Thought disorder in mania has been described as "reversible" (Andreasen & Grove, 1986), as disturbances are typically severe during acute phases of illness (e.g., hospital admission) and remit completely following treatment (Andreasen & Grove, 1986; Docherty, Schnur, & Harvey, 1988; Harrow & Marengo, 1986; Marengo & Harrow, 1987). Remission of positive thought disorder has also been observed in schizophrenic patients (Andreasen & Grove, 1986), suggesting that some subtypes tend to follow an episodic course, independent of diagnosis. Consistently, forms of thought disorder that are characteristic of schizophrenia have been shown to have greater stability over time, including higher-level disturbances (i.e. 0.50, 0.75) on the TDI (Metsänen et

al., 2006) and idiosyncratic verbalizations (Braff et al., 1988; Harrow & Marengo, 1986; Metsänen et al., 2006).

Summary. Research has consistently shown differences in the form and severity of thought disorder across diagnostic groups using both the TLC and TDI. Broadly, thinking in schizophrenia is characterized by odd, impoverished, and internally-driven speech. Classically "schizophrenic" thought disorder tends to have greater stability over time, with slight increases during acute phases of illness. The disconnected and elaborated thinking disturbances seen in mania tend to be episodic, emerging during acute phases and typically remitting completely with treatment. As measured by the TDI, combinatory thinking is indicative of acute psychological distress, while "thought disorganization" (e.g., vagueness, perseveration, inappropriate distance, confusion, looseness, fluidity, absurd responses, and incoherence) appears to reflect a more stable trait. It has been posited that the stable thought disturbances common to schizophrenia are pathognomonic of underlying pathophysiology, while sporadic forms are secondary to clinical and situational factors (Levy et al., 2010; Walberg et al., 2001).

Despite the high degree of within-subjects variability across studies, these data demonstrate a clear association between thought disorder and diagnosis. The nature of this relationship is less clear. It is possible that these findings reflect an association between thought disorder and certain clinical or personal features related to diagnosis, such as symptoms, medication, or social functioning. In this case, diagnosis contains important information about the nature of thought disorder that warrants further study. An alternative explanation is that thought and language characteristics play an influential role in clinical diagnosis, thereby making evidence of their relationship tautological.

The Significance of Affective Experience in the Etiology of Thought Disorder

Central to Bleuler's early conceptualization of schizophrenia was the intrinsic link between thinking and affect. For him, the core of psychosis was in the fragmented associations among thoughts and feelings, or "splitting." Since this time, interest in the relationship between affect and thought disorder has continued to drive research and clinical work.

The connection between thinking and affect was observed by Johnston & Holzman (1979), who described exacerbations of thought disorder during times of increased stress in both psychiatric patients and healthy individuals. Their observations stimulated interest in the role of stress sensitivity as a marker of psychosis liability; they questioned "is thought disorder unique to schizophrenics, or is everyone susceptible to it if subjected to enough stress? Or does such susceptibility vary among individuals (–) some requiring little stress of thinking to become disordered, others requiring greater amounts, and still others remaining quite immune to such disorganization no matter what the stress" (p. 54)? They speculated that "thought disorder reflects a predisposition in some vulnerable people to react to stress by becoming less able to conceptualize, focus attention, and reason logically" and "that persons with manifest thought disorder require less stress than do normal people for their thinking to be affected" (p. 54). Unfortunately, Johnston and Holzman never sought empirical answers to these questions. However, they provide a compelling theory about the nature of psychopathology that has contemporary relevance.

Perhaps the earliest study of the impact of stress on TD was conducted by Shimkunas (1972), who found that emotionally-salient questions elicited greater thought

disorder (as measured by global severity ratings) in schizophrenia when they had personal relevance (e.g., "Tell me about your saddest memory") than general discussion of emotional topics. This area of research was later revisited in a series of studies by Docherty and colleagues, who examined the impact of affective valence on the clarity of communication. They introduced the concept of "affective reactivity" of speech, demonstrating that schizophrenic patients exhibited significantly greater referential failures during negatively valenced topics compared to positive or neutral topics (Docherty et al., 1994; Docherty, Sledge, & Wexler, 1994; Docherty, 1996; Docherty & Herbert, 1997; Docherty, Hall, & Gordinier, 1998). Consistent findings were later replicated by other researchers (Haddock et al., 1995; Tai, Haddock, & Bentall, 2004).

Research on affective reactivity of language in mood disorders is limited. However, there is evidence that negatively valenced topics exacerbate referential failures in manic patients (Tai, Haddock, & Bentall, 2004). Depressed patients, on the other hand, have not been found to exhibit affective reactivity of language, although their rate of referential failures is typically higher than controls (Docherty, 1996; Rubino et al., 2011; Tai, Haddock, & Bentall, 2004). These studies have not compared affective reactivity in mood disorders with and without psychotic features. Familial patterns of affective reactivity were examined in two studies, results of which indicated that the speech of unaffected relatives of schizophrenic patients is not influenced by affective valence (Docherty, Sledge, & Wexler, 1994; Docherty, 1996).

Although affective reactivity in schizophrenia and mania has been well-established, a high degree of within-group variance exists. Moderators of affective reactivity of language have been implicated. It has been suggested that women are more

susceptible to disordered thinking in times of stress (Metsänen et al., 2005). Further, affective reactivity tends to be greater in patients with positive schizophrenia compared to negative schizophrenia (Docherty et al., 1994; Docherty, Sledge, & Wexler, 1994; Docherty & Herbert, 1997; Docherty, Hall, & Gordinier, 1998). There is also evidence that family psychiatric history moderates the relationship between affective reactivity and thought disorder in schizophrenia. Specifically, negative affect has been shown to exacerbate thought disorder (as measured by the TLC) in patients with a family history of psychosis, but not those without a family history of psychosis (Docherty, Rhinewine, Labhart, & Gordinier, 1998). Despite compelling preliminary evidence, research examining moderators of affective reactivity is limited. Further study is needed to gain a broader understanding of moderating factors and replicate previous results.

Familial Basis of Thought Disorder and Affective Reactivity

Both thought disturbances and affective reactivity have been shown to have familial underpinnings. This has been supported by evidence of an over-represented aggregation of these traits within families (Docherty et al., 1998; Levy et al., 2010). In family studies, psychopathology of relatives has been shown to predict the expression of thought disorder and degree of affective reactivity, as well as the interaction between them.

Familial-high risk studies have documented thought disorder in children and adolescents who go on to develop psychotic disorders (Bearden et al., 2011; Gooding et al., 2012; Metsänen et al., 2004, 2007; Ott, Roberts, Rock, & Erlenmeyer-Kimling, 2002). Further, TD is common in clinically unaffected relatives of manic and schizophrenic patients, differentiating them from relatives of healthy controls (Hain et al.,

1995; Johnston & Holzman, 1979; Shenton et al., 1989). Interestingly, the type of severity of thought disorder has been shown to cluster in families (Berenbaum, Oltmanns, & Gottesman, 1985; Shenton et al., 1989).

As the majority of these studies are biology-focused, findings have largely been interpreted as evidence of genetic underpinnings. However, given the correlational nature of this research, conclusions drawn from the data are largely speculative. Adoption studies have helped to disentangle the genetic and environmental contributions to TD. Wahlberg and colleagues (1997) examined the likelihood of TD in adoptees, based on genetic-risk (presence or absence of psychotic disorder in biological mother) and communication deviance (CD) in adopted parents. Results suggested a significant interaction effect, with high-risk adoptees in high CD environments exhibiting the greatest TD. Interestingly, high-risk adoptees in low CD families had significantly lower TD, suggesting potential protective effects of environment. Results of have demonstrated co-familiality of TD independent of shared environmental factors. In a subsequent study, Wahlberg and colleagues (2000) replicated these results using the TDI, finding that the interaction between high-risk status and CD in adopted parents distinctly predicted idiosyncratic thinking in adoptees.

Taken together, results from family and adoption studies suggest that both genetic and environmental influences are involved in the development of TD. The familial basis of TD has also been demonstrated from a social-learning perspective. The seminal work of Singer and Wynne (1965) provides compelling support for the role of familial communication patterns in shaping the development of TD and psychosis. Their Family Studies research program at the NIMH grew out of Wynne's earlier hypothesis about the

social-transactional nature of thought disorder, specifically that “the fragmentation of experience, the identity diffusion, the disturbed modes of perception and communication, and certain other characteristics of the acute reactive schizophrenic's personality structure are to a significant extent derived, by processes of internalization, from characteristics of the family social organization. Also internalized are the ways of thinking and of deriving meaning, the points of anxiety, and the irrationality, confusion, and ambiguity that were expressed in the shared mechanisms of the family social organization (Wynne, 1958).”

Their research explored how unusual ways in which parents perceived, interpreted, and reasoned about the world interfered with their ability to establish shared attention and construct mutual meaning with their children. Wynne and Singer posited that these disturbed social interactions compromised the child’s development of cohesive, stable mental representations of the self and world, thereby putting them at increased risk of psychosis. To study this, they devised a system for classifying and scoring dimensions of communication deviance based on verbal responses to projective tests (i.e. the Thematic Apperception Test and Rorschach).

From this, they identified a pattern of CD that reliably predicted the later emergence and severity of psychosis in children, broadly characterized by vague, fragmented, and contradictory communicating. Interestingly, subtle hindrances to shared meaning were more detrimental to the listener (i.e. psychologically) than overt disruptions. This pattern was defined by several CD categories, including: (1) closure problems, in which the speaker leaves the story hanging or fails to acknowledge a major perceptual element of the stimulus (e.g., "Well, this could be the son of this elderly lady who's… looks as though he's told his mother, if that’s his mother, some distressing news

about something"); (2) disruptive behavior, in which the speaker interrupts the task, by asking irrelevant questions or attributing personal, self-referential meaning to the stimuli (e.g., “Reminds me of my son contemplating whether he should play the guitar or not...”); (3) peculiar language and logic, in which the speaker uses odd phrasing, peculiar reasoning, or repetitiveness (e.g., “Maybe it isn't his mother because he wearing an overcoat”).

Collectively, this research suggests that certain forms of TD are part of a developmental process, involving both biological and environmental influence. However, the relative effects of these mechanisms, and the nature of their interactions is unclear.

Moderators and Correlates of Thought Disorder

Demographic Factors.

Age and Gender. Few studies have examined the effects of age and gender on thought disorder presentation. Harvey and colleagues (1997) examined thought disorder (TLC) in a cross-sectional study of schizophrenic patients, who ranged in age from 19 to 96. Poverty of speech was more prevalent and severe in older adults (i.e. > 64), while “disconnected” speech (e.g., circumstantiality, loss of goal, incoherence, derailment, etc.) tended to characterize younger patients. In a younger sample (M = 32.59), Spohn and colleagues (1986) found that thought disorder severity (TD_R) increased with age (r = .323, $p < .01$). Taken together, it is possible that the severity of thought disorder increases across young adulthood and begins to attenuate in later life. However, these studies do not account for age-at-onset and chronicity of illness, which could affect cross-sectional relationship of thought disorder and age.

Evidence on the relationship between gender and thought disorder has been mixed. Several studies have found thought disorder to be similar in men and women (Atalay & Atalay, 2006; Johnston & Holzman, 1979; Perry et al., 1995). While a significant correlation between TD_R and gender (women exhibited higher TD) was evidenced by Spohn and colleagues (1986), this association attenuated significantly after controlling for duration of lifetime hospitalizations and global symptom severity. This could suggest that clinical factors mediate the effect of gender on TDI; however, this relationship may also be an artifact of sampling bias, as the proportion of women (27%) was considerably less than that of men.

Race, Social Class, and Education. Several studies have examined effect of cultural factors on the measurement of thought disorder. Haimo and Holzman (1979) found that total TDI scores did not differ across race, regardless of whether ratings were based on Rorschach (TD_R) or WAIS (TD_w) protocols. This suggests that the TDI differentiates characteristics of cultural dialect from thought disorder. The relationship between socioeconomic status (SES) and TD was less clear. In both schizophrenia and control groups, SES was positively correlated with TD_R scores. SES was similarly related to TD_w scores in the schizophrenia group; however, in the control group, lower SES was associated with higher TD_w scores. The explanation for this inconsistency is unclear. However, findings suggest that TDI scores based on the WAIS may be more sensitive to SES than scores based on the Rorschach. These results can be compared to the findings of Mazumdar and colleagues (1994) who found that poverty of speech on the TLC was over-represented in subjects from a rural compared to non-rural background. Higher educational status was related to more frequent instances of distractible speech,

illogicality, clanging, and neologisms, while perseveration was more common in less educated subjects. Although not assessed formally, it is possible that verbal output is greater in groups with higher education and SES, in turn contributing to a higher frequency of disturbances. Overall, demographic features do not appear strongly related to the severity of thought disorder, particularly when using TD_R scores.

Clinical Factors.

***Medication*.** The efficacy of neuroleptic medication on thought disorder severity has been evidenced by several studies. Hurt and colleagues (1983) assessed changes in TD_R scores in patients with schizophrenia who were randomly assigned to receive a high or low dose of haloperidol, placebo, or no treatment. While thought disorder remained stable in both comparison groups across the study period, patients in the treatment condition exhibited a significant reduction in TD_R scores. Specifically, they showed a rapid improvement within the first three days, with the rate of change lessening as the study period progressed. Despite the notable reduction in TD_R scores, thought disorder remained evident in the drug-treated groups at discharge. A notable limitation of this study was the administration of only three cards of Rorschach, on which the reliability and validity of TDI is unestablished. In a later study, Spohn and colleagues (1986) demonstrated a reduction in TD_R severity with haloperidol over a 10-week period, with residual symptoms persisting throughout the study period. Post-hoc analyses revealed that severe forms of thought disorder were more responsive to treatment, while low level pathology tended to remain stable. Between-subjects variability was also evidenced by Gold and Hurt (1990) in a randomized-control trial of haloperidol. While thought disorder improved over the 10-week study period, there was significant variability in the

rate of change across patients, which was not explained by baseline severity level. As posited by Spohn and colleagues, certain forms of thought disorder may be more responsive to treatment than others. However, there is also evidence that neuroleptic medication causes a significant reduction in verbal productivity (Bilder et al., 1992), suggesting that the treatment-related reduction in thought disorder may be a function of decreased verbal output.

Unfortunately, potential mediators and moderators of psychotropic treatment response have not been formally examined. Further, there is limited research on the effects of other medications on thought disorder, including second generation antipsychotics. A significant treatment effect has been evidenced in a small sample receiving various combinations of typical and atypical antipsychotics, as well as mood stabilizers and anticholinergics (Goldberg, Dodge, Aloia, Egan, & Weinberger, 2000). However, this study did not examine the comparative effects of each drug. Levy et al. (1993) demonstrated that methylphenidate significantly increased thought disorder severity (TDI) in schizophrenic patients, but had no effect on healthy controls. However, a combination of chlorpromazine and methylphenidate has been shown to significantly improve language performance and verbal fluency (Bilder et al., 1992).

Psychiatric Symptoms. Research on the covariance of thought disorder and other symptom dimensions has been inconclusive. Results of factor-analytic studies have been highly variable, with TD loading onto positive (i.e. hallucinations and delusions; Andreasen & Olsen, 1982) and disorganized (i.e. bizarre behavior, inappropriate affect; Picardi et al., 2012) factors, as well as an independent domain (Leask et al., 2009). It is unclear whether these inconsistencies are a function of methodological bias or reflect

actual variation. However, the use of categorical data in these studies could obscure the true latent structure of symptoms. Several correlational studies have examined relationships among thought disorder and other symptom domains. Harrow and Marengo (1986) found that thought disorder severity was positively correlated with delusions and unrelated to hallucinations in patients with schizophrenia, non-schizophrenia psychoses, and non-psychotic disorders. Thought disorder has also been shown to correspond longitudinally with clinician-rated scores on the Brief Psychiatric Rating Scale, namely thought disorder (i.e. hallucinations, unusual thought content, and conceptual disorganization) and hostility-suspicion subscales (Hurt, Holzman, & Davis, 1983). Cuesta and Peralta (1993) found global symptomatology (Strauss-Carpenter Scale) was associated with poverty of speech, poverty of speech content, and blocking on the TLC.

Illness Duration. Changes in thought disorder have been evidenced in relation to the course of illness. Johnston and Holzman (1979) found differences in TD_R among recent-onset and chronic schizophrenia patients. The chronically ill group exhibited more severe thought disorder, with considerably more absurd and incoherent responses. Thought disorder severity has been shown to increase as a function of illness duration (Maeda et al., 2007; Spohn et al., 1986) as well as number of hospitalizations (Marengo & Harrow, 1997; Spohn et al., 1986). However, inconsistent evidence also exists. For instance, Cuesta and Peralta (1993) found that rate of hospitalization was associated with perseveration on the TLC, but unrelated to all other items. This null finding may be a function of measurement approach, as the restricted range of TLC ratings could obscure true relationships. Collectively, these studies imply that the severity of TD may increase across the course of chronic illness. However, these data are drawn from cross-sectional

research, which makes it difficult to draw conclusions about the longitudinal course of thought disorder.

Summary

Thought disorder is a complex and dimensional phenomenon. Akin to its clinical presentation, the etiology of TD is presumed to be similarly diverse, involving interactions among biological, environmental, and psychological factors. Although genetic and neurological underpinnings have been implicated, a precise pathogenic model of thought disorder remains elusive. Efforts to isolate pathophysiological mechanisms are likely confounded by sources of thought disorder variability that are not biologically-based. Thus, explaining the relative contributions of environmental predictors is critical to the search for neurobiological mechanisms. While demographic, developmental, clinical, and state-related factors have all been shown to influence thought disorder, the combined effects of these predictors have not been examined. Thus, future research on integrative models of thought disorder is needed to better explain variability at the clinical level and, in turn, inform etiological mechanisms.

The familial transmission of thought disorder is well-established empirically, suggesting a strong genetic component involved in its etiology. Further study of the familiality of thought disorder would greatly enhance the search for susceptibility genes. In light of accumulated evidence of shared risk among psychiatric disorders (Aukes et al., 2012, Mortensen et al., 2010), examining dimensional systems of classifying family history, which account for variability in diagnosis and sub-clinical symptomatology (e.g., depression that does not cross the threshold of major depressive disorder) is warranted. Capturing the complexity of family history may provide a clearer picture of the familial

aggregation of thought disorder and help to distinguish genetic from environmental mechanisms.

In sum, in order to gain a clearer understanding of psychotic phenomena, we must regain an appreciation for the "richness of psychopathology" (Brockington, 1992). The incorporation of dimensional conceptualizations of psychosis into theoretical and empirical paradigms is critical to this aim. Research into the correlates and moderators of dimensional constructs, including demographic and psychological factors, is critically needed. Approaching research from a dimensional perspective has the potential to promote a more complex understanding of psychosis.

Study Purpose and Hypotheses

Bleuler saw thought disorder as the core defining feature of psychotic phenomena, reflective of the "splitting of the psychic functions" that occurred when, in the process of thinking, one's ideas and feelings disconnect, becoming fragmented and competing functions. Unfortunately, interest in thought disorder as the conceptual core of psychosis was lost with rise of the modern DSM system, paralleling the shift towards a more simplistic, categorical way of defining psychiatric disorders.

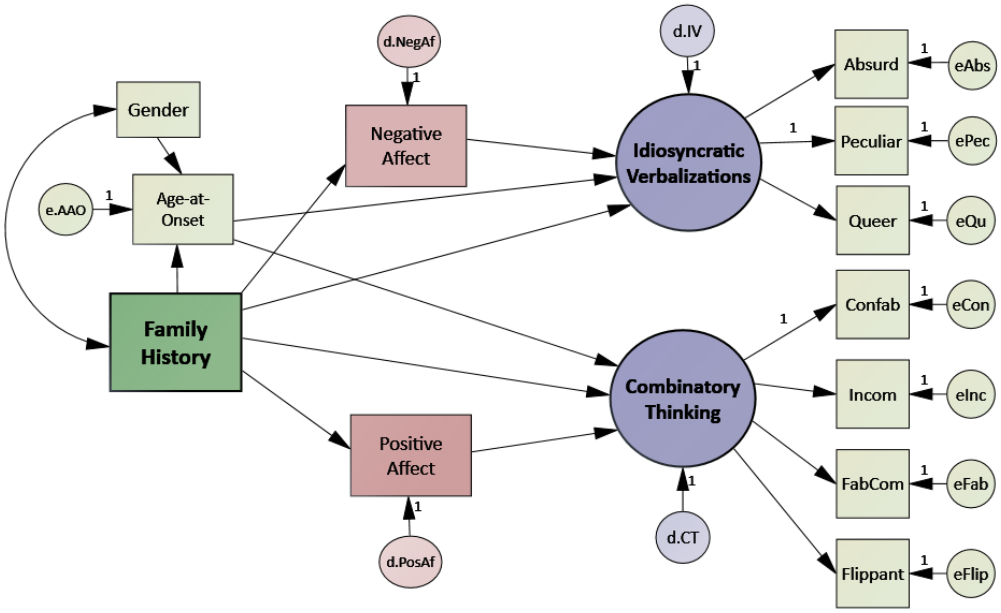
This study sought to explore thought disorder from a dimensional perspective, with the aim of disentangling qualitative heterogeneity and diverse sources of influence transdiagnostically. The two primary objectives were to: (1) to demonstrate the feasibility and comparative utility of a dimensional approach to psychopathology research; (2) to examine a multifactorial, integrative model of thought disorder in a large, transdiagnostic sample (i.e. schizophrenia, schizoaffective, and bipolar disorder). Specifically, the direct,

indirect, and combined effects of a diverse set of empirically-supported predictors were examined, which include family psychiatric history, sex, age-at-onset, and affective state.

Overview of Proposed Model

The path diagram presented in Figure 1 illustrates the hypothesized relationships among predictors. The model reflects a hybrid approach, which integrates categorical and dimensional frameworks. This approach has been recommended by several authors as a strategy for comparing dimensional and categorical classification (Krueger & Piasecki, 2002; Peralta & Cuesta, 2003). The inclusion of categorical diagnosis as an independent, between-subjects variable is warranted for several reasons. First, because the domain-specific approach has not yet been empirically-validated, examining the relationship between symptom dimensions and traditional diagnoses is informative (NIMH, 2009). This strategy is also valuable from a conceptual standpoint, as it enables integration and evaluation of existing research findings within a dimensional framework. The proposed model connects two primary domains of extant literature: (1) factors that explain heterogeneity at the diagnostic level and (2) factors that explain heterogeneity at the domain level (i.e. thought disorder).

Figure 1
Proposed Structural Equation Model



Family history is a predictor of proband diagnosis, reflecting evidence of symptom aggregation within families (Aukes et al., 2012; Rasic et al., 2013). Age-at-onset is also influenced by family history, supported by evidence of earlier onset of schizophrenia and bipolar illness in probands with a family history of psychosis (Gorwood et al., 1995). However, this relationship is moderated by sex. In non-familial cases, onset is earlier in men than women, while no sex differences are evident in familial psychosis cases (Esterberg & Compton, 2012; Gorwood et al., 1995; Malaspina et al., 2000). Sex also moderates the association between family history and diagnosis, as the prevalence of affective and psychotic disorders tends to be higher in the relatives of women compared to men (Atalay & Atalay, 2006; Sham et al., 1994).

The second part of the model illustrates correlates and moderators of thought disorder. A direct path is depicted between family history and thought disorder, reflecting

evidence of familial transmission. Family history and thought disorder are also associated indirectly through diagnosis. Although not conceptually meaningful, the examination of diagnosis as a mediator provides a comparison between categorical and dimensional frameworks by explaining the proportion of variance in thought disorder independently explained by diagnosis.

Affective state is also included as a predictor of TD, with positive and negative affect differentially influencing idiosyncratic verbalizations and combinatory thinking. Positive and negative affect mediate the relationships between family history and thought disorder domains. This reflects evidence that patients with a family history of psychosis exhibit greater exacerbations of thought disorder in response to negative affect.

Primary Research Questions

(1) To what extent is variability in thought disorder explained by affective state?

Hypotheses:

1a. Higher positive affect will be associated with increased combinatory thinking.

1b. Greater negative affect will be associated with more severe idiosyncratic verbalizations.

(2) To what extent does family psychiatric history explain variability in clinical presentation?

Hypotheses:

2a. Probands with a family history of psychosis will have significantly higher scores on the idiosyncratic verbalizations factor.

2b. family history will be a significant predictor of affective valence; probands with family history of psychosis will have significantly lower positive affect than those without.

2c. The direct effect of family history on thought disorder will be partially mediated by affective state (i.e. valence/intensity)

(3) What is the comparative utility of categorical diagnoses within a dimensional and multifactorial model?

Hypotheses:

3a. Variance in TD will be inadequately explained by DSM diagnosis alone.

3b. Accounting for family history, age-at-onset, and affective state will significantly improve the explanatory power of diagnosis.

METHODS

Description of Dataset

Data for the proposed study were drawn from a larger project on co-familial traits in psychotic disorders collected by Dr. Deborah Levy at the Psychology Research Laboratory / Mailman Research Center at McLean Hospital. The full dataset includes patients with schizophrenia, schizoaffective disorder, and bipolar psychosis, their first-degree relatives, and healthy controls. Authorization to share data was granted by the Institutional Review Boards at both McLean Hospital and the University of Louisville.

Analyses were conducted on a subsample of patients (n = 322) with schizophrenia (n = 79), schizoaffective disorder (n = 140), and bipolar psychosis (n = 103) who entered into the study between 1996 and 2010. Data collected through assessment of their first-degree relatives (n = 439) were coded and included in the analysis.

At the time of data collection, written informed consent was obtained from all study participants in accordance with the IRB guidelines of McLean Hospital. Probands (PRO) were recruited from an outpatient mental health clinic; those included in the study met DSM-IV criteria for schizophrenia (SZ), schizoaffective disorder (SZA), or bipolar disorder with psychotic features (BP). The Structured Clinical Interview for DSM-IV Disorders (SCID; Spitzer, Williams, Gibbon, & First, 1994) was administered to all participants by a trained interviewer. Consensus diagnoses were made by experienced clinicians based on information obtained from the SCID, in conjunction with available hospital records. All probands who participated in the study met the following inclusion criteria:

(1) Age 18 to 65-years-old

(2) No diagnosed central nervous system disease

(3) No substance abuse within the past year

(4) Estimated verbal IQ ≥ 70 based on the Vocabulary subtest of the WAIS-R

(5) Fluent English speaker

(6) Absence of tardive dyskinesia

Measures

Thought Disorder. The Thought Disorder Index (TDI; Johnston & Holzman, 1979) provides a system for identifying, categorizing, and evaluating the severity of disordered thinking as expressed in language. TDI scoring can be based on any verbal sample, including the Wechsler Adult Intelligence Scale (WAIS), but is most commonly derived from verbatim responses to the Rorschach Inkblot Test (Rorschach, 1921). The Rorschach is believed to elicit greater instances of thought disorder than more structured methods (Johnston & Holzman, 1979), such as interviews (e.g., TLC) and non-projective tests (e.g., WAIS).

As presented in Appendix A, the TDI includes 23 categories of thought disturbances, most of which were originally defined by Rappaport that fall along a continuum of severity, ranging from mild to severe (.25, .50, .75, and 1.00). The .25 level reflects subtle instances of cognitive slippage, which are commonly observed in healthy individuals, particularly in times of anxiety, stress, and fatigue (Johnston & Holzman, 1986). Disturbances at the .50 level "convey[s] an impression of loss of mooring, shaky reality contact, emotional overreaction, and distinct oddness" (Johnston & Holzman, 1986, p. 490). Significant instability in thinking and perceiving is represented at the .75 level, while responses at the 1.0 level indicate a complete break from reality.

A global score can be estimated by summing all instances of thought disorder, weighted by severity level. Johnston and Holzman (1979) developed the following formula for calculating total TDI scores on the Rorschach (TD_R):

$$TD_R = \frac{0.25\,(A) + 0.50\,(B) + 0.75\,(C) + 1.00\,(D)}{\text{Total \# Rorschach Responses}} \times 100$$

The weighted sum of scores is divided by the total number of Rorschach responses to control for verbal productivity and multiplied by 100. The authors chose to control for verbal output using number of responses, as it correlated similarly with weighted sum of scores as the exact word count and is less demanding to calculate (Holzman, Shenton, & Solovay, 1986).

Reliability. TDI total score has been shown to have good inter-rater reliability, with correlations ranging from .80 to .93 (Coleman et al., 1993; Johnston & Holzman, 1979; Solovay et al., 1987) and an intra-class correlation (ICC) of .74 (Coleman et al., 1993). Inter-rater reliability estimates for severity ranged from .86 to .93 (ICC = .77) at the .25 level, .56 to .75 (ICC = .72) at the .50 level, and .50 to 1.0 at the.75-level (ICC = .77) (Coleman et al., 1993). Due to low frequency of responses at the 1.0 level, correlations could not be calculated. In terms of qualitative factors, ICCs were .86 for Irrelevant Intrusions, .76 for Combinatory Thinking and .58 for Idiosyncratic Verbalizations. Confusion and Fluid Thinking factors could not be calculated due to low frequency of related responses. It was noted that larger discrepancies among rating teams were found in more disordered protocols due to the number of individual scores assigned.

Factor Structure. The theoretical foundation of the TDI encompasses four qualitative dimensions of thought disorder, each defined by a subset of conceptually related categories. These domains include: (1) *associative looseness*, in which responses

appear to be driven by internal processes instead of demands of the task; (2) *combinatory thinking*, in which percepts, ideas, or images are joined in an inappropriate, incongruous or unrealistic manner; (3) *disorganized responses*, in which a lack of clarity of thought and sense of confusion are displayed; (4) *deviant verbalizations*, in which word usage is odd, idiosyncratic, or undecipherable (Solovay, Shenton, & Holzman, 1987, p. 18). Unfortunately, the theoretical structure proposed by Johnston and Holzman (1979) has never been tested empirically.

The factor structure of the TDI has been examined in only one study. Solovay and colleagues (1987) conducted a principle components analysis in a small sample (n = 97) of patients with schizophrenia and mania. The resulting model specified six factors, which were labeled: (1) combinatory thinking; (2) idiosyncratic verbalizations; (3) autistic thinking; (4) fluid thinking; (5) absurdity; (6) confusion (Shenton et al., 1987). The idiosyncratic verbalizations factor, which was comprised of peculiar and queer responses accounted for the largest proportion of variance (17.7%). This was followed by the combinatory thinking factor (9.2%), which subsumed playful confabulations, incongruous combinations, flippancy, and fabulized combinations. While the names of these factors are shared with the original, conceptually-based domains, their definitions are not equivalent.

Affective State. Positive and negative affect was measured through lexical analysis of transcribed Rorschach protocols using the Linguistic Inquiry and Word Count (LIWC, Pennebaker, 2001). The LIWC is an automated program that quantifies the proportion of words in a given text that fall into a set of pre-defined dimensions, which tap into various psychological processes (e.g., cognition, affect, personality), content

areas (e.g., food, space, family), and parts of speech (e.g., pronouns, articles, past tense). At the core of the program is the LIWC dictionary, which defines the composition and structure of linguistic dimensions. The dictionary was initially constructed within the context of exploring written expression of emotion, but has since undergone several revisions and expansions. The most recent version, LIWC2007, recognizes over 4,000 words and word stems that reflect 72 linguistic categories.

The LIWC calculates several affect variables, which are arranged hierarchically. The overarching category, affective processes (915 words), is the sum of two discrete dimensions: positive affect (e.g., love, nice sweet; 406 words) and negative affect (e.g., hurt, ugly, nasty; 499 words). Negative affect is comprised of three narrower categories: (1) anxiety (e.g., worried, fearful, nervous; 91 words); (2) anger (e.g., hate, kill, annoyed; 184 words); (3) sadness (e.g., crying, grief, sad; 101 words). While subcategories of positive affect (i.e. optimism, positive feeling) were included in the original dictionary, they were excluded from the latest revision due to low base rates. The remaining affect dimensions have been shown to be reliable indices. Pennebaker et al. (2007) demonstrated strong internal consistency of words within each affect category, with Cronbach alpha statistics ranging from .87 (anxiety) to .97 (positive emotion).

The external validity of affect dimensions has also been evidenced. Pennebaker & Francis (1996) found a high degree of correspondence between LIWC scales and related ratings assigned by four independent judges. Strong correlations were reported for positive (r = .41) and negative (r = .31) affect, as well as subcategories of anxiety (r = .38) and anger (r = .22). However, there was significant discrepancy between LIWC and judges' ratings of sadness. The LIWC counts of affective processes have also been

shown to accurately capture responses to emotionally-provocative stimuli (Kahn et al., 2007). The LIWC has been used extensively in psychological research. This approach has been valuable for exploring affective processes in psychotic disorders, as cognitive, communicative, and symptomatic factors are known to confound self-reported measures of emotion (Cohen et al., 2008).

In the current study, the LIWC was used to quantify the proportion of negative and positive affect words in transcribed Rorschach protocols. This methodology allowed for evaluation of affect and TD based on the same responses. All administrations were tape recorded and later transcribed verbatim by a professional transcriptionist with considerable experience working with Rorschach protocols. To prepare for LIWC analysis, electronic transcripts were edited to include only text that was spoken by the patient (i.e. examiner instructions/queries, transcriptionist notes were removed). Transcripts were then entered into the LIWC program to calculate the proportion of negative and positive affect words. Because the word "like" is used frequently in Rorschach responses when describing the blot (e.g., "It looks *like*"), we chose to remove it from the LIWC dictionary to control for erroneous inflation of the positive affect scores.

Family Psychiatric History. Two methods were used to collect family history data: (1) direct diagnostic evaluation of relatives (i.e. family study); (2) informant-reports of psychiatric histories of all known relatives, collected through interviews with one or more relatives and probands who were able to do so. Relatives who participated in the study were administered the Structured Clinical Interview for DSM-IV by experienced interviewers. The SCID has been shown to be a highly consistent instrument, with

excellent inter-rater (Skre et al., 1991) and test-retest reliability (Williams et al., 1992). Further, it has high specificity for most diagnoses, with the exception of substance use and antisocial personality disorders, which tend to be underreported (Andreasen et al., 1977).

Axis I conditions were diagnosed by consensus of expert clinicians, based on interview information and available medical records. The presence of Cluster A personality disorders (paranoid, schizoid, schizotypal) were evaluated in the same manner and classified into one of three groups: (1) Definite (DEF), full criteria were met; (2) Probable (PROB), 3 to 4 criteria were met; (3) Possible (POSS), 1 to 2 criteria were met.

The Family Informant Schedule and Criteria (FISC; Mannuzza & Fyer, 1990) was used to collect family history data. The FISC is a structured interview used to determine the presence of DSM diagnoses in family members, based on information provided by one or more informants. The interview process begins by identifying all of the probands first- and second-degree relatives, which helps to increase efficiency and accuracy of data collection. Information assessed through the FISC closely parallels traditional diagnostic criteria, but tends to be less stringent to account for the lower specificity of informant reports.

The family history method has been shown to reliably diagnose most disorders when a structured interview, such as the FISC, is used. Andreasen et al. (1977) demonstrated good inter-rater reliability for psychotic and mood disorders, with kappa coefficients ranging from .80 to 1.0. A later meta-analysis of seven family history studies reported moderate to high inter-rater reliability of schizophrenia, mania, depression, and substance abuse, while anxiety disorders were less consistently diagnosed (Hardt &

Franke, 2007). The family history method is associated with high specificity for all disorders, but sensitivity tends to be lower and more variable across diagnoses (Andreasen et al., 1977; Andreasen et al., 1986; Fogelson et al., 2004; Hardt & Franke, 2007).

Broadly, this approach has greater sensitivity for broader versus narrower categories. For example, Andreasen et al. (1986) found that sensitivity was low for schizophrenia (31%), but considerably higher for psychotic disorders (69%). The family history method also tends to have greater sensitivity for disorders characterized by externalizing symptoms, such as those of bipolar disorder (59 – 100%), compared to internalizing symptoms, as expressed in depression (50 – 62%; Andreasen et al., 1986; Li et al, 1997). Generally, observable symptoms (e.g., mania, delusions) are more accurately reported by informants, while subtle signs (e.g., onset, duration) and symptoms (e.g., guilt, self-depreciation) tend to be identified with less precision.

Although the family history approach lacks the specificity and precision of the family study method, it is superior in terms of scope. That is, it assesses complete pedigrees, including relatives who are deceased or unavailable, thereby providing a more comprehensive picture of family psychiatric history.

Familial/Sporadic Distinction. The familial/sporadic distinction has been used to distinguish differences in neurobiology, cognitive functioning, and clinical features. Results of neuroimaging studies have evidenced differences in ventricular volume (Reveley, Reveley, & Murray, 1984; Schwarzkopf et al., 1991), subcortical white matter density (Zetzsche et al., 2008), laterality (Malaspina & Friedman, 1998; Roy, Flaum, Gupta, Jaramillo, & Andreasen, 1994), and neuronal activity (Malaspina & Harkavy-

Friedman, 2004) between familial and sporadic subtypes. Neurocognitive functioning has also been shown to differentiate groups. Greater impairments in attention, reasoning, and visual-motor ability are typically observed in familial cases (Erol, Bayram, Kosger, & Mete, 2012; Hallmayer et al., 2005; Sautter, McDermott, & Cornwell, 1994; Wolitzky et al., 2006), although conflicting data also exist (Chen, Lu, & Lung, 2011). In terms of clinical characteristics, it is well-established that patients with a family history of psychosis have a significantly earlier age-at-onset than those with no family history (Esterberg & Compton, 2012; Gorwood et al., 1995; Malaspina et al., 2000). Evidence on symptom differences has been much less consistent, as familial schizophrenia has been associated with greater negative symptom severity (Malaspina et al., 1996, 2000; Verdoux et al., 1996), lower negative symptom severity (Baron et al., 1992), and greater positive symptom severity (Kendler & Hays, 1982; Roy et al., 1994; Sautter & McDermott, 1994). While the family history method has yielded some compelling findings, inconsistencies among studies suggest that this approach may not adequately capture heterogeneity.

Broadening the Distinction. The subdivision of schizophrenia into familial and sporadic cases is founded in the assumption that genetic liability to schizophrenia is discrete. When this approach gained recognition in the 1970s, isolating a susceptibility gene for schizophrenia was a viable research target. However, contemporary models suggest that the genetic underpinnings of schizophrenia are highly diverse, involving interactions among various genes and environmental factors (Svrakic et al., 2013). Further, there is increasing evidence that genetic liability to psychiatric disorders is shared, which refutes the notion of schizophrenia-specific risk on which the

familial/sporadic distinction is based. Population-based studies evidence familial co-aggregation of schizophrenia, bipolar disorder, and major depressive disorder. Schizophrenia and bipolar disorder have the highest rate of family clustering (Aukes et al., 2012; Laursen & Labouriau, 2005; Mortensen, Pedersen, & Pedersen, 2010; Rasic, Hajek, Alda, & Uher, 2013), while a family history of major depression is associated with a two-times greater risk of schizophrenia than the general population (Aukes et al., 2012; Rasic et al., 2013).

Despite the utility of epidemiological research, the indiscriminate diagnostic groupings (e.g., bipolar with and without psychotic features) inherent to this approach may lead to an underestimation of familial transmission of symptoms that present across diagnoses. Cross-sectional studies have demonstrated clustering of psychosis in bipolar pedigrees (Potash et al., 2001), as well as familial aggregation of mood symptoms in schizophrenia and schizoaffective disorders (McGrath et al., 2004). These findings parallel pathophysiological evidence, as a recent study found that distinct genetic variants cluster within families and relate to the manifestation of specific symptoms (Hatzimanolis et al., 2013).

The significance of family history in the study of psychopathology has been increasingly supported by genetic and epidemiological research. This evidence also substantiates the use of family history as a clinical research strategy. However, in order to increase predictive power of family history, the traditional approach must be revised in accordance with contemporary evidence. Specifically, the definition should be expanded beyond the familial/sporadic distinction to account for a more dimensional view of family history. Presently, research exploring a broader definition of family history is limited.

Family history of affective illness has been demonstrated as a protective factor in schizophrenia, as it has been associated with better intellectual and neurocognitive functioning (Anglin et al., 2009), as well as better overall prognosis (Pope & Lipinski, 1978). A significantly greater prevalence of depressive symptoms has been evidenced in schizophrenic patients with familial depression (Babinkostova & Stefanovski, 2011; Kendler & Hays, 1983; McGrath et al., 2004; Subotnick et al., 1997).

Coding of Family History. Despite their similar functions, the family study and family history methods have unique strengths and weaknesses as research tools. Given the current focus on familial aggregation of psychopathology, heuristically, we sought to capture the breadth of family history, while maximizing methodological rigor. Thus, both SCID and FISC data were used to define the pattern of illness in families. To maximize the quality of data, the operationalization of family history was restricted to first-degree relatives. Information on second-degree relatives was excluded because: (1) significantly fewer second-degree relatives were assessed directly; (2) informant reports of family history tend to be less reliable for more distant relatives with whom they presumably have more limited contact.

Probands were assigned to one of four family history categories: (1) psychotic; (2) affective; (3) mixed; (4) other. Previous research has compared affective and psychotic family history groups, evidencing differences in age-at-onset (Kendler & Hays, 1983; McGrath et al., 2004), symptom presentation (Kendler et al., 2004; Peralta & Cuesta, 2007; Subotnik et al., 1997), and cognitive functioning (Anglin et al., 2009). While these findings support the use of family history subtypes, the specific criteria used for classification have been highly inconsistent across studies. Because the relative utility of

these different approaches has not been established, we chose to examine two separate definitions of family history, one that employs stringent criteria to maximize the specificity of diagnostic categories (Narrow) and a second that has lower thresholds for group inclusion and thus has greater sensitivity. Specific criteria for each system are outlined in Table 1.

Table 1
Family History Classification Criteria for Narrow and Broad Approaches

	Narrow	**Broad**
PSYCHOTIC (FH-PSY)	(a) At least one relative diagnosed with: ▪ Schizophrenia ▪ Psychosis NOS ▪ Delusional Disorder ▪ Schizoid/Schizotypal (Full Criteria)	(a) At least one relative diagnosed with: ▪ Schizophrenia ▪ Psychosis NOS ▪ Delusional Disorder ▪ Schizoid/Schizotypal (2+ Criteria)
AFFECTIVE (FH-AFF)	(a) At least one relative diagnosed with: ▪ MDD, Recurrent ▪ Bipolar disorder; **AND** (b) No family history of psychosis	(a) At least one relative diagnosed with: ▪ MDD, Single or Recurrent Episode ▪ Bipolar disorder; **AND** (b) No family history of psychosis
MIXED (FH-MIX)	(a) At least one relative diagnosed with: ▪ Schizoaffective disorder ▪ Bipolar psychosis; **OR** (b) Family history of: ▪ Psychosis (narrow criteria); **AND** ▪ Mood disorder (narrow criteria)	(a) At least one relative diagnosed with: ▪ Schizoaffective disorder ▪ Bipolar psychosis; **OR** (b) Family history of: ▪ Psychosis (broad criteria); **AND** ▪ Mood disorder (broad criteria)
OTHER (FH-O)	(a) Family history of: ▪ MDD, Single Episode ▪ Schizoid/Schizotypal Traits ▪ Other Axis I condition ▪ No Diagnoses	(a) Family history of: ▪ Other Axis I condition ▪ No diagnoses

Note: FH = family history; NOS = not otherwise specified; MDD = major depressive disorder;

Analysis Plan

Specified Model. The predicted model specified both latent factor and path analysis components. Thought disorder dimensions were specified as latent constructs. However, because the factor structure of the TDI has not been well-established, the measurement model was tested separately, prior to estimation of the full model. Six

alternative measurement models were tested based on: (1) the conceptually-based factors established by Johnston and Holzman (1979); (2) the empiric factors derived from Principal Components Analysis (Solovay et al., 1987); (3) a one-factor model. The best-fitting model was included in the structural analysis. The structural model included 10 hypothesized paths between two latent factors and five observed variables: family history, age-at-onset, sex, negative affect, and positive affect. The model depicted in Figure 1 has 39 estimated parameters and 40 degrees of freedom; however, the exact properties of the final model were determined by the results of the factor analyses.

Analysis Decisions. Data preparation and descriptive analyses were conducted using SPSS v22.0 (SPSS IBM, Armonk, NY, 2013). AMOS v22.0 (AMOS IBM, Armonk, NY, 2013) was used to test structural equation models (SEM). Statistical analyses were based on covariance matrices using maximum likelihood (ML) estimation, provided the assumptions of univariate and multivariate normality were met. Latent factors were scaled using the marker variable approach, which is suggested for hybrid models with endogenous factors (Kline, 2011; Sass, 2011). Analyses were carried out following a multistep process for estimating models with measurement and structural components (Anderson & Gerbing, 1988; Klein, 2011). The testing sequence included the following steps: (1) Evaluate whether the measurement model is consistent with the data; (2) Respecify the measurement model with empirically and conceptually supported modifications; (3) Test all omitted paths in the structural model and retain those that are significant; (4) Test all specified paths and correlations; (5) Estimate the full model.

Model Fit Criteria. Model fit was evaluated based on multiple fit indices, as has been recommended by several authors (Brown, 2006; Hu & Bentler, 1999; Kline, 2011).

The chi-square statistic is one indicator of model fit, in which a non-significant value indicates adequate fit. Despite its utility, the chi-square statistic is sensitive to large sample size, multivariate non-normality, and strong correlations among indicators, which have been associated with inflated Type I error rate (Kline, 2011). Given this limitation, additional goodness-of-fit indices were used in conjunction with chi-square to evaluate model fit, namely the Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and the Root Mean Square Error of Approximation (RMSEA).

CFI and TLI are incremental fit indices that estimate the relative improvement of the specified model compared to the independence (null) model (Kline, 2010). Both indices yield estimates ranging from 0 to 1.0. The TLI yields similar estimates as the CFI but imposes stricter penalties on model complexity. A value of .95 is generally accepted as a cutoff for good model-fit (Hu & Bentler, 1999), while values >.90 suggest adequate fit (Marsh, Hau, & Wen, 2004).

RMSEA is an absolute fit index that compares the specified model to a perfectly-fitting model. Estimates, which range from 0 to 1.0, indicate the proportion of variance unaccounted for, and thus smaller values are preferred. A value of .06 has been suggested as a cutoff for good model-fit, with a 90% confidence interval encompassing an approximate range of < .05 – .08 (Hu & Bentler, 1999). However, RMSEA has been criticized for penalizing models with small sample size and few degrees of freedom (Marsh, Hau, & Wen, 2004). To offset potential bias, the *p* of Close Fit (PCLOSE) statistic was interpreted in conjunction with RMSEA. PCLOSE tests the null hypothesis that RMSEA equals .05 and thus non-significant estimates (i.e., >.05) indicate a close-

fitting model. RMSEA can also be used to compare nested models along with the chi-square difference test (Sass, 2011).

The chi-square difference test was used to compare the relative fit of nested models. A significant difference indicates that the more parsimonious (i.e. > *df*) model is preferred. For non-nested models, comparisons were based on the Akaike Information Criterion (AIC) and Bayesian Information Criterion (BIC). Lower values of the AIC and BCC indicate better model-fit, while a discrepancy of 10 or greater is evidence of a significant difference between models (Burnham & Anderson, 2002).

Sample Size and Statistical Power. Given the complexity of structural equation modelling techniques, the stability of parameter estimates is reliant on large sample sizes (Kline, 2011). Unfortunately, there is no definitive approach for calculating sample size a priori. In practice, sample size requirements are based on model complexity, although exact guidelines are inconsistent. A generally accepted rule of thumb is 10 to 20 cases per estimated parameter (Kline, 2010). However, there is some evidence that as few as 5 cases per parameter is an acceptable ratio, particularly when using a robust estimation method, such as ML estimation (Tanaka, 1987). Based on these guidelines, the minimum sample size needed to estimate the proposed model is 175, although a more conservative requirement is 360 cases.

Preliminary Analyses.

***Demographic Characteristics*.** The sample includes 322 individuals diagnosed with schizophrenia (n = 103), schizoaffective disorder (n = 140), and bipolar disorder (n = 79). Demographic information is summarized in Table 2. Participants were predominantly white (91.9%) and had a mean age of 37.98 years (SD = 9.78), which was

consistent across diagnostic groups, (F = 2.77, p = .064. Men (n = 163, 50.6%) and women (n = 159, 49.4%) were equally represented in the overall sample. However, the sex distribution differed across diagnostic groups, with a significantly lower proportion of women diagnosed with SZ than SZA or BP. The Hollingshead Two-Factor Index (Hollingshead, 1957) was used to assess socioeconomic status (SES) based on parental education and income. Participants with schizophrenia had significantly lower SES than those with bipolar disorder; no differences emerged with respect to the SZA group, χ^2 = 7.69, p = .021. Compared to those with bipolar disorder, individuals with schizophrenia and schizoaffective disorder had significantly fewer years of education and lower verbal IQ scores (estimated from the Vocabulary subtest of the WAIS-III) (Wechsler, 1997).

Table 2
Demographic Characteristics of the Sample, Stratified by Proband Diagnosis

	Schizophrenia (n = 103)	**Schizoaffective (n = 140)**	**Bipolar (n = 79)**
Age, *M* (SD)	39.02 (9.62)	38.46 (9.39)	35.78 (10.41)
Education, *M* (SD)	13.73 (2.59)	14.19 (2.12)	15.38 (2.39)
Verbal IQ, *M* (SD)	99.33 (11.96)	100.89 (11.79)	106.58 (11.11)
Sex, *% male*	69 (67.0%)	65 (46.4%)	29 (36.7%)
Race, *n (%)*			
White	92 (89.3%)	130 (92.9%)	74 (93.7%)
Black	5 (4.9%)	5 (3.6%)	3 (3.8%)
Asian	2 (2.0%)	0 (0%)	1 (1.3%)
Multiracial/Other	4 (4.8%)	5 (3.6%)	1 (1.3%)
SES*, *n (%)*			
I – III	84 (82.2%)	123 (89.1%)	76 (96.2%)
IV – V	17 (16.8%)	15 (10.9%)	3 (3.8%)

Note: M = Mean; *SD* = standard deviation; IQ = Intelligence Quotient; SES = socioeconomic status
***** n = 318

***Clinical Characteristics*.** Clinical characteristics, stratified by diagnosis, are presented in Table 3. Participants had a mean age-at-onset of 23.49 (SD = 7.42), which was consistent across diagnoses, F=1.28, p = .278. However, because of age differences at the time of testing, the mean duration of illness was lower in BP compared to both

other groups ($F = 5.33$, $p = .005$). Individuals with SZ and SZA exhibited higher scores on the Brief Psychiatric Rating Scale (Overall & Gorham, 1962) than those with BP, $F=32.47$, $p < .001$, indicating significantly greater symptom severity. Global Assessment of Functioning (GAS; Endicott, Spitzer, Fleiss, & Cohen, 1976) scores were also notably lower in the SZ and SZA groups compared to BP, $F=55.46$, $p < .001$. Taken together, these results suggest that, on average, participants with SZ and SZA experienced more chronic and severe forms illness.

Table 3.
Clinical Characteristics of the Sample, Stratified by Proband Diagnosis

	Schizophrenia (n = 103)	**Schizoaffective (n = 140)**	**Bipolar (n = 79)**
GAS, *M* (SD)	35.65 (10.01)	38.64 (9.64)	51.06 (11.69)
Age-at-Onset, *M* (SD)	23.02 (6.89)	23.19 (6.75)	24.66 (9.05)
Duration of Illness, *M* (SD)	16.13 (10.15)	15.28 (9.43)	11.55 (9.58)
BPRS Total, *M* (SD)	50.17 (15.10)	46.36 (14.23)	34.32 (9.20)
Chlorpromazine Equiv., *M* (SD)	633.18 (432.34)[a]	631.00 (609.05)[b]	406.68 (741.83)[c]
Medication, n (%)			
None	0 (-)	2 (1.43)	6 (7.59)
Neuroleptics only	27 (26.21)	14 (10.0)	1 (1.27)
Neuroleptics/Antidepressants	24 (23.31)	55 (39.29)	20 (25.32)
Mood Stabilizer	0 (-)	1 (.714)	12 (15.19)
Mood Stabilizer/Neuroleptics	28 (27.18)	46 (32.86)	26 (32.91)
Anxiolytic	12 (11.65)	20 (14.29)	6 (7.59)
Antiparkinson	16 (15.53)	19 (13.57)	2 (2.53)

Note: M = Mean; *SD*= standard deviation; GAS = global assessment of symptoms

[a] N = 98
[b] N = 135
[c] N = 48

Family History Characteristics. One-hundred and ninety-two (59.6%) probands had at least one family member participate in the study. Table 4 provides a summary of the relatives interviewed by proband diagnosis. The BP group had the fewest family members participate per proband (56/79; 70.9%) compared to SZ (147%) and SZA (139%). Interestingly, considerably more parents of SZ and SZA participants were recruited than BP participants. A total of 400 first-degree relatives completed the SCID-I.

Of this group, 21 (5.25%) were diagnosed with a non-affective psychotic disorder (e.g., schizophrenia), 17 (4.25%) with an affective psychotic disorder (e.g., schizoaffective disorder), 77 (19.25%) with a major mood disorder (e.g., bipolar, recurrent major depression), and 30 (7.5%) with a minor mood disorder (e.g., single depressive episode, cyclothymia). One-hundred and thirty-nine relatives (34.75%) did not meet criteria for any Axis I disorder. In terms of Axis II disorders, 17 (4.25%) met full criteria (i.e. *Definite*) paranoid personality disorder, 16 (4.0%) for schizotypal personality disorder, and 3 (<1%) for schizoid personality disorder.

Table 4.
Relatives Interviewed, Stratified by Proband Diagnosis

	Schizophrenia	**Schizoaffective**	**Bipolar**
Mother, n (%)	40 (26.5)	54 (27.7)	4 (7.1)
Father, n (%)	23 (15.2)	37 (19.0)	3 (5.4)
Sister, n (%)	54 (35.8)	66 (33.8)	32 (57.1)
Brother, n (%)	31 (20.5)	34 (17.4)	16 (28.6)
Son/Daughter, n (%)	3 (2.0)	4 (2.1)	1 (2.0)
Total	**151 (37.56)**	**195 (48.51)**	**56 (13.93)**

Missing Values and Outliers.

All participants in the sample had full data for each of the 23 TDI categories. Complete Rorschach transcripts were available for all but 7 participants; 4 protocols were not audio-recorded (due to technical difficulties) and 3 could not be located. Because verbatim responses are required for the LIWC, these cases were excluded from analysis. Further, 8 participants did not provide family history information and 5 were missing age-at-onset values. Taken together, a total of 302 participants had complete data for all variables of interest.

TDI category scores were substantially skewed, reflecting the high frequency of zero usage for each type of thought disorder. Thus, factor analyses were based on

category scores that were: (1) weighted by their respective severity level; (2) controlled for number of responses; (3) mathematically transformed using a base-10 logarithmic function to approximate a normal distribution. Several categories could not be adequately corrected due to extreme infrequencies (neologism, idiosyncratic symbolism, contamination) and were excluded from analysis.

LIWC variables were moderately skewed, such that there were high concentrations of scores near zero and several extreme values at the upper-range. Extreme values were carefully inspected and deemed to reflect true measurement variance; thus, all outliers were retained for analysis (a more detailed description of extreme values is presented below). Square root transformations were employed to normalize distributions of LIWC variables.

Variable Descriptives.

Table 5.
Descriptive Data for Model Variables, M (SD)

	All (N = 302)	**Schizophrenia** (N = 96)	**Schizoaffective** (N = 133)	**Bipolar** (N = 73)
TDI				
Total Score	20.07 (21.85)	27.94 (29.96)	19.68 (16.94)	10.41 (10.42)
Idio. Verb.	4.50 (5.03)	5.68 (4.91)	5.08 (5.41)	1.90 (3.30)
Comb. Think.	3.33 (2.85)	3.37 (2.87)	3.69 (3.10)	2.62 (2.18)
Responses	19.71 (6.96)	19.20 (8.20)	19.95 (6.81)	19.96 (5.27)
LIWC				
NegAff	.388 (.381)	.424 (.537)	.383 (.306)	.351 (.229)
Anxiety	.039 (.076)	.042 (.088)	.041 (.077)	.032 (.053)
Anger	.154 (.203)	.171 (.274)	.151 (.163)	.139 (.156)
Sadness	.064 (.103)	.074 (.140)	.061 (.078)	.055 (.082)
PosAff	1.50 (.709)	1.49 (.778)	1.50 (.675)	1.48 (.686)
PREDICTORS				
Age-at-Onset	23.45 (7.33)	22.99 (6.87)	23.16 (6.73)	24.56 (8.80)

Note: TDI = Thought Disorder Index; NegAff = negative affect; PosAff = positive affect

The FISC interview was administered to 398 relatives and 294 probands. Of the 130 probands for whom no relatives were interviewed, 122 completed the FISC and were

deemed to be reliable informants. The remaining 8 probands were unable to provide accurate responses and thus, family history could not be assessed. FISC data were collected for 1,041 first-degree relatives, in addition to those family members who completed the SCID. Collectively, family history information was obtained for 1,435 relatives, the equivalent of 4 to 5 relatives per proband. Using these data, probands were assigned to one of four family history groups: (1) psychosis; (2) mood; (3) mixed; (4) other. As described above, two classification schemes were examined, one with greater specificity (Narrow) and one with greater sensitivity (Broad). Table 6 presents the frequency distributions for each of these systems, stratified by proband diagnosis. As intended, in the Narrow system, a noticeably higher proportion of cases fall into the Other category, with fewer assigned to the Psychosis, Mood, and Mixed groups.

Table 6.
Family History Group Frequencies, n (%)

	NARROW				BROAD			
Category	**SZ** (n=97)	**SZA** (n=133)	**BP** (n=75)	**Total**	**SZ** (n=97)	**SZA** (n=133)	**BP** (n=75)	**Total**
Psychosis	15 (15.5)	15 (11.3)	6 (8.0)	50 (16.4)	18 (18.6)	14 (10.5)	4 (5.3)	36 (11.8)
Mood	35 (36.1)	54 (40.6)	37 (49.3)	96 (31.5)	20 (20.6)	27 (20.3)	25 (33.3)	103 (33.8)
Mixed	8 (8.2)	27 (20.3)	9 (12.0)	74 (24.3)	32 (33.0)	64 (48.1)	27 (36.0)	92 (30.2)
Other	39 (40.2)	37 (27.8)	23 (30.7)	85 (27.9)	27 (27.8)	28 (21.1)	19 (25.3)	74 (24.3)

LIWC Validity.

While the LIWC has been demonstrated as a valid measure of affective state in the general population (Groom & Pennebaker, 2002), its use in psychiatric samples has not been well-established. Further, this is the first study, to the best of our knowledge, to use lexical analysis to examine emotion word use in Rorschach responses. Thus, preliminary analyses were conducted to establish that the LIWC was indeed tapping into the construct of affective state in the current sample. Specifically, we assessed: (1) the

convergence of LIWC domains with established measures of affect (convergent validity); (2) differences in affect scores across diagnostic groups (concurrent validity); (3) the characteristics of extreme outliers (extreme value analysis).

Convergent Validity. Clinical ratings of depressed mood (BPRS; Brief Psychiatric Rating Scale) and scores of self-reported social and physical anhedonia (CAS; Chapman Anhedonia Scales) were collected for a subset of participants (N = 191). In theory, the constructs tapped by these instruments are similar to that of the LIWC and thus should be observable statistically. However, given the differences in content and methodology, we expected only modest correlations between LIWC domains and clinical measures. As seen in Table 7, Social Anhedonia was significantly associated with overall Negative Affect ($r = .199$, $p < .05$), as well as the subdomain of Anxiety, while physical anhedonia correlated significantly with Anger subdomain. Clinician-rated depression was unrelated to all LIWC categories.

While this could be due to the restricted range of the BPRS Depression item, the absence of significant correlations is consistent with previous research demonstrating that clinician ratings of affect are divergent from measures of subjective experience and outward expression (Halari, Mehrotra, Sharma, & Kumari, 2006). This is supported by the current results, in which BPRS Depression did not correlate significantly with self-report anhedonia scales. Consistent with expectations, Positive Affect was unrelated to clinical measures of negative affect, which lends support for discriminate validity. However, Positive Affect also correlated modestly with Negative Affect; it is unclear whether this is indicative of poor construct validity or methodological overlap (i.e. word count). Overall, results suggest that the LIWC is a valid measure of negative affect in the

current sample, while further evidence is needed to determine the validity of Positive Affect.

Table 7
Correlations among LIWC Categories and Clinical Measures

	LIWC Categories						Clinical Measures		
	Affect	**PosAff**	**NegAff**	**Anx**	**Anger**	**Sad**	**BPRS**	**SocAnh**	**PhyAnh**
Affect	1								
PosAff	.916**	1							
NegAff	.512**	.165*	1						
Anxiety	.354**	.102	.674**	1					
Anger	.361**	.082	.770**	.335*	1				
Sadness	.268**	.165*	.378*	.153*	.160*	1			
BPRS Dep	-.076	-.076	.020	-.001	.031	.053	1		
SocAnh	.112	.027	.191**	.166*	.123	.020	.031	1	
PhysAnh	-.005	-.086	.118	.050	.179*	-.070	.107	.572**	1

Note: LIWC = Linguistic Inquiry and Word Count; PosAff = positive affect; NegAff = negative affect; Anx = anxiety; BPRS = Brief Psychiatric Rating Scale; SocAnh = Chapman Social Anhedonia Scale; PhyAnh = Chapman Physical Anhedonia Scale

Listwise N = 191
* $p < .05$
** $p < .01$

Concurrent Validity. Construct validity was explored further by assessing differences in LIWC scores across diagnostic groups. Because the valence (i.e. depression, mania) and severity (e.g., criteria for duration/intensity) of affect are characteristics that differentiate disorders, we used diagnosis as a criterion for affective state. To increase the specificity of groups, individuals with past mood disorders (e.g., BP I, in full remission) or current mixed episodes were excluded from analysis. It was expected that those diagnosed with depression (i.e. SZA depressed type, BP MRE depressed, or SZ with MDD) would have higher Negative Affect, Anxiety, and Sadness scores and lower PosEmo scores than those without. Affective differences have also been evidenced among subtypes of schizophrenia, with negative affect significantly elevated in those with chronic compared to acute illness (Suslow, Roestel, Ohrmann, & Arolt, 2003).

Thus, we expected that individuals diagnosed with chronic SZ would exhibit greater Negative Affect, Anger, and Sadness than those with recent-onset or residual symptoms.

Mean scores of LIWC variables for diagnostic groups are presented in Table 8. Results of a one-way ANOVA indicated significant differences among groups on Total Affect, $F(3, 287)=3.34$, $p=.02$, Negative Affect, $F(3, 287)=5.51$, $p=.001$, Anger, $F(3, 287)=4.05$, $p=.008$, and Sadness, $F(3,287)=3.95$, $p=.009$. Post-hoc pairwise comparisons revealed that SZ chronic group exhibited significantly greater Negative Affect, Anger, and Sadness than the SZ and Manic groups, but did not differ from the Depressed group. No significant differences were found between the Manic and Depressed groups.

Table 8.
Means and Standard Deviations for LIWC Affect Variables Across Diagnostic Groups

LIWC	**SZ** (N = 42)	**SZ, Chronic** (N = 57)	**Manic** (N = 144)	**Depressed** (N = 48)
Total Affect*	1.71 (.743)[1]	2.24 (1.22)[1]	1.91 (.748)	1.89 (.855)
PosAff	1.33 (.662)	1.58 (.834)	1.47 (.654)	1.39 (.706)
NegAff*	.283 (.211)[2]	.563 (.659)[2]	.352 (.251)[2]	.420 (.390)
Anxiety	.035 (.048)	.049 (.107)	.032 (.056)	.061 (.101)
Anger*	.108 (.120)	.243 (.351)	.143 (.161)	.161 (.189)
Sadness*	.039 (.055)	.099 (.173)	.055 (.074)	.050 (.070)

* <.05
[1] SZ, Chronic > SZ
[2,3,4] SZ Chronic > SZ, Manic

Extreme Value Analysis. To supplement the empirical assessment of construct validity, we conducted a qualitative analysis of extreme scores (>3.2 SD above the mean) on each LIWC variable. Because, in theory, these values represent the most extreme affective states in the sample, we would expect them to be highly salient in the respective Rorschach protocols. Table 9 presents a summary of extreme-scoring cases for each LIWC variable. Across Negative Affect categories, extreme scores were found to consistently correspond to protocols in which a striking degree of negative affect was

expressed. This group of individuals exhibited a strong tendency to respond to the task in a self-referential manner. Several described feeling frightened, saddened, or overwhelmed by the blot, typically because of distressing personal associations. For example, in describing a percept, one woman (#5538) stated, "When I was younger, I was sexually abused... and this like reminds me... of… These are my feet, this is my crotch. An' that's the beast in there. It's really embarrassing." Others gave responses that were personalized and affectively elaborated, but had little bearing on the task at hand (see #3726 in Table 9). There was some indication that LIWC scores accurately differentiated dimensions of negative affect. For instance, the direct expression of fear, embarrassment, and worry was pronounced for those with high scores on Anxiety. However, there were other instances in which extreme-value protocols appeared to reflect generalized distress instead of specific dimensions of negative affect (e.g., #5313). Further, the range of affect expressed in protocols was often quite broad, with some participants exhibiting extreme scores on more than one dimension. These observations suggest that, in the current sample, Negative Affect is a more robust and valid measure of negative affect than the individual subdomain scores.

Consistent with convergent and concurrent validity analyses, the qualitative examination of extreme-values did not provide compelling support for the construct validity of Positive Affect. There were two extreme-scoring cases in this category, one of which did convey strong positive feelings in reaction to the stimuli (#3099). The second cases also had extreme scores on Negative Affect, Anxiety, and Anger (#8900). This individual's style of responding on the Rorschach can be best characterized as paradoxical, offering descriptions such as, "that could be something at peace or

something at war" and "peaceful animals just show-, or peaceful people just showing their enemies." Despite the high frequency of both Positive Affect and Negative Affect words, the overall tone of this protocol is negative. This pattern of scores was observed in several other cases, whose Positive Affect and Negative Affect values were both significantly elevated (i.e. 2.5 – 3.2 SDs above the mean), although they did not meet criteria for extreme values.

Table 9.
Qualitative Analysis of Extreme Values for LIWC Categories

ID	Value (z)	Rorschach Excerpts
NEGATIVE AFFECT		
8900	3.61 (8.50)	▪ Could be uh... somebody at war. (Um hm.) 'Cause it doesn't look happy to me... looks like it gets worse.
3726	3.41 (7.98)	▪ Your orders were to kill on sight and.. and level the whole Dresden country to ashes. An' that's what they did to me. ▪ And people were using me in nineteen seventy five. (People were using you?) Yes, the key year for the, for the battle of the planet. And.. I was caught in the crossfire of it.
5538	2.26 (4.94)	▪ Kinda looks like two people havin' a conversation. But it's.. mm.. bad. Their hearts are torn out. ▪ Well there's two people.. standin' there.. the part in the red.. in the red.. in the middle.. sorta tearin' out each other's hearts. I dunno. I could be wrong
ANXIETY		
8900	.71 (8.60)	▪ These are scary, they're, they're pictur-, they're pictures of uh.. (SIGHS) That could be something at peace or something at war, I don't know. ▪ An' the doctor do-, doctor has to probably put all, together all these... interpretations because... isn't that a little too overwhelming to look at a-, every day, for everybody?
5538	.57 (6.82)	▪ When I was younger, I was sexually abused... and this like reminds me... of… These are my feet, this is my crotch. An' that's the beast in there. It's really embarrassing. That's what it reminds me of. I'm sorry. ▪ I don't know. I don't know. I don't do so well on these... I don't wanna screw anything up.
9318	.43 (5.02)	▪ This is scary. I'd be scared to find out what this really means. [I'm] findin' the dark things.
ANGER		
8900	1.80 (8.62)	▪ Well, something was on fire, but it wasn't dead, and they want like, g-, they want, the devil wants most, a lotta people dead.
3726	1.58 (7.12)	▪ ...an' a man gave me a book, an' then somethin' smashed through my phone. (Hm.) An'.. I think it was supposed, supposed to kill. Didn't kill me. It's a very evil place. (What's evil about it?) It's evil, just take my word.

2371	.84 (3.43)	▪ Anonymity. (Anonymity?) Yeah. (PP) I keep seeing erotic imagery, and it's makin' me sick.
SADNESS		
3501	1.10 (10.21)	▪ Tears... the tears were about the same distance from my eyes as the uh.. parts that looked like tears... made me sad looking at that one.
5313	.44 (3.71)	▪ Looks like two people coming together in a violent collision... looks like blood
3726	.42 (3.51)	▪ 'Cause the living EC Master.. does want, doesn't want me to have any happiness. He wants me kneel, kneeled down... to a rock.
POSITIVE AFFECT		
8900	4.09 (3.76)	▪ I liked that one, I liked that one. Completely happy, that's when you're a child, play the violin-- ... That's a cello, an' uh.. they're tryin' to bring, if there's hate, the hate is subdued by uh, by music. ▪ Um.. it's peaceful to me. It has color in it, lighter colors, like discord, like the fighting is gonna stop someday.
3099	3.93 (3.53)	▪ A spaceship. That's a great-lookin' spaceship. Not a Vulcan spaceship but ... definitely a nice spaceship. ▪ Sounds like a parade or something. That's a good feeling that gives me. Wow. Good feelings. I like that one. Great feelings. It's very emotional for me

Note: ID = Participant ID#, Value = LIWC Raw Score; Z = z-score; Dx = Axis I Diagnosis; SZ = Schizophrenia; Chron = Chronic; SZA = Schizoaffective Disorder; BP = Bipolar
Text in parentheses indicates examiner comment

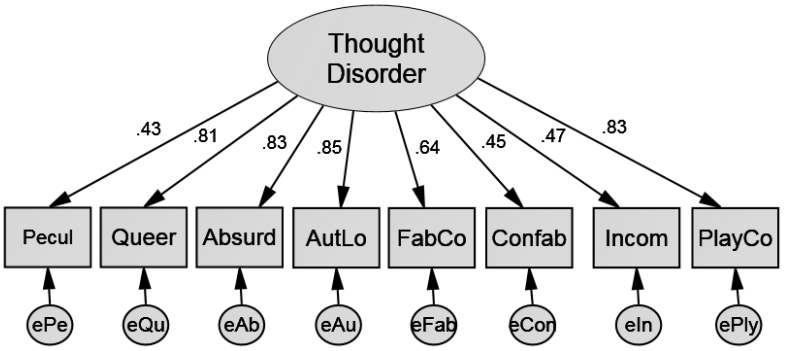
Summary. Taken together, results of these analyses demonstrate Negative Affect as a construct valid measure, which taps into the latent domain of negative affect. Results did not provide compelling support for the validity of Positive Affect in the current sample. Given that Positive Affect has been shown to be valid in research on non-clinical samples, it is possible that the scale functions differently in a psychiatric population. Discrepant findings may also relate to the unique methodology of the current study, as language elicited by the Rorschach is likely quite different from other sampling approaches (i.e. structured interviews). As discussed elsewhere, the task itself tends to "pull for" negative affect, which offers another possible explanation for the functional differences between LIWC scales. Regardless of the explanation, Positive Affect was excluded from further analyses.

Primary Analyses

Measurement Model

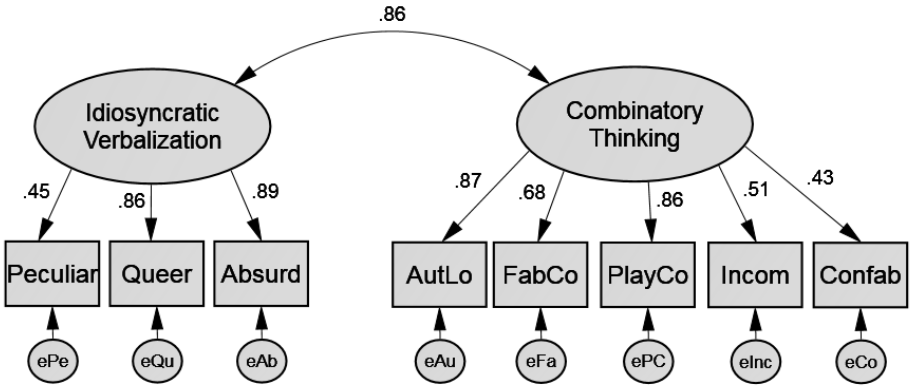
Model 1. Model 1 was estimated in the full sample, results of which indicated poor model-fit [χ^2 (20) = 117.28 p <.001, TLI = .882, CFI = .916, RMSEA = .127, RMSEA CI_{90} = .105 - .150, PCLOSE < .001]. Large residual covariances were observed among measured variables. However, given the theoretical basis of this model (i.e., one unitary factor explaining TD), attempts to respecify the model were not made.

Model 1



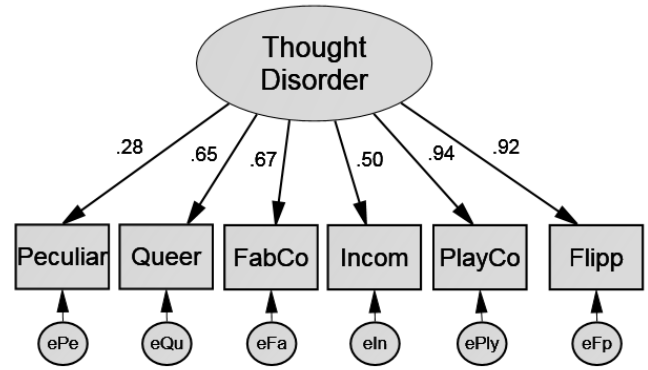
Model 2. Model 2 yielded marginal fit between implied estimates and observed data [χ^2 (19) = 61.93, p <.001, TLI = .945, CFI = .963, RMSEA = .087, RMSEA CI_{90} = .063 - .111, PCLOSE = .006]. However, modification indices revealed large residual covariances between Peculiar and Confabulation scores. The respecification of a correlation between ePe and eCon resulted in a significant improvement to model fit. However, factors were highly correlated (r = .86), suggesting that they are not separable constructs.

Model 2



Model 3. Model 3 yielded a fair degree of consistency between model-implied estimates and observed data [χ^2 (9) = 37.38, p < .001, TLI = .946, CFI = .967, RMSEA = .102, RMSEA CI_{90} = .070 - .137, PCLOSE = .006]. Large residual covariances were observed between eQu and Pe, which is consistent with the specified 2-factor PCA model (Model 4).

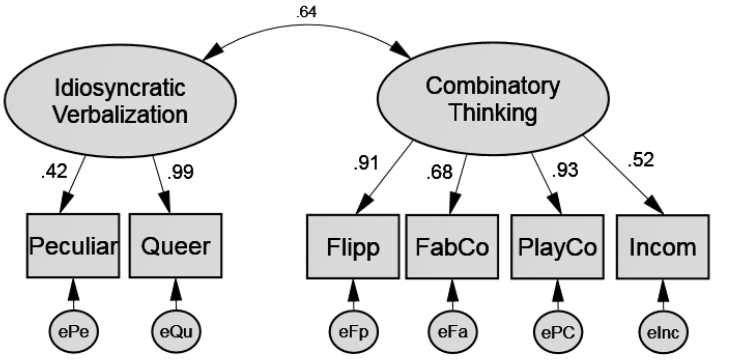
Model 3



Model 4. The 2-factor PCA-derived model was a close fit to the observed data [χ^2 (8) = 3.69, p = .884, TLI = 1.01, CFI = 1.00, RMSEA = .00, RMSEA CI_{90} = .00 - .32, PCLOSE = .985]. Residual covariances among indicators were insignificant. There was a modest correlation between factors (r = .64), indicative of related yet separable factors.

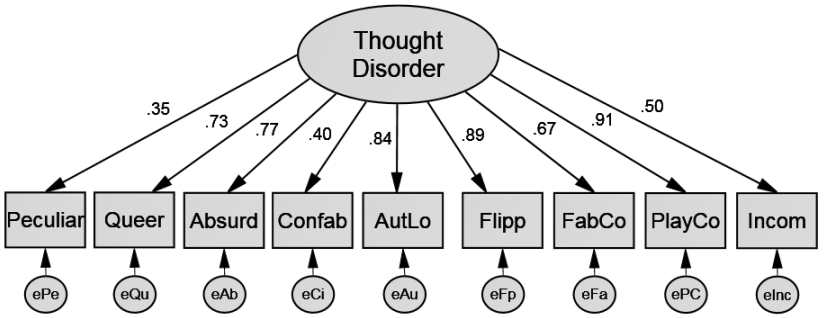
Despite statistical support for this model, it has fewer than three indicators per factor, which increases the likelihood of empirical under-identification, meaning that estimates may be biased or invalid.

Model 4



Model 5. Model 5 was a poor fit to the observed data [χ^2 (27) = 211.90, p < .001, TLI = .846, CFI = .884, RMSEA = .151, RMSEA CI_{90} = .132 - 170, PCLOSE < .001]. Specifically, there were large residual covariances were among several indicators, including Playful Confabulation and Flippant, as well as Absurd and Queer.

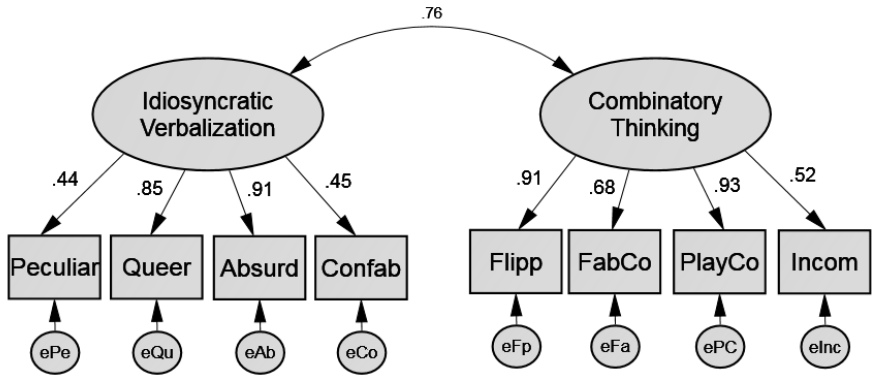
Model 5



Model 6. The specification of Model 6 was informed by estimates from the corresponding one-factor model (Model 5), as well as results of the PCA and

conceptually-based models. Model 6 yielded estimates that were highly consistent with observed data [χ^2 (19) = 26.66, p = .767, TLI = .991, CFI = .994, RMSEA = .035, RMSEA CI_{90} = .00 - .065, PCLOSE = .767]. The correlation between factors was strong (r = .76), but indicative of separable constructs.

Model 6



Model Comparison. Of the six models tested, there were two (Model 4 and Model 6) that demonstrated an adequate degree of fit to the data. Statistical comparison of these models based on AIC and BIC criteria (Table 10), indicates that Model 4 is preferred over Model 6. The strict penalty for model complexity inflicted by these estimates should be taken into consideration, given the considerable difference in number of parameters between the two models. Further, Model 4 does not meet minimum guidelines for empirical identification due to the number of indicators per factor, which increases the risk of unstable estimates (Brown, 2006; Kline, 2011). This risk may be exacerbated when estimated within the more complex structural model. Because Model 6 was a good fit to the data and met all conditions for theoretical identification, it was used to estimate the full model.

Table 10.
Statistical Comparison of TDI Factor Models

	χ^2	*df*	TLI	CFI	RMSEA (90% CI)	P_{CLOSE}	Δ RMSEA	χ^2_{DIFF} (*df*)	AIC	BIC
Model 1	117.28	20	.882	.916	.127 (.105 - .150)	<.001	–	–	–	–
Model 2	54.89	19	.956	.970	.077 (.053 - .101)	.031	.050	62.39 (1)***	88.89	153.06
Model 3	38.77	9	.942	.965	.104 (.072 - .139)	.004	–	–	62.78	119.41
Model 4	5.16	8	1.01	1.00	.00 (.00 - .47)	.959	.104	33.60 (1)***	31.16	80.23
Model 5	205.21	27	.849	.887	.147 (.129 - .167)	<.001	–	–	241.21	308.18
Model 6	26.66	19	.991	.994	.035 (.00 - .065)	.767	.112	178.55 (8)***	60.66	124.83

Note: χ^2 = chi-square test; *df* = degrees of freedom; TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; 90% CI = 90% confidence interval; P_{CLOSE} = *p* of Close Fit; χ^2_{DIFF} = Chi-Square Difference Test; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion
** < .01
*** < .001

Structural Model

The structural model included a series of analyses, which were carried out sequentially. Specifically, we: (1) examined the comparative sensitivity of family history groupings; (2) tested omitted paths; (3) tested specified paths; (4) evaluated the overall model; (5) examined covariates; (6) evaluated the relative utility of categorical diagnosis within the baseline model.

Comparative Sensitivity of Family History Coding Schemes. To evaluate the sensitivity of family history categories to detect differences in clinical characteristics we: (1) conducted multiple-group path analysis to test invariance of structural paths in familial and sporadic groups, comparing across Broad and Narrow schemes; (2) assessed the relative effects of different family history groupings on endogenous variables using effect coding.

Multiple Group Analysis. We assessed whether differences in structural relationships between familial (i.e. family history of psychosis) and sporadic (i.e. no family history of psychosis) groups were better captured using Narrow or Broad family

history criteria. Path coefficients were calculated separately for each familial and sporadic group and the magnitude of differences were compared across coding systems. As presented in Table 11, the relationship between Negative Affect and Idiosyncratic Thinking differed as a function of family history using both Narrow and Broad criteria. Specifically, greater Negative Affect was associated with more severe Idiosyncratic Thinking for those with a family history of psychosis, but not those without. Earlier age-at-onset was also predictive of higher Idiosyncratic Thinking scores in the familial, but not sporadic, group. While this trend was consistent across coding approaches, the moderation effect did not reach statistical significance when using the Broad criteria.

Table 11
Comparative Sensitivity of Family History Coding Schemes

	Narrow					Broad				
	Psychosis (n=124)		No Psychosis (n=181)		Pairwise Comp	Psychosis (n=159)		No Psychosis (n=146)		Pairwise Comp
Parameter	**Est.**	**S.E.**	**Est.**	**S.E.**	***z***	**Est.**	**S.E.**	**Est.**	**S.E.**	***z***
Sex → AAO	.139	1.25	.092	1.13	–	.112	1.07	.099	1.32	–
AAO → NegAff	-.019	.007	-.031	.003	–	-.025	.006	-.004	.003	–
Sex → NegAff	.012	.093	.138	.039	–	.063	.076	.097	.042	–
NegAff → IV[a, b]	.407***	.044	.036	.039	-2.63***	.370***	.041	-.016	.038	-3.04***
NegAff → CT	.166	.025	-.018	.046	–	.135	.026	-.013	.05	–
AAO → IV[c]	-.248***	.003	-.044	.001	2.14**	-.171***	.003	-.093	.001	–
AAO → CT	-.120	.002	-.053	.002	–	-.089	.002	-.073	.002	–
Sex → IV	-.129	.038	-.035	.021	–	-.135	.034	.001	.019	–
Sex → CT	-.097	.026	.019	.024	–	-.092	.024	.035	.026	–

Note: Est. = standardized estimate; S.E. = standard error; *z* = *z*-score; FH = family history; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking
** < .01
*** < .001

Effect Coding. We compared different family history groupings, in terms of the magnitude of their associations with model variables, namely Age-at-Onset, Negative Affect, Idiosyncratic Thinking, and Combinatory Thinking. Effects coding was used to assess differences among (1) Non-affective psychosis; Mood/Other; Affective psychosis;

(2) All psychosis; Other; Mood only; (3) All psychosis; Mood only/Other for both Broad and Narrow definitions of family history. Based on existing literature, we expected family history to account for variability in Age-at-Onset, Negative Affect, and Idiosyncratic Thinking. Table 12 presents the standardized effects, goodness-of-fit statistics, and relative-fit indices for each of the six models estimated. No differences were found among affective psychosis, mood/other, and affective psychosis (B1, N1) regardless of whether Broad or Narrow criteria were used. The familial/sporadic distinction (B3, N3) explained significant variability in Negative Affect, with higher scores associated with family history of psychosis using both Narrow and Broad criteria. The second family history variable, which drew comparisons among affective / non-affective psychosis, other, and mood categories, captured no differences when Narrow criteria were used. However, when defined by Broad criteria, this set of contrasts explained significant variability in both Age-at-Onset and Negative Affect. Further, it yielded the strongest correlation (albeit, not statistically significant) with Idiosyncratic Thinking of the six family history variables. While differences in fit statistics were negligible overall, the corresponding B2 model yielded the lowest chi-square, AIC, and BIC values, as well as the highest TLI and CFI values, all of which are associated with goodness-of-fit. Given these findings, all subsequent analyses of family history were based on B2.

Table 12
Family History Effect Coding

	FH	→AAO	→NA	→IV	→CT	χ^2 (*46*)	TLI	CFI	RMSEA (90% CI)	P_{Close}	AIC	BIC
Broad	B1	-.052	.041	-.015	.051	100.08	.941	.959	.062 (.046 - .079)	.109	164.08	283.13
	B2	.118*	-.123*	-.070	.021	97.68	.944	.961	.061 (.044 - .078)	.137	161.69	280.74
	B3	.047	-.127*	-.047	.029	98.87	.943	.960	.061 (.045 - .078)	.123	162.87	281.92
N	N1	-.079	-.005	-.005	.051	101.47	.940	.958	.063 (.046 - .080)	.095	165.47	316.52

N2	.048	-.103	-.030	.028	98.25	.943	.961	.061 (.044 - .078)	.139	162.25	281.30
N3	.040	-.139*	-.067	.007	100.11	.942	.959	.062 (.046 - .079)	.109	164.11	283.16

Note: FH = family history; DSM Dx = DSM-IV diagnosis; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking; χ^2 = chi-square test; TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; P_{CLOSE} = p of Close Fit; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion

FH 1 = Psychosis (-1); Mood/Other (0); Affective Psychosis (1)
FH 2 = All Psychosis (-1); Other (0); Mood Only (1)
FH 3 = All Psychosis (-1); No Psychosis (1)

* < .05
** < .01
*** < .001

Baseline Model

Omitted Paths. Following the sequence recommended by Bollen (1989), the five paths omitted from the a priori model were tested first. As shown in Table 13, none of these parameters reached statistical significance and thus were not retained in the model.

Table 13
Estimation of Omitted Paths

Parameter	**Standardized Estimate**	**S.E.**	***p*-value**
AAO → NegAff	-.014	.003	.810
Sex → FH	.047	.094	.409
Sex → NegAff	.067	.044	.238
Sex → IV	-.089	.021	.140
Sex → CT	-.038	.018	.517

Note: S.E. = standard error; FH = family history; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking

Specified Paths. We then evaluated the nine structural paths that were specified a priori. Table 14 presents the standardized estimates and significance levels for each of these parameters. As predicted, family history of psychosis was significantly associated with earlier Age-at-Onset and more intense Negative Affect in probands. Interestingly, although family history had no direct effect on either TD domain, earlier Age-at-Onset and greater Negative Affect were significant predictors of Idiosyncratic Thinking

severity. None of these variables (i.e. family history, Age-at-Onset, or Negative Affect) explained a significant proportion of variance in Combinatory Thinking. While we hypothesized a positive association between Positive Affect and Combinatory Thinking, this effect could not be formally evaluated. Consistent with expectations, there was a modest association between sex and Age-at-Onset (male → earlier Age-at-Onset), although this effect did not reach statistical significance.

Table 14.
Estimation of Specified Paths

Parameter	**Standardized Estimate**	**S.E.**	***p*-value**
Sex → AAO	.104	.838	.066
FH → AAO*	.118	.510	.038
AAO → IV*	-137	.001	.026
AAO → CT	-.083	.001	.164
FH → NegAff*	-.126	.027	.028
FH → IV	-.074	.013	.217
FH → CT	.024	.011	.686
NegAff → IV**	.273	.021	<.001
NegAff → CT	.092	.023	.122
FH ↔ Sex	.047	.024	.410

*Note :*S.E. = standard error; FH = family history; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking

Model Fit. The fit of the overall model was then evaluated. Fit statistics indicated poor convergence between implied parameters and observed data [χ^2 (57) = 368.93, p < .001, TLI = .744, CFI = .813, RMSEA = .134, RMSEA CI_{90} = .121 - .147, PCLOSE = <.001], due largely to substantial residual covariance among latent factors Idiosyncratic Thinking and Combinatory Thinking. The respecification of correlated disturbance terms resulted in a significant improvement to model fit [χ^2 (56) = 142.63, p = .009, TLI = .928, CFI = .948, RMSEA = .071, RMSEA CI_{90} = .057 - .086, PCLOSE = .009, Δ RMSEA = .063, χ^2_{DIFF} (1) = 226.30]. Figure 2 depicts the final model, with standardized estimates and significance levels presented for measurement and structural parameters.

Figure 2.
Final SEM Model with Standardized Estimates

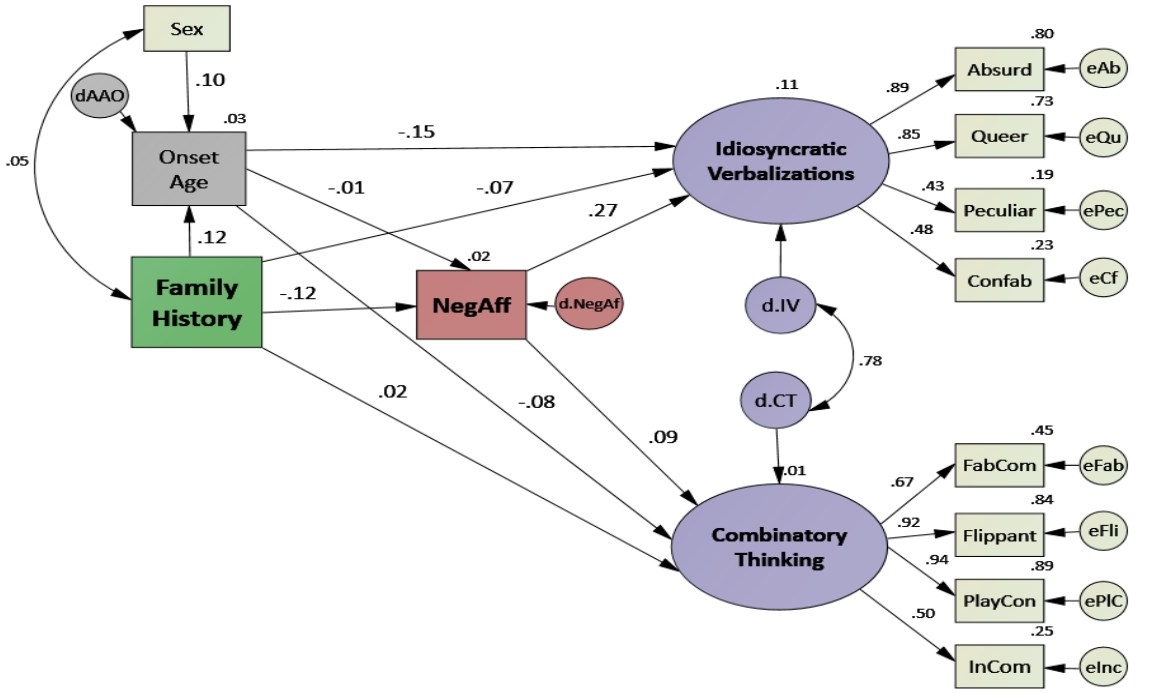


Table 15.
Model Fit Indices for SEM Model

	χ^2	*df*	TLI	CFI	RMSEA (90% CI)	P_{CLOSE}	Δ RMSEA	χ^2_{DIFF} (1)
Specified Model	368.93	57	.744	.813	.134 (.121 - .147)	<.001	–	–
d.IV ↔ d.CT	142.63	56	.928	.948	.071 (.057 - .086)	.009	.063	226.30

Note: χ^2 = chi-square test; *df* = degrees of freedom; TLI = Tucker-Lewis Index; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation; P_{CLOSE} = p of Close Fit; χ^2_{DIFF} = Chi-Square Difference Test

Moderating Effect of Sex

To assess whether structural relationships among variables differed as a function of sex, we estimated parameters for men and women separately and compared them using the Critical Ratio of Differences test. Table 16 presents standardized path coefficients for each group. Of the nine pairwise comparisons conducted, the direct effect of Age-at-Onset on Negative Affect was the only parameter moderated by sex. For men, later

illness onset was associated with greater negative affect, while the opposite was true for women: earlier onset predicted more intense negative affect.

Table 16
Pairwise Comparisons of Structural Parameters by Sex

	Males			Females			Pairwise Comparison	
Parameter	**Estimate**	**S.E.**	***p***	**Estimate**	**S.E.**	***p***	**C.R.**	**Difference**
FH → AAO	.111	.581	.160	.127	.845	.122	.487	.611
AAO → NegAff	.129	.069	.103	-.114	.055	.167	-2.14	-2.88***
AAO → IV	-.141	.003	.101	-.123	.001	.161	.754	1.03
AAO → CT	-.099	.002	.241	-.078	.002	.357	.390	.711
FH → NegAff	-.159	.036	.044	-.097	.041	.241	.450	.511
FH → IV	-.120	.019	.161	.012	.014	.885	1.21	1.21
FH → CT	-.045	.015	.596	.099	.016	.241	1.21	1.21
NegAff → IV	.209	.044	.020	.363	.040	.002	.409	.791
NegAff → CT	.059	.033	.484	.127	.032	.132	.542	.737

Note: FH = family history; DSM Dx = DSM-IV diagnosis; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking
* < .05
** < .01
*** < .001

Covariate Analysis

Because previous research has indicated age, SES, education, and chlorpromazine equivalent as possible covariates of TD, we estimated the effects of these variables on the baseline model. Overall, the inclusion of covariates did not significantly change the nature of structural relationships among model variables. Negative Affect had a modest, positive association with CPZ, but was independent of age, SES, and education. No significant effects were found between covariates and TD domains, with the exception of a small, negative correlation between SES and Combinatory Thinking. We speculated that this relationship was spurious, reflecting variance in verbal fluency common to these variables. That is, verbal fluency is strongly related to SES and was applied as a correction to TDI category scores. Thus, we re-estimated this effect after controlling for

speech fluency (i.e. word count), which substantially attenuated the effect of SES on Combinatory Thinking (r = .08, p = .124).

Affective State and Thought Disorder

Consistent with our hypothesis, the intensity of negative affect significantly predicted the severity of Idiosyncratic Thinking, but was unrelated to Combinatory Thinking. Specifically, each 1-SD increase in Negative Affect was associated with a .273-SD increase in Idiosyncratic Thinking, after controlling for all other variables in the model. Said differently, Negative Affect accounted for 7.45% of the variance in Idiosyncratic Thinking. Unfortunately, we were unable to examine the association between Combinatory Thinking and positive affect due to validity issues with the LIWC Positive Affect category.

Family History

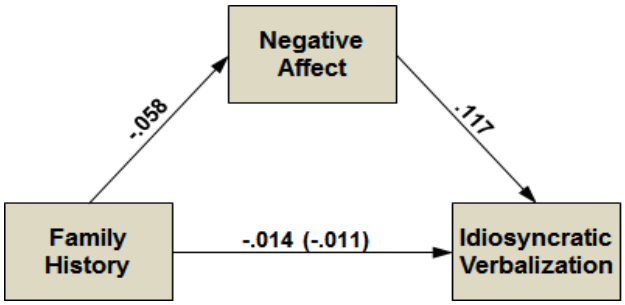
The model sought to address the extent to which family psychiatric history accounted for variability in clinical characteristics. Our prediction that family history of psychosis would be associated with more severe Idiosyncratic Thinking was not supported by the data. In fact, family history was unrelated to both qualitative and quantitative variability in TD. With respect to differences in affective state, family history of psychosis was associated with significantly greater negative affect compared to other groups. Family history accounted for variability in Age-at-Onset, as familial psychosis predicted significantly earlier Age-at-Onset compared to other family history groups.

Mediation of Negative Affect. To test the prediction that the relationship between family history and Idiosyncratic Thinking would be mediated by Negative Affect, we calculated the effect decomposition associated with the mediator. Figure 3

shows the direct and indirect effects among these variables. The inclusion of Negative Affect reduced the strength of the relationship between family history and Idiosyncratic Thinking, accounting for 27.2% of the total effect. The statistical significance of the mediated effect was evaluated using the Sobel Test, results of which indicated that the indirect effect of family history on Idiosyncratic Thinking through Negative Affect did not differ significantly from zero (z = -1.88, p = .059). However, because this is a highly conservative test that performs best with large sample sizes (i.e. >500), it is possible that there was not enough power to detect an effect.

Figure 3

Mediation of NegAff on the Relationship between FH and IV



Note: all values are unstandardized estimates; parentheses indicate indirect effects

Categorical Diagnosis

To address hypotheses on the comparative utility of categorical and dimensional classification, we assessed the degree to which DSM diagnosis accounted for variability in TD factors independently, as well as its covariance within the baseline model. When estimated as the only predictor, diagnosis accounted for 9% of variance in Idiosyncratic Thinking, with higher scores on this factor significantly associated with a diagnosis of schizophrenia (r = -.31). Diagnosis was virtually unrelated to the severity of Combinatory

Thinking, explaining none of the variance on this factor. DSM diagnosis was then entered into the baseline model, specified as a predictor of Age-at-Onset, Negative Affect, Idiosyncratic Thinking, and Combinatory Thinking; the direct effects of family history and sex on diagnosis were also estimated. As found in the diagnosis-only model, schizophrenia was associated with significantly greater Idiosyncratic Thinking scores compared to the other groups (r = -.26, p < .001), but diagnosis was unrelated to Combinatory Thinking. Overall, the inclusion of diagnosis in the model explained an additional 7% of the variance of Idiosyncratic Thinking. Inconsistent with expectations, diagnosis did not account for differences in Age-at-Onset or Negative Affect, nor was it predicted by family history. There was a small effect of sex on diagnosis, such that females were more likely to be diagnosed with bipolar disorder and men with schizophrenia.

Table 17
Structural Coefficients of DSM Diagnosis within the Baseline Model

Parameter	**Standardized Estimate**	**S.E.**
FH → DSM Dx	.095	.052
Sex → DSM Dx	.240***	.083
DSM Dx → AAO	.059	.578
DSM Dx → NegAff	-.056	.030
DSM Dx → IV	-.272***	.016
DSM Dx → CT	-.071	.012

Note: FH = family history; DSM Dx = DSM-IV diagnosis; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking
* < .05
** < .01
*** < .001

We then conducted a multigroup analysis to determine whether structural relationships differed as a function of diagnosis. However, because sample sizes were relatively small and unequal across groups, parameters could not be estimated and

compared reliably. Thus, we decided to collapse the three diagnostic categories into two broader groups: non-affective (SZ) and affective psychoses (SZA, BP). Family history of psychosis was predictive of earlier onset and greater Negative Affect in the full sample; however, when estimates were calculated for each group separately, this effect was found to be specific to non-affective psychosis, as was the association between Age-at-Onset and Negative Affect. Within the non-affective psychosis group, there were also significant effects of family history (psychosis) on Idiosyncratic Thinking and Age-at-Onset on Combinatory Thinking, which were not present in the affective psychosis group. Due to difference in structural relationships, the model explained considerably more variance in Idiosyncratic Thinking for probands diagnosed with non-affective (24%) compared to affective psychosis (6%).

Table 18.
Pairwise Comparison of Structural Parameters by Affective and Non-Affective Psychosis Groups

	Psychosis		Affective Psychosis		Pairwise Comp
Parameter	**Estimate**	**S.E.**	**Estimate**	**S.E.**	**z**
FH → AAO	.167*	.718	.021	.717	-1.38
Sex → AAO	.083	.601	.159	.579	0.51
FH → NegAff	-.188*	.041	-.045	.031	1.68*
AAO → NegAff	.039	.004	-.068	.004	-0.90
Sex → NegAff	.067	.035	.143	.025	0.25
CPZ → NegAff	.182*	.000	.116	.000	-1.29
NegAff → IV	.323***	.035	.103	.054	-1.30
NegAff → CT	.147	.028	-.014	.044	-1.10
AAO → IV	-.198*	.002	-.003	.002	1.69*
FH → IV	-.155*	.019	.129	.019	2.45**
FH → CT	-.022	.015	.109	.016	1.076
AAO → CT	-.159*	.002	.035	.002	1.589
Sex → CT	-.003	.013	-.043	.013	-0.317
Sex → IV	-.043	.015	-.100	.016	-0.382
CPZ → CT	.027	.000	-.203*	.000	-1.79*
CPZ → CT	.13	.000	-.199*	.000	-2.74***

Note: FH = family history; AAO = age-at-onset; NegAff = negative affect; IV = idiosyncratic thinking; CT = combinatory thinking; CPZ = chlorpromazine equivalent
* < .05
** < .01
*** < .001

Summary of Results

Confirmatory factor analysis was conducted to test a series of alternative TDI models. The best-fitting model (Model 6) indicated two distinct factors, which resembled idiosyncratic and combinatory dimensions. The idiosyncratic factor was defined by TDI categories characterized by overly-abstract, self-referential, and stilted responses. In contrast, categories loading on the combinatory factor reflect a style of responding that is conceptually organized, but perceptually-dominated, over-embellished and thematically bizarre.

Figure 4 presents a synthesized illustration of significant results. Overall, the baseline model, with the specification of CPZ as a covariate of Negative Affect, accounted for 11% of the total variance of Idiosyncratic Thinking and 3% of variance of Combinatory Thinking. Negative Affect emerged as the strongest predictor of Idiosyncratic Thinking *(r* = .27). This effect was comparable in men (r = .22) and women (*r* = .36), but differed as a function of Family History; Negative Affect had a significantly stronger effect on Idiosyncratic Thinking in those with a Family History of psychosis (*r* = .37) compared to those without (*r* = .02). Family History also moderated the effect of Age-at-Onset on Idiosyncratic Thinking, such that earlier onset predicted more severe scores in the familial (*r* = -.17), but not sporadic, group (*r* = .09). None of these predictors (Negative Affect, Age-at-Onset, and Family History) were significantly related to Combinatory Thinking, with no sex-specific effects.

While Age-at-Onset had no effect on Negative Affect in the full sample, this relationship was significantly moderated by sex, with earlier onset associated with greater Negative Affect in women (*r* = -.11), but lower Negative Affect in men (*r* = .13). Family

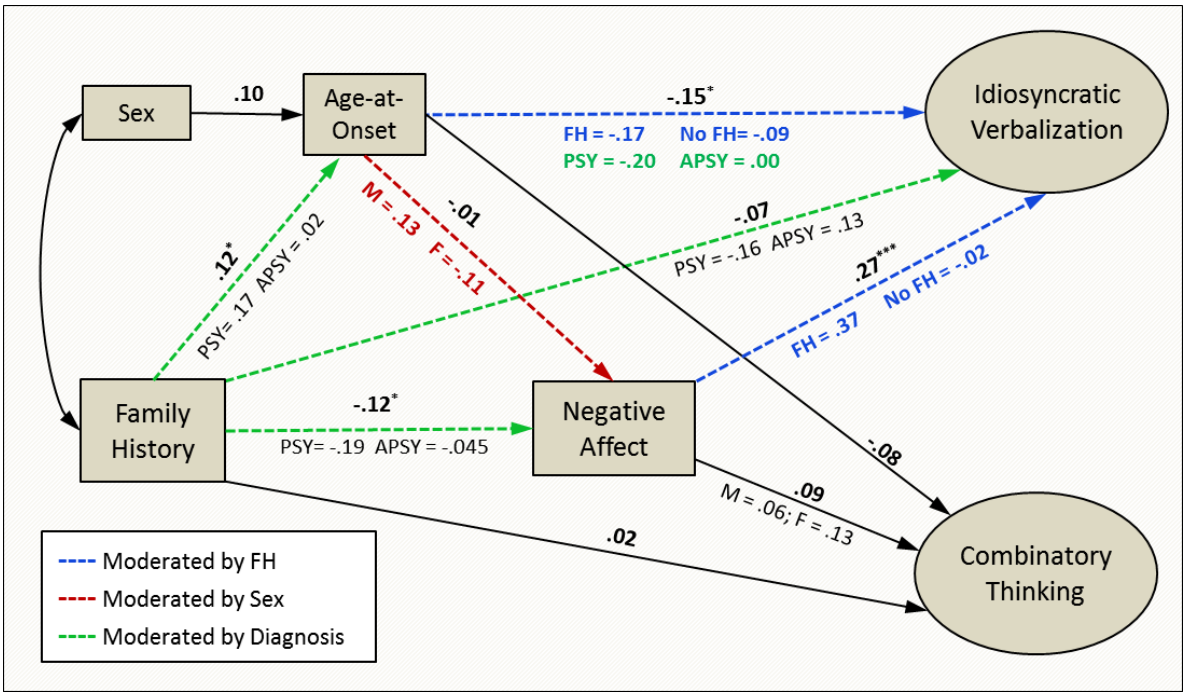
History of psychosis was also a significant predictor of Negative Affect (r = -.12) in the full sample; however, when estimated for each sex separately, was found to be a male-specific effect (M: r = -.16; F: r = .08). Negative Affect was positively correlated with CPZ dose, the magnitude of which was greater in women (r = .19) than men (r = .08). Due to sex differences in structural relationships, the variance in Idiosyncratic Thinking explained by the model was greater for women (16%) compared to men (8%). Similarly, given the moderating effects of Family History, variance in Idiosyncratic Thinking was better explained for those with a Family History of psychosis (19%) than those without (1%). There were no differences in Combinatory Thinking among groups.

DSM diagnosis was a significant predictor of Idiosyncratic Thinking, explaining 7% of unique variance when entered into the full model compared to 9% of the variance when estimated independently. Thus, the portion of variance explained by diagnosis was largely independent of other predictors in the model; this includes Family History, Age-at-Onset, and Negative Affect, which, contrary to expectations, were found to be unrelated to diagnosis. This was also true of Combinatory Thinking, which we expected, based on previous research, to be associated with bipolar disorder, but did not differ by diagnostic group. Sex had a moderate effect on diagnosis, such that women were significantly more likely to hold a diagnosis of bipolar disorder and men SZ. However, the effects of diagnosis on model variables did not differ as a function of sex.

Structural relationships were found to differ between non-affective (schizophrenia) and affective psychosis (schizoaffective and bipolar) diagnoses. Overall, there was a pattern of more robust effects in the schizophrenia group compared to affective psychosis group. These differences in magnitude are likely due to the generally

higher severity of TD in the SZ group, contributing to a broader range of TDI scores. Family History of psychosis was significantly associated with earlier onset and greater Negative Affect, and Idiosyncratic Thinking in probands with non-affective psychosis, but had no effect for those with a diagnosis of affective psychosis. Similarly, the effects of Age-at-Onset and Negative Affect on Idiosyncratic Thinking were specific to schizophrenia, as was the relationship between CPZ and Negative Affect. Due to the differences in structural relationships, the model accounted for considerably more variance in Idiosyncratic Thinking for probands diagnosed with non-affective (24%), compared to affective (6%), psychosis.

Figure 4



DISCUSSION

Bleuler saw thought disorder as the core defining feature of psychotic phenomena, reflective of the "splitting of the psychic functions" that occurred when, in the process of thinking, one's ideas and feelings disconnect, becoming fragmented and competing functions. This view was echoed by Meehl (1962), who called thought disorder the "diagnostic bell ringer" for schizophrenia, which for him, was exemplified by the comment, "naturally I'm growing my father's hair." While Meehl cautioned that the presence of a single symptom is inadequate grounds on which to infer a nosology, he saw thought disorder as a "rare exception," which itself was pathognomonic of schizophrenia.

Interest in thought disorder as the conceptual core of psychosis diminished with rise of the modern DSM system, shifting focus to more clearly-defined and readily-observed indicators that would enhance the reliability of psychodiagnosis. The last several decades have seen mounting criticism of the DSM framework, prompting a movement to reform our scientific paradigm. At current, the pendulum appears to be swinging back towards a dimension-based approach to classification and study.

With this shift, revisiting the significance of thought disorder in the conceptualization and study of psychosis is not only warranted, but timely. Theoretical and empirical foundations of thought disorder have supported it as a construct with a high degree of specificity (e.g., compared to hallucinations, which are believed to have highly diverse causal pathways). Thus, research in this area has the potential to elucidate robust etiological links, which, in turn, could inform individualized, effective intervention

approaches. The primary aim of the current study was to begin to disentangle the phenomenological and etiological heterogeneity of TD, through the application of a dimension-based, multifactorial, transdiagnostic approach. Above all, results illustrated the immense complexity of TD, in terms of both qualitative variability and the diverse, interactional sources of influence.

Much research on TD, particularly through the 1970s and 1980s, focused on its significance as a diagnostic marker. More recent work has explored broader scientific questions (e.g., regarding the cognitive and psychological underpinnings of TD), but has done so within the confines of diagnostic categories. We were interested in exploring the association between diagnosis and TD in order to better understand what diagnosis is, and is not, telling us about TD. Consistent with previous research, we found that diagnosis did account for differences in TD, with the SZ group exhibiting the highest severity of idiosyncratic thinking compared to SZA or BP. However, the interactions among demographic and clinical variables illustrated a much more complex and interesting picture of TD. Of note is the prominent role of negative affect, reminding us of the fundamental link thinking and feeling, even among individuals historically believed to have no emotion.

The data demonstrate a pattern of associations among family psychiatric history, age-at-onset, negative affect, and idiosyncratic thinking, suggestive of a developmental process specific to idiosyncratic thinking. This parallels findings from a diverse body of research that has explored the familial nature of thought disorder (Johnston & Holzman, 1979; Docherty et al., 1999; Wynne & Singer, 1965). Across genetics, family linkage, and social-learning studies, using a range of measurement approaches, familial forms of

TD have consistently been characterized by idiosyncratic ideation, language, and reasoning. This has been explained from biological, psychological, and social perspectives, although the exact mechanisms remain unclear. While the current study does not help to parse out the sources of influence underlying the familial aggregation of Idiosyncratic Thinking, it does shed light on the broader developmental process.

While family psychopathology is not analogous to family TD, they have been found to be highly correlated and both predictive of proband TD (Singer & Wynne, 1965). Family History had no direct effect on Idiosyncratic Thinking in our sample, but shared an interesting association with Negative Affect. In those with a Family History of psychosis, Negative Affect was not only more intense, but strongly associated with the severity of Idiosyncratic Thinking. In order to interpret this, we must first consider the meaning of Negative Affect as it was measured in this study—in the content of Rorschach responses.

Traditionally, affective content on the Rorschach, whether attributed affective qualities to the inkblot (e.g., "a menacing bat") or in reference to one's own emotional state, is thought to reflect a marked loss of distance from the stimuli, indicative of severe cognitive disturbance (Rappaport et al., 1946). In individuals with psychosis, this has been shown to occur in the context of self-referential responses, in which the intrusion of affect is tied to the "intermingling" of personal material, interfering with the perceptual process (Harrow & Prosen, 1978). While the influence of intermingling was not empirically assessed, our qualitative analysis revealed a striking patterns of distressing, personalized responses in those protocols with extreme Negative Affect scores.

The pattern observed among Family History, Negative Affect, and Idiosyncratic Thinking can be interpreted from a social-psychodynamic framework. From this perspective, cognitive and affective capabilities develop in childhood, as a function of the parent-child relationship. It is these interpersonal transactions that shape the way in which we perceive and make sense of surroundings, form concepts, process and modulate affect, and construct an understanding of the self and others. When these early relationships do not provide the opportunity to establish a shared perspective or present inaccurate or inconsistent feedback about perceptual and emotional experience, the child may not develop an organized, stable representation of the self and world. The work of Wynne and Singer (1965) provides compelling support for this theory, linking parental disturbances in focusing attention, communicating, and interpersonal relating to the development of TD, as well as psychosis risk more broadly.

Disorganization in family relations and communication is also a predictor of affective disturbances, associated with poor modulation of negative affect in both individuals with and without psychosis (Read & Gumley, 2008; Morris et al., 2007). Communication deviance within families has been found to predict a range of affect-related outcomes, including poor eye contact, avoidance of emotions, and higher levels of distress (Miklowitz & Stackman, 1992). Families characterized as having a high level of CD have also been shown to exhibit greater cognitive deterioration during periods of emotional arousal, including difficulty with attention and concentration (Lewis et al., 1981).

This interpretation aligns with Bleuler's early conceptualization, in which the loosening of associations between thought and affective processes, and the dominance of

inner life was the core of psychosis (Bleuler, 1911; Harrow & Prosen, 1978). A common notion is echoed in Vygotsky's model of cognitive development, which he believed to be a largely social process, founded in cooperative and collaborative dialogue with caregivers. It was also at the core of his theory of psychosis, in which he describes "a separation of […] emotional expressions from the concepts with which they are closely associated. Taken together, this provides a strong theoretical framework through which to understand the relationship between distressing affect and TD.

This interpretation of the data also corresponds with the stress sensitivity theory of psychosis, which has been one of the leading etiological models for many years (Walker & Diforio, 1997). Related research has consistently demonstrated that individuals with psychosis have a lower threshold for and heightened response to stress (Docherty et al., 1996). This is particularly true in familial psychosis, which has been linked to higher responsivity to emotional, interpersonal, and perceptual stimuli (Lancaster et al., 2010; Myin-Germeys & VanOs, 2006). In regards to the relationship between affect and TD, Docherty (1996) found that, when faced with emotionally provocative stimuli, individuals with a Family History of psychosis exhibited heightened reactivity, resulting in significant deterioration in the clarity of their language. While we cannot assume that a Family History of psychosis is analogous with disorganization of family communication or attachment, the commonalities among these two lines of research suggest that they are overlapping constructs.

The Role of Age-at-Onset and Sex.

Age-at-onset also had an interesting pattern of associations within the model, lending further support to the developmental interpretation. Earlier onset age was

associated with a higher severity of idiosyncratic disturbances, which is consistent with a large body of evidence linking early onset to an overall more severe form of psychosis, including higher levels of TD (Kao et al., 2010). Interestingly, earlier onset has been found to characterize familial psychosis, a finding that was replicated in the current data (Husted, Greenwood, & Bassett, 2006; Kendler & MacLean, 1990). Individuals with a Family History of psychosis have been shown to have a significantly earlier age-at-onset than those with no Family History (Esterberg & Compton, 2012; Gorwood et al., 1995; Malaspina et al., 2000). While generally explained as evidence of genetic liability, the relationship between Family History and onset can also be interpreted within the context of a psychosocial process. That is, the magnitude of relational and attachment disorganization may be more pronounced in families with psychosis, which could lead the child to develop less stable, cohesive mental representations that put them at heightened risk for psychosis earlier in life.

Age-at-onset did not differ as a function of sex, which was unexpected given the well-established evidence that men develop psychosis an average of 5 to 10 years earlier than women; while this finding is most consistently reported in schizophrenia research (Goldstein & Lewine, 2000), it has also been replicated in samples with affective psychosis (Kennedy et al., 2005; Welham, Thomis, & McGrath, 2004) and sub-clinical psychosis (Spawen et al., 2003). Perhaps the most surprising finding that emerged from the data was the sex difference in the Age-at-Onset on Negative Affect in men and women. The intensity of negative affective was associated with later onset in men and earlier onset in women. We know that, in males, schizophrenia is characterized by earlier onset and restricted affect (Anglin et al., 2009; Lewine, 1981; Resend, Viglione, & de

Lima Argimon, 2009; Salem & Kring, 1998). Women, on the other hand, typically have a later onset, along with more intense experience and expression of affect (Anglin et al., 2009). However, women with earlier onset been shown to present with clinical features similar to typical-onset men (Gureje & Bamidele, 1998). While this would suggest earlier onset to be predictive of lower emotionality in both men and women, findings come from diagnosis-based research, which may not apply to the current sample.

Another possible explanation for this relates to the course of psychosis. In schizophrenia, negative affect has been shown to increase the longer one has lived with symptoms and spent hospitalized; this includes anger, fear, and guilt (Suslow et al., 2003). Because men tend to experience earlier onset and a more severe, persistent course of psychosis, we might expect to see more intense negative affect in men compared to women over time.

Strengths and Limitations

The methodological approaches used in this research are, in many ways, unique. The use of the LIWC to assess negative affect in Rorschach responses was novel; while preliminary support for construct validity was demonstrated, we recognize that questions may remain about psychometrics. Second, as argued throughout this paper, the information contained in categorical diagnosis is inherently limited. Thus, despite the rigor with which family psychiatric history was assessed, it is a flawed marker of family differences. Given the limited specificity of groups, we must be careful not to over-interpret the significance of Family History in the model. While results suggest that *something* is going on in psychotic families, what exactly that is, is purely speculative. Parsing out the specific mechanisms at work would be a worthwhile aim for future

research. Given recent work on early adversity and attachment disorganization, exploring the effects of these factors within the developmental model would be particularly interesting.

with potential implications for how we interpret and generalize the current findings.

Finally, results are based on a predominantly white, well-educated, middle- to upper-class sample, which is not representative of the broader population. This is an important caveat, given that psychosis is more prevalent in low-income areas. Clinical features of psychosis have also been shown to differ in minority groups, including a higher preponderance of paranoia in African American men, which is linked to the degree of discrimination experienced. If poverty and discrimination are indeed risk factors of psychosis, as research suggests, then the developmental pathways underlying TD might look very different across sociocultural groups.

Overall, these limitations are outweighed by the strengths of the study, including the large sample size, equal representation of men and women, and novel methodological approach.

Implications & Future Directions

The broad implications of this research relate to how we approach the classification and study of psychosis. Broad diagnostic categories collapse important aspects of individual difference and variation, leading critical relationships to be overlooked. The dimensional framework presented a complex, interactional view of the data, revealing a pattern of relationships among variables that pointed to a developmental process. Such a nuanced, multidimensional perspective is more representative of human experience and thus better apt to elucidate links between psychotic phenomena and

diverse sources of influence. Shifting towards a constructivist paradigm has the potential to facilitate deeper understanding of the complex nature of psychosis. Related to this, the approach embodied in the current study supports a phenomenological, developmental, and psychological perspective of psychosis—something that has historically been underrepresented and undervalued in science and clinical work.

With regards to clinical work, dimensional research has the potential to provide detailed, precise information about the structure and nature of psychosis, which is a critical step in the development of individualized treatments. At present, antipsychotic medication remains the first, often only, treatment for psychosis, despite accumulating evidence that calls into question the efficacy and long-term safety of these drugs (Bolla, Lehtinen, Cullberg, & Ciompi, 2009; Harrow & Jobe, 2007). This reactive, one-size-fits-all approach of treatment is a direct consequence of the categorical, medical model, which has worked to simplify how we conceptualize and treat psychosis.

The dimensional framework has potential to shift attention back to a phenomenological view and bring with it an emphasis on proactive, person-centered approaches to treatment. Our findings draw attention to possible mechanisms of TD that would be feasible targets of psychosocial treatment. This includes affective reactivity, which, if it indeed has an exacerbating influence on TD, could be a viable mechanism of change for future interventions. Given a host of empirically-supported psychosocial interventions for affect regulation currently available, adapting this approach for TD would be a feasible and potentially fruitful endeavor.

Another important, albeit indirect, implication relates to the implementation of prevention efforts. The current results add to an already large body of evidence

suggesting that children in families with psychosis are at increased risk for a range of psychological problems (Miklowitz & Stackman, 1992; Wynne, 1994;). Regardless of the relative influences of biological and environmental causes, psychosis, like any other psychological, social, or medical problem one might face, is an undeniable source of stress for the individual and their family. Further, because people with psychosis tend to have a higher sensitivity to stress and lack the personal resources needed to cope adaptively, the experience of psychosis itself has the potential to perpetuate further psychosocial stress. Unfortunately, traditional treatment models often fail to appreciate and adequately address the personal, social, and relational ramifications of psychosis, such as the impact of hospitalization on one's family (including children) and personal identity (Holden & Lewine, 1982).

Not surprisingly, family interventions have been associated with remarkable outcomes, in terms of reducing subjective distress, reestablishing social roles, and reducing relapse (Alahen et al., 2000). The overarching perspective of these approaches is that psychosis develops within the social system, through a complex, interactive process, reciprocally determined by the interplay of self-environment factors (Aderhold & Gottwalz, 2004). With this is the assumption that psychosis is phenomenologically complex and understandable in the context of the unique dynamics of the family system.

Despite the effectiveness of family treatment, such approaches are not widely available. Addressing such issues more directly across mental health services, in addition to increasing availability of family-based treatment, could help to reduce the psychosocial risk factors for children in families with psychosis.

Conclusion

Thought disorder is the central defining feature of psychosis. Thus, disturbances in thinking represent a unique and promising pathway through which to understand the nature of psychotic phenomena. Exploring this from a dimensional perspective has potential to contribute to a more comprehensive and integrative model of psychosis, which has implications for both science and clinical work.

REFERENCES

American Psychiatric Association. (1980). Diagnostic and Statistical Manual of Mental Disorders, Third Edition: DSM-III. Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision: DSM-IV-TR. Washington, DC: American Psychiatric Association.

Anderson, J.C. & Gerbing, D.W. (1988). Structural equation modeling in practice: A review and recommended two-step approach. *Psychological Bulletin, 103,* 411 – 423.

Andreasen, N. (1979). Thought, language, and communication disorders. I. Clinical assessment, definition of terns, and evaluation of their reliability. *Archives of General Psychiatry*, 36, 1315 – 1321.

Andreasen, N. (1979b). Thought, language, and communication disorders. II. Diagnostic significance. *Archives of General Psychiatry*, 36, 1325–1330.

Andreasen, N. (1986). Scale for the Assessment of Thought, Language, and Communication (TLC). *Schizophrenia Bulletin*, 12, 473–82.

Andreasen, N., Endicott, J., Spitzer, R., & Winokur, G. (1977). The family history method using diagnostic criteria: Reliability and validity. *Archives of General Psychiatry*, 34, 1229–1235.

Andreasen, N., & Grove, W. (1986). Thought, language, and communication in schizophrenia: Diagnosis and prognosis. *Schizophrenia Bulletin*, 12, 348–357.

Andreasen, N., & Olsen, S. (1982). Negative v positive schizophrenia: definition and validation. *Archives of General Psychiatry*, 39, 789 – 784.

Andreasen, N. J. C., & Powers, P. S. (1974). Overinclusive Thinking in Mania and Schizophrenia. *The British Journal of Psychiatry*, 125(5), 452–456. doi:10.1192/bjp.125.5.452

Andreasen, N., Rice, J., Endicott, J., Reich, T., & Coryell, W. (1986). The family history approach to diagnosis: how useful is it? *Archives of General Psychiatry* 43(1), 421–429.

Anglin, D., & Stanford, A. (2009). Family history of affective illness in schizophrenia patients: Symptoms and cognition. *Schizophrenia Research*, 110, 24–27. doi:10.1016/j.schres.2009.02.020.

Atalay, F. & Atalay, H. (2006). Gender differences in patients with schizophrenia in terms of sociodemographic and clinical characteristics. German Journal of Psychiatry, 1 (–)7.

Aukes, M.F., Laan, W., Termorshuizen, F., Buizer-Voskamp, J.E., Hennekam, E.M., Smeets, H.M., … Kahn, R.S. (2012). Familial clustering of schizophrenia, bipolar disorder, and major depressive disorder. *Genetics in Medicine : Official Journal of the American College of Medical Genetics*, 14(3), 338–41. doi:10.1016/gim.2011.16

Babinkostova, Z., & Stefanovski, B. (2011). Family history in patients with schizophrenia and depressive symptoms. Depression, 228, 219–228.

Bachman, P. & Cannon, T. Cognitive and neuroscience aspects of thought disorder. In: Holyoak KJ, Morrison RG, eds. The Cambridge Handbook of Thinking and Reasoning. Cambridge University Press; 2005.

Barch, D. M., Bustillo, J., Gaebel, W., Gur, R., Heckers, S., Malaspina, D., … Carpenter, W. (2013). Logic and justification for dimensional assessment of symptoms and related clinical phenomena in psychosis: relevance to DSM-5. *Schizophrenia Research*, 150(1), 15–20. doi:10.1016/j.schres.2013.04.027

Baron, M., Gruen, R., & Asnis, L. (1982). Schizophrenia: a comparative study of patients with and without family history. *The British Journal of Psychiatry*, 140(5), 516–517. doi:10.1192/bjp.140.5.516

Baron, M., Gruen, R., & Romo-Gruen, J. (1992). Positive and negative symptoms. Relation to familial transmission of schizophrenia. *The British Journal of Psychiatry, 161,* 610 – 614.

Barrera, A., McKenna, P. J., & Berrios, G. E. (2005). Formal thought disorder in schizophrenia: an executive or a semantic deficit? *Psychological Medicine*, 35(1), 121–32.

Bearden, C., Wu, K., Caplan, R., & Cannon, T. (2011a). Thought disorder and communication deviance as predictors of outcome in youth at clinical high risk for psychosis. *Journal of the American Psychiatry*, 1–19. doi:10.1016/j.jaac.2011.03.021.

Bentler, P.M. & Bonett, D.G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88,* 588 – 606.

Berenbaum, H., Oltmanns, T. F., & Gottesman, I. I. (1985). Formal thought disorder in schizophrenics and their twins. *Journal of Abnormal Psychology*, 94(1), 3–16.

Bilder, R.M., Lipschutz-Broach, L., Reiter, G., Geisler, S.G., Mayerhoff, D.I., & Lieberman, J.A. (1992). Intellectual deficits in first-episode schizophrenia: Evidence for progressive deterioration. *Schizophrenia Bulletin*, 18, 437 – 448.

Braff, D.L., Glick, I.D., & Griffin, P. (1983). Thought disorder and depression in psychiatric patients. *Comprehensive Psychiatry*, 24, 57 – 64.

Braff, D.L., Glick, I.D., Johnson, M.H., & Zisook, S. (1988). The clinical significance of thought disorder across time in psychiatric patients. *Journal of Nervous and Mental Disease*, 176, 213 – 220.

Breakey, W.R., & Goodell, H. (1972). Thought disorder in mania and schizophrenia evaluated by Bannister's grid test for schizophrenic thought disorder. *The British Journal of Psychiatry : The Journal of Mental Science*, *120*(557), 391–5.

Brockington, I. (1992). Schizophrenia: Yesterday's concept. *European Psychiatry*, 7, 203–207.

Brown (2006) *Confirmatory Factor Analysis for Applied Research*. New York: Guilford Press.

Buchanan, R., & Carpenter, W. (1994). Domains of psychopathology: an approach to the reduction of heterogeneity in schizophrenia. *The Journal of Nervous and Mental Disease*, 184, 193 – 204.

Burbridge, J.A., & Barch, D.M. (2002). Emotional valence and reference disturbance in schizophrenia. *Journal of Abnormal Psychology*, 111(1), 186–191. doi:10.1037//0021-843X.111.1.186

Chen, Y.-C., Lu, Y.-C., & Lung, F.-W. (2011). Confirmatory factor analysis in neurophysiological and neuropsychological dimensions of schizophrenia. *The International Journal of Neuroscience*, 121(9), 528–35. doi:10.3109/00207454.2011. 582239

Cohen, A.S., Minor, K.S., Baillie, L.E., & Dahir, A.M. (2008). Clarifying the linguistic signature: Measuring personality from natural speech. *Journal of Personality Assessment,* 90, 559 – 563.

Coleman, M.J., Carpenter, J.T., Waternaux, C., Levy, D.L., Shenton, M.E., & Perry, J. (1993). The Thought Disorder Index : A Reliability Study. *Psychological Assessment*, 5(3), 336–342.

Cuesta, M.J., & Peralta, V. (1993). Does formal thought disorder differ among patients with schizophrenic, schizophreniform and manic schizoaffective disorders? *Schizophrenia Research*, 10, 151 – 158.

Cuesta, M.J., & Peralta, V. (1999). Thought disorder in schizophrenia: Testing models through confirmatory factor analysis. *European Archives of Psychiatry and Clinical Neuroscience*, 249(2), 55–61.

Dalen, P., & Hays, P. (1990). Aetiological heterogeneity of schizophrenia: the problem and the evidence. *The British Journal of Psychiatry*, 157(1), 119–122. doi:10.1192/bjp.157.1.119

DeVylder, J., & Lukens, E. (2013). Family history of schizophrenia as a risk factor for axis I psychiatric conditions. *Journal of Psychiatric Research*, 47(2), 181–7. doi:10.1016/j.jpsychires.2012.09.023

Docherty, N. (2012). On identifying the processes underlying schizophrenic speech disorder. *Schizophrenia Bulletin*, 38(6), 1327–35. doi:10.1093/schbul/sbr048

Docherty, N., Grosh, E., & Wexler, B. (1996). Affective reactivity of cognitive functioning and family history in schizophrenia. Biological Psychiatry, 39, 59 – 64.

Docherty, N., Hall, M., & Gordinier, S. (1998). Affective reactivity of speech in schizophrenia patients and their nonschizophrenic relatives. *Journal of Abnormal Psychology*, 107(3), 461–7.

Docherty, N. M., Evans, I. M., Sledge, W. H., Seibyl, J. P., & Krystal, J. H. (1994). Affective reactivity of language in schizophrenia. *The Journal of Nervous and Mental Disease*, 182(2), 98–102.

Docherty, N., Schnur, M., & Harvey, P. D. (1988). Reference performance and positive and negative thought disorder: a follow-up study of manics and schizophrenics. *Journal of Abnormal Psychology*, 97(4), 437–42.

Dutta, R., Greene, T., Addington, J., McKenzie, K., Phillips, M., & Murray, R.M. (2007). Biological, life course, and cross-cultural studies all point toward the value of dimensional and developmental ratings in the classification of psychosis. *Schizophrenia Bulletin*, 33(4), 868–76. doi:10.1093/schbul/sbm059

Erol, A., Bayram, S., Kosger, F., & Mete, L. (2012). Executive functions in patients with familial versus sporadic schizophrenia and their parents. *Neuropsychobiology*, 66(2), 93–9. doi:10.1159/000337738

Esterberg, M. L., & Compton, M. T. (2009). The psychosis continuum and categorical versus dimensional diagnostic approaches. Current Psychiatry Reports, 11(3), 179–84.

Esterberg, M., & Compton, M. (2012). Family history of psychosis negatively impacts age at onset, negative symptoms, and duration of untreated illness and psychosis in first-episode psychosis patients. *Psychiatry Research*, 197(1-2), 23–8. doi:10.1016/j.psychres.2012.03.001

Esterberg, M.L., Trotman, H.D., Holtzman, C., Compton, M.T., & Walker, E.F. (2010). The impact of a family history of psychosis on age-at-onset and positive and negative symptoms of schizophrenia: a meta-analysis. *Schizophrenia Research*, 120(1-3), 121–30. doi:10.1016/j.schres.2010.01.011

Exner, J.E. (1969). The Rorschach systems. New York: Grune and Stratton

First, M.B., Spitzer, R.L, Gibbon M., & Williams, J.B.W: Structured Clinical Interview for DSM-IV-TR Axis I Disorders, Research Version, Patient Edition. (SCID-I/P) New York: Biometrics Research, New York State Psychiatric Institute, November 2002.

Fusar-Poli, P., Bechdolf, A., Taylor, M.J., Bonoldi, I., Carpenter, W.T., Yung, A.R., & McGuire, P. (2013). At risk for schizophrenic or affective psychoses? A meta-analysis of DSM/ICD diagnostic outcomes in individuals at high clinical risk. *Schizophrenia Bulletin*, 39(4), 923–32. doi:10.1093/schbul/sbs060

Gold, J. M., & Hurt, S. W. (1990). The effects of haloperidol on thought disorder and IQ in schizophrenia. *Journal of Personality Assessment*, 54(1-2), 390–400. doi:10.1080/00223891.1990.9674002

Goldberg, T. E., Dodge, M., Aloia, M., Egan, M. F., & Weinberger, D. R. (2000). Effects of neuroleptic medications on speech disorganization in schizophrenia: biasing associative networks towards meaning. *Psychological Medicine*, 30(5), 1123–30.

Goldberg, T. E., & Weinberger, D. R. (2000). Thought Disorder in Schizophrenia: A Reappraisal of Older Formulations and an Overview of Some Recent Studies. *Cognitive Neuropsychiatry*, 5(1), 1–19. doi:10.1080/135468000395790

Gorwood, P., Leboyer, M., Jay, M., Payan, C., & Feingold, J. (1995). Gender and age at onset in schizophrenia: impact of family history. *American Journal of Psychiatry*, 152(2), 208–12.

Gottesman, I. I. & Gould, T.D. (2003). The Endophenotype Concept in Psychiatry: Etymology and Strategic Intentions. *American Journal of Psychiatry*, 160(4), 636–645. doi:10.1176/appi.ajp.160.4.636

Griffiths, T. D., Sigmundsson, T., Takei, N., Rowe, D., & Murray, R. M. (1998). Neurological abnormalities in familial and sporadic schizophrenia. *Brain: A Journal of Neurology*, *121*, 191–203.

Gureje, O., & Bamidele, R. W. (1998). Gender and schizophrenia: association of age at onset with antecedent, clinical and outcome features. *The Australian and New Zealand Journal of Psychiatry*, *32*(3), 415–23. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/9672733

Haddock, G., Wolfenden, M., Lowens, I., Tarrier, N., & Bentall, R.P. (1995). Effect of emotional salience on thought disorder in patients with schizophrenia. *The British Journal of Psychiatry : The Journal of Mental Science*, 167(5), 618–20.

Hain, C., Maier, W., Hoechst-Janneck, S., & Franke, P. (1995). Subclinical thought disorder in first-degree relatives of schizophrenic patients: Results from a matched-pairs study with the Thought Disorder Index. *Acta Psychiatrica Scandinavia*, 92, 305 – 309.

Hallmayer, J. F., Kalaydjieva, L., Badcock, J., Dragović, M., Howell, S., Michie, P. T., … Jablensky, A. (2005). Genetic evidence for a distinct subtype of schizophrenia characterized by pervasive cognitive deficit. *The American Journal of Human Genetics*, 77(3), 468–476.

Hardt, J., & Franke, P. (2007). Validity, reliability and objectivity of the family history method in psychiatry: a meta-analysis. *European Psychiatry : The Journal of the Association of European Psychiatrists*, 22(1), 49–58. doi:10.1016/j.eurpsy.2006.10.003

Harrow, M., Marengo, J., & McDonald, C. (1986). The early course of schizophrenic thought disorder. *Schizophrenia Bulletin,* 12(2), 208–24.

Harrow, M., & Marengo, J. T. (1986). Schizophrenic thought disorder at followup: its persistence and prognostic significance. *Schizophrenia Bulletin*, 12(3), 373–93.

Harrow, M., & Quinlan, D. Is disordered thinking unique to schizophrenia? (1977). *Archives of General Psychiatry*, 34, 15-21.

Harvey, P.D., Earle-Boyer, E.A., & Weiglus, M.S. (1984). The consistency of thought disorder in mania and schizophrenia: An assessment of acute psychotics. *The Journal of Nervous and Mental Disease*, 172, 458 – 463.

Harvey, P. D., Lenzenweger, M. F., Keefe, R. S., Pogge, D. L., Serper, M. R., & Mohs, R. C. (1992). Empirical assessment of the factorial structure of clinical symptoms

in schizophrenic patients: formal thought disorder. *Psychiatry Research*, 44(2), 141–51.

Hatzimanolis, A, McGrath, J.A., Wang, R., Li, T., Wong, P.C., Nestadt, G., … Avramopoulos, D. (2013). Multiple variants aggregate in the neuregulin signaling pathway in a subset of schizophrenia patients. *Translational Psychiatry*, 3(February), e264. doi:10.1038/tp.2013.33

Holden, D. F., & Lewine, R. R. J. (1982). How Families Evaluate Mental Health Professionals, Resources, and Effects of Illness. *Schizophrenia Bulletin*, *8*(4), 626–633. http://doi.org/10.1093/schbul/8.4.626

Holzman, P.S., Levy, D.L, & Johnston, M.H. (2005). The Use of the Rorschach Technique for Assessing Formal Thought Disorder. In R. F. Bornstein & J. M. Masling (Eds.). Scoring the Rorschach: Seven Validated Systems. (pp. 1-24). Mahwah, NJ: Lawrence Erlbaum Associates.

Holzman, P.S., Shenton, M.E., & Solovay, M.R. (1986). Quality of thought disorder in differential diagnosis. *Schizophrenia Bulletin,* 12(3), 360–71.

Horn, H., Federspiel, A., Wirth, M., Muller, T.J., Wiest, R., Wang, J., & Strik, W. (2009). Structural and metabolic changes in language areas linked to formal thought disorder. *British Journal of Psychiatry*, 194, 130 – 138. doi: 10.1192/bjp.bp.107.045633

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling*, 6, 1–55.

Hurt, S.W., Holzman, P.S., & Davis, J.M. (1983). Thought disorder: The measurement of its changes. *Archives of General Psychiatry*, 40, 1281 – 1285.

Jablensky, A. (2006). Subtyping schizophrenia: implications for genetic research. *Molecular Psychiatry,* 11(9), 815–36. doi:10.1038/sj.mp.4001857

Jampala, V.C., Taylor, M.A, & Abrams, R. (1989a). The diagnostic implications of formal thought disorder in mania and schizophrenia: a reassessment. *The American Journal of Psychiatry,* 146(4), 459–63.

Johns, L.C. & van Os, J. (2001). The continuity of psychotic experience in the general population. *Clinical Psychology Review*, 21, 1125 - 1141.

Joober, R., Rouleau, G.A., Samarthji, L., Bloom, D., Lalonde, P., Labelle, A., & Benkelfat, C. (2005). Increased prevalence of schizophrenia spectrum disorders in relatives of neuroleptic-non-responsive schizophrenic patients. *Schizophrenia Research, 77,* 35 – 41.

Kao, Y.-C., Liu, Y.-P., Altamura, A., Conley, R., Buchannan, R., Aleman, A., … Opjordsmoen, S. (2010). Effects of age of onset on clinical characteristics in schizophrenia spectrum disorders. *BMC Psychiatry*, *10*(1), 63. http://doi.org/10.1186/1471-244X-10-63

Kendler, K., & Hays, P. (1982). Familial and sporadic schizophrenia: a symptomatic, prognostic, and EEG comparison. *American Journal of Psychiatry*, 139(12), 1557–1562.

Kendler, K., & Hays, P. (1983). Schizophrenia subdivided by the family history of affective disorder: A comparison of symptomatology and course of illness. *Archives of General Psychiatry*, 10, 468.

Kendler, K. S., & MacLean, C. J. (1990). Estimating familial effects on age at onset and liability to schizophrenia. I. Results of a large sample family study. *Genetic Epidemiology*, 7(6), 409–17. doi:10.1002/gepi.1370070603

Kinney, D. K., Holzman, P. S., Jacobsen, B., Jansson, L., Faber, B., Hildebrand, W., … Zimbalist, M. E. (1997). Thought disorder in schizophrenia and control adoptees and their relatives. *Archives of General Psychiatry*, 54, 475–479.

Kline, R. B. Principles and Practice of Structural Equation Modeling, Third Edition. New York: The Guilford Press; 2010.

Krueger, R. F., & Piasecki, T. M. (2002). Toward a dimensional and psychometrically-informed approach to conceptualizing psychopathology. *Behaviour Research and Therapy*, 40(5), 485–99.

Laursen, T., & Labouriau, R. (2005). Family history of psychiatric illness as a risk factor for schizoaffective disorder: A Danish register-based cohort study. *Archives of General Psychiatry*, 62, 841 (–)848.

Leask, S. J., Vermunt, J. K., Done, D. J., Crow, T. J., Blows, M., & Boks, M. P. (2009). Beyond symptom dimensions: schizophrenia risk factors for patient groups derived by latent class analysis. *Schizophrenia Research*, 115(2-3), 346–50. doi:10.1016/j.schres.2009.09.017

Levy, D.L., Coleman, M.J., Sung, H., Ji, F., Matthysse, S., Mendell, N.R., & Titone, D. (2010). The Genetic Basis of Thought Disorder and Language and Communication Disturbances in Schizophrenia. *Journal of Neurolinguistics*, 23(3), 176. doi:10.1016/j.jneuroling.2009.08.003

Levy, D.L., Smith, M., Robinson, D., Jody, D., Lerner, G., Alvir, J., … Mayerhoff, D.I. (1993). Methylphenidate increases thought disorder in recent onset schizophrenics, but not in normal controls. *Biological Psychiatry*, 34(8), 507–14.

Lewine, R.R. (1981). Sex differences in schizophrenia. Timing or subtypes? Psychological Bulletin, 90, 432 (–)444.

Lewis, S.W., Reveley, A.M., Reveley, M.A., Chitkara, B., & Murray, R. M. (1987). The familial/sporadic distinction as a strategy in *Schizophrenia Research*. *The British Journal of Psychiatry*, 151(3), 306–313. doi:10.1192/bjp.151.3.306

Lilienfeld, S.O., Wood, J.M., Garb, H.N. The scientific status of projective techniques, *Psychological Science in the Public Interest* v. 1, pp. 27–66, 2000.

Maeda, K., Kasai, K., Uetsuki, M., Hata, A., Araki, T., Rogers, M.A., … Iwanami, A. (2007). Increased positive thought disorder with illness duration in patients with schizophrenia. *Psychiatry and Clinical Neurosciences*, 61(6), 687–90. doi:10.1111/j.1440-1819.2007.01730.x

Maier, W., Rietschel, M., Lichtermann, D., & Wildenauer, D. B. (1999). Family and genetic studies on the relationship of schizophrenia to affective disorders. *European Archives of Psychiatry and Clinical Neuroscience*, 249 Suppl , 57–61.

Malaspina, D. (2000). Relation of familial schizophrenia to negative symptoms but not to the deficit syndrome. *American Journal of Psychiatry*, 157(6), 994–1003. doi:10.1176/appi.ajp.157.6.994

Malaspina, D., & Friedman, J. H. (1998). Psychobiological heterogeneity of familial and sporadic schizophrenia. *Biological Psychiatry*, 6(97), 489 – 496.

Mannuzza, S., Fyer, A.J., Endicott, J., & Klein, D.F. Family informant schedule and criteria (FISC). New York, NY: Anxiety Disorders Clinic, New York Psychiatric Institute; 1985.

Marengo, J. T., & Harrow, M. (1997). Longitudinal courses of thought disorder in schizophrenia and schizoaffective disorder. *Schizophrenia Bulletin*, 23(2), 273–85.

Marsh, H.W., Hau, K.T., and Wen, Z. (2004). In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's findings. *Structural Equation Modeling,* 11 (3), 320-41

Matthysse, S., & Holzman, P. S. (1988). Comment on Berenbaum, Oltmanns, and Gottesman (1985): "Formal thought disorder in schizophrenics and their twins". *Journal of Abnormal Psychology*, 97(1), 105–9.

Mazumdar, P. K., Chaturvedi, S. K., & Gopinath, P. S. (1994). Thought disorder and its correlation with clinicodemographic variables in schizophrenia. Psychopathology, 27, 37–42.

McGrath, J.A., Nestadt, G., Liang, K.-Y., Lasseter, V.K., Wolyniec, P.S., Fallin, M.D., … Pulver, A.E. (2004). Five latent factors underlying schizophrenia: analysis and relationship to illnesses in relatives. *Schizophrenia Bulletin*, 30(4), 855–73.

Metsänen, M. (2007). Thought disorder as a predictive sign of mental disorder: a study of high-risk and low-risk adoptees in the Finnish Adoptive Family Study of Schizophrenia. *Acta Universitatis Ouluensis.*

Metsänen, M., Wahlberg, K.-E., Hakko, H., Saarento, O., & Tienari, P. (2006). Thought Disorder Index: a longitudinal study of severity levels and schizophrenia factors. *Journal of Psychiatric Research*, 40(3), 258–66. doi:10.1016/j.jpsychires.2005.03.004

Metsänen, M., Wahlberg, K.-E., Saarento, O., Tarvainen, T., Miettunen, J., Koistinen, P., … Tienari, P. (2004). Early presence of thought disorder as a prospective sign of mental disorder. *Psychiatry Research*, 125(3), 193–203. doi:10.1016/j.psychres.2004.01.002

Miklowitz, D. J. (1994). Family risk indicators in schizophrenia. *Schizophrenia Bulletin*, 20(1), 137–49.

Miklowitz, D. ., & Stackman, D. (1992). Communication deviance in families of schizophrenic and other psychiatric patients: current state of the construct. *Progress in Experimental Personality & Psychopathology Research*, *15*, 1–46.

Mortensen, P.B., Pedersen, M.G., & Pedersen, C. B. (2010). Psychiatric family history and schizophrenia risk in Denmark: which mental disorders are relevant? *Psychological Medicine*, 40(2), 201–10. doi:10.1017/S0033291709990419

Murray, R., & Jones, P. (1996). Sporadic schizophrenia—another male preserve? *European Psychiatry*, 286–288.

Nakamura, M., Nestor, P.G., Levitt, J.J., Cohen, A.S., Kawashima, T., Shenton, M.E., & McCarley, R.W. (2008). Orbitofrontal volume deficit in schizophrenia and thought disorder. *Brain*, 131, 180 – 195. doi: 10.1093/brain/awm265

National Institute of Mental Health. NIMH strategic plan. Bethesda: National Institute of Mental Health; 2008.

National Institute of Mental Health. RDoC Draft 3.1 June 2011 (Internet). http://www.nimh. nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml

Nestor, P.G., Shenton, M.E., Wible, C., Hokama, H., O'Donnell, B.F., Law, S., McCarley, R.W.. A neuropsychological analysis of schizophrenic thought disorder. *Schizophrenia Research*. 1998;29(3):217–25.

O'Callaghan E., Larkin C., Kinsella A., & Waddington J.L. (1991). Familial, obstetric, and other clinical correlates of minor physical anomalies in schizophrenia. *American Journal of Psychiatry, 148*, 479–483.

Oltmanns, T. F., Murphy, R., Berenbaum, H., & Dunlop, S. R. (1985). Rating verbal communication impairment in schizophrenia and affective disorders. *Schizophrenia Bulletin*, 11(2), 292–9.

Ott, S. L., Roberts, S., Rock, D., Allen, J., & Erlenmeyer-Kimling, L. (2002). Positive and negative thought disorder and psychopathology in childhood among subjects with adulthood schizophrenia. *Schizophrenia Research*, 58(2-3), 231–239.

Owen, M., Lewis, S., & Murray, R. (1989). Family history and cerebral ventricular enlargement in schizophrenia. A case control study. *The British Journal of Psychiatry, 154,* 629 – 634.

Pennebaker, J. W. (2001). Linguistic inquiry and word count. Mahwah, NJ: Lawrence Erlbaum Associates.

Pennebaker, J. W., & King, L. A. (1999). Linguistic styles: Language use as an individual difference. *Journal of Personality and Social Psychology*, 77, 1296–1312.

Pennebaker, J. W., Mehl, M. R., & Niederhoffer, K. G. (2003). Psychological aspects of natural language use: Our words, our selves. Annual Review of Psychology, 54, 547.

Peralta, V., Cuesta, M.J., & de Leon, J. (1992). Thought disorder in schizophrenia: A factor analytic study. *Comprehensive Psychiatry*, 33(2), 105 – 110.

Peralta, V., & Cuesta, M. (2003). The diagnosis of schizophrenia : Old wine in new bottles. *International Journal of Psychology and Psychological Therapy*, 3(1911), 141–152.

Picardi, A., Viroli, C., Tarsitani, L., Miglio, R., de Girolamo, G., Dell'Acqua, G., & Biondi, M. (2012). Heterogeneity and symptom structure of schizophrenia. *Psychiatry Research*, 198(3), 386–94. doi:10.1016/j.psychres.2011.12.051

Potash, J. B., Willour, V. L., Chiu, Y. F., Simpson, S. G., MacKinnon, D. F., Pearlson, G. D., … McInnis, M. G. (2001). The familial aggregation of psychotic symptoms in bipolar disorder pedigrees. *The American Journal of Psychiatry*, 158(8), 1258–64.

Pope, H. G., & Lipinski, J. F. (1978). Diagnosis in schizophrenia and manic-depressive illness: a reassessment of the specificity of "schizophrenic" symptoms in the light of current research. *Archives of General Psychiatry*, 35(7), 811–28.

Ragin, A.B., & Oltmanns, T. F. (1987). Communicability and thought disorder in schizophrenics and other diagnostic groups. A follow-up study. *The British Journal of Psychiatry*, 150(4), 494–500. doi:10.1192/bjp.150.4.494

Rasic, D., Hajek, T., Alda, M., & Uher, R. (2013). Risk of Mental Illness in Offspring of Parents With Schizophrenia, Bipolar Disorder, and Major Depressive Disorder: A Meta-Analysis of Family High-Risk Studies. *Schizophrenia Bulletin*, 1–11. doi:10.1093/schbul/sbt114

Read, J., Mosher, L. & Bentall, R. (2004). Models of madness. Hove: Brunner-Routledge.

Resnick, H. S., & Oltmanns, T. F. (1984). Hesitation patterns in the speech of thought-disordered schizophrenic and manic patients. *Journal of Abnormal Psychology*, *93*(1), 80–6.

Reveley, A.M., Reveley, M.A., & Murray, R. M. (1984). Cerebral ventricular enlargement in non-genetic schizophrenia: a controlled twin study. *The British Journal of Psychiatry : The Journal of Mental Science,* 144, 89–93.

Ritsner, M., Ratner, Y., Gibel, A., & Weizman, R. (2005). Familiality in a five-factor model of schizophrenia psychopathology: findings from a 16-month follow-up study. *Psychiatry Research*, 136(2-3), 173 – 179. doi:10.1016/j.psychres.2005.04.006

Ritsner, M., Ratner, Y., Gibel, A., & Weizman, R. (2007). Positive family history is associated with persistent elevated emotional distress in schizophrenia: evidence from a 16-month follow-up study. *Psychiatry Research*, *153*(3), 217–23. doi:10.1016/j.psychres. 2006.07.003

Rorschach, H. (1921). Psychodiagnostik. Leipzig, Germany: Ernst Bircher Verlag.

Roy, M.A., Flaum, M.A., Gupta, S., Jaramillo, L., & Andreasen, N.C. (1994). Epidemiological and clinical correlates of familial and sporadic schizophrenia.

Acta Psychiatrica Scandinavica, 89(5), 324–328. doi:10.1111/j.1600-0447.1994.tb01523.

Royal College of Psychiatrists (2006) Consensus Statement on the Use of High Dose Antipsychotic Medication. (Council Report CR136). London: Royal College of Psychiatrists.

Rubino, I. A., D'Agostino, L., Sarchiola, L., Romeo, D., Siracusano, A., & Docherty, N. M. (2011). Referential failures and affective reactivity of language in schizophrenia and unipolar depression. *Schizophrenia Bulletin*, 37(3), 554–60. doi:10.1093/schbul/sbp108

Salem, J., & Kring, A. (1998). The role of gender differences in the reduction of etiologic heterogeneity in schizophrenia. *Clinical Psychology Review*, 18(7), 795–819.

Sans-Sansa, B., McKenna, P. J., Canales-Rodríguez, E. J., Ortiz-Gil, J., López-Araquistain, L., Sarró, S., … Pomarol-Clotet, E. (2013). Association of formal thought disorder in schizophrenia with structural brain abnormalities in language-related cortical regions. *Schizophrenia Research*, 146(1-3), 308–13. doi:10.1016/j.schres.2013.02.032

Sass, D.A. (2011). Testing measurement invariance and comparing latent factor means within a confirmatory analysis framework. *Journal of Psychoeducational Assessment*, 29, 347 – 363.

Sautter, F. (1994). The short-term course of familial and nonfamilial schizophrenic-spectrum disorder. *Journal of Psychiatric Research*, 28(1), 97 – 106.

Sautter, F. J., McDermott, B. E., Cornwell, J., Johnson, J., Borges, A., Wilson, A. F., … Foundas, A. L. (1995). A preliminary study of the neuropsychological heterogeneity of familial schizophrenia. *Schizophrenia Research*, 18, 1–7.

Scherr, M., Hamann, M., Schwerthöffer, D., Froböse, T., Vukovich, R., Pitschel-Walz, G., & Bäuml, J. (2012). Environmental risk factors and their impact on the age of onset of schizophrenia: Comparing familial to non-familial schizophrenia. *Nordic Journal of Psychiatry*, 66(2), 107–14. doi:10.3109/08039488.2011.605171

Schwarzkopf, S.B., Nasrallah, H.A., Olson, S.C., Bogerts, B., McLaughlin, J.A., & Mitra, T. (1991). Family history and brain morphology in schizophrenia: An MRI study. *Psychiatry Research*: Neuroimaging, 40(1), 49–60. doi:10.1016/0925-4927(91)90028-O

Sham, P. C., Jones, P., Russell, a, Gilvarry, K., Bebbington, P., Lewis, S., … Murray, R. (1994). Age at onset, sex, and familial psychiatric morbidity in schizophrenia. Camberwell Collaborative Psychosis Study. *The British Journal of Psychiatry* : The Journal of Mental Science, 165(4), 466–73.

Shimkunas, A.M. (1972). Demand for intimate self-disclosure and pathological verbalizations in schizophrenia. *Journal of Abnormal Psychology*, 80(2), 197 – 205.

Singer, M. T. & Wynne, L. (1965). Thought Disorder and Family Relations of Schizophrenics: Results and Implications. *Archives of General Psychiatry*, 12(2), 201 – 212. doi:10.1001/archpsyc.1965.01720320089010

Skre, I., Onstad, S., Torgersen, S., & Kringlen, E. (1991). High interrater reliability for the Structured Clinical Interview for DSM-III-R Axis I (SCID-I). *Acta Psychiatrica Scandinavica*, 84(2), 167–73.

Solovay, M.R., Shenton, M.E., Gasperetti, C., Coleman, M., Kestnbaum, E., Carpenter, J.T., & Holzman, P.S. (1986). Scoring manual for the Thought Disorder Index. *Schizophrenia Bulletin*, 12(3), 483–96.

Solovay, M.R., Shenton, M.E., & Holzman, P.S. (1987). Comparative studies of thought disorders: I. Mania and schizoaffective. *Archives of General Psychiatry*, 44, 13–20.

Spauwen, J., Krabbendam, L., Lieb, R., Wittchen, H.-U., van Os, J., Cottler, L. B., … Lachner, G. (2003). Sex differences in psychosis: normal or pathological? *Schizophrenia Research*, *62*(1-2), 45–49. http://doi.org/10.1016/S0920-9964(03)00063-X

Spohn, H. E., Coyne, L., Larson, J., Mittleman, F., Spray, J., & Hayes, K. (1986). Episodic and residual thought pathology in chronic schizophrenics: effect of neuroleptics. *Schizophrenia Bulletin*, 12(3), 394–407.

Stirling, J., Hellewell, J., Blakey, A., & Deakin, W. (2006). Thought disorder in schizophrenia is associated with both executive dysfunction and circumscribed impairments in semantic function. *Psychological Medicine*, 36(4), 475–84. doi:10.1017/S0033291705006884

Strauss, J. S., Carpenter, W. T., & Bartko, J. J. (1974). The diagnosis and understanding of schizophrenia, Part III. Speculations on the processes that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin*, 11, 61–75.

Subotnik, K.L., Nuechterlein, K.H., Asarnow, R.F., Fogelson, D.L., Goldstein, M.J., & Talovic, S.A. (1997). Depressive symptoms in the early course of schizophrenia: relationship to familial psychiatric illness. *The American Journal of Psychiatry*, 154(11), 1551–6.

Sutker, P.B., & Adams, H.E. (Eds.). (2002). Comprehensive Handbook of Psychopathology. Boston: Kluwer Academic Publishers. doi:10.1007/b107807

Svrakic, D. M., Zorumski, C. F., Svrakic, N. M., Zwir, I., & Cloninger, C. R. (2013). Risk architecture of schizophrenia: the role of epigenetics. *Current Opinion in Psychiatry,* 26(2), 188–95. doi:10.1097/YCO.0b013e32835d8329

Tai, S., Haddock, G., & Bentall, R. (2004). The effects of emotional salience on thought disorder in patients with bipolar affective disorder. *Psychological Medicine*, 34(5), 803–9.

Tabarés-Seisdedos, R., Balanzá-Martínez, V., Salazar-Fraile, J., Selva-Vera, G., Leal-Cercós, C., & Gómez-Beneyto, M. (2003). Specific executive/attentional deficits in patients with schizophrenia or bipolar disorder who have a positive family history of psychosis. *Journal of Psychiatric Research,* 37(6), 479–486. doi:10.1016/S0022-3956(03)00071-2

Tanaka, J.S. (1987). "How big is big enough?": Sample size and goodness of fit in structural equation models with latent variables. *Child Development, 58,* 134 – 146.

Tsuang, M. T., Lyons, M. J., & Faraone, S. V. (1990). Heterogeneity of schizophrenia. Conceptual models and analytic strategies. *The British Journal of Psychiatry*, 156(1), 17–26. doi:10.1192/bjp.156.1.17

Væver, M. S., Licht, D. M., Møller, L., Perlt, D., Jørgensen, A., Handest, P., & Parnas, J. (2005). Thinking within the spectrum: schizophrenic thought disorder in six Danish pedigrees. *Schizophrenia Research*, 72(2-3), 137–49. doi:10.1016/j.schres.2004.04.001

Van Os, J., Gilvarry, C., & Bale, R. (1999). A comparison of the utility of dimensional and categorical representations of psychosis. *Psychological Medicine*, 29, 595–606.

Verdoux, H., van Os, J., Sham, P., Jones, P., Gilvarry, K., & Murray, R. (1996). Does familiality predispose to both emergence and persistence of psychosis? A follow-up study [published erratum appears in Br J Psychiatry 1996 Jul;169:116]. *The British Journal of Psychiatry*, 168(5), 620–626. doi:10.1192/bjp.168.5.620

Wahlberg, K., & Wynne, L. (2000). Thought disorder index of Finnish adoptees and communication deviance of their adoptive parents. *Psychological Medicine*, 30(1), 127–136.

Walker, E., Shapiro, D., Esterberg, M., & Trotman, H. (2010). Neurodevelopment and Schizophrenia: Broadening the Focus. *Current Directions in Psychological Science*, 19(4), 204–208. doi:10.1177/0963721410377744

Walker, E. & Shaye, J. (1982). Familial schizophrenia. A predictor of neuromotor and attention dysfunction in schizophrenia. *Archives of General Psychiatry, 39,* 1153 – 1160.

Welham, J. L., Thomis, R. J., & McGrath, J. J. (2004). Age-at-first-registration for affective psychosis and schizophrenia. *Schizophrenia Bulletin*, *30*(4), 849–53. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/15954194

Wilcox, J.A,, Ramirez, A.L., & Baida-Fragoso, N. (2000). The prognostic value of thought disorder in psychotic depression. *Annals of Clinical Psychiatry : Official Journal of the American Academy of Clinical Psychiatrists*, 12(1), 1–4.

Williams, J. B., Gibbon, M., First, M. B., Spitzer, R. L., Davies, M., Borus, J., … Rounsaville, B. (1992). The Structured Clinical Interview for DSM-III-R (SCID). II. Multisite test-retest reliability. *Archives of General Psychiatry*, 49(8), 630–6.

Wolitzky, R., Goudsmit, N., Goetz, R R., Printz, D., Gil, R., Harkavy-Friedman, J., & Malaspina, D. (2006). Etiological heterogeneity and intelligence test scores in patients with schizophrenia. *Journal of Clinical and Experimental Neuropsychology*, 28(2), 167–177. doi:10.1080/13803390500360315

Wright, A.G.C., Krueger, R.F., Hobbs, M.J., Markon, K.E., Eaton, N.R., & Slade, T. (2013). The structure of psychopathology: Toward an expanded quantitative empirical model. *Journal of Abnormal Psychology*, 122(1), 281 – 294. doi: 10.1037/a0030133.

Wynne, L. C., & Singer, M. T. (1963). Thought disorder and family relations of schizophrenics. I. A research strategy. *Archives of General Psychiatry*, *9*, 191–8. Retrieved from http://www.ncbi.nlm.nih.gov/pubmed/14002138

Yang, J., Visscher, P. M., & Wray, N. R. (2010). Sporadic cases are the norm for complex disease. *European Journal of Human Genetics* : EJHG, 18(9), 1039–43. doi:10.1038/ejhg.2009.177

Zetzsche, T., Preuss, U.W., Frodl, T., Leinsinger, G., Born, C., Reiser, M., … Meisenzahl, E. M. (2008). White matter alterations in schizophrenic patients with pronounced negative symptomatology and with positive family history for

schizophrenia. *European Archives of Psychiatry and Clinical Neuroscience, 258*(5), 278–84. doi:10.1007/s00406-007-0793-4

APPENDIX A

Thought Disorder Index

	Level of Deviance			
	.25	.50	.75	1.0
ASSOCIATIVE LOOSENING	**Inappropriate Distance** Responses seem to be dictated insufficiently by the task at hand; rather, they are heavily influenced by personal associations **Clang** The response is limited to a single, clear-cut usage or rhyming or alliteration. Determined by sound rather than word usage. **Perseveration** A poor-form response that is repeated at least three times on consecutive cards **Relationship Verbalization** Subject either repeats a response previously giver of offers a new response, but relates the present response to the former one.	**Looseness** Subject responds with ideas that are either unrelated or arbitrarily or tangentially related. Associations are embellished in an idiosyncratic manner and may flow rapidly with no focus of conversation. The original point is lost.	**Fluidity** A loss of object constancy due to the subject's perception of the world in a highly unstable way.	
DISORGANIZED THINKING	**Vagueness** Response conveys no clear meaning; can include both meandering speeches as well as excessively short, cryptic statements that carry hardly any specific information **Word-Finding Difficulty** The search for a word that the subject appears to know but on which s/he is blocking	**Confusion** Subject does not appear to be sure what s/he is saying, thinking, or perceiving or appears to be disoriented about time, place, or person		**Incoherence** A response that is not only unrelated to the task, but is completely impossible for the examiner to understand in any context

	Level of Deviance			
	.25	.50	.75	1.0
COMBINATORY THINKING	**Incongruous Combination** Single details of a blot that are contiguous with each other are merged into a single response. Participant imparts too much reality to the images.	**Fabulized Combination** Percept and related ideas are condensed into conclusions that violate reality; considerations about relationships between images, blot qualities, and objects.	**Confabulation** Subject carries to an extreme an elaborative ideational tendency that extends the percept beyond the bounds of reality constraints.	**Contamination** Two separate, unrelated percepts are merged into one.
		Idiosyncratic Symbolism The use of concrete images or blot color to represent abstract ideas is scored when such symbolism is idiosyncratic and is given with an air of reality	**Autistic Logic** Subject justifies a response by rationalizing it with a "because" statement that is illogical or based on private autistic reasoning processes rather than on conventional, logical reasoning.	
IDIOSYNCRATIC VERBALIZATION	**Peculiar** Odd words or phrases that may have a recognizable meaning but do not fit the context in which they are used; quaint, idiosyncratic, or private meaning	**Queer** On continuum with peculiar but more severe. Examiner is generally uncertain about what is meant by the word or phrase	**Absurd** Almost totally arbitrary; bears little resemblance to objective reality. Scorer is unable to form an idea about the source of the response.	**Neologisms** New word responses that could be actual words, except that they are not.

APPENDIX B

Summary of Literature on Diagnostic Differences in Thought Disorder Characteristics

Study	Measure	DIAGNOSTIC GROUP			
		Schizophrenia	**Schizoaffective**	**Mania**	**Depression**
Breakey & Goodell (1972)	BGT	↑under-inclusion	(–)	↑conceptual over-inclusion, idiosyncratic thinking	(–)
Andreasen & Powers (1974)	OST; PT	↑under-inclusive thinking	(–)	↑over-inclusive thinking	(–)
Andreasen (1979b)	TLC	↑poverty of speech, poverty of speech content	(–)	↑pressure of speech, derailment, tangentiality, illogicality, loss of goal	↑poverty of speech, ↑circumstantiality
Andreasen (1984)	TLC	↑poverty of speech, ↑poverty of speech content	(–)	↑tangentiality, ↑derailment, ↑incoherence, ↑illogicality, ↑pressure of speech	(–)
Harvey et al. (1984)	TLC	↑negative TD	(–)	↑positive TD	(–)
Harrow & Marengo (1986)	OST; PT	↑rate of relapse 4 years post hospitalization compared to other psychotic and non-psychotic disorders			
Resnick & Oltmanns (1984)	TLC	↑global TD	(–)	↑pressure of speech	(–)
Shenton et al. (1987)	TDI	↑idiosyncratic/autistic thinking, ↑absurdity, ↑confusion	*Depressed*: resembled SZ *Manic*: resembled manic	↑combinatory thinking	(–)
Solovay et al. (1987)	TDI	↑disorganization, confusion, deviant verbalizations	(–)	↑combinatory with "humor, flippancy, and playfulness."	(–)
Ragin & Oltmanns (1987)	TLC	Poverty of speech, derailment, loss of goal similar to manics; ↑reduction in derailment & loss of goal over time than manic		↑pressure of speech	↓TD than all other groups
Braff et al. (1988)	PT	Severity of idiosyncratic thinking equivalent to depressed group at baseline	(–)	(–)	↑reduction in idiosyncratic thinking at discharge compared to SZ
Jampala et al. (1989)		↑tangentiality, neologisms, private word	(–)	↑flight of ideas	(–)

		usage, paraphasias, driveling (i.e. poverty of content), non-sequiturs (i.e. looseness)			
Cuesta & Peralta (1993)	TLC SAPS SANS	↑poverty of speech, ↑poverty of speech content, ↑illogicality, ↑tangentiality, ↑perseveration, ↑global alogia (SANS)	*Manic:* ↑pressure of speech, ↑clanging	(–)	(–)
Docherty et al. (1996)	CDI	↑unclear references	(–)	↑speech output, ↑ambiguous word meanings	(–)
Marengo & Harrow (1997)	Severity Ratings	↓ rates of TD remission over 7-year period compared to SZA group	↑association between the course of TD and psychotic symptoms	↑ rates of TD remission in non-SZ psychosis (MDD, bipolar, NOS) than SZ and SZA	
Wilcox et al. (2000)	TLC	(–)	(–)	(–)	↑TD prevalence in psychotic- vs. non-psychotic depression Negative TD most prominent in both groups
Vaever et al. (2005)	TDI	↑idiosyncratic symbolism, fluidity, absurdity, confabulation, autistic logic, contamination; ↑total score compared to those with "other" diagnoses and those without mental illness.	(–)	(–)	(–)

Note: BGT = Bannister's Grid Test; OST = Object Sorting Test; PT = Proverbs Test; TLC = Thought, Language, & Communication Scale; TDI = Thought Disorder Index; SANS = Scale for the Assessment of Negative Symptoms in Schizophrenia; SAPS = Scale for the Assessment of Positive Symptoms in Schizophrenia; CDI = Communication Disturbances Index; (–) = group not included in study

CURRICULUM VITAE

Mara A. Hart

PERSONAL INFORMATION

Office Address:
Department of Psychological and Brain Sciences
University of Louisville
Louisville, KY 40292
Phone: (502) 852-3395

E-mail: mara.hart@louisville.edu

Home Address:
148 N. Birchwood Avenue #1
Louisville, KY 40206
Phone: (914) 299-9097

EDUCATION

Ph.D., Clinical Psychology, 2011-Present, University of Louisville, Louisville, KY
Dissertation Title: *Exploring a Multifactorial Model of Thought Disorder: Application of a Dimensional, Transdiagnostic Approach*
Advisor: Richard R. J. Lewine, Ph.D.

M.A., Clinical Psychology, 2013, University of Louisville, Louisville, KY

M.S., Clinical Psychology, 2009, Loyola College, Baltimore, MD

B.A., Psychology, 2006, Loyola College, Baltimore, MD

HONORS AND AWARDS

2015 Excellence in Clinical Work, Department of Psychological and Brain Sciences

CLINICAL EXPERIENCE

August 2015 - Present **Integrative Interventions Team, Co-leader/Peer Supervisor**
University of Louisville Psychological Services Center, Louisville, KY

Assist in the facilitation of a specialty clinic in integrative psychotherapy at the Psychological Services Center (PSC), a psychology clinic that provides a range of services to the greater community on a sliding fee scale. The Integrative Interventions Team specializes in the evaluation and treatment of adults with severe psychological, behavioral, and interpersonal difficulties (e.g., psychoses, affective disorders, etc.), which are generally compounded by immense social stress and significant histories of trauma and adversity. As co-leader, I help to plan, organize, and run weekly group meetings for supervision and didactics. Additionally, I provide individual supervision to all new team members on a weekly basis and supervise several cases seen by senior student therapists.

Supervisor: Rich Lewine, Ph.D.

August 2012 - August 2015 **Integrative Interventions Team, Student Clinician**
University of Louisville Psychological Services Center, Louisville, KY

Provided assessment and psychotherapy services for adults experiencing complex psychosocial problems using an integrative and person-focused approach to case

conceptualization that draws together relevant psychological, social, and biological mechanisms. Received weekly group and individual supervision that fostered conceptual and practical understanding of a broad range of psychological theories, principles, and empirical evidence. Gained experience with a diverse array of intervention models and techniques, drawing from cognitive-behavioral, psychodynamic, humanistic, interpersonal, narrative, existential and emotion-focused paradigms. Provided peer supervision of psychotherapy cases for junior students on a weekly basis.

Supervisor: Rich Lewine, Ph.D.

August 2011 – August 2015 **Adult and Child Assessment Service, Student Clinician**
University of Louisville Psychological Services Center, Louisville, KY

Conducted evaluations for adults and children for a wide-range of referral questions. Child assessment experience included evaluations for eligibility in the advance placement program at Jefferson County Public Schools. Adult cases were referred from a range of sources, including outpatient psychiatry, neurology, primary care, and the court system, for questions related to diagnosis, neurocognitive functioning, as well as disability and competency. Assessment batteries were tailored to fit the needs of client and referral questions, and included semi-structured and structured interviews, intellectual and achievement testing, neuropsychological assessment, projective techniques, standardized personality measures, and measures of functional ability, as well as self and observer reports.

Supervisors: Bernadette Walter, Ph.D.
David Winsch, Ph.D.
Rich Lewine, Ph.D.

July 2013 – July 2014 **Secure Care Unit Treatment Team, Practicum Student**
Central State Psychiatric Hospital, Louisville, KY

A year-long, 20-hour per week practicum, in which I worked as part of a multidisciplinary treatment team on an all-male secure-care unit that served patients in long-term forensic care, those admitted with a history of violence or sexual acting-out, as well as referrals from jail for treatment and competency evaluations. Primary responsibilities included intake evaluations, suicide and violence risk assessment, neuropsychological, personality, and cognitive testing, in addition to evaluations of mental status, malingering, and independent living skills. Met with patients for short-term individual therapy and relapse prevention planning, in addition to planning and facilitating a weekly therapy group.

Supervisors: James Gedra, Psy.D.
Abby Stiff-Miller, Psy.D.

August 2011 – October 2013 **Geriatric Neuropsychology and Psychotherapy Team, Student Clinician**
University of Louisville Geriatric Clinic, Louisville, KY

Assessment: Conducted weekly neuropsychological evaluations in consultation with primary care physicians to assist in the diagnosis of dementia syndromes. Generated diagnostic reports which integrated data from cognitive and psychological assessments with information collected from patients, families, and medical records. Feedback was provided to patients and families, as well as to

referring physicians. Select cases were presented at interdisciplinary care plan meetings. Provided peer supervision of assessment training, administration, report writing, and procedural/administrative tasks

Psychotherapy: Provided psychotherapy to older adults and couples experiencing late-life issues such as cognitive changes, depression, anxiety, grief, and interpersonal problems. Worked with caregivers of older adults who expressed feelings of burden, depression, or anxiety related to these responsibilities.

Supervisor: Benjamin Mast, Ph.D.

May 2012 - August 2012 **Emergency Psychiatric Services, Practicum Student**
University of Louisville Hospital, Louisville, KY

Interviewed patients triaged into the psychiatric emergency room during morning treatment team, a multidisciplinary meeting that included psychiatry, social work, substance abuse counseling, nursing, and medical students. Collaborated in assessment of presenting problems and level of risk to determine need for hospitalization. Conducted phone interviews with family/friends to gain collateral information when necessary to inform treatment decisions. Contributed to conceptualization and disposition planning.

Supervisors: Rich Lewine, Ph.D.
Rif El-Mallakh, M.D.

August 2008 - May 2009 **GIFTS LLC Psychological Services, Masters Extern**
Baltimore, MD

Administered clinical interviews, standardized psychological assessments, and projective tests to children, adolescents, and adults; scored and interpreted data in order to synthesize outcomes; wrote formal psychological reports and conducted feedback sessions with clients and families.

Supervisor: Akintude Morakinyo, Ph.D.

August 2008 - May 2009 **Baltimore City Public Schools, Masters Extern**
HOPE Health Systems, Baltimore, MD

Worked with elementary and middle school students in the Baltimore City Public School System. Led and co-facilitated group, individual, and family therapy sessions; conducted classroom observations and provided feedback to teachers and staff; assisted with crisis situations, providing interventions to students in distress, in addition to support and education to staff.

Supervisors: Akintude Morakinyo, Ph.D.
Mary King, LCSW

OUTREACH AND EDUCATIONAL PRESENTATIONS

Stress Less on Your Road to Academic Success, Workshop series developed for the University of Louisville Women's Center (November 2015)

Managing Stress and Promoting Wellness, workshop given for the Cardinal Covenant scholarship program (October 2015)

Negative Thinking and College Success, invited talk for freshman students in General Studies course (September 2015)

Understanding the Dimensionality of Psychosis: Implications for Recovery-Oriented Treatment, educational talk presented to staff at Central State Hospital (June 2014)

Stress Management and Your Career, invited talk for members of the Dress for Success organization (March 2013)

RESEARCH INTERESTS

Understanding the phenomenological and developmental complexity of severe psychological disturbances, specifically by studying unique dimensions and features of such experiences, as related to interactions among psychosocial factors and individual characteristics.

RESEARCH EXPERIENCE

July 2011 – Present **University of Louisville Affect and Cognition Lab, Doctoral Student**
Supervisor: Rich Lewine, Ph.D.

A Study of Thinking and Feeling in Schizophrenia and Schizoaffective Disorder
PI: Rich Lewine

Collaborated in developing study concept, methodology, and preparing IRB protocol. Recruited subjects from an acute inpatient psychiatric unit; administered study assessments: *Rorschach Inkblot test, Wechsler Test of Adult Reading, Beck Depression Inventory, Beck Anxiety Inventory, Positive and Negative Affect Schedule, Big Five Inventory.* Scored measures, managed database, and supervised undergraduate research assistants. Analyzed and interpreted data; presented findings

May 2010 – June 2011 **University of Maryland, College Park, MD, Clinical Research Associate**
Supervisor: Jack Blanchard, Ph.D.

Collaboration to Advance Negative Symptom Assessment in Schizophrenia
PIs: Jack Blanchard, Ann Kring (Berkeley), Bill Horan (UCLA), Raquel Gur (UPenn)

National Institute of Mental Health-funded, multisite project to evaluate the reliability and validity of a new negative symptom assessment instrument, the Clinical Assessment Interview for Negative Symptoms (CAINS). Recruited participants from the University of Maryland Medical System community mental health clinics and Baltimore Veterans Administration. Administered structured diagnostic and symptom interviews, neuropsychological and performance-based assessments. Scored and managed data; coded interviews from other sites to evaluate inter-rater reliability. Attended weekly assessment supervision and didactics

Investigating Cognitive Models of Negative Symptoms in Schizophrenia
PIs: Shannon Couture, Ph.D. & Melanie Bennett, Ph.D.

VA-funded pilot study examining how perceptions of one's self, illness, and environment relate to negative symptoms. Presented study to clinic staff at recruitment sites; recruited participants, collected and managed data. Conducted diagnostic interviews, symptom assessments, cognitive testing, questionnaires, and distress-tolerance/persistence tasks

Adapting Motivational Interviewing for Persons with Schizophrenia
PI: Melanie Bennett, Ph.D.

VA-funded pilot study focused on adapting motivational interviewing to suit

individual differences in persons diagnosed with schizophrenia. Carried out administrative tasks, prepared study protocol and consent documents for Institutional Review Board approval

RESEARCH ASSESSMENT EXPERIENCE

Structured Clinical Interview for DSM Disorders, Brief Psychiatric Rating Scale, Scale for Assessment of Negative Symptoms, Scale for the Assessment of Positive Symptoms, MATRICS Consensus Cognitive Battery, Brief Cognitive Assessment Tool for Schizophrenia, UCSD Performance-based Skills Assessment – Brief, Simpson-Angus Scale, Calgary Depression Scale for Schizophrenia, Chapman Anhedonia Scales, Internalized Stigma of Mental Illness Inventory, Rorschach Inkblot Test, Thought Disorder Index

PUBLICATIONS

Waford, R., Lewine, R., Robertson, C., & Hart, M. (submitted). Negative and positive affect are differentially associated with characteristics of thought disorder in schizophrenia. Manuscript submitted to the Journal of Abnormal Psychology.

Hart, M. & Lewine, R. (pending submission). Toward a dimensional and integrative understanding of psychopathology: Implications for a multifactorial, clinical model of thought disorder

Hart, M. & Lewine, R. (pending submission). The nature of cognitive impairment in schizophrenia: A multiple-groups confirmatory factor analysis of the WAIS-R

Hart, M. & Lewine, R. (unpublished manuscript). The moderating effect of family history on the relationship between executive dysfunction and thought disorder in schizophrenia.

Lewine, R. & Hart, M. (unpublished manuscript). David Robeson Morgan: A case study that raises questions about the role of frontal lobes in complex cognitive processes.

PROFESSIONAL PRESENTATIONS

Martin, M., Lewine, R., & Hart, M. (October 2015). *Typical and atypical gender roles in individuals with schizophrenia and schizoaffective disorder.* Poster presented at the 2015 annual meeting of the Society for Research in Psychopathology, New Orleans, LA.

Hart, M. & Lewine, R. (October 2015). *Personalized associations and affective responsivity as predictors of thought disorder on the Rorschach*. Abstract accepted for the 2015 annual meeting of the Society for Research in Psychopathology, New Orleans, LA (unable to present).

Hart, M., Waford, R., Robertson, C., & Lewine, R. (September 2014). *Exploring the relationship between affect and thought disorder in schizophrenia*. Poster presented at the 2014 annual meeting of the Society for Research in Psychopathology, Evanston, IL.

Waford, R., Robertson, C., Hart, M., & Lewine, R. (September 2014). *Does affective valence moderate thought disorder severity in schizophrenia and schizoaffective disorder* ? Poster presented at the 2014 annual meeting of the Society for Research in Psychopathology, Evanston, IL.

Hart, M., & Lewine, R. (September 2013). *Examining neuropsychological heterogeneity in schizophrenia through a broadened definition of family history.* Abstract accepted for the 2013 annual meeting of the Society for Research in Psychopathology, Oakland, CA (unable to present).

Hart, M. & Lewine, R. (October 2012). *Reexamining the relationship between domains of neurocognitive functioning and thought disorder in schizophrenia.* Poster presented at the annual meeting of Society for Research in Psychopathology, Ann Arbor, MI.

Waford, R., Robertson, C., Hart, M., & Lewine, R. (October 2012). *Does affective valence moderate thought disorder severity in schizophrenia and schizoaffective disorder?* Poster presented at the annual meeting of Society for Research in Psychopathology, Ann Arbor,

MI.

Robertson, C., Lewine, R., Waford, R., & Hart, M. (October 2012). *Examining the efficacy of subgrouping by FFM Personality Traits in Schizophrenia: An exploratory investigation.* Poster presented at the annual meeting of the Society for Research in Psychopathology, Ann Arbor, MI.

Hart, M., Bennett, M., Couture, S., Blanchard, J. (September 2011). *The relationship between persistence, neurocognitive performance, and negative symptoms in schizophrenia.* Poster presented at the annual meeting of Society for Research in Psychopathology, Boston, MA.

Hart, M., Petrow, A., Bennett, M. (May 2011). *Internalized stigma in a sample of individuals with schizophrenia.* Poster presented at the annual University of Maryland School of Medicine Research Day, Baltimore, MD.

TEACHING EXPERIENCE

Fall 2015; Spring 2015; Fall 2014	**Introduction to Psychology,** *Teaching Assistant* Attended lectures and proctored biweekly examinations for two course sections of 300 students; scored all examinations; generated grade distributions and statistics; uploaded and managed grades through online grade management system, Blackboard.
Spring 2013	**Psychology of Learning**, *Lab Instructor* Planned and facilitated weekly lab meetings for two sections of 30 students; activities included conducting experiments in a virtual rat-training program, watching video clips and discussing illustrated learning principles; graded weekly written assignments and term papers; managed online course page through Blackboard
Fall 2012	**Forensic Psychology**, *Teaching Assistant* Attended all lectures, graded tests and papers for 150 students
	Psychology of Personality, *Teaching Assistant* Attended lectures and learned course material; met with students on an individual basis as needed to review exams or discuss course material; graded exams/assignments for class of 150 students
Spring 2012	**Experimental Psychology**, *Lab Instructor* Attended all lectures, planned and facilitated weekly lab meetings for two sections of 30 students; facilitated group exercises and activities related to the scientific method; conducting test review sessions; graded weekly written assignments and term papers; managed online course page through Blackboard
Fall 2011	**Psychology of Personality**, *Teaching Assistant* Attended all lectures; provided additional support to students on an individual basis*;* collaborated in the development of exams; scored all exams and managed grade database

SERVICE EXPERIENCE

2012 – 2015	Peer Mentor for incoming graduate students
2012	Conducted free memory screenings for the community for National Memory Screening Day
2012 – 2014	Assisted in planning and coordination of admissions interviews for program applicants

2011	Society for Research in Psychopathology: 2011 Student Contributor, Publication Committee
2005- 2006	Volunteered at My Sister's Place (Baltimore, MD), a day program that provides shelter, resources, and support to women and children struggling with homelessness and domestic abuse. Responsibilities included planning and conducting activities, serving meals, and assisting with administrative tasks.
2004 - 2005	Volunteered at Frederick Ozanam House (Baltimore, MD), a half-way-house for men working toward independent living after being incarcerated, institutionalized, or homeless. Responsibilities included planning and carrying out activities and social events, as well as providing support and companionship to members.
2003 - 2004	Volunteered at Baltimore Reads Inc. (Baltimore, MD) as a tutor and mentor for inner-city elementary school children experiencing academic and emotional difficulties, in addition to significant environmental stressors (e.g., poverty, neighborhood violence, family problems)

PROFESSIONAL ACTIVITIES AND AFFILIATIONS

American Psychological Association - *student member*
Division 12, Society of Clinical Psychology
Division 29, Division of Psychotherapy
Kentucky Psychological Association - *student member*
Society for Research in Psychopathology - *student member*
International Society for Psychological and Social Approaches to Psychosis - *student member*
Maryland Psychological Association for Graduate Students - *student member*

RELEVANT WORK EXPERIENCE

November 2009 - May 2010	Special Education Teacher *Kennedy Krieger Institute, Baltimore, MD* Created, facilitated, and evaluated lesson plans in compliance with Maryland State Department of Education standards; administered and interpreted informal student assessments; composed and presented Individualized Educational Plans (I.E.P.s); collaborated with professional staff in order to implement, monitor, and evaluate the effectiveness of Behavioral Intervention Plans
May 2007 - November 2009	Teaching Assistant *Kennedy Krieger Institute, Baltimore, MD* Supported lead teacher in lesson planning, instruction, student assessment, and classroom organization; responsible for grading assignments, recording behavioral data, and maintaining student records; implemented behavioral and crisis intervention plans; worked as part of a multidisciplinary team representing education, social work, psychology, psychiatry, occupational therapy, speech pathology, music therapy, and nursing to ensure coordination of services for students
August 2006 - May 2007	Therapeutic Behavior Aide *Kennedy Krieger Institute, Baltimore, MD* Worked one-on-one with a student with Fetal Alcohol Syndrome in a non-public special education high school; responsible for adapting

lesson plans and assignments to fit her needs and abilities; assisted in the development and implementation of behavioral intervention plans and tracked behavioral data; collaborated with teachers, social worker, psychologist, and psychiatrist to coordinate care

PROFESSIONAL REFERENCES

Richard R. J. Lewine, Ph.D.
Department of Psychological and Brain Sciences
University of Louisville
Louisville, KY 40292
(502) 852-3243
rich.lewine@louisville.edu

Benjamin Mast, Ph.D.
Department of Psychological and Brain Sciences
University of Louisville
Louisville, KY 40292
(502) 852-3280
b.mast@louisville.edu

Jack J. Blanchard, Ph.D.
Department of Psychology
University of Maryland
College Park, MD 20742
(301) 405-8438
jblancha@umd.edu