THE PLACEMENT OF HYPOMANIA WITHIN A STRUCTURAL MODEL OF PSYCHOPATHOLOGY INCLUDING THOUGHT DISORDER AND INTERNALIZING SPECTRA

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The Placement of Hypomania within the Thought Disorder and Internalizing Spectra of Psychopathology

Schizotypy refers to traits or symptoms similar to schizophrenia, but in a diminished form, and is thought to reflect a liability for schizophrenia-spectrum disorders (Chapman, Chapman, Raulin, & Edell, 1978; Meehl, 1962). Like schizophrenia, schizotypy is heterogeneous, and can be separated into at least three symptom clusters: positive, negative, and disorganized (Kwapil & Barrantes-Vidal, 2015; Raine et al., 1994; Tandon, Nasrallah & Keshavan, 2009). Positive schizotypy refers to subclinical positive symptoms of schizophrenia spectrum disorders such as attenuated delusion-like beliefs and hallucinations (Claridge, 1997; Lenzenweger, 2006; Meehl, 1962; 1990). Negative schizotypy refers to subclinical negative symptoms such as social anhedonia and constricted affect (Lenzenweger & Dworkin, 1996; Kwapil, Gross, Silvia, & Barrantes-Vidal, 2013). Disorganized schizotypy includes symptoms, like disorganized speech, that reflect difficulties maintaining linear thought processes (Hewitt & Claridge, 1989; Kerns & Becker, 2008).

Another potential risk factor for the development of schizophrenia-spectrum disorders and other serious mental illness is hypomania (Eckblad & Chapman, 1986). Hypomania refers to elevated mood states similar to mania, but lower in intensity and duration (American Psychiatric Association, 2013). Early work on schizotypy included hypomania among the risk factors for schizophrenia (Eckblad & Chapman, 1986).

Recently, efforts have been made to structurally model psychopathology from a dimensional perspective (e.g., Forbush & Watson, 2013; Kotov et al., 2011; Kotov et al., 2017; Wright et al., 2012). For example, the recently developed Hierarchical Taxonomy of Psychopathology (HiTOP) has six broad spectra including internalizing, disinhibited

externalizing, antagonistic externalizing, thought disorder, detachment, and somatoform (Kotov et al., 2017). In this model, positive schizotypy traits (i.e., psychoticism) are included on the thought disorder spectrum while depression, anxiety, and obsessive compulsive disorder symptoms are included on the internalizing spectrum, and negative schizotypy traits (e.g., anhedonia, withdrawal, etc.) are included on the detachment spectrum. The somatoform spectrum contains disorders of somatization such as illness anxiety disorder, while the disinhibited and antagonistic externalizing spectra contains disorders like substance use disorders and attention-deficit/hyperactivity disorder (ADHD), respectively. One major unanswered question in these taxonomies is to which spectrum/spectra bipolar spectrum disorders belong. The HiTOP model provisionally includes mania/hypomania on both the internalizing and thought disorder spectra and suggests that more research is needed to understand where it belongs in the taxonomy (Kotov et al., 2017). Thus, it is not clear whether hypomania is better understood as a facet of internalizing pathology or of a thought disorder.

One way to help determine the relation that hypomania has with other forms of psychopathology is to examine the differential relations that they have with external validators, such as personality variables. Maladaptive personality is linked to a wider range of psychopathology (Clark, 2005), but the relationship may differ between different personality factors and different psychopathology. Thus, it may be helpful to identify underlying personality traits that are more likely to be linked to one form of psychopathology over another. As a result differing relationships could reveal trends in how hypomania is related to other psychopathology.

The debate about whether positive schizotypy and hypomania belong on the same spectra in structural models mirrors the debate about whether schizophrenia-spectrum disorders can be discriminated from bipolar-spectrum disorders (Kotov et al., 2011; Wright et al., 2012). The

distinction between schizophrenia and bipolar disorder dates back to Kraepelin, who proposed the dichotomy (Kraepelin, 1896). However, it is not clear if this is still an adequate distinction, or if a more nuanced model is appropriate. Kraepelin himself later doubted whether these disorders could in fact be discriminated (Lake & Hurwitz, 2007; Kraepelin, 1920). Recent molecular genetic research has found that schizophrenia-spectrum and bipolar spectrum disorders share some genetic susceptibility (Berrettini, 2003; Cardno & Owen 2014; Schulze et al., 2014), and behavioral genetic studies have found that schizophrenia-spectrum and bipolar-spectrum co-aggregate within families (Craddock, O'Donovan, & Owen 2005), suggesting there may not be a genetic distinction between the two disorders. Moreover, some studies have proposed a transdiagnostic psychotic phenotype consisting of a high number of manic symptoms (Boks, Leask, Vermunt, & Kahn, 2007; Kendler et al., 1998; Peralta & Cuesta, 2003).

In addition to behavioral and molecular genetic studies, schizophrenia and bipolar show similar developmental patterns and risk factors. For instance, both have an average age of onset in late adolescence/early adulthood (Lewinsohn, Seeley, Roberts, & Allen, 1997; Sham, MacLean, & Kendler, 1994). Both disorders have similar risk factors such as paternal age, urbanicity, and childhood adversity such as a loss of parent (Laursen, Munk-Olsen, Nordentoft, & Mortensen, 2007). Moreover, both disorders are associated with drug use (Demjaha, MacCabe, & Murray, 2011) and both show similar developmental brain structure abnormalities (Hallahan et al., 2011).

Along with a similarity in etiology, schizophrenia and bipolar disorder have similar phenomenological characteristics (Murray et al., 2004). The majority of patients with schizophrenia experience affective symptoms similar to those in bipolar disorder (Boks, Leask, Vermunt, & Kahn, 2007), and studies examining those at high risk for psychosis show that these

affective symptoms play an important role in the development of the disorder (Owens & Johnstone, 2006). Some theorists have suggested that depression and manic symptoms are included in the structure of schizophrenia-spectrum disorders, along with traditional positive, negative, and disorganized symptoms (Fonseca-Pedrero et al., 2011; Picardi et al., 2012; Ruggeri et al., 2005; Van Der Does et al., 1995). Neurobiological research has revealed that dopamine dysregulation is implicated in both disorders (Owen, O'Donovan, & Harrison, 2005), and many dopamine-related pharmacological treatments that are beneficial for one disorder tend to be helpful for the other. These include most atypical antipsychotics that block dopamine 2 (D2) receptors to reduce positive symptoms while simultaneously stabilizing mood symptoms—primarily mania (Stahl & Grady, 2014). Taken together, these results suggest that schizophrenia and bipolar disorder have similar genetic and environmental risk factors, clinical presentations, and prognoses.

Despite these similarities, there are also important differences observed between schizophrenia and bipolar disorder. One of the primary differences between bipolar disorder and schizophrenia is the effectiveness of lithium. Lithium has long been the frontline treatment for bipolar disorder, with its benefits being more conducive to a primarily manic presentation compared to a primarily depressive one (Stahl & Grady, 2014). A recent meta-analysis concluded that there is no quality evidence that indicates lithium, on its own, is an efficacious treatment for people with psychotic symptoms (Leucht, Helfer, Dold, Kissling, & McGrath, 2015). This same meta-analysis indicated that there was minimal "low quality" (p. 2) evidence that it is even partially effective as an augmenter to antipsychotic medication. This difference in treatment response suggests that the disorders may be distinct.

In addition to the lack of lithium effectiveness in schizophrenia, other differences between bipolar disorder and schizophrenia support the Kraepelinian dichotomy. The LMAN2L rs2271893 gene variant is associated with schizophrenia, with conflicting evidence of a relationship with bipolar disorder (Chen et al., 2013; Lim et al., 2014). Children eventually diagnosed with schizophrenia show more motor, language, and cognition deficits compared to their peers who are eventually diagnosed with bipolar disorder, which suggests differences in development (Cannon et al., 2002). People with schizophrenia tend to have lower IQ scores than people with bipolar disorder (Gilvarry et al., 2000) and more severe executive functioning deficits (Qureshi & Frangou, 2002). Thus, there are still key differences between schizophrenia and bipolar disorder that suggest they may be distinct disorders.

As mentioned, the debate regarding the differentiation of positive schizotypy from hypomania may show similar patterns to the debate regarding the Kraepelinian dichotomy distinguishing schizophrenia and bipolar disorder. Like the latter, positive schizotypy and hypomania may have many similarities both in clinical presentations and nomological networks. Despite this possibility, few studies have simultaneously examined positive schizotypy and hypomania together. The studies that have included both have typically found a moderate correlation between measures of hypomania and positive schizotypy (Chapman, Chapman, & Raulin 1978; Mahon, Perez-Rodriguez, Gunawardane, & Burdick, 2013; Morvan et al., 2011). Moreover, individuals with bipolar disorder show higher levels of positive schizotypy (Rybakowski & Klonowska, 2011). There is evidence that people experiencing high levels of psychological distress are prone to both positive schizotypy and hypomania (Preti et al., 2015) as well as a common relationship with non-pathological personality characteristics such as artistic

creativity (Rawlings & Locarnini, 2008). Thus, more research is needed to examine whether and how schizotypy and hypomania are related.

In contrast to evidence showing that positive schizotypy and hypomania are similar, other research suggests differences between the constructs. Although people with bipolar disorder score higher on measures of schizotypy compared to healthy controls, they still score significantly lower than people with schizophrenia (Rossi & Daneluzzo, 2002). This may indicate that, although similar, there are different liabilities wherein schizotypy represents an increased liability for schizophrenia, while hypomania represents more of an increased liability for bipolar disorder.

One direct way for determining whether constructs can be discriminated from each other is confirmatory factor analysis (Rubio, Berg-Weger & Tebb, 2001). However, to my knowledge, only one study has measured both schizotypy and mania in a confirmatory factor analysis. This study found that positive schizotypy, negative schizotypy, and mania loaded on to separate factors, but shared a single general factor (Preti et al., 2015). It is important to note, however, that this study used only a single measure of mania and single measure of schizotypy. Thus, these results may represent the separation of those measures rather than differences in the latent constructs. In addition, multiple other studies using exploratory factor analyses have found that a two-factor structure, separating hypomania and positive schizotypy (Preti et al., 2015), and hypomania and negative schizotypy (Akiyama et al., 2005) fits the data better than a single-factor structure.

There has been previous work suggests that hypomania may be more related to internalizing psychopathology than psychosis. Most notably, hypomania and the presence of a major depressive episode are both included as criteria for bipolar II disorder in the DSM-5.

Empirically, past research has shown that depression is associated, though not very strongly, with hypomania (Meyer, 2002). In fact, the coexistence of these two symptom sets has been seen in community longitudinal research. Patients with major depressive disorder and those with primarily hypomanic symptoms of bipolar disorder tend to show similar patterns in the future development of additional mood and anxiety disorders and the utilization of mental health resources (Päären et al., 2014). A qualitative analysis of interviews conducted with bipolar II disorder patients showed a common theme that many patients were aware of hypomania usually leading to depression (Fletcher, Parker, & Manicavasagar, 2013). However, hypomania and depression are in some ways diametrically opposed constructs since depression is primarily a negative affective state while hypomania is, typically, primarily a positive affective state.

One major difference between depression and mania (or hypomania) is the response to certain treatments. Lithium, as mentioned before, is effective in treating symptoms of bipolar disorder, but it is not as effective at reducing unipolar depressive symptoms. Selective serotonin reuptake inhibitors (SSRIs) are rarely given to bipolar disorder patients for fear that the alleviation of a depressive episode may lead to a manic/hypomanic state (Stahl & Grady, 2014). The difference between depression and hypomania has also been shown empirically. Patients who have previously only experienced a hypomanic state show different manic symptom patterns than those who have experienced both a hypomanic state and a depressed state (Benazzi & Akiskal, 2003), reflecting the differences between depression and hypomania

Similar to hypomania, positive schizotypy is also associated with depression. It is well-known that many people with frank psychosis have experienced a depressive episode at some point (Martin, Cloninger, Guze, & Clayton, 1985). This co-occurrence also exists, but to a lesser extent, for depression and positive schizotypy. One may hypothesize that depression would be

more strongly related to negative schizotypy due to the overlap of anhedonia in negative schizotypy, but previous research has shown that depression is more strongly associated with positive than negative schizotypy (Emsley et al., 1999; Drake et al., 2004; Lysaker, Bell, Bioty, & Zito, 1995). When tested empirically, negative affect was moderately associated with positive schizotypy, furthering the support for this relationship even at a subclinical level.

A second symptom commonly considered as part of internalizing pathology is anxiety. Although the evidence linking hypomania and generalized anxiety is relatively sparse, the comorbidity rate of generalized anxiety disorder and bipolar disorder is more established. Some estimates show that roughly one in five people with bipolar I disorder experiences generalized anxiety at some point (Simon et al., 2004), though bipolar I disorder showed a higher rate than bipolar II disorder, for which hypomania is a key feature. Other estimates are as high 30%-40% for generalized anxiety disorder and significantly higher when including other anxiety disorders (Boylan et al., 2004; Pini et al., 1999). This is considerably higher than the rates of anxiety disorders in people without bipolar disorder. One study examining the link between hypomania and generalized anxiety reported that young adults rate higher levels of anxiety during a time period the authors refer to as "dark hypomania," (primarily irritable and risk taking) though importantly not during "bright hypomania" (primarily active with elevated mood; Brand et al., 2015, p. 25). Finally, some symptom components (e.g., hyperarousal), are important characteristics of both anxiety and hypomania. This potentially indicates that the association between hypomania and generalized anxiety involves multiple other factors.

In addition to generalized anxiety, social anxiety is also considered to be a part of the internalizing spectrum. Although less researched than the relations among anxiety, depression, and mania, some research suggests that there may be a subset of people with social anxiety who,

when on medication, show hypomanic symptoms (Valença et al., 2005). These patients exhibit similar clinical features to patients with bipolar II disorder. Some debate that this is not actually a comorbidity, but instead a subtype of bipolar disorder where social anxiety is the primary symptom (Himmelhoch, 1998). In this perspective, only when removing the presence of anxious symptoms are clinicians and scores on psychological tests able to capture the presence of hypomania (i.e., the patients were always hypomanic, but social anxiety masked the symptoms). Others argue that social anxiety may precede hypomania, but very few people exhibit social anxiety symptoms during hypomanic episodes (Perugi et al., 2001). Regardless of what the relationship may be, previous studies have been retrospective and used mainly descriptive statistics. This is to say that an empirical rigorous test of the connection between social anxiety and hypomania is still needed, but that there is evidence that a significant association exists.

In contrast to hypomania, a long line of research suggests that schizotypy is related to social anxiety (Brown, Silvia, Myin-Germeys, Lewandowski, & Kwapil, 2008; Pallanti, Quercioli, & Hollander, 2004; Raine, Lencz, & Mednick, 1995). Early conceptualizations of schizotypy included social anxiety as a sign of schizotypy (Meehl, 1962; 1990). This is reflected in that excessive social anxiety is a symptom of schizoptypal personality disorder in the DSM-5 (American Psychiatric Association, 2013). The exact relations between social anxiety and schizotypy are unclear. Some research suggests that it may be similar to, but distinct from, social anhedonia, which is a prominent negative schizotypy symptom (Cicero et al., 2015). In contrast, social anxiety may be more closely related to the "affective instability" often seen in positive schizotypy (Brown et al., 2008, p. 131). This difference was seen in a comparison of social anxiety and social anhedonia. Those with social anxiety were higher in positive schizotypy while those with social anhedonia were higher in negative schizotypy (Brown, Silvia, Myin-Germeys,

& Kwapil, 2008). In a follow-up study, social anxiety was associated with positive schizotypy (Brown et al., 2008). Thus, I expected to find that positive schizotypy, but not hypomania, would be positively correlated with social anxiety.

As mentioned, negative affect—including anxiety and depression—is associated with positive schizotypy, and past research has shown that this association is stronger for positive schizotypy than negative schizotypy (Lewandowski et al., 2006). Negative affect is also considered to be one of the emerging symptoms when psychotic symptoms are first developing (Ulloa et al., 2000), and could potentially be related to the persistence of these initial positive symptoms (Debbané, Van der Linden, Gex-Fabry, & Eliez, 2009; Escher, Romme, Buiks, Delespaul, & Van Os, 2002). Despite this evidence, the association appears to be moderate at best.

Another disorder associated with internalizing pathology that is related to both bipolar disorder and psychotic spectrum disorders is obsessive-compulsive disorder (OCD). Multiple studies have shown that OCD and bipolar disorders are often comorbid, with estimates as high as 35% of patients with bipolar disorder also meeting criteria for OCD (e.g., Angst, 1998; Kruger et al., 1995; Perugi et al., 2001; see Amerio, Odone, Liapis, & Ghaemi, 2014 for a meta-analysis). Due to this high comorbidity, psychopathologists have suggested that there may be a subtype of OCD that includes mania (Hantouche et al., 2003). Those with comorbid bipolar disorder and OCD have significantly more functional impairments and more hospitalizations compared to those with only OCD alone (Zutshi, Kamath, & Reddy, 2007). Thus, one would expect to find that hypomania would be positively correlated with OCD symptoms.

Similar to the association between hypomania and OCD, positive schizotypy and OCD often co-occur (Poyurovsky & Koran, 2005) and the two are highly related (Chmielewski &

Watson, 2008). In a study of adults with OCD, half had at least mild schizotypal symptoms with about a quarter being moderate or higher (Sobin et al., 2000). This could be interpreted, similar to the hypomanic association, as a subtype of OCD patients who experience clinically relevant psychotic symptoms. For those high in schizotypy, positive psychotic symptoms seem to cluster with OCD symptoms more strongly than at lower levels of schizotypy, potentially supporting an OCD subtype of positive schizotypy (Suhr, Spitznagel, & Gunstad, 2006). OCD symptoms are also common in people with schizophrenia, with estimates suggesting that as many as 60% of people with schizophrenia have OCD symptoms (Berman et al., 1998). This is intuitive from a phenomenological perspective, since schizotypal symptoms such as irrational beliefs can be very similar to the types of irrational beliefs about danger seen in those with OCD. This may be due to similar neurobiological pathways in both disorders (Tibbo & Warneke, 1999). Although obsessions must be recognized as irrational to be considered on the OCD-spectrum, people with subclinical delusion-like beliefs also have insight that their beliefs may not be true. Like hypomania, I expected to find that positive schizotypy would be positively correlated with obsessive-compulsive disorder symptoms.

An important point to note regarding the association of hypomania within other forms of mental illness is the possible connection between hypomania and externalizing psychopathology. Specifically, research has demonstrated the possibility that ADHD and (hypo)mania are associated. One argument supporting this perspective is that people who go on to develop bipolar disorder are often first diagnosed with ADHD in childhood (Biederman et al., 1998; Carlson, 1998; Geller et al., 1998). This may represent a high misdiagnosis rate or a high comorbidity, but either way indicates that the two are clinically similar. Despite this, research supporting the

perspective of hypomania being externalizing is relatively unconvincing. Therefore, the current study will focus more on hypomania's associations with internalizing and thought disorders.

As mentioned previously, efforts have been made to structure psychopathology in a way consistent with the emerging dimensional approach to mental illness. Perhaps the leading set of studies in this new approach is Hierarchical Taxonomy of Psychopathology (HiTOP). This group has already published a preliminary analysis compiling research on the six spectra mentioned above (Kotov et al., 2017), and currently has several other projects in preparation. The goal of HiTOP is to solve the issues inherent in the current classification system of psychopathology as definable categories. The empirical approach taken in efforts like this is important to better classify different psychopathologies while simultaneously acknowledging the similarities and co-occurrences (Kotov et al., 2017).

One important aspect of HiTOP's first publication (Kotov et al., 2017) is the placement of mania within the six spectra. Although other disorders and their symptoms, such as substance use or schizophrenia, are clearly understood to belong to a particular spectrum, mania's position is still considered ambiguous. Evidence exists indicating that mania, and a corresponding diagnosis of a bipolar disorder, is associated with other internalizing disorders (Blanco et al., 2015; Forbush & Watson, 2013; Keyes et al., 2013; Kotov et al., 2015; Watson, 2005; Watson et al., 2012). This evidence is not comprehensive, however, as other research has pointed to mania existing within the thought disorder spectrum that includes schizophrenia and other psychotic disorders (Caspi et al., 2014; Keyes et al., 2013; Kotov et al., 2011). In this line of research, the similarities between mania and the positive symptoms of schizophrenia indicate overlapping features. The HiTOP model acknowledges that crossloading is possible, and potentially even expected when examining "such interstitial constructs" (Kotov et al., 2017, p. 463).

Interestingly, despite much research examining mania and how it fits into the larger structure of psychopathology, there has been relatively little research examining hypomania's position in that same structure. HiTOP acknowledges bipolar I and II together as a syndrome/disorder associated with the subfactor *mania*, though research does not appear to directly examine hypomania to determine if it in fact shares common characteristics with both internalizing and thought disorder (as mania does). It is important to know this, however, as the integration of differing levels of severity into a comprehensive model of psychopathology is necessary to ensure that the model can be applied in a variety of contexts and situations.

OCD is another example of an interstitial construct similar to mania. Within the HiTOP model, OCD is thought of as a subset of the fear cluster within internalizing disorders. Despite a long line of research supporting OCD as an internalizing disorder (e.g., Keyes et al., 2013; Krueger & Markon, 2006; Miller et al., 2008), there is some evidence that OCD shares common features with subfactors of the thought disorder spectrum (Caspi et al., 2014; Chmielewski & Watson, 2008). Moreover, OCD could function similarly to mania in that it shares common features of multiple spectra rather than fitting neatly into one dimension.

In addition to the structural relations of different psychopathologies, the association between psychopathology and normal personality traits is also important to examine. Specifically, the five-factor model of personality, including extraversion, agreeableness, conscientiousness, neuroticism, and openness (e.g., Costa & McCrae, 1992; Goldberg, 1993), shows important relations with different forms of psychopathology.

Maladaptive personality is linked with psychopathology (Clark, 2005). In fact, the five-factor model of personality has been linked to virtually every psychopathology from anxiety and depression (Kotov, Gamez, Schmidt, & Watson, 2010) to schizophrenia and other severe mental

illness (Schroeder, Naber, & Huber, 2014) to personality disorders (Samuel & Widiger, 2008; Saulsman & Page, 2004). This research consistently finds that extraversion, agreeableness, and conscientiousness are all negatively associated with psychopathology (i.e., as these traits increase, psychopathology decreases) while neuroticism is positively associated with psychopathology (Clark, 2005; Kotov et al., 2010, Saulsman & Page, 2004). Perhaps the strongest connection is between anxiety and neuroticism, as neuroticism is essentially the trait version of the anxiety state. Openness is not generally considered to be as relevant in many of the associations between personality factors and psychopathology (Kotov et al., 2010, Samuel & Widiger, 2008; van der Heijden et al., 2013, Watson, Clark, & Chmielewski, 2008). This could represent a flaw in measurement of openness (e.g., that it does not capture pathological openness) or a differential way that openness affects experiences compared to other personality factors. The current research aims to replicate these findings as a way to validate this structural model of psychopathology.

Current Study

The primary goal of the current research was to examine where hypomania fits within structural models including positive schizotypy, negative schizotypy, depression, anxiety, social anxiety, and obsessive-compulsive disorder. Based on previous research, the main tests examined comparisons between models that allowed hypomania to be associated with positive schizotypy, depression, both, or neither. Due to discrepancies in the literature surrounding OCD, a similar set of comparisons were conducted, and combined with comparisons that included hypomania in different classifications. A secondary goal was to examine how personality factors differential relate to different forms of psychopathology, in order to externally validate the placement of hypomania within the structural model.

Method

Participants

Participants were 949 (M_{age} = 20.48, SD = 3.98) undergraduates from a large, public university. They participated in exchange for partial class credit. The sample was ethnically diverse, with 40.6% Asian, 17.3% White, 3.1% Hispanic, 1.6% Pacific Islander, 1.3% Black, 27.7% Multiethnic, and 8.3% Other. The sample was a majority female (74.3%). Originally, there were 1104 participants, but 155 were excluded for scoring above five on a scale for infrequent responding. This scale contains items that are highly unlikely to be endorsed such, "I have never spoken to a person wearing eye glasses." Higher scores indicate a higher likelihood of careless responding.

Materials

Positive Schizotypy. The first measure of positive schizotypy was the Magical Ideation Scale (MagicId; Eckblad & Chapman, 1983). The MagicId is a 30-item, true-false measure that assesses unrealistic beliefs of causality (e.g., "The hand motions that strangers make seem to influence me at times"). The second measure of positive schizotypy was the Perceptual Aberration Scale (PerAb; Chapman, Edell, & Chapman, 1980). The PerAb is a 35-item, true-false scale designed to measure psychotic-like distortions such as, "I have sometimes felt that some part of my body no longer belongs to me." Both the MagicId and the PerAb have notable support for their reliability and validity (Edell, 1995). In the current sample, the MagicId and PerAb had Cronbach's α's of .82 and .90, respectively. The third and fourth measures of positive schizotypy were the Unusual Perceptual Experiences (SPQ-UPE) and the Magical Ideation (SPQ-MI) subscales of the Schizotypal Personality Questionnaire (SPQ; Raine, 1991). The SPQ-UPE contains 9 items measuring abnormal sensations (e.g., "I often hear a voice

speaking my thoughts aloud") and had a Cronbach's α = .77 in the current study. The SPQ-MI comprises 7 items assessing odd beliefs (e.g., "Are you sometimes sure that other people can tell what you are thinking") and had a Cronbach's α = .75 in the current study. The SPQ is one of the most widely-used measures of schizotypy and has shown good factor structure and validity (e.g., Cicero, 2015; Compton Goulding, Bakeman, & McClure-Tone, 2009; Fonseca-Pedrero, et al., 2014; Fossati et al., 2003)

Negative Schizotypy. The first measure of negative schizotypy was the Social Anhedonia scale (SocAn; Chapman et al., 1976). This measure contains 40 true-false items that assess a general lack of pleasure and enjoyment from social interactions (e.g. "I am usually content just to sit alone, thinking and daydreaming"). The Social Anhedonia scale predicts future development of psychosis (e.g., Gooding, Tallent, & Matts, 2005) and showed good reliability in the current sample (α = .82).

Second and third measures of negative schizotypy were the Constricted Affect and No Close Friends subscales of the SPQ (Raine, 1991). The Constricted Affect scale is comprised of 8 items assessing a lack of emotional range (e.g., "I rarely laugh or smile). The No Close Friends subscale is 9 items and measures a lack of closeness in relationships (e.g., "I attach little importance to having close friends"). These scales have been shown to load strongly onto a negative schizotypy latent factor (e.g., Cicero, 2015, Preti et al., 2015), and are highly associated with SCID ratings of negative symptoms (Raine, 1991). In the current study the Constricted Affect and No Close Friends subscales had Cronbach's α = .78 & .81, respectively.

Hypomania. The first measure of hypomania was the Hypomanic Personality Scale (HPS; Eckblad & Chapman, 1986). The HPS is a 48-item true/false questionnaire used to identify persons with hypomanic personality traits (Eckblad & Chapman, 1986). An example of a true-

keyed item is, “Sometimes ideas and insights come to me so fast that I cannot express them all.” Studies have shown the HPS to have good cross-cultural equivalency (Meyer, 2002). In this sample the HPS had a Cronbach’s α = .91. The second measure of hypomania was the Cyclothymia subscale of the Temperament Evaluation of Memphis, Pisa, Paris, and San Diego Autoquestionnaire (TEMPS-A; Akiskal et al., 2005). The Cyclothymia factor of the TEMPS-A contains 12 items with statements such as, “The way I see things is sometimes vivid, but at other times lifeless.” This subscale showed excellent internal consistency (Cronbach’s α = .91; Akiskal et al., 2005) in past studies and good reliability in the current sample (Cronbach’s α = .87). A third measure of hypomania was the Mood Disorder Questionnaire (MDQ; Hirschfeld et al., 2000), which is a 13-item, yes/no measure that can be used as a screener for bipolar disorders (Hirschfeld et al., 2000). With a cutoff score of 7, the MDQ has good sensitivity and very good specificity (Hirschfeld et al., 2000). The scale has items that examine energy level (e.g., “Has there ever been a period of time when… you were not your usual self and you were much more talkative or spoke faster than usual?”), mood (e.g., “…you felt much more self-confident than usual), and irritability (e.g., “…you were so irritable that you shouted at people or started fights or arguments?). In the current study, the MDQ had a Cronbach’s α = .85.

Depression. The first measure of Depression was the Center for Epidemiological Studies-Depression Questionnaire (CES-D; Radloff, 1977). The CES-D is a 20-item scale that measures common symptoms of depression such as sleep disturbances and eating habits. Participants rate each item on a scale from 0 (*Rarely or none of the time*) to 3 (*Most or all of the time*). The CES-D is primarily used to screen for current episodes of depression or assess levels of depressive symptoms (Waugh, Meyer, Youngstrom, & Scott, 2014). In the current sample, the CES-D had a Cronbach’s α = .96. A second measure of depression was the Depression Subscale of the

Depression Anxiety Stress Scale (DASS-Depression; Lovibond & Lovibond, 1993). The DASS is a 42-item self-report questionnaire for which participants rate statements from 0 (*did not apply to me at all*) to 3 (*applied to me very much, or most of the time*; Lovibond & Lovibond, 1993). Each of the three subscales of the DASS contains 14 items. Multiple studies have found support for excellent internal consistency of the Depression subscale (Lovibond & Lovibond, 1993; Lovibond & Lovibond, 1995; Antony, Bieling, Cox, Enns, & Swinson, 1998). Items address common symptoms of depression such as, "I felt that life wasn't worthwhile." In the current study, the depression subscale of the DASS showed excellent internal consistency ($\alpha = .93$).

A third measure of depression was the Inventory of Depression and Anxiety Symptoms (IDAS) General Depression Scale. The IDAS is a 64-item measure, 20 of which make up the General Depression scale (Watson et al., 2007). Participants rate statements on a scale from 1 (*not at all*) to 5 (*extremely*) regarding how much the statement applied to them over the past two weeks. The General Depression scale showed excellent reliability (Cronbach's $\alpha = .92$) in the current study and correlates well with other measures of depression (Watson et al., 2007). A fourth measure of depression was the Physical Health Questionnaire-9 (PHQ-9; Kroenke, Spitzer, & Williams, 2001). The PHQ-9 is a 9-item checklist used to screen for depression or evaluate depressive symptoms. Participants rate how often they have felt a certain symptom over the past two weeks from 0 (*not at all*) to 3 (*nearly every day*). Items address common symptoms such as lack of interest, feeling down, sleep disturbances, and changes in appetite. The scale has good test-retest reliability (Kroenke, Spitzer, & Williams, 2001), and showed excellent internal consistency in the current sample ($\alpha = .90$).

Finally, a fifth measure of depression was the Patient Reported Outcomes Measurement Information System-Depression (PROMIS-B). The PROMIS is a measure developed by the

National Institute of Health (NIH) that utilized item response theory to create a pool of questions measuring patient-reported outcomes across a variety of domains, with research finding that the scales show strong reliability and validity (see Cella et al., 2010 for a summary). Specifically, the depression scale of the PROMIS measures negative mood, decreased positive affect, information processing deficits, negative views of the self, and negative social cognition. In the current study, the depression scale had a Cronbach's $\alpha = .96$.

Anxiety. The first measure of anxiety was the Anxiety subscale of the Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1993). The DASS Anxiety subscale contains 14 items typically experienced during anxiety (e.g., "I felt scared without any good reason). Participants rate items on a Likert scale from 0 to 3, in terms of how much that statement applies to them. The anxiety subscale is highly correlated with the Beck Anxiety Inventory (Lovibond & Lovibond, 1995) and showed good reliability in the current study ($\alpha = .89$).

A second measure of Anxiety was the Patient Reported Outcomes Measurement Information System-Anxiety (PROMIS-A). As mentioned before, the PROMIS is a large-scale measure designed by the NIH to measure a variety of health related domains. The anxiety scale includes items assessing fear, anxious misery, hyperarousal, and somatic symptoms related to arousal (Cella et al., 2010). In the current study, this scale showed excellent reliability ($\alpha = .95$).

A third measure of anxiety was the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). The PSWQ is a 16-item Likert scale for which participants rate from 0 (*never*) to 6 (*almost always*) how much an item applies to them. The scale is designed to measure pervasive worry (e.g., "I am always worrying about something") as an independent trait. The PSWQ has high internal consistency and test-retest reliability (Meyer et al., 1990). In the current study, the PSWQ had a Cronbach's $\alpha = .92$.

Social Anxiety. The first measure of social anxiety was the Social Anxiety subscale of the IDAS (Watson et al., 2007). The Social Anxiety subscale of the IDAS contains five items that are traditionally experienced during anxiety such as, "I was worried about embarrassing myself socially." The scale has good reliability across a wide range of populations and correlates with other measures of social anxiety (Watson et al., 2007). In the current sample, the IDAS showed good reliability ($\alpha = .87$). A second measure of social anxiety was the Excessive Social Anxiety subscale of the SPQ (SPQ-ESA; Raine, 1991). SPQ-ESA is an 8-item subscale that evaluates anxiety in situations involving interaction with others (e.g., "I feel very uncomfortable in social situations involving unfamiliar people"). The ESA subscale had good reliability in the current study ($\alpha = .86$).

Obsessive-Compulsive Disorder. The first measure of obsessive-compulsive disorder symptoms was the Obsessive Compulsive Inventory-Revised (OCI-R; Foa et al., 2002). The OCI-R is an 18-item questionnaire designed to measure distress from OCD symptoms. Participants rate the severity of symptoms (e.g., "I need things to be arranged in a certain way) over the past month on a scale from 0 (*not at all*) to 4 (*extremely*). Both the total score and subscale scores have good internal consistency in clinical samples (Foa et al., 2002), but only the total scale shows adequate test-retest reliability for an undergraduate sample (Hajcak, Huppert, Simons, & Foa, 2004). Another study found that the OCI-R was correlated with other measures of OCD, while also discriminating from other anxiety disorders (Abramowitz & Deacon, 2006). In the current sample, the OCI-R had excellent internal consistency ($\alpha = .95$). A second measure of OCD symptoms was the Vancouver Obsessional Compulsive Inventory (VOCI; Thordarson et al., 2004). The VOCI is a 55-item measure aimed at assessing a variety of obsessions, compulsions, and other characteristics of OCD (e.g. "I am excessively concerned about getting

germs and disease"). Items are rated on a 5-point scale from "not at all" to "very much" regarding how true the statement is. The VOCI total score has very good internal consistency and excellent test-retest reliability (Thordarson et al., 2004). In this sample, the VOCI had a Cronbach's $\alpha = .94$.

Big Five Personality. Personality was measured using Goldberg's (1992) Big-Five Factor Markers from the International Personality Item Pool (Goldberg et al., 2006). This measure is correlated with other measures of the Big-Five and has a wide research base supporting its validity (e.g., Briley, Domiteaux, & Tucker-Drob, 2014; Gnambs, 2014; Gow, Whiteman, Pattie, & Deary, 2005; Saucier & Goldberg, 2002; Zheng et al., 2008). The five factors and their reliabilities in the current study are as follows: Extraversion $\alpha = .89$, Agreeableness $\alpha = .89$, Conscientiousness $\alpha = .82$, Neuroticism $\alpha = .90$, Openness $\alpha = .84$.

Procedure

Participants completed the study online in a single session taking an average of 60 minutes with the online program Qualtrics. Participants completed the study in the following order: Center for Epidemiological Studies Depression Scale Revised, Inventory of Depression and Anxiety Symptoms, Obsessive Compulsive Inventory- Revised, the Patient Health Questionnaire-9, Patient Reported Outcomes Measurement Information System- Anxiety Scale, Patient Reported Outcomes Measurement Information System- Depression Scale, The Schizotypal Personality Questionnaire, The Survey of Attitudes and Experiences (including the Perceptual Aberration Scale, Magical Ideation Scale, and Social Anhedonia Scale), The Temperament Evaluation of the Memphis, Pisa, Paris, and San Diego-autoquestionnaire, The Mood Disorder Questionnaire, the Vancouver Obsessional Compulsive Inventory, the Penn State

Worry Questionnaire, The Hypomanic Personality Scale, and the International Personality Item Pool.

Data Analyses. All models were tested with M-Plus Version 7.4 (Muthen & Muthen, 1998-2016). Data were extracted with Maximum Likelihood parameter estimates (ML). The primary comparison of model fit was Bayesian Information Criterion (BIC), as recommended by current literature (Vrieze, 2012). In addition to BIC, Sample-Size Adjusted BIC (SABIC) and Akaike Information Criterion (AIC) were used to evaluate comparative fit (i.e., one model against another). Root Mean Squared Error of Approximation (RMSEA), Comparative Fit Index (CFI), Tucker-Lewis Index (TLI), and Standardized Root Mean Squared Residual (SRMSR) were used to evaluate absolute fit. Following previous work, the models were considered acceptable if (a) CFI and TLI > .90, and (b) RMSEA and SRMSR < .10. Model fit was considered excellent if (a) CFI and TLI > .95, and (b) RMSEA and SRMSR < .05. For all analyses, I allowed the errors of subscales from the same measure to correlate with each other.

Thirteen models were tested to determine the best fitting model. (1) A seven-factor model with positive schizotypy, negative schizoytypy, hypomania, depression, anxiety, social anxiety, and OCD loading on separate factors. This model tested whether hypomania, positive schizotypy, and OCD were distinct constructs that are not part of common higher order factors. (2) A six-factor model with hypomania scales and positive schizotypy scales loading onto the same factor. This model examined whether hypomania could be discriminated from positive schizotypy (i.e., if this model fit better than model 1, I would conclude that hypomania could not be discriminated from positive schizotypy). (3) A six-factor model with hypomania scales and depression scales loading onto the same factor. This model examined whether hypomania could be discriminated from depression. (4) A six-factor model with hypomania scales loading onto

both the positive schizotypy scale and the depression scale. This model tested whether hypomania is a part of both positive schizotypy and depression. (5) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and hypomania) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model tested whether hypomania best fits as being part of both a higher order thought disorder spectrum and a higher-order internalizing spectrum. (6) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, negative schizotypy, and hypomania) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model examined whether hypomania should be a part of both higher order factors while also including negative schizotypy as a thought disorder component. (7) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, hypomania, and OCD) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model tested whether both OCD and hypomania can be considered part of a higher order thought disorder spectrum and higher order internalizing spectrum. (8) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, negative schizotypy, hypomania, and OCD) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model tested the same possibility as model 8, but with negative schizotypy as a part of thought disorder. (9) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and hypomania) and internalizing (depression, anxiety, social anxiety, and OCD). This model examined if hypomania should solely be included as part of thought disorder with OCD exclusively part of internalizing. (10) A seven-factor model with one higher-order factor, internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model examined hypomania as only being a component of a higher-order internalizing spectrum, thereby eliminating the higher order thought disorder component.

(11) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and negative schizotypy) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). This model tested the possibility of hypomania exclusively loading on internalizing while positive and negative schizotypy encompass the thought disorder spectrum. (12) A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and OCD) and internalizing (depression, anxiety, social anxiety, and OCD). This model examined if hypomania forms its own factor entirely without being part of a higher order factor. OCD was examined as a part of both thought disorder and internalizing. (13) A seven-factor model with one higher-order factor, internalizing (depression, anxiety, and social anxiety). In this model, OCD and hypomania were both considered independent, leaving positive schizotypy, negative schizotypy, OCD and hypomania as first-order factors.

Results

Zero-Order Correlations. I first examined the zero-order correlations among all the variables in the current study. As can be seen in Table 1, the variables within a certain psychopathology were all strongly correlated with each other (e.g., measures of positive schizotypy with other measures of positive schizotypy). All psychopathology measures were significantly associated with all other psychopathology measures with the exception of the Mood Disorder Questionnaire (hypomania) with the Social Anhedonia scale (negative schizotypy), which was nonsignificant.

Structural Model of Psychopathology. The primary goal of the current research was to determine where hypomania fits into a structural model of psychopathology. As can be seen in Table 2, Model 1 (in which all forms of psychopathology were independent, without the presence of higher order factors), Model 4 (in which hypomania was considered to be a part of

the positive schizotypy and depression simultaneously), and Model 7 (in which OCD and hypomania crossloaded onto both a thought disorder and an internalizing higher-order factor fit the data well according to the absolute fit indices). Model 7 was retained as the best fitting model for three reasons. First, it had the lowest BIC, which was my primary indicator of the best fitting model (Vrieze, 2012). Second, Model 7 was the most parsimonious of the three well-fitting models because it required the estimation of only 90 parameters rather than 103 and 100 parameters for Model 1 and Model 4, respectively. Third, Model 7 (Figure 1) is the most consistent with previous research, which has nearly universally found the existence of higher-order internalizing and thought disorder factors. A complete list of coefficients and associations for Model 7 can be seen in Tables 3-5.

Personality Variables on Psychopathology. A series of regressions was conducted in which each of the five personality variables were regressed on each of the seven first-order factors. A summary of results can be seen in Table 6, with a visual representation in Figure 2. After using a Bonferroni correction for multiple comparisons, significance was determined to be alpha $\leq .001$. Negative schizotypy was negatively associated with extraversion, agreeableness, conscientiousness, and openness and positively associated with neuroticism. Positive schizotypy was negatively associated with agreeableness and conscientiousness, and positively associated with neuroticism. Hypomania was negatively associated with extraversion and conscientiousness, and positively associated with neuroticism. OCD was negatively associated with extraversion, agreeableness, and conscientiousness, and positively associated with neuroticism. Depression was negatively associated with extraversion and conscientiousness, and positively associated with neuroticism. Anxiety was negatively associated with extraversion and conscientiousness, and positively associated with neuroticism. Finally, social anxiety was

negatively associated with extraversion, agreeableness, and conscientiousness, and positively associated with neuroticism. Note that conscientiousness was negatively associated with every psychopathology while neuroticism was positively associated with every psychopathology. Openness was only negatively associated with negative schizotypy.

Discussion

Recent attempts to structurally model psychopathology have largely shown that psychopathologies tend to cluster into higher-order dimensions or spectra (Kotov et al., 2017). To date, the question regarding where hypomania fits into these dimensions is unanswered. Thus, the primary goal of the current research was to determine where hypomania fits within the structure of psychopathology. The best fitting model included a first-order hypomania factor that crossloaded both on the higher-order internalizing factor and the higher-order thought disorder factor. This result is consistent with previous research suggesting hypomania shares similarities with both depression and schizophrenia (Blanco et al., 2015; Caspi et al., 2014; Forbush & Watson, 2013; Keyes et al., 2013; Kotov et al., 2011; Kotov et al., 2015; Watson, 2005; Watson et al., 2012). Specifically, mood disturbances are central to both internalizing disorders and hypomania (Achenbach, 1966; American Psychiatric Association, 2013).

Similarities between hypomania and thought disorder include increased grandiosity with frequent psychotic symptoms experienced by those in a manic/hypomanic state (Dunayevich & Keck, 2000; Goodwin & Jamison, 1990; Turkington & Kingdon, 1996; Verduox et al., 1998; Wigman et al., 2012). In addition, the overlap of mania with thought disorder and internalizing disorders is underscored by the DSM-5 diagnosis of schizoaffective disorder, which is essentially a combination of a psychotic-spectrum disorder and a mood disorder (American Psychiatric Association, 2013).

The OCD first-order factor also crossloaded on both the internalizing and thought disorder factors. This finding is consistent with some previous research that indicates the obsessions in OCD share similar qualities to delusions in schizophrenia-spectrum disorders, while also showing similarities to anxiety and other internalizing disorders (Chmielewski & Watson, 2008; Keyes et al., 2013; Krueger & Markon, 2006; Miller et al., 2008; Poyurovsky & Koran, 2005, Sobin et al., 2000).

The current research found that subscales of common constructs factored together. This led to a well-fitting seven-factor structure of the data wherein positive schizotypy, negative schizotypy, hypomania, depression, anxiety, social anxiety, and obsessive-compulsive disorder were separate factors (i.e., Model 1). Models containing fewer factors did not fit the data as well. For example, Models 2-4 all tested hypomania scales as loading onto depression, positive schizotypy, or both; none of these models fit the data well. For this reason, the seven-factor model was retained and then used to test the higher-order structure of the data. Overall, this suggests that these seven constructs (i.e., positive schizotypy, negative schizotypy, hypomania, OCD, anxiety, depression, and social anxiety) can be discriminated from each other. This finding is especially important for the interpretation of hypomania, and suggests that it is distinct from both positive schizotypy and internalizing pathology, which is consistent with previous research. As mentioned, the best fitting model included the first-order hypomania factor cross loading on both the thought disorder and internalizing second-order factors. This finding is consistent with previous postulations that hypomania is associated with both positive schizotypy (Caspi et al., 2014; Keyes et al., 2013; Kotov et al., 2011) and depression (Blanco et al., 2015; Forbush & Watson, 2013; Keyes et al., 2013; Kotov et al., 2015; Watson, 2005; Watson et al., 2012). This finding is consistent with the HiTOP model, which provisionally had hypomania crossloading on

both the thought disorder and internalizing spectra (Kotov et al., 2017). Like the HiTOP model, this finding partially supports the Kraepelinian dichotomy. On the subclinical level, positive schizotypy and hypomania appear to be highly related and part of the same spectrum that likely functions through similar processes. Yet at the same time, hypomania's crossloading on depression indicates that the mood component can separate the two and show differential relations with other psychopathology.

As previously mentioned, the connection between bipolar disorders and schizophrenia spectrum disorders has been widely examined since Kraepelin proposed the dichotomy (Kraepelin, 1896). This dichotomy has been questioned throughout the years (Lake & Hurwitz, 2007), but has been maintained through the current diagnostic system (American Psychiatric Association, 2013). Research in behavioral genetics (Berrettini, 2003; Cardno & Owen 2014; Craddock, O'Donovan, & Owen 2005; Schulze et al., 2014), phenomenology (Boks, Leask, Vermunt, & Kahn, 2007; Murray et al., 2004; Owens & Johnstone, 2006), and risk factors and development (e.g., Hallahan et al., 2011; Lewinsohn, Seeley, Roberts, & Allen, 1997; Sham, MacLean, & Kendler, 1994; Laursen, Munk-Olsen, Nordentoft, & Mortensen, 2007) has also pointed to the possibility that the two disorders are highly related, if not part of the same spectrum. At the same time, research in those areas has suggested that they are in fact distinct (Cannon et al., 2002; Chen et al., 2013; Leucht, Helfer, Dold, Kissling, & McGrath, 2015; Lim et al., 2014; Qureshi & Frangou, 2002). The current research supports a perspective indicating overlapping symptomatology, with clear distinctions. Hypomania was tested as solely belonging to the thought disorder spectrum or as part of positive schizotypy as a first-order factor. In both of these scenarios, the model did not fit the data as well, indicating that hypomania (and

presumably mania) cannot be considered synonymous with positive schizotypy (or positive symptoms in schizophrenia).

In addition to being highly related to, but distinct from, positive schizotypy, hypomania has the same relation to depression. As mentioned, depressive symptoms are a core feature of bipolar disorders in traditional nosologies (American Psychiatric Association, 2013). The two share similar developmental characteristics (Betts, Williams, Najman, & Alati, 2016; Payá et al., 2013; Zammit et al., 2004) and are considered to have reciprocal cycling between the two types of symptoms in bipolar disorders (American Psychiatric Association, 2013). However, there are several reasons that hypomania and depression are distinct constructs. First, by definition, they involve experiences that exist on opposite sides of the affect spectrum. Second, they appear to have different biological mechanisms, represented by their differential response to psychopharmacological treatments (Stahl & Grady, 2014). The current results indicate that hypomania and depression are in fact related, as is currently reflected in the DSM-5. Models in which hypomania was either not related to depression or solely related to depression did not fit the data well. Similar to the relation between positive schizotypy and hypomania, depression and hypomania seem to be distinct, but highly related, constructs.

In addition to hypomania loading on both the thought disorder and internalizing spectra, the best fitting model included OCD on both spectra. This finding was somewhat surprising because most recent structural models have suggested that OCD either loads along with internalizing or forms a distinct factor (e.g., Kotov et al., 2011; Kotov et al., 2017; Wright et al., 2012) though others have mentioned that it might be partially related to thought disorder (Caspi et al., 2014; Watson, Wu, & Cutshall, 2004).

Perhaps the best reflection of OCD's changing position within the larger framework of psychopathology can be seen through how it has changed in the DSM. In the DSM-IV, OCD was considered to be part of anxiety disorders (American Psychiatric Association, 2000), which had clearly been determined to be part of the internalizing spectrum. In this sense, OCD has traditionally been viewed as an internalizing disorder. By the release of the DSM-5, OCD's position had changed and currently exists as its own section that is considered distinct from anxiety (American Psychiatric Association, 2013). This represents the changing perspective of OCD, and allows for the possibility that its placement along with anxiety disorders is inaccurate. Results of the current study indicate that it may in fact be connected to anxiety and other internalizing psychopathology while also being related to the psychotic disorders spectrum.

Though OCD has been shown in the past to load solely onto the internalizing spectrum, these data support the notion that OCD symptoms (or at least the obsessive portions) are also closely related to thought disorder symptoms such as odd thinking, a primary component seen in positive schizotypy. One potential explanation for the difference between the current results and the previous findings is that previous structural attempts have used full-blown psychosis instead of subclinical psychotic processes. OCD symptoms may be closely related to subclinical thought disorder but differ from thought disorder symptoms seen in those who have converted to a schizophrenia spectrum disorder.

There are marked similarities between unusual beliefs experienced with positive schizotypy and the obsessions that are essential for a diagnosis of OCD. For instance, both require unrealistic perceptions of cause and effect. In fact, the obsessions seen in OCD could sometimes be considered unusual beliefs (e.g., contamination beliefs with health outcomes that are highly unlikely). However, delusions are deeply-held beliefs that are maintained when clear

contradictory evidence is presented. Obsessions, on the other hand, are recognized by the individual as not rational, but are nevertheless very compelling. To treat both, replacement beliefs are usually utilized (Clark & Beck, 2010; Nelson, 2005), though exposure is typical used for obsessions (Abramowitz et al., 2011; Foa, Deacon, & Whiteside, 2005). To the best of my knowledge, there is no evidence of treatment efficacy for exposure in delusions.

One possibility for the similarity between obsessions and delusions is thought-action fusion. Thought-action fusion is the belief that simply thinking about an action is identical to actually completing that action, and is common in people with OCD (Berle & Starcevic, 2005; Rachman, 1993; Salkovskis, 1985; Shafran, Thordarson, & Rachman, 1996). In fact, some notions of OCD posit that thought-action fusion is a driving mechanism in maintaining the symptoms associated with the disorder. Thought-action fusion has also recently been studied in people with schizophrenia, with results indicating that people with schizophrenia endorse thought-action fusion likelihood to a greater extent that comparison participants, and thoughts associated with unethical behaviors were associated with delusional beliefs (Berle, Blaszczynski, Einstein, & Menzies, 2006). Given its delusion-like presentation, individuals high on measures of positive schizotypy may also experience this phenomenon, but potentially to a lesser extent. It is important to note, however, that in the previously-mentioned study, there were still low levels of endorsement overall for thought-action fusion beliefs in the chronic schizophrenia sample. This may indicate that thought-action fusion is one of multiple different shared mechanisms for the overlap between schizophrenia-spectrum disorders and OCD. Specific measures of thought-action fusion were not included in this study, however, future studies may examine to what extent this phenomenon is driving this association.

Despite the finding that OCD crossloaded on the thought disorder spectrum and the phenomenological similarities, there are several important distinctions between the disorders. First, the current study indicates that OCD also crossloads with internalizing psychopathology. This is consistent with previous research findings demonstrating that OCD loads on the internalizing spectrum in structural analyses. Second, though obsessions and delusions are similar as mentioned previously, there are notable differences. Third, even with potentially overlapping symptoms, there are clear distinctions. For instance, the experience of hallucinations within psychotic disorders is frequently a key diagnostic feature but is not present in typical OCD (American Psychiatric Association, 2013). Fourth, there are important differences in functioning and quality of life or morbidity in people with schizophrenia compared to people with schizophrenia and comorbid OCD symptoms. People in the latter group are usually lower functioning, have more hospitalizations, and more psychosocial difficulties (Berman et al., 1998; Niendam, Berzak, Cannon, & Bearden, 2009; Poyurovsky et al., 2001; Samuel et al., 1993). Comparing individuals with each disorder independently shows that those with schizophrenia experience a lower quality of life compared to those with OCD (Koran, Thienemann, & Davenport, 1996; Spitzer et al., 1995). One reason schizophrenia-spectrum disorders are associated with higher morbidity and lower quality of life than are OCD-related disorders is that people with schizophrenia often also have negative symptoms and cognitive deficits that seem to drive the lowered quality of life in people. Finally, a first-order model in which the OCD indicators loaded along with positive schizotypy did not fit the data well and fit worse than a model with OCD and positive schizotypy loading on separate factors. Thus, it appears that OCD can clearly be discriminated from psychotic disorders. Future research may further explicate these distinctions using general population and clinical samples.

In addition to the findings related to OCD, another important finding of the current research is the replication of findings supporting an internalizing spectrum. As is supported widely in the literature, depression, anxiety, and social anxiety loaded onto a second-higher internalizing factor (Achenbach, 1966; Achenbach et al., 1991; Blanco et al., 2015; Lahey et al., 2004; Kotov et al., 2017). The current study adds to cross-cultural literature by replicating these findings in an ethnically diverse sample, suggesting that this structure could hold up across ethnicities (Krueger et al., 2003).

Interestingly, negative schizotypy did not load onto a higher-order factor. Its indicators, though generally considered to be part of a broader schizotypy construct (Kwapil & Barrantes-Vidal, 2015), also did not combine to create a total schizotypy first-order factor. The differentiation between negative schizotypy and positive schizotypy is well-documented (Chmielewski & Watson, 2008; Kwapil & Barrantes-Vidal, 2015; Raine et al., 1994; Tandon, Nasrallah & Keshavan, 2009), however, given that they both are associated with an increased risk of schizophrenia, the continued support indicating that they also belong to different higher-order factors is important. In the HiTOP model, negative schizotypy is not directly mentioned, but schizoid personality disorder is part of separate higher-order spectrum referred to as *detachment*, though it crossloads onto the thought disorder spectrum (Kotov et al., 2017). At the same time, the individual symptoms making up negative schizotypy are all included on the detachment spectrum. The current study did not directly measure other forms of detachment. Future research could include multiple measures of negative schizotypy along with multiple measures of detachment to determine whether negative schizotypy loads along with detachment or forms a distinct factor.

Regarding the analyses involving personality measures, the current results are consistent with some previous studies, but inconsistent with others. For example, researchers have long debated whether openness to experience is relevant to psychopathology, with some researchers suggesting that it should be associated with increased positive schizotypy (Ross, Lutz, & Bailley, 2004; Edmundson, Lynam, Miller, Gore, & Widiger, 2011). In the current research, openness to experience was only, and negatively, associated with negative schizotypy. In recent conceptualizations of personality pathology such as the Personality Inventory for the DSM-5, openness is excluded entirely (American Psychiatric Association, 2013). In these models, maladaptive openness is replaced by psychoticism, which is often used interchangeably with positive schizotypy. The lack of significant findings for relations with openness provides more support for removing openness from personality pathology conceptualizations.

A second finding related to personality was that neuroticism and conscientiousness are both associated with all seven first-order factors. This could represent an increased liability based on these traits, or reflect common resulting behaviors from the difficulty of suffering from a mental illness. Other than neuroticism and conscientiousness, the other factors from the five-factor personality model showed differential relations across psychopathologies. As anticipated, neuroticism was most strongly associated with internalizing psychopathology, specifically anxiety. In line with past research, extraversion was strongly associated with both negative schizotypy and social anxiety (Krueger & Markon, 2014). Negative schizotypy encompasses social anhedonia, which is distinct but related to social anxiety (Brown, Silvia, Myin-Germeys, Lewandowski, & Kwapil, 2008). Though they differ, the behavioral result of both may be similar in that they lead one to avoid social situations, in other words, being less extroverted.

Importantly, however, social anxiety is generally considered to be internalizing pathology, while social anhedonia would be related to social withdrawal and detachment (Kotov et al., 2017).

As with all studies, this study had limitations that may need to be addressed in future studies. The first is that this study examined only two spectra: internalizing and thought disorder. Some evidence suggests that hypomania may in fact be associated with externalizing pathology as well such as attention deficit hyperactivity disorder (ADHD; see Brody, 2001 for a review). Hypomania may be related to ADHD, and externalizing pathology more generally, in a multitude of ways. First, increased activity is arguably the defining feature of both ADHD, hyperactive type, and (hypo)mania (Biederman et al., 1998; Geller et al., 1998). Second, both disorders are also associated with increased impulsivity (Barkley, 1997; Brody, 2001). Despite clear knowledge that they are separate disorders with different pathophysiological and neurological differences (Blumberg et al., 2003; Dickstein et al., 2005; Pliszka et al., 2000), there exists enough overlap to reason that they could both be part of externalizing pathology.

The distinction between ADHD and the bipolar disorders is important because people with ADHD, particularly children, are often misdiagnosed as bipolar disorder and vice versa (Biederman et al., 1998; Carlson, 1998; Geller et al., 1998). Given the differences in appropriate treatment, differential diagnosis at early stages is essential. In addition to ADHD, hypomania may be related to other types of externalizing pathology such as oppositional defiant disorder (ODD) and intermittent explosive disorder (IED). Both of these disorders have components that mirror those seen in people with mania or hypomania, such as those mentioned above. Future research could also include measures of externalizing pathology to see if hypomania loads along with those symptoms or crossloads with them in addition to internalizing and/or thought disorder.

A second limitation of this study was the use of undergraduate students, who may differ from a clinical population. Since the HiTOP model examines traits and symptoms at multiple levels, one would assume that subclinical representations of psychopathology would be structured similarly to clinical presentations. Although psychosis is currently conceptualized as a spectrum, with varying levels that can be modeled in general samples (van Os, Linscott, Myin-Germeys, Delespaul, & Krabbendam, 2009; Yung et al., 2009), undergraduates may not be representative of the general population. Undergraduate students likely have higher SES and higher IQs than a community sample, though research shows that undergraduates show levels of psychopathology that are comparable to those in the same age-range that are not attending college (Blanco et al., 2008). Future research could examine where hypomania fits in with the broader structure of psychopathology in clinical samples or samples drawn from the general population.

Another limitation of the current study is the use of self-report questionnaires as opposed to more intensive interviews. Though these particular self-report measures have shown strong reliability and validity in other studies (e.g., Akiskal et al., 2005; Chapman, Edell, & Chapman, 1980; Lovibond & Lovibond, 1993), the presence of multiple sources of information would inevitably increase the confidence with which results are interpreted. This would also allow for comparisons between self-perceptions of severity versus clinical opinions, potentially helping to merge subclinical and clinical models.

Finally, an aspect of this study that is both a limitation and a strength was the presence of many measures regarding psychopathology administered at one time. Since the study was unproctored, it is possible that infrequent or inattentive responding may have influenced the results. To mitigate this risk, the infrequency subscale of the Wisconsin Schizotypy Scales was

included to assess for careless or invalid responding. Using a cutoff of five (where higher indicates infrequent responding), we excluded 155 participants suspected of invalid responding, leading to our final sample size. Though this is roughly 14% of the total sample, we were still able to maintain sufficient power and have more confidence in the interpretation of results. Despite this attempt, it is still impossible to know for sure if the validity of answers was consistent across the study.

The use of many measures can also be viewed as strength. In any structural equation modeling, the use of multiple observed variables to determine a latent variable is preferred over a single indicator—if the indicators are considered good measures—and usually contributes to more valid conclusions by reducing measurement error (Hayduk & Littvay, 2012). By using multiple indicators, we can be confident that results are not representative of a singular scale that is more subject to bias, but rather represents the psychopathology itself. This is also important in understanding relations between psychopathologies. For instance, to examine whether anxiety and depression belong to the same higher-order factor, using only the DASS (which has a subscale of each) would likely yield a stronger association than using multiple measures from different sources converging on each construct independently. Thus, the correlation between the scale scores could be related to method covariance rather than a true covariance between the constructs.

A second strength is the measuring of symptoms instead of diagnoses. A large body of research is conducted in this way (e.g., Achenbach, 1966; Clark & Watson, 1991), and many of the aforementioned structural papers used this approach. This is important for two reasons. First, diagnoses have been shown to be less reliable than symptom scores, which is likely related to the fact that diagnoses are dichotomous (Chmielewski, Clark, Bagby, & Watson, 2015; Markon,

Chmielewski, & Miller, 2011; Regier et al., 2013). The second reason is that the current structure used to diagnose is possibly flawed in itself. In fact, the changing of this system is a major goal of HiTOP and other related efforts. Since the argument made in many of these studies is that the DSM is insufficient to accurately capture the true nature of mental illness, using diagnoses as defined by the DSM would be counterproductive. Moreover, many disorders are multidimensional with symptoms that overlap across disorders. A study that uses symptoms instead of diagnoses would allow for symptoms to load onto different factors, independent of the spectrum onto which their associated disorder loads.

Finally, and related to the previous points, a third strength is that the measures in the current study examined symptoms of mental illness at differing levels of severity. By capturing clinical and subclinical dysfunction, results can be interpreted as representing a wider range of potential problems. In this sense, the models retained could be applied to multiple contexts, though more research should be conducted to ensure that associations are the same across severity of psychopathology.

This research advances our understanding of psychopathology in two key ways. First, it adds further clarification to the Kraepelinian dichotomy, supporting the assertion that hypomania and positive schizotypy (and more broadly bipolar disorder and schizophrenia) are distinct but highly related constructs. This is important for two reasons including its clinical utility and empirical advancement. Clinically, the understanding that the two constructs are distinct, despite sometimes presenting nearly identically, can help guide decision-making. Regarding empirical advancement, that hypomania loads onto the thought disorder spectrum with positive schizotypy and the internalizing spectrum with depression indicates that it should be considered in future examinations of each of these two forms of psychopathology.

Second, these results further support the emerging perspective that OCD and schizophrenia-spectrum disorders are related. Obsessions and delusions, though notably different, may have similar pathways of development and maintenance. This should help inform future biological, psychological, and behavioral research by providing a baseline framework suggesting that the association is likely meaningful and that continued research is needed.

The results of this study are far from the final goal of comprehensively understanding and modeling mental illness. Some important areas of continued research that are needed include more work identifying common and differential biomarkers of different forms of psychopathology, understanding genetic susceptibilities that may be shared or different across psychopathologies, examining how other experiences such as ecological factors contribute to the development of different forms of mental illness, and using increasingly complex statistical analyses to determine the empirical relations between diagnoses and symptoms. Examples of statistical analyses that could be utilized to answer these issues include network analyses—where different forms of psychopathology can be seen as more closely linked to certain other forms—or growth modeling, whereby we may be able to determine if signs of one psychopathology early on are predictive of a course that enables the eventual development of another. For instance, unusual beliefs that are not of full clinical concern in adolescence may be indicative of an increased susceptibility to develop obsessions leading to a diagnosis of OCD. In addition to the areas above, expanding the methodology of the current study to include more measures from different spectra, as well as testing if a subclinical model can be integrated into a clinical model, should be primary goals of future research.

Conclusion

The primary goal of the current research was to examine whether hypomania is better conceptualized as part of the internalizing spectrum, the thought disorder spectrum, or a combination of both. These results suggest that hypomania loads on both spectra, which is consistent with the provisional structure of the recently-developed HiTOP model. In addition, the results of the current research suggest that OCD symptoms also crossload on the thought disorder and internalizing spectra. The differential relations between personality traits and different types of psychopathology also help to understand the relations among psychopathology and "normal" personality traits.

Table 1. Bivariate correlations for all the scales used in the current study

	1	2	3	4	5	6	7	8	9	10	11	12
Positive Schizotypy Measures												
1) PerAb	1											
2) MagicId	.686**	1										
3) SPQ-MI	.481**	.650**	1									
4) SPQ-UPE	.615**	.647**	.679**	1								
Negative Schizotypy Measures												
5) SPQ-NCF	.339**	.282**	.384**	.494**	1							
6) SPQ-CA	.394**	.301**	.365**	.521**	.766**	1						
7) SocAn	.438**	.334**	.250**	.308**	.580**	.471**	1					
Hypomania Measures												
8) HPS	.314**	.379**	.336**	.403**	.245**	.254**	.093*	1				
9) MDQ	.207**	.288**	.259**	.316**	.153**	.190**	.071	.348**	1			
10) TEMPS-Cyc	-.381**	-.401**	-.361**	-.507**	-.476**	-.469**	-.305**	-.461**	-.395**	1		
OCD Measures												
11) VOCI	-.539**	-.451**	-.428**	-.529**	-.421**	-.480**	-.313**	-.398**	-.237**	.487**	1	
12) OCI-R	.487**	.411**	.332**	.432**	.367**	.374**	.344**	.246**	.247**	-.432**	-.607**	1

Table 1. (Continued) Bivariate correlations for all the scales used in the current study

	1	2	3	4	5	6	7	8	9	10	11	12
Depression Measures												
13) IDAS-Dep	.332**	.262**	.243**	.343**	.417**	.422**	.336**	.213**	.248**	-.490**	-.436**	.507**
14) DASS-Dep	.345**	.271**	.250**	.291**	.344**	.375**	.294**	.180**	.281**	-.405**	-.418**	.445**
15) CES-D	.312**	.272**	.260**	.349**	.353**	.370**	.272**	.221**	.338**	-.454**	-.411**	.426**
16) PHQ	.336**	.273**	.280**	.389**	.442**	.454**	.344**	.247**	.271**	-.537**	-.438**	.504**
17) PROMIS-B	.286**	.228**	.210**	.317**	.437**	.445**	.324**	.139**	.191**	-.471**	-.382**	.434**
Anxiety Measures												
18) DASS-Anx	.378**	.351**	.315**	.338**	.274**	.309**	.256**	.253**	.322**	-.377**	-.443**	.491**
19) PSWQ	.112**	.120**	.114**	.218**	.323**	.312**	.168**	.196**	.165**	-.341**	-.378**	.352**
20) PROMIS-A	.328**	.263**	.252**	.364**	.400**	.424**	.280**	.229**	.243**	-.484**	-.453**	.508**
Social Anxiety Measures												
21) SPQ-ESA	.196**	.203**	.314**	.427**	.643**	.618**	.212**	.226**	.165**	-.440**	-.412**	.319**
22) IDAS-Soc	.312**	.265**	.242**	.355**	.420**	.450**	.316**	.197**	.253**	-.410**	-.423**	.510**
Personality Measures												
23) Extraversion	-.107**	-.017	-.009	-.111**	-.542**	-.522**	-.383**	.074*	.038	.188**	.215**	-.189**

Table 1. (Continued) Bivariate correlations for all the scales used in the current study

	1	2	3	4	5	6	7	8	9	10	11	12
24) Agreeableness	-.243**	-.141**	-.015	-.032	-.211**	-.189**	-.500**	.073*	.051	.050	.116**	-.191**
25) Conscientiousness	-.231**	-.152**	-.066*	-.101*	-.112**	-.170**	-.163**	-.030	-.113**	.184**	.092*	-.125**
26) Neuroticism	.196**	.172**	.155**	.241**	-318**	-311**	.213**	.199**	.177**	-.428**	-.395**	.365**
27) Openness	-.098*	-.017	.101*	.083*	-.149**	-.147**	-.227**	.183**	.150**	.009	.080*	-.105**

	13	14	15	16	17	18	19	20	21	22	23	24
Depression Measures												
13) IDAS-Dep	1											
14) DASS-Dep	.590**	1										
15) CES-D	.673**	.703**	1									
16) PHQ	.780**	.578**	.694**	1								
17) PROMIS-B	.740**	.572**	.648**	.743**	1							
Anxiety Measures												
18) DASS-Anx	.492**	.808**	.637**	.478**	.392**	1						
19) PSWQ	.512**	.300**	.338**	.477**	.453**	.285**	1					
20) PROMIS-A	.722**	.513**	.590**	.669**	.701**	.468**	.596**	1				

Table 1. (Continued) Bivariate correlations for all the scales used in the current study

	13	14	15	16	17	18	19	20	21	22	23	24
Social Anxiety Measures												
21) SPQ-ESA	.378**	.306**	.319**	.401**	.393**	.237**	.464**	.446**	1			
22) IDAS-Soc	.698**	.479**	.481**	.606**	.551**	.452**	.405**	.595**	.462**	1		
Personality Measures												
23) Extraversion	-.299**	-.234**	-.192**	-.288**	-.330**	-.157**	-.354**	-.305**	-.546**	-.409**	1	
24) Agreeableness	-.104**	-.115**	-.014	-.050	-.087*	-.093*	.032	-.039	.011	-.110**	.342**	1
25) Conscientiousness	-.248**	-.269**	-.205**	-.230**	-.249**	-.182**	.043	-.142**	-.068*	-.232**	.238**	.356**
26) Neuroticism	.517**	.363**	.384**	-502**	.488**	.295**	.585**	.542**	.370**	.417**	-.356**	-.106**
27) Openness	-.057	-.061	.057	.010	-.068*	-.022	-.052	-.013	-.100*	-.083*	.438**	.567**

	25	26	27
25) Conscientiousness	1		
26) Neuroticism	-.202**	1	
27) Openness	.350**	-.097*	1

Table 2. Model fit statistics

Model	Parameters	AIC	BIC	SABIC	χ^2	df	RMSEA	Low	High	CFI	TLI	SRMR
1	103	111642.056	112142.163	111815.040	1060.845	172	0.074	0.07	0.078	0.93	0.906	0.051
2	97	111863.147	112334.122	112026.055	1293.936	178	0.081	0.077	0.085	0.913	0.886	0.064
3	97	112007.121	112478.096	112170.029	1437.910	178	0.086	0.082	0.091	0.901	0.872	0.072
4	100	111705.335	112190.875	111873.281	1130.123	175	0.076	0.072	0.080	0.925	0.901	0.050
5*	90	112165.866	112602.852	112317.017	1610.654	185	0.090	0.086	0.094	0.888	0.860	0.068
6*	89	112191.023	112623.155	112340.495	1637.812	186	0.091	0.087	0.095	0.886	0.859	0.070
7	**93**	**111688.227**	**112139.780**	**111844.417**	**1127.016**	**182**	**0.074**	**0.070**	**0.078**	**0.926**	**0.906**	**0.052**
8	92	111770.030	112216.727	111924.540	1210.819	183	0.077	0.073	0.081	0.919	0.898	0.060
9*	89	112167.417	112599.548	112316.889	1614.206	186	0.090	0.086	0.094	0.888	0.861	0.069
10*	88	112248.764	112676.040	112396.556	1697.552	187	0.092	0.088	0.096	0.882	0.854	0.072
11*	88	112248.764	112676.040	112396.556	1697.552	187	0.092	0.088	0.096	0.882	0.854	0.072
12*	92	112028.879	112475.577	112183.389	1469.668	183	0.086	0.082	0.090	0.899	0.873	0.060
13*	90	112165.866	112602.852	112317.017	1610.654	185	0.090	0.086	0.094	0.888	0.860	0.068

Model 1: A seven-factor model with positive schizotypy, negative schizoytypy, hypomania, depression, anxiety, social anxiety, and OCD loading on separate factors. **Model 2**: A six-factor model with hypomania scales and positive schizotypy scales loading onto the same factor. **Model 3**: A six-factor model with hypomania scales and depression scales loading onto the same factor. **Model 4**: A six-factor model with hypomania scales loading onto both the positive schizotypy scale and the depression scale. **Model 5**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and hypomania) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 6**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, negative schizotypy, and hypomania) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 7**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, hypomania, and OCD) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 8**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy, negative schizotypy, hypomania, and OCD) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 9**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and hypomania) and internalizing (depression, anxiety, social anxiety, and OCD). **Model 10**: A seven-factor model with one higher-order factor, internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 11**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and negative schizotypy) and internalizing (depression, anxiety, social anxiety, OCD, and hypomania). **Model 12**: A seven-factor model with two higher-order factors, thought disorder (positive schizotypy and OCD) and internalizing (depression, anxiety, social anxiety, and OCD). **Model 13**: A seven-factor model with one higher-order factor, internalizing (depression, anxiety, and social anxiety).

*Models would not converge without freeing residual error correlations between SPQ-NCF and SPQ-CA with SPQ-ESA

Table 3. Standardized estimates and standard errors for Model 7

	Positive Schizotypy	Negative Schizotypy	Hypomania	Depression	Anxiety	Social Anxiety	OCD
PerAb	.753 (.022)						
MagicID	.803 (.019)						
SPQ-UPE	.821 (.019)						
SPQ-MI	.724 (.023)						
SPQ-CA		.798 (.030)					
SPQ-NCF		.873 (.027)					
SocAn		.644 (.027)					
HPS			.566 (.028)				
TEMPS-Cyc			.825 (.022)				
MDQ			.507 (.029)				
CESDR				.808 (.014)			
DASS-Dep				.715 (.018)			
IDAS-Dep				.877 (.010)			
PHQ				.875 (.010)			
PROMIS-B				.835 (.012)			
DASS-Anx					.633 (.023)		
PROMIS-A					.851 (.014)		
PSWQ					.640 (.023)		
SPQ-ESA						.580 (.027)	
IDAS-Soc						.781 (.021)	
OCIR							.772 (.019)
VOCI							.791 (.019)
All values are significant at $p < .001$							

Table 4. First-order factor loading estimates and standard errors for higher-order factors for Model 7

	Thought Disorder	Internalizing
Positive Schizotypy	.958 (.041)	
Hypomania	.470 (.051)	.459 (.048)
OCD	.500 (.050)	.514 (.048)
Depression		.927 (.009)
Anxiety		.972 (.011)
Social Anxiety		.924 (.019)
Negative Schizotypy		
All values are significant at $p < .001$		

Table 5. Higher-order factor relations to each other and negative schizotypy (estimates and standard errors)

	Thought Disorder	Internalizing
Internalizing	.524 (.039)	
Negative Schizotypy	.533 (.037)	.628 (.028)
All values are significant at $p < .001$		

Table 6. Personality variable regressions on each psychopathology

	Positive Schizotypy	Negative Schizotypy	Hypomania	Depression	Anxiety	Social Anxiety	OCD
Extraversion	-.081 (.039)*	-.571 (.029)***	-.284 (.041)***	-.394 (.031)***	-.410 (.032)***	-.564 (.031)***	-.383 (.036)***
Agreeableness	-.134 (.038)***	-.367 (.035)***	-.096 (.040)*	-.109 (.036)**	-.086 (.038)*	-.153 (.039)***	-.164 (.038)***
Conscientiousness	-.187 (.037)***	-.249 (.036)***	-.247 (.037)***	-.273 (.034)***	-.213 (.036)***	-.246 (.037)***	-.215 (.036)***
Neuroticism	.313 (.035)***	.445 (.034)***	.581 (.029)***	.609 (.023)***	.662 (.022)***	.646 (.025)***	.580 (.027)***
Openness	.023 (.038)	-.206 (.037)***	.008 (.040)	-.060 (.037)	-.054 (.038)	-.124 (.039)**	-.074 (.038)
Estimate (Standard Error) * p < .05, ** p < .01, *** p < .001							

Figure 1. Model 7 structure

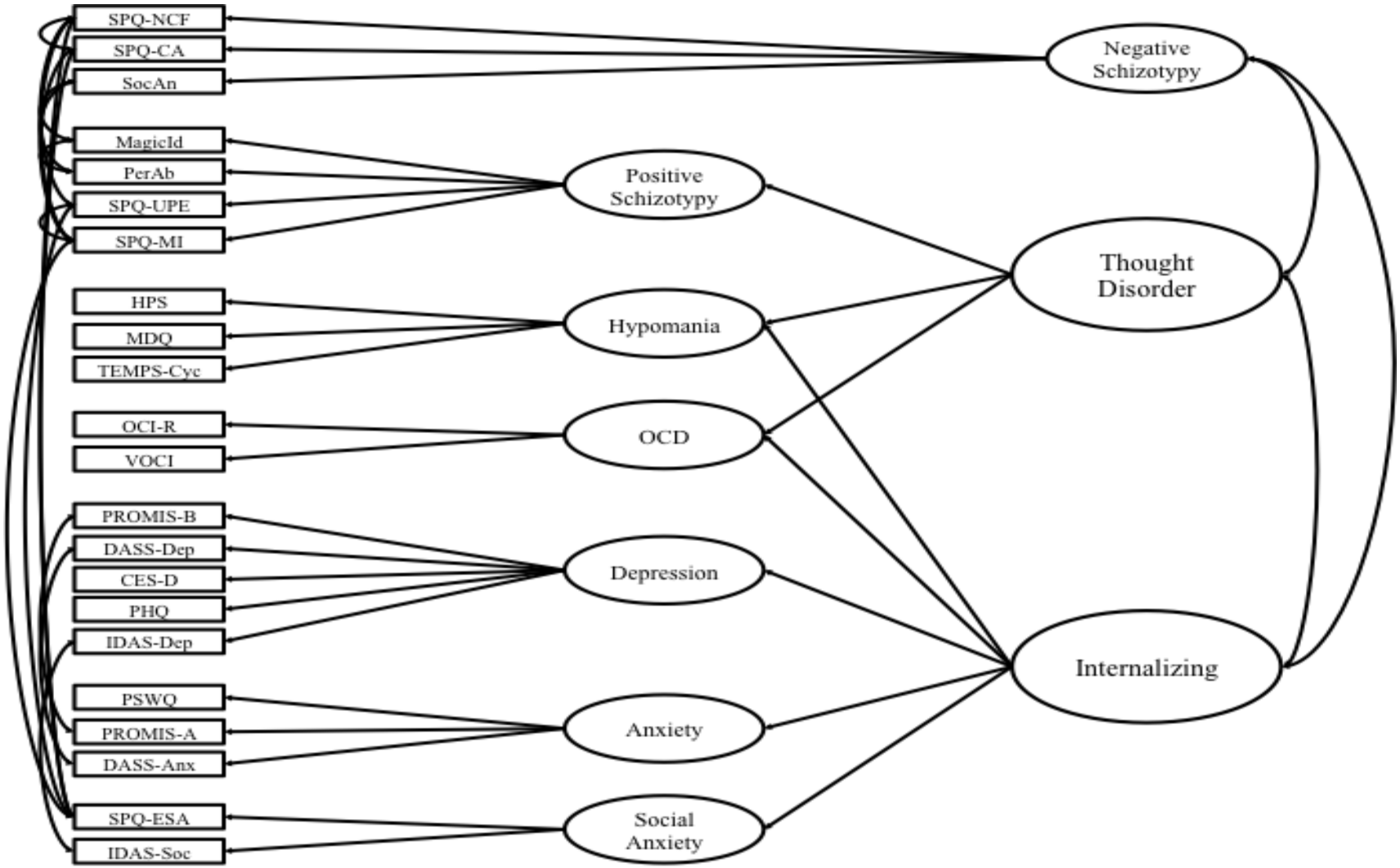
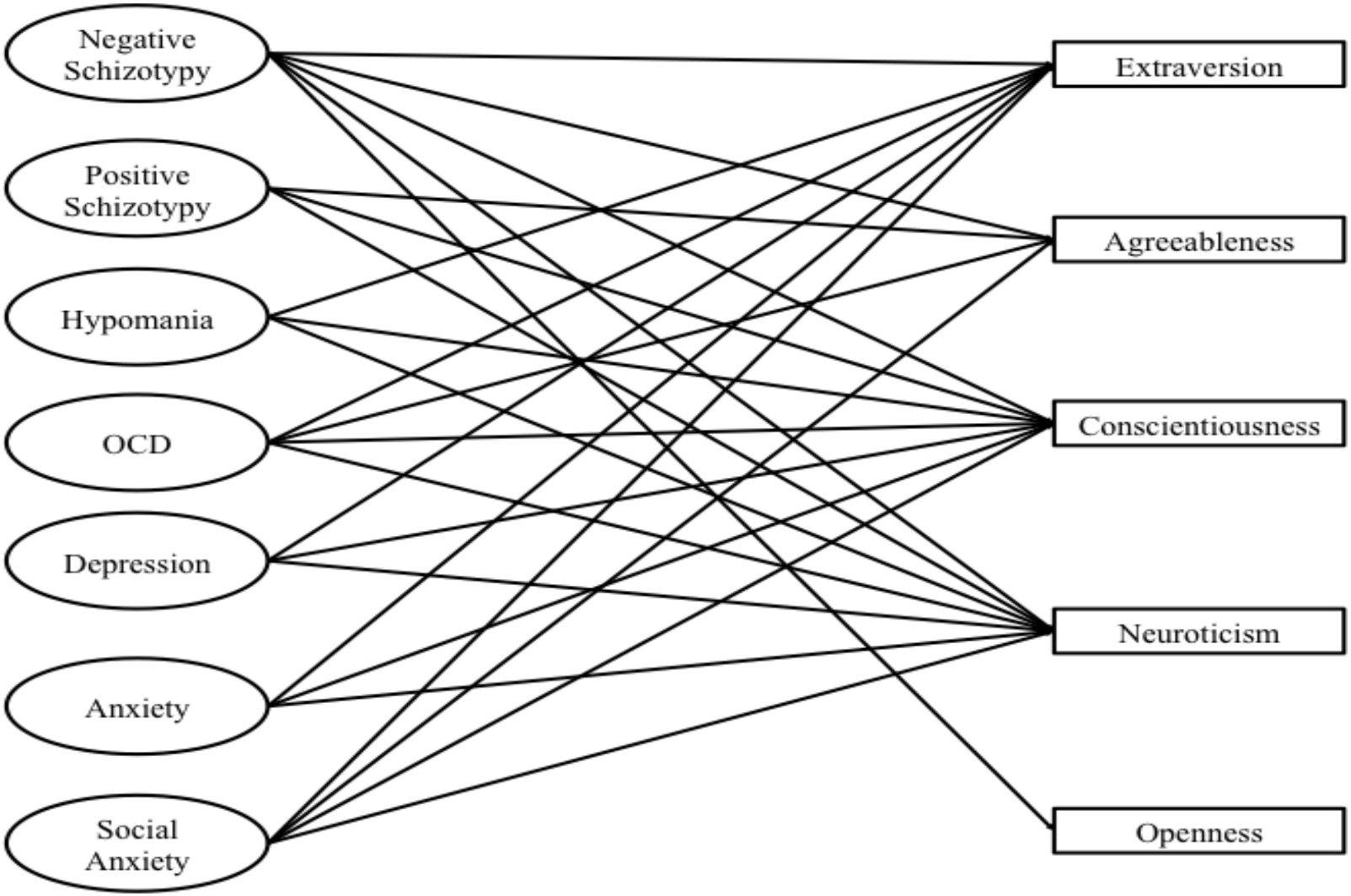


Figure 2. Personality variable regressions



References

Abramowitz, J. S. & Deacon, B. J., (2006). Psychometric properties and construct validity of the Obsessive-Compulsive Inventory--Revised: Replication and extension with a clinical sample. *Journal of Anxiety Disorders, 20*(8), 1016-1035.

Abramowitz, J. S., Deacon, B. J., & Whiteside, S. P. H., (2011). *Exposure therapy for anxiety: Principles and practice*. New York: Guilford Press.

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: a factor analytic study. *Psychological Monographs*, *80*, 1-37.

Achenbach, T. M., Howell, C. T., Quay, H. C., Conners, C. K., & Bates, J. E. (1991). National survey of problems and competencies among four-to sixteen-year-olds: parents' reports for normative and clinical samples. *Monographs of the Society for Research in Child Development, 56*, i-130.

Akiskal, H. S. & Akiskal, K. K., (2005). TEMPS: Temperament evaluation of Memphis, Pisa, Paris and San Diego. *Journal of Affective Disorders, 85*, 1–2.

Akiyama, T., Tsuda, H., Matsumoto, S., Miyake, Y., Kawamura, Y., Noda, T., Akiskal, K. K., & Akiskal, H.S., (2005). The proposed factor structure of temperament and personality in Japan: Combining traits from TEMPS-A and MPT. *Journal of Affective Disorders, 85*, 93-100.

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed. Text revision). Washington, DC: Author.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed). Washington, DC: Author.

Amerio, A., Odone, A., Liapis, C. C., & Ghaemi, S. N., (2014). Diagnostic validity of comorbid

bipolar disorder and obsessive– compulsive disorder: A systematic review. *Acta Psychiatrica Scandinavica, 129*, 343–358. doi: 10.1111/acps.12250.

Angst, A. (1998). The emerging epidemiology of hypomania and bipolar II disorder. *Journal of Affective Disorders, 50*, 143– 151.

Antony, M. M., Bieling, P. J., Cox, B. J., Enns, M. W., & Swinson, R. P., (1998). Psychometric properties of the 42-item and 21-item versions of the Depression Anxiety Stress Scales in clinical groups and a community sample. *Psychological Assessment, 10*(2), 176-181.

Barkley, R. (1997). *ADHD and the Nature of Self Control*. Guilford, New York.

Benazzi, F. & Akiskal, H. S., (2003). Refining the evaluation of bipolar II: Beyond the strict SCID-CV guidelines for hypomania. *Journal of Affective Disorders, 73*, 33– 38.

Berle, D., Blaszczynski, A., Einstein, D. A., & Menzies, R. G., (2006). Thought-action fusion in schizophrenia: A preliminary investigation. *Behaviour Change, 23*(4), 260-269.

Berle, D., & Starcevic, V., (2005). Thought–action fusion: Review of the literature and future directions. *Clinical Psychology Review, 25*, 263–284.

Berman, I., Merson, A., Viegner, B., Losonczy, M.F., Pappas, D., Green, A.I., (1998). Obsessions and compulsions as a distinct cluster of symptoms in schizophrenia: a neuropsychological study. *Journal of Nervous and Mental Disease, 186*, 150–156.

Berrettini, W. (2003). Evidence for shared susceptibility in bipolar disorder and schizophrenia. *American Journal of Medical Genetics, 123*, 59-64.

Betts, K. S., Williams, G. M., Najman, J. M., & Alati, R., (2016). Predicting spectrums of adult mania, psychosis and depression by prospectively ascertained childhood neurodevelopment. *Journal of Psychiatric Research, 72*, 22-29

Biederman, J., Klein, R. G., Pine, D. S., & Klein, D. F., (1998). Resolved: Mania is mistaken for

ADHD in prepubertal children. *Journal of the American Academy of Child and Adolescent Psychiatry, 37,* 1091–1096.

Blanco, C., Okuda, M., Wright, C., Hasin, D. S., Grant, B. F., Liu, S. M., & Olfson, M., (2008). Mental health of college students and their non-college-attending peers: Results from the National Epidemiologic Study on Alcohol and Related Conditions. *Archives of General Psychiatry, 65*(12), 1429-1437. doi: 10.1001/archpsyc.65.12.1429.

Blanco, C., Wall, M. M., He, J. P., Krueger, R. F., Olfson, M., Jin, C. J., ... & Merikangas, K. R. (2015). The space of common psychiatric disorders in adolescents: comorbidity structure and individual latent liabilities. *Journal of the American Academy of Child & Adolescent Psychiatry, 54*, 45-52.

Blumberg, H. P., Martin, A., Kaufman, J., Leung, H. C., Skudlarski, P., Lacadie, C., Fulbright, R. K., Gore, J. C., Charney, D. S., Krystal, J. H., & Peterson, B. S., (2003). Frontostriatal abnormalities in adolescents with bipolar disorder: Preliminary observations from functional MRI. *American Journal of Psychiatry, 160*, 1345–1347.

Boks, M. P. M., Leask, S., Vermunt, J. K., & Kahn, R. S., (2007). The structure of psychosis revisited: The role of mood symptoms. Schizophrenia Research. doi:10.1016/j.schres.2007.02.017.

Boylan, K. R., Bieling, P. J., Marriott, M., Begin, H., Young, T., & MacQueen, G. M., (2004). Impact of comorbid anxiety disorders on outcome in a cohort of patients with bipolar disorder. *Journal of Clinical Psychiatry, 65*(8), 1106-1113.

Brand, S., Foell, S., Bajoghli, H., Keshavarzi, Z., Kalak, N., Gerber, M., Schmidt, N. B., Norton, P. J., & Holsboer-Trachsler, E., (2015). “Tell me, how bright your hypomania is, and I tell you, if you are happily in love!” — Among young adults in love, bright side

hypomania is related to reduced depression and anxiety, and better sleep quality. *International Journal of Psychiatry and Clinical Practice, 19*, 24–31.

Briley, D. A., Domiteaux, M., & Tucker-Drob, E. M., (2014). Achievement-relevant personality: Relations with the Big Five and validation of an efficient instrument. *Learning and Individual Differences, 32*, 26-39.

Brody, J. F. (2001). Evolutionary recasting: ADHD, mania and its variants. *Journal of Affective Disorders, 65*(2), 197-215.

Brown, L. H., Silvia, P. J., Myin-Germeys, I., Lewandowski, K. E., & Kwapil, T. R., (2008). The relationship of social anxiety and social anhedonia to psychometrically identified schizotypy. *Journal of Social and Clinical Psychology, 27*(2), 127-149.

Cannon M., Caspi A., Moffitt T. E., et al. (2002). Evidence for early childhood, pan-developmental impairment specific to schizophreniform disorder: Results from a longitudinal birth cohort. *Archives of General Psychiatry, 59*, 449–456.

Cardno, A. G., & Owen, M. J. (2014). Genetic relationships between schizophrenia, bipolar disorder and schizophrenia. *Schizophrenia Bulletin, 40*, 504-515.

Carlson, G. A. (1998): Mania and ADHD: Comorbidity or confusion. *Journal of Affect Disorders, 51*, 177–187.

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, C. J., Harrington, H., Israel, S., … Moffitt, T. E. (2014). The p factor: one general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science, 2*, 119-137.

Cella, D., Riley, W., Stone, A., Rothrock, N., Reeve, B., Yount, S., Amtmann, D., Bode, R., Buysse, D., Choi, S., Cook, K., Devellis, R., DeWalt, D., Fries, J. F., Gershon, R., Hahn, E. A., Lai, J. S., Pilkonis, P., Revicki, D., Rose, M., Weinfurt, K., Hays, R., & PROMIS

Cooperative Group, (2010). The Patient-Reported Outcomes Measurement Information System (PROMIS) developed and tested its first wave of adult self-reported health outcome item banks: 2005-2008. *Journal of Clinical Epidemiology, 63*(11), 1179-1194. doi: 10.1016/j.jclinepi.2010.04.011.

Chapman, L. J., Chapman, J. P., & Raulin, M. L., (1976). Scales for physical and social anhedonia. *Journal of Abnormal Psychology, 85*, 374–382.

Chapman, L. J., Chapman, J. P., Raulin, M. L., & Edell, W. S. (1978). Schizotypy and thought disorder as a high risk approach to schizophrenia. In G. Serban (Ed.), *Cognitive defects in the development of mental illness* (pp. 351-360). New York: Brunner/Mazel.

Chapman, L. J., Edell, W. S., & Chapman, J. P. (1980). Physical anhedonia, perceptual aberration, and psychosis proneness. *Schizophrenia Bulletin, 6*(4), 639-653.

Chen, D. T., Jiang, X., Akula, N., Shugart, Y. Y., Wendland, J. R., Steele, C. J. M., Kassem, L., …, Strauss, J., (2013). Genome-wide association study meta-analysis of European and Asian-ancestry samples identifies three novel loci associated with bipolar disorder. *Molecular Psychiatry, 18*, 195-205. doi:10.1038/mp.2011.157.

Chmielewski, M., Clark, L. A., Bagby, R. M., & Watson, D. (2015). Method matters: Understanding diagnostic reliability in DSM-IV and DSM-5. *Journal of Abnormal Psychology, 124*, 764-769.

Chmielewski, M., & Watson, D. (2008). The heterogeneous structure of schizotypal personality disorder: Item-level factors of the Schizotypal Personality Questionnaire and their associations with obsessive-compulsive disorder symptoms, dissociative tendencies, and normal personality. *Journal of Abnormal Psychology, 117*, 364-376.

Cicero, D. C. (2015). Measurement invariance of the Schizotypal Personality Questionnaire in

Asian, Pacific Islander, White, and Multiethnic populations. *Psychological Assessment*, *28*, 351-361. http://dx.doi.org/10.1037/pas0000180.

Claridge, G. (1997) *Schizotypy: Implications for Illness and Health*. Oxford University Press.

Clark, D. A., & Beck, A. T., (2010). *Cognitive therapy of anxiety disorders: Science and practice*. New York: Guilford Press.

Clark, L. A. (2005). Temperament as a unifying basis for personality and psychopathology. *Journal of Abnormal Psychology, 114*, 505-521. http://dx.doi.org/10.1037/0021-843X.114.4.505.

Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology, 100*, 316- 336.

Compton, M. T., Goulding, S. M., Bakeman, R., & McClure-Tone, E. B., (2009). Confirmation of a four-factor structure of the schizotypal personality questionnaire among undergraduate students. *Schizophrenia Research, 111*, 46–52.

Costa, P. T., & McCrae, R. R. (1992). Revised NEO personality inventory (NEO PI-R) and NEO five-factor inventory (NEO FFI): Professional manual. *Psychological Assessment Resources*.

Craddock, N., O'Donovan, M. C., & Owen, M. J., (2005). Genetics of schizophrenia and bipolar disorder: dissecting of psychosis. *Journal of Medical Genetics, 42*, 193-204.

Debbané, M., Van der Linden, M., Gex-Fabry, M., & Eliez, S., (2009). Cognitive and emotional associations to positive schizotypy during adolescence. *Journal of Child Psychology and Psychiatry, 50*(3), 326–334. doi:10.1111/j.1469-7610.2008.01961.x.

Demjaha, A., MacCabe, J. H., & Murray, R. M., (2012). How genes and environmental factors determine the different neurodevelopmental trajectories of schizophrenia and bipolar disorder. *Schizophrenia Bulletin, 38*(2), 209-214. doi:10.1093/schbul/sbr100.

Dickstein, D. P., Garvey, M., Pradella, A. G., Greenstein, D. K., Sharp, W. S., Castellanos, F. X., Pine, D. S., & Leibenluft, E., (2005). Neurologic examination abnormalities in children with bipolar disorder or attention-deficit/hyperactivity disorder. *Biological Psychiatry, 58*, 517-524. doi:10.1016/j.biopsych.2004.12.010.

Drake, R. J., Pickles, A., Bentall, R. P., Kinderman, P., Haddock, G., Tarrier, N., & Lewis, S. W., (2004). The evolution of insight, paranoia and depression during early schizophrenia. *Psychological Medicine, 34*(2), 285– 292.

Dunayevich, E., & Keck, P. E. (2000). Prevalence and description of psychotic features in bipolar mania. *Current Psychiatry Reports, 2*, 286–290. doi:10.1007/s11920-000-0069-4.

Eckblad, M., & Chapman, L. J. (1983). Magical ideation as an indicator of schizotypy. *Journal of Consulting and Clinical Psychology, 51*, 215-225. doi:10.1037//0022-006X.51.2.215.

Eckblad, M., & Chapman, L. J. (1986). Development and validation of a scale for hypomanic personality. *Journal of Abnormal Psychology, 95*(3), 214-222.

Edell, W. S. (1995). The psychometric measurement of schizotypy using the Wisconsin Scales of psychosis-proneness. In G. Miller (Ed.), *The Behavioral High-Risk Paradigm in Psychopathology*. Series in Psychopathology. Springer, New York, NY: Pringer-Verlag.

Edmundson, M., Lynam, D. R., Miller, J. D., Gore, W. L., & Widiger, T. A. (2011). A five factor measure of schizotypal personality traits. *Assessment, 18*, 321–334.

Emsley, R. A., Oosthuizen, P. P., Joubert, A. F., Roberts, M. C., & Stein, D. J., (1999). Depressive and anxiety symptoms in patients with schizophrenia and schizophreniform

disorder. *Journal of Clinical Psychiatry*, *60*(11), 747–751.

Escher, S., Romme, M., Buiks, A., Delespaul, P., & Van Os, J., (2002). Independent course of childhood auditory hallucinations: A sequential 3-year follow-up study. *British Journal of Psychiatry – Supplement, 43*, 10–18.

Fletcher, K., Parker, G., & Manicavasagar, V., (2013). A qualitative investigation of hypomania and depression in bipolar II disorder. *Psychiatry Quarterly, 84*, 455-474.

Foa, E. B., Huppert, J. D., Leiberg, S., Langner, R., Kichic, R., Hajcak, G., & Salkovskis, P. M., (2002). The Obsessive-Compulsive Inventory: Development and validation of a short version. *Psychological Assessment, 14*, 485-496.

Foa, E. B., Liebowitz, M. R., Kozak, M. J., Davies, S., Campeas, R., Franklin, M. E., et al., (2005). Randomized placebo-controlled trial of exposure and ritual prevention, Clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, *162*(1), 151-161.

Fonseca-Pedrero, E., Fumero, A., Paino, M., de Miguel, A., Ortuño-Sierra, J., Lemos-Giráldez, S., & Muñiz, J., (2014). Schizotypal Personality Questionnaire: New sources of validity evidence in college students. *Psychiatry Research, 219*(1), 214-220.

Fonseca-Pedrero, E., Paino, M., Lemos-Giráldez, S., Sierra-Baigrie, S., Ordóñez-Camblor, N., & Muñiz, J., (2011). Early psychopathological features in Spanish adolescents. *Psiocothema, 23*(1), 87-93.

Forbush, K. T., & Watson, D. (2013). The structure of common and uncommon mental disorders. *Psychological Medicine, 43*, 97-108.

Fossati, A., Raine, A., Carretta, I., Leonardi, B., & Maffei, C., (2003). The three-factor model of schizotypal personality: Invariance across age and gender. *Personality & Individual Differences, 35*, 1007–1019.

Geller, B., Williams, M., Zimerman, B., Frazier, J., Beringer, L., & Warner, K. L., (1998). Prepubertal and early adolescent bipolarity differentiate from ADHD by manic symptoms, grandiose delusions, ultra-rapid or ultradian cycling. *Journal of Affective Disorders, 51*, 81–91.

Gilvarry, Catherine & Takei, Nori & Russell, Ailsa & Rushe, Teresa & Hemsley, David & Murray, Robin. (2000). Premorbid IQ in patients with functional psychosis and their first-degree relatives. *Schizophrenia Research, 41*, 417-29. doi:10.1016/S0920-9964(99)00092-4.

Gnambs, T. (2014). A meta-analysis of dependability coefficients (test-retest reliabilities) for measures of the Big Five. *Journal of Research in Personality, 52*, 20-28.

Goldberg, L. R. (1993). The structure of phenotypic personality traits. *American Psychologist, 48*, 26-34.

Goldberg, L. R., Johnson, J. A., Eber, H. W., Hogan, R., Ashton, M. C., Cloninger, C. R., & Gough, H. C., (2006). The International Personality Item Pool and the future of public-domain personality measures. *Journal of Research in Personality, 40*, 84-96.

Gooding, D. C., Tallent, K. A., & Matts, C. W., (2005). Clinical status of at-risk individuals 5 years later: Further validation of the psychometric high-risk strategy. *Journal of Abnormal Psychology, 114*, 170–175.

Goodwin, F. K. & Jamison, K. R. (1990). *Manic-depressive illness*. Oxford: Oxford University Press.

Gow, A. J., Whiteman, M. C., Pattie, A., & Deary, I. J. (2005). Goldberg's 'IPIP' Big-Five factor markers: Internal consistency and concurrent validation in Scotland. *Personality and Individual Differences, 39*, 317-329.

Hajcak, G., Huppert, J. D., Simons, R. F., & Foa, E. B., (2004). Psychometric properties of the OCI-R in a college sample. *Behaviour Research and Therapy, 42*(1), 115-123.

Hallahan B., Newell J., Soares, J. C., Brambilla, P., Strakowski, S. M., Fleck, D. E., Kieseppä, T. Altshuler, L. L., …, McDonald, C., (2011). Structural magnetic resonance imaging in bipolar disorder: an international collaborative mega-analysis of individual adult patient data. *Biological Psychiatry, 69*, 326–335.

Hantouche, E. G., Angst, J., Demonfaucon, C., Perugi, G., Lancrenon, S., & Akiskal, H. S., (2003). Cyclothymic OCD: a distinct form? *Journal of Affective Disorders, 75*, 1-10.

Hayduk, L. A., & Littvay, L., (2012). Should researchers use single indicators, best indicators, or multiple indicators in a structural equation models? *BMC Medical Research Methodology, 12*(159).

Hewitt, J. K., & Claridge, G. S. (1989). The factor structure of schizotypy in a normal population. *Personality and Individual Differences*, 10, 323– 329.

Himmelhoch, J. M. (1998). Social anxiety, hypomania and the bipolar spectrum: data, theory and clinical issues. *Journal of Affective Disorders, 50*, 203– 213.

Hirschfeld, R. M. A., Williams, J. B. W., Spitzer, R. L., Calabrese, J. R., Flynn, L., Keck, P. E. Jr, Lewis, L., McElroy, S. L., Post, R. M., Rapport, D. J., Russell, J. M., Sacks, G. S., & Zajecka, J., (2000). Development and validation of a screening instrument for bipolar spectrum disorder: The mood disorder questionnaire. *American Journal of Psychiatry, 157*, 1873–1875.

Kendler, K. S., Karkowski, L. M., Walsh, D., (1998). The structure of psychosis: latent class analysis of probands from the Roscommon Family Study. *Archives of General Psychiatry, 55*, 492–499.

Kerns, J. G., & Becker, T. M. (2008). Communication disturbances, working memory, and emotion in people with elevated disorganized schizotypy. *Schizophrenia Research*, 100, 172–180.

Keyes, K. M., Eaton, N. R., Krueger, R. F., Skodol, A. E., Wall, M. M., Grant, B. F., . . .Hasin, D. S. (2013). Thought disorder in the meta-structure of psychopathology. *Psychological Medicine, 43*, 1673-1683.

Koran, L. M., Thienemann, M. L., & Davenport, R., (1996). Quality of life for patients with obsessive-compulsive disorder. *American Journal of Psychiatry, 153*(6), 783-788.

Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin, 136*, 768–821.

Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., . . . Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology, 126*(4), 454-477. doi:10.1037/abn0000258.

Kotov, R., Perlman, G., Gámez, W., & Watson, D. (2015). The structure and short-term stability of the emotional disorders: A dimensional approach. *Psychological Medicine, 45*, 1687-1698.

Kotov, R., Ruggero, C. J., Krueger, R. F., Watson, D., Yuan, Q., & Zimmerman, M., (2011). New dimensions in the quantitative classification of mental illness. *Archives of General Psychiatry, 68*(10), 1003-1011.

Kraepelin, E. (1896). Dementia-Praecox and Paraphrenia. Psychiatrie Reprinted (1971). Robert E.Krieger, Huntington, NY.

Kraepelin E. (1920). Die Erscheinungsformen des Irreseins [The manifestations of insanity]. *Z ges Neurol Psychiatr*, *62*, 1–29.

Kroenke, K., Spitzer, R. L., Williams, J. B., (2001). The PHQ-9: Validity of a brief depression severity measure. *Journal of General Internal Medicine, 16*(9), 606-613.

Krueger, R. F., Chentsova-Dutton, Y. E., Markon, K. E., Goldberg, D. P., & Ormel, J. (2003). A cross-cultural study of the structure of comorbidity among common psychopathological syndromes in the general health care setting. *Journal of Abnormal Psychology, 107*, 216-227.

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2*, 111-133.

Krueger, R. F., & Markon, K. E., (2014). The role of the DSM-5 personality trait model in moving toward a quantitative and empirically based approach to classifying personality and psychopathology. *Annual Review of Clinical Psychology, 10*, 477-501.

Krüger, S., Cooke, R. G., Hasey, G. M., Jorna, T., & Persad, E., (1995). Comorbidity of obsessive compulsive disorder in bipolar disorder. *Journal of Affective Disorders, 34*(2), 117–120. doi: 10.1016/0165-0327(95)00008-B.

Kwapil, T. R., & Barrantes-Vidal, N. (2015). Schizotypy: Looking back and moving forward. *Schizophrenia Bulletin, 41*(2), 366-373 doi:10.1093/schbul/sbu186.

Kwapil, T. R., Gross, G. M., Silvia, P. J., & Barrantes-Vidal, N., (2013). Prediction of psychopathology and functional impairment by positive and negative schizotypy in the Chapmans' ten-year longitudinal study. *Journal of Abnormal Psychology, 122*(3), 807-815. doi: 10.1037/a0033759.

Lahey, B. B., Applegate, B., Waldman, I. D., Loft, J. D., Hankin, B. L., & Rick, J. (2004). The structure of child and adolescent psychopathology: generating new hypotheses. *Journal of Abnormal Psychology, 113*, 358-385.

Lake, C.R. & Hurwitz, N. (2007). Schizoffective disorder merges schizophrenia and bipolar disorders as one disease – there is no schizoaffective disorder. *Current Opinion in Psychiatry, 20*, 365-379.

Laursen, T. M., Munk-Olsen, T., Nordentoft, M., & Mortensen, P. B., (2007). Increased mortality among patients admitted with major psychiatric disorders: A register-based study comparing mortality in unipolar depressive disorder, bipolar affective disorder, schizoaffective disorder, and schizophrenia. *Journal of Clinical Psychiatry, 68*(6), 899-907.

Lenzenweger, M., (2006). Schizotypy: An organizing framework for schizophrenia research. *Current Directions in Psychological Science, 15*(4), 162-166.

Lenzenweger, M. F., & Dworkin, R. H. (1996). The dimensions of schizophrenia phenomenology: Not one or two, at least three, perhaps four. *The British Journal of Psychiatry, 168*, 432– 440. doi:10.1192/bjp.168.4.432.

Leucht, S., Helfer, B., Dold, M, Kissling, W., & McGrath, J. J., (2015). Lithium for

schizophrenia. *Cochrane Database of Systematic Reviews, 10*. doi: 10.1002/14651858.CD003834.pub3.

Lewandowski, K. E., Barrantes-Vidal, N., Nelson-Gray, R. O., Clancy, C., Kepley, H. O., & Kwapil, T. R., (2006). Anxiety and depression symptoms in psychometrically identified schizotypy. *Schizophrenia Research, 83*(2), 225-235.

Lewinsohn, P. M., Seeley, J. R., Roberts, R. E., & Allen, N. B., (1997). Center for Epidemiologic Studies Depression Scale (CES-D) as a screening instrument for depression among community-residing older adults. *Psychology and Aging, 12*(2), 277–287.

Lim, C. H., Zain, S. M., Reynolds, G. P., Zain, M. A., Roffeei, S. N., Zainal, N. Z., Kanagasundram, S., & Mohamed Z., (2014). Genetic association of LMAN2L gene in schizophrenia and bipolar disorder and its interaction with ANK3 gene polymorphism. *Progress in Neuropsychopharmacology & Biological Psychiatry, 3*(54), 157-62. doi: 10.1016/j.pnpbp.2014.05.017.

Lovibond, S. H., & Lovibond, P. F. (1993). Manual for the Depression Anxiety Stress Scales (DASS). *Psychology Foundation Monograph*.

Lovibond, P. F., & Lovibond, S. H. (1995). The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. *Behaviour Research and Therapy, 33*, 335-342.

Lysaker, P. H., Bell, M. D., Bioty, S. M., & Zito, W. S., (1995). The frequency of associations between positive and negative symptoms and dysphoria in schizophrenia. *Comprehensive Psychiatry, 36*(2), 113– 117.

Mahon, K., Perez-Rodriguez, M. M., Gunawardane, N., Burdick, K. E., (2013). Dimensional endophenotypes in bipolar disorder: Affective dysregulation and psychosis proneness. *Journal of Affective Disorders, 151*, 695–701.

Markon, K. E., Chmielewski, M., & Miller, C. J. (2011). The reliability and validity of discrete and continuous measures of psychopathology: a quantitative review. *Psychological Bulletin, 137*, 856-879.

Martin, R. L., Cloninger, C. R., Guze, S. B., & Clayton, P. J., (1985). Mortality in a follow-up of 500 psychiatric outpatients. *Archives of General Psychiatry, 42*(1), 47–54. doi:10.1001/archpsyc.1985.01790240049005.

Meehl, P. E. (1962). Schizotaxia, schizotypy, and schizophrenia. *American Psychologist, 17*, 827-838.

Meehl, P. E. (1990). Toward an integrated theory of schizotaxia, schizotypy, and schizophrenia. *Journal of Personality Disorders, 4*, 1–99.

Meyer, T. D., (2002). Correlates of the hypomanic personality scale: results from a family sample. *Personality and Individual Differences, 32*(1), 3-14.

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D., (XXX). Development and validation of the Penn State Worry Questionnaire. Behaviour Research and Therapy, 28(6), 487-495.

Miller, M. W., Fogler, J. M., Wolf, E. J., Kaloupek, D. G., & Keane, T. M. (2008). The internalizing and externalizing structure of psychiatric comorbidity in combat veterans. *Journal of Traumatic Stress, 21*, 58-65.

Morvan, Y., Tibaoui, F., Bourdel, M. C., Lôo, H., Akiskal, K. K., Akiskal, H. S., Krebs, M. O., (2011). Confirmation of the factorial structure of temperamental autoquestionnaire

(TEMPS-A) in non-clinical young adults and relation to current state of anxiety, depression and to schizotypal traits. J*ournal of Affective Disorders, 131*, 37–44.

Murray, R. M., Sham, P., van Os, J., Zanelli, J., Cannon, M., & McDonald, C., (2004). A developmental model for similarities and dissimilarities between schizophrenia and bipolar disorder. *Schizophrenia Research, 71*, 405-416.

Muthen, L. K., & Muthen, B. O. (1998-2016). *Mplus User's Guide. Seventh Edition.* Los Angeles, CA: Muthen & Muthen.

Nelson, H. E. (2005). *Cognitive-behavioural Therapy with Delusions and Hallucinations: A Practice Manual*. (2nd ed). Nelson Thornes.

Niendam, T. A., Berzak, J., Cannon, T. D., & Bearden, C. E., (2009). Obsessive compulsive symptoms in the psychosis prodrome: Correlates of clinical and functional outcome. *Schizophrenia Research, 108*, 170-175. doi:10.1016/j.schres.2008.11.023.

Owen, M. J., O'Donovan, M. C., & Harrison, P. J., (2005). Schizophrenia: a genetic disorder of the synapse. *BMJ, 330*, 158-159.

Owens, D. G. & Johnstone, E. C., (2006). Precursors and prodromata of schizophrenia: Findings from the Edinburgh High Risk Study and their literature context. *Psychological Medicine, 36*, 1501–1514.

Päären, A., Bohman, H., von Knorring, A., von Knorring, L., Olsson, G., & Jonsson, U., (2014). Hypomania spectrum disorder in adolescence: A 15-year follow-up of non-mood morbidity in adulthood. *BMC Psychiatry*, *14*(9).

Pallanti, S., Quercioli, L., & Hollander, E. (2004). Social anxiety in outpatients with schizophrenia: A relevant cause of disability. *American Journal of Psychiatry, 161*, 53–58.

Payá, B., Rodríguez-Sánchez, J. M., Otero, S., Muñoz, P., Castro-Fornieles, J., Parellada, M., Gonzalez-Pinto, A., Soutullo, C., Baeza, I., Rapado-Castro, M., Sáenz-Herrero, M., Moreno, D., & Arango, C., (2013). Premorbid impairments in early-onset psychosis: differences between patients with schizophrenia and bipolar disorder. *Schizophrenia Research, 146,* 103-110.

Peralta, V. & Cuesta, M. J., (2003). The nosology of psychotic disorders: a comparison among competing classification systems. *Schizophrenia Bulletin, 29,* 413–425.

Perugi, G., Akiskal, H. S., Toni, C., Simonini, E., & Gemignani, A., (2001). The temporal relationship between anxiety disorders and (hypo)mania: a retrospective examination of 63 panic, social phobic and obsessive-compulsive patients with co-morbid bipolar disorder. *Journal of Affective Disorders, 67,* 199–206.

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,* 386-394. doi:10.1016/j.psychres.2011.12.051.

Pini, S., Dell'Osso, L., Mastrocinque, C., Marcacci, G., Papasogli, A., Vignoli, S., Pallanti, S., & Cassano, G., (1999). Axis I comorbidity in bipolar disorder with psychotic features. *British Journal of Psychiatry, 175,* 467-471.

Pliszka, S. R., Liotti, M., & Woldorff, M. G., (2000). Inhibitory control in children with attention-deficit/hyperactivity disorder: Event-related potentials identify the processing component and timing of an impaired right-frontal response-inhibition mechanism. *Biological Psychiatry, 48,* 238 –246.

Poyurovsky, M., Hramenkov, S., Isakov, V., Rauchverger, B., Modai, I., Schneidman, M., Fuchs, C., Weizman, A., (2001). Obsessive–compulsive disorder in hospitalized patients with chronic schizophrenia. *Psychiatry Research, 102*, 49–57.

Poyurovsky, M., & Koran, L. M. (2005). Obsessive-compulsive disorder (OCD) with schizotypy vs. schizophrenia with OCD: Diagnostic dilemmas and therapeutic implications. *Journal of Psychiatric Research, 39*, 399–408.

Preti, A., Corrias, I., Gabbrielli, M., Lai, V., Muratore, T., Pintus, E., Pintus, M., Sanna, S., Scanu, R., Tronci, D., …, Giovanni Carta, M., (2015). The independence of schizotypy from affective temperaments: A combined confirmatory factor analysis of SPQ and the short TEMPS-A. *Psychiatry Research, 225*, 145-456.

Qureshi, S. & Frangou, S., (2002). The neurobiology of bipolar disorder. *Journal of Affective Disorders, 72*, 209–226.

Rachman, S. (1993). Obsessions, responsibility and guilt. *Behaviour Research and Therapy, 31*, 149–154.

Radloff, (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385–401.

Raine, A. (1991). The SPQ: a scale for the assessment of schizotypal personality based on DSM-III-R criteria. *Schizophrenia Bulletin, 17*, 555-564. doi:doi: 10.1093/schbul/17.4.555.

Raine, A., Lencz, T., & Mednick, S.A., (1995). *Schizotypal personality*. New York: Cambridge University Press.

Raine, A., Reynolds, C., Lencz, T., Scerbo, A., Triphon, N., & Kim, D. (1994). Cognitive-perceptual, interpersonal, and disorganized features of schizotypal personality. *Schizophrenia Bulletin, 20*, 191–201. http://dx.doi.org/10.1093/schbul/20.1.191.

Rawlings, D., & Locarnini, A., (2008). Dimensional schizotypy, autism, and unusual word associations in artists and scientists. *Journal of Research in Personality, 42*, 465-471.

Regier, D. A., Narrow, W. E., Clarke, D. E., Kraemer, H. C., Kuramoto, S. J., Kuhl, E. A., & Kupfer, D. J. (2013). DSM-5 field trials in the United States and Canada, part II: test-retest reliability of selected categorical diagnoses. *American Journal of Psychiatry, 168*, 1186-1194.

Ross, S. R., Lutz, C. J., & Bailley, S. E. (2004). Psychopathy and the five factor model in a noninstitutionalized sample: A domain and facet level analysis. *Journal of Psychopathology and Behavioral Assessment, 26*, 213–223.

Rossi, A. & Daneluzzo, E., (2002). Schizotypal dimensions in normal and schizophrenic patients: a comparison with other clinical samples. *Schizophrenia Research, 54*, 67–75.

Rubio, D. M., Berg-Weger, M., & Tebb, S. S. (2001). Using structural equation modeling to test for multidimensionality. *Structural Equation Modeling, 8*, 613-626. doi:10.1207/S15328007SEM0804_06

Ruggeri, M., Koeter, M., Schene, A., Bonetto, C., Vazquez-Barquero, J. L., Becker, T., Knapp, M., Knudsen, H. C., Tansella, M., Thornicroft, G., EPSILON Study Group, (2005). Factor solution of the BPRS-expanded version in schizophrenic outpatients living in five European countries. *Schizophrenia Research, 75*, 107–117.

Rybakowski, J. K. & Klonowska, P., (2011). Bipolar mood disorder, creativity and schizotypy: an experimental study. *Psychopathology, 44*, 296–302.

Salkovskis, P. (1985). Obsessional-compulsive problems: A cognitive–behavioural analysis. *Behaviour Research and Therapy, 23*, 571–583.

Samuel, J., Nesdadt, G., Wolyniec, P., Adler, L., Liang, K., Pulver, A., (1993). Obsessive–compulsive symptoms in schizophrenia. *Schizophrenia Research, 9,* 139.

Samuel, D. B., & Widiger, T. A., (2008). A meta-analytic review of the relationships between the five-factor model and DSM-IV-TR personality disorders: A facet level analysis. *Clinical Psychology Review, 28,* 1326-1342.

Schroeder, K., Naber, D., & Huber, C. G., (2014). Maladaptive personality traits increase subjectively during the course of schizophrenia spectrum disorders. *The Journal of Nervous and Mental Disease, 202*(4), 319-323. doi: 10.1097/NMD.0000000000000125.

Schulze, T. G., Akula, N., Breuer, R., Steele, J., Nalls, M. A., Singleton, A. B., Degenhardt, F. A., Nothen, M. N., Cichon, S., Rietschel, M., The Bipolar Genome Study, & McMahon, F. J., (2014). Molecular genetic overlap in bipolar disorder, schizophrenia, and major depressive disorder. *World Journal of Biological Psychiatry, 15*(3), 200-208. doi:10.3109/15622975.2012.662282.

Saucier, G. & Goldberg, L. R., (2002). Assessing the Big Five: Applications of 10 psychometric criteria to the development of marker scales. *Big Five Assessment*, 29-58. Goettingen, Germany: Hogrefe & Huber.

Saulsman, L. M., & Page, A. C., (2004). The five-factor model and personality disorder empirical literature: A meta-analytic review. *Clinical Psychology Review, 23,* 1055-1085.

Shafran, R., Thordarson, D. S., & Rachman, S., (1996). Thought–action fusion in obsessive-compulsive disorder. *Journal of Anxiety Disorders*, *10*, 379–391.

Sham, P. C., MacLean, C. J., & Kendler, K. S., (1994). A typological model of schizophrenia based on age at onset, sex and familial morbidity. *Acta Psychiatrica Scandinavica, 89,* 135–141. doi:10.1111/j.1600-0447.1994.tb01501.x.

Simon, N. M., Otto, M. W., Wisniewski, S. R., Fossey, M., Sagduyu, K., Frank, E., Sachs, G. S., Nierenberg, A. A., Thase, M. E., & Pollack, M. H., (2004). Anxiety disorder comorbidity in bipolar disorder patients: Data from the first 500 participants in the Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD). *American Journal of Psychiatry, 161*(12), 2222-2229.

Sobin, C., Blundell, M. L., Weiller, F., Gavigan, C., Haiman, C., & Karayiorgou, M. (2000). Evidence of a schizotypy subtype in OCD. *Journal of Psychiatric Research, 34*, 15–24.

Spitzer, R. L., Kroenke, K., Linzer, M., Hahn, S. R., Williams, J. B., deGruy, F. V. 3rd, Brody, D., & Davies, M., (1995). Health-related quality of life in primary care patients with mental disorders. Results from the PRIME-MD 1000 Study. *JAMA, 274*(19), 1511-1517.

Stahl, S. M. & Grady, M. M., (2014). *Stahl's essential psychopharmacology: The prescriber's guide* (5th ed.). Cambridge, UK ; New York: Cambridge University Press.

Suhr, J. A., Spitznagel, M. B., & Gunstad, J., (2006). An obsessive-compulsive subtype of schizotypy. *The Journal of Nervous and Mental Disease, 194*(11), 884-886. doi: 10.1097/01.nmd.0000243929.45895.d9.

Tandon, R., Nasrallah, H.A., & Keshavan, M.S. (2009). Schizophrenia, “just the facts” 4 Clinical features and conceptualization. *Schizophrenia Research, 110,* 1-23.

Thordarson, D. S., Radomsky, A. S., Rachman, S., Shafran, R., Sawchuck, C. N., & Ralph Hakstian, A., (2004). The Vancouver Obsessional Compulsive Inventory (VOCI). *Behaviour Research and Therapy, 42*(11), 1289-1314.

Tibbo, P. & Warneke, L., (1999). Obsessive-compulsive disorder in schizophrenia: Epidemiologic and biologic overlap. *Journal of Psychiatry and Neuroscience, 24*(1), 15-24.

Turkington, D. & Kingdon, D. (1996). Using a normalising rationale in the treatment of schizophrenic patients. In G. Haddock, & P. S. Slade, (Eds.), *Cognitive-behavioural interventions with psychotic disorders,* 103–115. London: Routledge.

Ulloa, R. E., Birmaher, B., Axelson, D., Williamson, D. E., Brent, D. A., Ryan, N. D., & Baugher, M., (2000). Psychosis in a pediatric mood and anxiety disorders clinic: Phenomenology and correlates. *Journal of the American Academy of Child and Adolescent Psychiatry, 39*(3), 337–345.

Valença, A. M., Nardi, A. E., Nascimento, I., Lopes, F. L., Freire, R. C., Mezzasalma, M. A., Veras, A. B., & Versiani, M., (2005). Do social anxiety disorder patients belong to a bipolar spectrum subgroup? *Journal of Affective Disorders, 86*, 11–18.

Van der Does, A.J., Dingemans, P.M., Linszen, D.H., Nugter, M.A., Scholte, W.F., (1995). Dimensions and subtypes of recent-onset schizophrenia. A longitudinal analysis. *The Journal of Nervous and Mental Disease, 183,* 681–687.

Van der Heijden, P. T., Rossi, G. M. P., van der Veld, W. M., Derksen, J. J. L., Egger, J. I. M., (2013). Personality and psychopathology: Higher order relations between the five factor model of personality and the MMPI-2 Restructured Form. *Journal of Research in Personality, 47,* 572-579. doi: 10.1016/j.jrp.2013.05.001.

Van Os, J., Linscott, R. J., Myin-Germeys, I., Delespaul, P., & Krabbendam, L. (2009). A systematic review and meta-analysis of the psychosis continuum: evidence for a psychosis proneness–persistence–impairment model of psychotic disorder. *Psychological Medicine, 39,* 179-195.

Vrieze, S. I. (2012). Model selection and psychological theory: a discussion of the differences between the Akaike information criterion (AIC) and the Bayesian information criterion (BIC). *Psychology Methods, 17*(2), 228-243.

Watson, D. (2005). Rethinking the mood and anxiety disorders: a quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology, 114*, 522-536.

Watson, D., Clark, L. A., & Chmielewski, M., (2008). Structures of personality and their relevance to psychopathology: II. Further articulation of a comprehensive unified trait structure. *Journal of Personality, 76*(6), 1545-1586. doi: 10.1111/j.1467-6494.2008.00531.x.

Watson, D., O'Hara, M. W., Naragon-Gainey, K., Koffel, E., Chmielewski, M., Kotov, R., ... Ruggero, C. (2012). Development and validation of new anxiety and bipolar symptom scales for an expanded version of the IDAS (the IDAS-II). *Assessment, 19*, 399-420.

Watson, D., O'Hara, M. W., Simms, L. J., Kotov, R., Chmielewski, M., McDade-Montez, E. A., ... Stuart, S. (2007). Development and validation of the Inventory of Depression and Anxiety Symptoms (IDAS). *Psychological Assessment, 19*, 253-268.

Watson, D., Wu, K. D., & Cutshall, C. (2004). Symptom subtypes of obsessive–compulsive disorder and their relation to dissociation. *Journal of Anxiety Disorders, 18*, 435–458.

Waugh, M. J., Meyer, T. D., Youngstrom, E. A., & Scott, J., (2014). A review of self-rating instruments to identify young people at risk of bipolar spectrum disorders. *Journal of Affective Disorders, 160*, 113-121.

Wigman, J. T. W., Vollebergh, W. A. M., Jacobs, N., Wichers, M., Derom, C., Thiery, E., Raaijmakers, Q. A. W., & van Os, J., (2012). Replication of the five-dimensional structure of positive psychotic experiences in young adulthood. *Psychiatry Research,*

197, 353-355.

Wright, A. G. C., Krueger, R. F., Hobbs, M. J., Markon, K. E., Eaton, N. R., & Slade, T., (2012). The structure of psychopathology: Toward an expanded quantitative empirical model. *Journal of Abnormal Psychology*, *122*(1), 281-294. doi: 10.1037/a0030133.

Yung, A. R., Nelson, B., Baker, K., Buckby, J. A., Baksheev, G., & Cosgrave, E. M., (2009). Psychotic-like experiences in a community sample of adolescents: Implications for the continuum model of psychosis and prediction of schizophrenia. *The Australian and New Zealand Journal of Psychiatry, 43*(2), 118-28. doi:10.1080/00048670802607188.

Zammit, S., Allebeck, P., David, A. S., Dalman, C., Hemmingsson, T., Lundberg, I., Lewis, G., (2004). A longitudinal study of premorbid IQ score and risk of developing schizophrenia, bipolar disorder, severe depression, and other nonaffective psychoses. *Archives of General Psychiatry, 61*, 354-360.

Zheng, L., Goldberg, L. R., Zheng, Y., Zhao, Y., Tang, Y., & Liu, L., (2008). Reliability and concurrent validation of the IPIP Big-Five factor markers in China: Consistencies in factor structure between Internet-obtained heterosexual and homosexual samples. *Personality and Individual Differences, 45*, 649-654.

Zutshi, A., Kamath, P., & Reddy, Y. C. J., (2007). Bipolar and nonbipolar obsessive-compulsive disorder: a clinical exploration. *Comprehensive Psychiatry, 48,* 245– 251. doi:10.1016/j.comppsych.2006.12.005.