Personality and Psychopathology in

Offspring of Mothers Diagnosed with Affective Illness

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Dedication

To my Mom and Dad,

for teaching me the value of an education,

and for making countless sacrifices in my pursuit of one.

Abstract

Offspring of mothers diagnosed with an affective illness are at increased risk for developing an affective spectrum disorder. The overarching goal of the present study is to investigate whether individual differences in cognition and personality among at-risk offspring promote or prevent the development of affective symptomatology in adolescence and young adulthood. Participants included siblings drawn from 98 families participating in a longitudinal study of the offspring of depressed mothers (Radke-Yarrow, 1998). Forty-two of the mothers in the study were diagnosed with unipolar depression, 26 with bipolar disorder I or II, and 30 were healthy comparisons. Ratings of offspring personality, cognitive style, and psychopathology were obtained from multiple measures across two time points in adolescence and young adulthood. History of a maternal affective disorder and offspring Neuroticism independently predicted elevated depressive symptoms in adolescence, while high Neuroticism and Extraversion predicted offspring mania. Offspring Neuroticism interacted with maternal diagnosis to predict risk for depression in young adulthood. Lower-order traits comprising Neuroticism showed unique associations with offspring affective symptoms both concurrently and prospectively. Overall, findings suggest that high Neuroticism is associated with increased risk for depressive and manic symptoms in adolescence and young adulthood, and this effect may be partially moderated by maternal psychopathology.

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Introduction

Offspring of mothers diagnosed with affective illnesses face a broad range of adversity early in life, placing them at risk for psychological difficulties across social, cognitive, and behavioral domains (for reviews see Chang, Steiner, & Ketter, 2003; Goodman & Tully, 2006). Impairment in critical areas of functioning can have cascading consequences for offspring, eventually contributing to the emergence of psychopathology in adolescence and early adulthood (Klimes-Dougan et al., 2010; Masten & Cicchetti, 2010). Current evidence supports increased rates of psychiatric diagnoses in offspring of parents with affective disorders (O-AD) compared to children of well parents (O-WELL; Beardslee, Keller, Lavori, Staley, & Sacks, 1993; DelBello & Geller, 2001; Hammen & Brennan, 2003; Weissman et al., 2006). O-AD appear to be at greatest risk for developing an affective spectrum disorder specifically, with offspring of parents diagnosed with bipolar depression (O-BD) four times more likely to develop a mood disorder than O-WELL (Lapalme, Hodgins, & LaRoche, 1997), and offspring of parents diagnosed with major depression (O-MD) two to three times more likely to develop depression than O-WELL (Hammen & Brennan, 2003; Weissman et al., 2006). Research on O-AD indicates that adolescence is a critical time for the development of affective pathology, as both unipolar and bipolar depression tend to emerge in the late childhood or teen years and increase in prevalence into early adulthood (Halligan, Murray, Martins, & Cooper, 2007; Lieb et al., 2002; Weissman et al., 2006).

Studies are now moving beyond questions of mere prevalence of psychopathology in O-AD, turning instead to process-oriented research. Questions of equifinality, in which

diverse pathways lead to the same outcome, and multifinality, in which similar pathways lead to different outcomes, are abundant in the field and highlight the need for research on risk and protective factors that may shape the course of complex developmental pathways (Cicchetti & Rogosch, 1996; Cicchetti & Toth, 1998). Guided by a developmental psychopathology perspective, research is beginning to embrace transactional models in which heritability, parenting practices, and environmental influences interact to shape patterns of adaptation over time (Cicchetti & Toth, 1998; Cummings & Davies, 1994; Goodman & Gotlib, 1999). Investigations into potential moderators of O-AD development are increasing as well. Individual differences in maternal behavior and affect have garnered significant attention in this regard (Goodman, 2007). In contrast, research has largely neglected offspring characteristics outside of common covariates like age, gender, and intelligence (IQ).

One area that warrants additional research is offspring cognitive style, which a number of prominent theories have highlighted as a core vulnerability factor in risk for depressive illness (Abramson, Seligman, & Teasdale, 1978; Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1983; Nolen-Hoesksema, 1991). Cognitive style is a broad-ranging construct that encompasses the inferences an individual makes about the causes, meaning, and consequences of life events (Abramson et al., 1989). Over several decades of work, cognitive style has come to include an individual's characteristic attitudes, self-worth or self-image, and attributional biases (Alloy, Abramson, Walshaw, & Nereen, 2006). Research investigating both unipolar and bipolar depression shows that maladaptive cognitive styles often color one's interpretation of life events, thereby

increasing vulnerability for affective illness (Alloy et al, 2006, 2009a). There is some

evidence that offspring of parents diagnosed with a depressive illness tend to exhibit

heightened cognitive vulnerability (e.g., Garber & Flynn, 2001). However, there is

limited research addressing whether parental psychopathology and cognitive vulnerability

interact to heighten risk for depression or mania in offspring. Studies are needed to

determine whether the presence of specific cognitive vulnerabilities can help identify

offspring at the greatest risk for developing later affective disorders. In light of this, the

current study aims to expand on existing literature by examining whether maladaptive

attributional biases predict depressive and manic symptomatology longitudinally in an at-

risk offspring sample.

A second area in which individual differences may be relevant to developmental

outcomes in O-AD involves temperament and personality. Research has repeatedly

shown that personality traits can be important predictors of a wide range of important life

outcomes (Ozer & Benet-Martinez, 2006; Shiner, Masten, & Roberts, 2003), suggesting a

need to incorporate personality variables in longitudinal studies. Developmental

psychopathologists have echoed this need by repeatedly calling for investigations into

whether child and adolescent temperament and personality moderate the relationship

between maternal affective diagnoses and offspring psychopathology (Gelfand & Teti,

1990; Goodman & Gotlib, 1999; Goodman, 2007). Individual differences in personality

have also been linked to maladaptive cognitive styles (Hankin, Lakdawalla, Carter,

Abela, & Adams, 2007), though little is known about how the two constructs are related

to one another. Studies are needed to examine whether these constructs are independent

or overlapping predictors of psychopathology, and to determine whether they interact with one another over the course of development. The present study will address this gap in the research by examining how individual differences in offspring personality traits, attributional biases, and maternal diagnoses interact to promote or protect against risk for affective psychopathology.

Personality in Mood Disorders

Throughout the course of development, human beings exhibit a wide range of individual differences in personality. Early in life, children are differentiated from one another by their emotional proclivities—one child is fussy or irritable, whereas another might be joyful and easily placated. By middle childhood, children vary in terms of an entirely new range of behaviors and adaptations, including self-regulatory styles, prosocial behaviors, creativity, and academic achievement. New developmental challenges emerge in adolescence, and so too do new patterns of individual differences in coping strategies, relational behaviors, and self-identity. Collectively, variation in these essential characteristics form the developing individual's *personality.*

Psychologists have long struggled to organize the broad range of individual differences encompassed by personality into a meaningful conceptual framework that can parse its most essential components and guide theoretically-informed empirical investigations. To date, most research in personality psychology has focused on traits, defined as "probabilistic descriptions of relatively stable, real patterns of emotion, motivation, cognition, and behavior" (DeYoung, 2015, p. 35). This definition highlights the ability of traits to describe or summarize an individual's average state behavior over

time. However, traits can also be considered as explanatory constructs, in that they represent the average functioning of the underlying psychological and biological mechanisms that motivate, or cause, goal-oriented behavior (DeYoung, 2015; Allen & DeYoung, in press). Individual differences in these underlying mechanisms (and therefore, in the traits we use to represent them) are shaped over the course of development by more distal factors, including both genetics and the individual's unique life experience (Shiner, 2010). Under certain conditions (e.g., when a child is both at risk for depression and raised in a negative rearing environment), the interaction of more distal factors may promote exacerbated functioning of the underlying mechanisms driving behavior, leading to extreme trait scores and increased vulnerability to psychopathology (Cicchetti & Toth, 1998).

Personality psychology's history has been marked by intense debate surrounding the structure of personality traits. However, there is now a relative consensus among developmental and adult personality researchers that a Five Factor Model that includes the traits of Extraversion, Neuroticism, Agreeableness, Conscientiousness, and Openness to Experience is best-suited to capturing shared covariance among more specific personality traits (Digman, 1990; McCrae & Costa, 1999; Shiner, 2010). The utility of the Five Factor Model, or the Big Five, is especially apparent in studies examining parental psychopathology and its influence on offspring characteristics. In contrast, prior to the last decade, most research evaluating offspring traits employed terms like "easy" or "difficult" temperament, which have since proven to be rather heterogeneous constructs that aggregate disparate and often unrelated behaviors (Rothbart, 1982; Rothbart, 2004).

The growing popularity of the Five Factor Model, augmented by developmental research that identifies clear analogues of the five domains early in life (e.g., De Pauw, Mervielde, & van Leeuwen, 2009; Shiner, 2010), has led to more robust findings regarding the prevalence of certain traits in O-AD. First, there is emerging evidence that the presence of a maternal affective disorder is associated with heightened offspring Neuroticism (or Negative Emotionality), though longitudinal research is needed to replicate this finding when offspring transition to late childhood and adolescence (Davis et al., 2007; Doucette, Horrocks, Grof, Keown-Stoneman, & Duffy, 2013; Doucette et al., 2014; Duffy et al., 2007; Farchione et al., 2007; Feldman et al., 2009; Melchior et al., 2012; Whiffen & Gotlib, 1989). Second, unipolar and bipolar diagnoses in caregivers appear to differentially predict levels of offspring Extraversion. A number of studies have found that O-MD are more likely than healthy controls to be low on Extraversion (Durbin, Klein, Hayden, Buckley, & Moerck, 2005; Olino et al., 2011). Studies examining Extraversion in O-BD are more mixed, with some research showing that O-BD tend to score higher on Extraversion-related traits (Nurnberger et al., 1988; Singh, DelBello, & Strakowski, 2008), other studies suggesting no relationship between O-BD and Extraversion (Jones et al., 2006; Nijjar, Ellenbogen, & Hodgins, 2014; Rothen et al., 2009), and still others showing differential patterns of scores depending on the specific facets of Extraversion under consideration (Chang Blasey, Ketter, & Steiner, 2003). Finally, there is some limited evidence to suggest that both O-MD and O-BD exhibit relative decreases in Effortful Control/ Conscientiousness, though more work is needed to confirm these initial findings (Singh et al., 2008).

Not coincidentally, the patterns of personality traits observed most frequently in O-AD are similar to the patterns that predict unipolar and bipolar depressive symptoms in normative populations. For example, a large meta-analysis by Kotov and colleagues (2010) recently concluded that major depression is consistently associated with high levels of Neuroticism and low levels of Conscientiousness, though both traits were also associated with a range of anxiety disorders, and may therefore be more fittingly conceptualized as broad indicators of internalizing psychopathology (Kotov, Gamez, Schmidt, & Watson, 2010; Clark, Watson, & Mineka, 1994). The relationship between Extraversion and major depression is more mixed. Traditionally, theorists have hypothesized that depression may reflect a hyposensitivity of the Behavioral Approach System (BAS; DePue & Iacono, 1989; Clark et al., 1994), a construct first proposed by Gray (1982) that is conceptually similar to Extraversion and tends to reflect sensitivity to incentives and rewards. In Kotov and colleagues' meta-analytic work, depression was negatively related to Extraversion, but the effect was smaller than anticipated and some of the included studies actually showed a positive association between the two constructs.

Research investigating the relationship between personality traits and bipolar depression is more limited. Several studies show a positive relationship between Neuroticism and bipolar disorder (Barnett et al., 2011; Evans et al., 2005; Jylhä et al., 2010; Nowakowska, Strong, Santosa, Wang, & Ketter, 2005; Quilty, Sellbom, Tackett, & Bagby, 2009; Young et al., 1995), though some research suggests that Neuroticism might be related specifically to depressive, as opposed to manic, features of the disorder (Murray, Goldstone, & Cunningham, 2007). A substantial body of work also links the

BAS and Extraversion to bipolar disorder. Alloy and Abramson (2010) have proposed a BAS-dysregulation model of bipolar disorder, in which individuals high on trait BAS are overly sensitive to environmental rewards or goal-relevant cues. Consistent with this model, individuals prone to mania are higher on self-report and behavioral measures of the BAS, and this finding is consistent even across fluctuations in mood states (Alloy, Abramson, Urošević, Bender, & Wagner, 2009a; Urošević, Abramson, Harmon-Jones, & Alloy, 2008). Moreover, individuals high on trait BAS, compared to people scoring in the moderate range, are both more likely to be diagnosed with bipolar disorder and to exhibit a more severe disease course (Alloy et al., 2009b). Studies using traditional measures of the Big Five to assess personality-BAS associations are more variable, with several studies showing a positive relationship between bipolar (mania, in particular) and Extraversion (Bagby et al., 1997; Barnett et al., 2011; Coulston et al., 2013; Middeldorp et al., 2011; Quilty et al., 2009; Tackett, Quilty, Sellbom, Rector, & Bagby, 2008), and others showing the reverse relationship or no relationship (Hirschfeld, Klerman, Keller, Andreasen, & Clayton, 1986; Jain, Blais, Otto, Hirshfeld, & Sachs, 1999; Jyhla et al., 2010; Kim, Joo, Kim, Lim, & Kim, 2011; Lewis, Scott, & Frangou, 2009).

Finally, several studies show associations between BD and high Openness and low Agreeableness, despite the fact that neither Openness nor Agreeableness appear to consistently vary in O-BD (Bagby et al., 1997; Murray et al., 2007; Nowakowska et al., 2005; Quilty et al., 2009). These findings suggest that high Openness and low Agreeableness could offer some specificity in predicting which O-BD might go on to

develop bipolar depression themselves, but research is needed to more clearly evaluate this hypothesis.

Research examining the role of personality traits in MD, BD, and those at risk for affective disorders is blossoming, but a number of serious limitations are present in the literature. First, longitudinal studies in high risk populations that include measures of both personality and psychopathology are needed in order to understand how initial trait vulnerabilities may be exacerbated under conditions of adversity, eventually leading to the emergence of psychopathology (Nigg, 2006). Prospective longitudinal research in offspring populations is also important for understanding whether specific traits may interact with familial psychopathology to offer specificity in predicting the type and form of psychopathology an individual is most likely to experience.

Second, studies that move beyond the level of the Big Five and evaluate specific lower-order traits are needed in order to parse frequently inconsistent findings. For instance, the trait most robustly linked to affective disorders, Neuroticism, is also a major risk factor for psychopathology more generally, and some argue that the trait therefore holds little etiological value (Ormel et al., 2013). However, emerging research shows that the lower-order facets of Neuroticism differentially predict unipolar and bipolar symptoms. For instance, the anger/irritability facet of Neuroticism is specifically associated with BAS hypersensitivity, and may therefore represent a vulnerability to manic/hypomanic symptoms (Urošević et al., 2008). Consistent with this hypothesis, Quilty, Pelletier, DeYoung, & Bagby (2013) recently found that two lower-order aspects of Neuroticism, Volatility and Withdrawal, differentially predict affective diagnoses.

Specifically, increased Volatility (equivalent to "irritable distress" in the developmental literature) and decreased Withdrawal ("anxious distress" in the developmental literature) differentiated bipolar mood disorders from unipolar mood disorders, suggesting that lower-order Neuroticism aspects may be more etiologically relevant to psychopathology than the broader Neuroticism domain.

Examination of lower-order traits may also help to explain the inconsistency of the relationship between Extraversion and affective disorders. For example, there is evidence to suggest that the positive emotionality facet of Extraversion, which is conceptually similar to the lower-order trait of Enthusiasm, is uniquely tied to major depression, whereas other facets are unrelated or only weakly related (Naragon-Gainey, Watson, & Markon, 2009; Quilty et al., 2013). This finding is consistent with early evidence showing that O-MD are lower than O-WELL on positive emotionality but not other dimensions of Extraversion, suggesting that low positive emotionality may uniquely predict risk for unipolar depressive symptoms within O-MD (Durbin et al., 2005). Investigations into the relationship between lower-order facets of Extraversion, BD, and O-BD, may likewise bring clarity to a literature characterized by inconsistency. In the limited research that has been done in offspring samples, O-BD score more highly on measures of activity and approach (both of which are considered part of Assertiveness, a lower-order Extraversion trait), as opposed to facets tapping more emotional aspects of Extraversion (Chang et al., 2003; Singh et al., 2008). Nonetheless, research in this area is limited, and studies in O-BD populations are needed to better clarify the role of various facets of Extraversion in modulating risk for bipolar symptomatology.

Attributional Style in Mood Disorders

Over the course of development, children and adolescents exhibit a range of individual differences in their attributional style, or the way they perceive and represent events occurring in the world around them (Shiner & Caspi, 2012). Often, these attributional biases can have long-term impacts on a child's adjustment. For instance, variability in attributional biases has been associated with outcomes as diverse as well-being, aggression, educational achievement, self-worth, and psychopathology (Ciarrochi, Heaven, & Davies, 2007; Dodge, 2006; Glasgow, Dornbusch, Troyer, Steinberg, & Ritter, 1997; Cole et al., 2008; Tennen, Herzberger, & Nelson, 1987). The most widespread model linking attributional biases to affective disorders is the reformulated learned helplessness model of depression, which posits that individuals prone to depression tend to exhibit a depressogenic attributional style, in which negative events or stressors are characterized as internal, or caused by the self as opposed to by the situation; stable, or unchanging and long-lasting as opposed to transient; and global, or due to factors that are present across most situations, as opposed to factors that characterize just the current context (Abramson et al., 1978; Abramson et al., 1989; Roesch & Weiner, 2001). There is a wealth of evidence linking depressogenic attributional styles to both concurrent and prospective depressive symptoms (Cole et al., 2008; Gladstone & Kaslow, 1995; Joiner & Wagner, 1995; Seligman, Kaslow, Alloy, & Peterson, 1984). Similarly, there is a rich literature examining cognitive vulnerabilities in individuals diagnosed with bipolar disorder (for a review, see Alloy et al., 2006). In a longitudinal study involving both unipolar and bipolar depressed patients Alloy, Reilly-Harrington, Fresco,

Whitehouse, and Zechmeister (1999) found that negative attributional biases interacted with negative life events to predict increases in depressive but not manic symptoms, whereas positive attributional biases interacted with positive life events to predict increases in manic, but not depressive symptoms. These findings stand in contrast to a second study by Reilly-Harrington, Alloy, Fresco, and Whitehouse (1999) in which negative attributional biases interacted with negative life events to predict increases in both depressive and manic symptoms in a sample of individuals diagnosed with bipolar disorder. Similarly, Knowles and colleagues (2007) recently found that both unipolar and bipolar patients in remission exhibit more pessimistic attributional styles than healthy controls. Clearly, more research is needed to clarify the relationship between attributional biases and mania, and to identify potential moderators that may help explain inconsistent findings in the literature (e.g., mood state, traits).

Finally, evidence indicates that the attributional biases prominent in individuals with affective diagnoses may also be present in children and adolescents at risk for psychopathology. Indeed, negative attributions are more prevalent in O-MD, suggesting that maladaptive mental representations may in part mediate the relationship between maternal depression and offspring psychopathology (Garber & Flynn, 2001; Garber & Robinson, 1997; Murray, Woolgar, Cooper, & Hipwell, 2001; Jaenicke et al., 1987). Unfortunately, few attempts to have been made to extend findings on attributional biases to offspring of parents with bipolar disorder. Only one study with a very small sample has evaluated this question, finding that O-BD report more negative attributions than children of mothers diagnosed with medical illnesses (Jaenicke et al., 1987).

The Present Study

Well-established bodies of literature now tie personality traits and cognitive style to the etiology of both depression and mania. However, there is little reason to believe that personality traits and cognitive style exert their influence on development in an independent fashion. Instead, the relationship between the two constructs is likely to be interactive, with traits influencing the attitudes, attributions, and mental representations that children form about the world around them, and these cognitive vulnerabilities likewise serving to modulate the influence of traits on long-term adjustment (Shiner & Caspi, 2012). Moreover, both traits and cognitive styles are likely to be influenced by familial risk factors, including the presence of maternal depressive disorders. Consistent with previous research, the hypotheses of the current study reflect the conclusions of studies showing that parental affective illness confers non-specific risk for psychopathology to offspring (Hodgins, Faucher, Zarac, & Ellenbogen, 2002; Lieb, Insensee, Höfler, Pfister, & Wittchen, 2002). As a result, we highlight the moderating influence of maternal pathology in general, focusing only on specific maternal diagnoses (unipolar/bipolar diagnoses) when extant research warrants it, or in an exploratory capacity.

Overall, the present study seeks to examine how maternal affective diagnoses and offspring personality characteristics (including traits and attributional biases) interact to predict affective symptomatology in a longitudinal sample of O-AD. The present study will pursue this goal through the following aims:

Aim 1: To examine the relationship between maternal affective diagnoses, offspring personality organization, and the emergence of offspring affective symptomatology both concurrently and prospectively.

- This aim will initially be evaluated at the level of the Big Five.

  Hypothesis 1: O-AD will exhibit heightened levels of Neuroticism. O-MD will exhibit lower scores on Extraversion, whereas O-BD will show the inverse pattern.

  Hypothesis 2: Both concurrently and prospectively, heightened Neuroticism and Extraversion will predict manic symptoms, whereas high Neuroticism and low Extraversion will predict depressive symptoms, and these relationships will be amplified in O-AD.

- This aim will also be evaluated using lower-order personality traits that comprise Neuroticism and Extraversion.

  Hypothesis 3: The lower-order personality trait Withdrawal (or anxious distress) will predict increased depressive symptomatology, and this relationship will be amplified in O-AD.

  Hypothesis 4: The lower-order personality trait Volatility (or irritable distress) will predict increased manic/hypomanic symptomatology, and this relationship will be amplified within O-AD.

  Hypothesis 5: The lower-order personality trait Enthusiasm (or affiliative Extraversion) will be inversely related to depressive symptomatology, and this relationship will be amplified in O-AD.

<u>Hypothesis 6</u>: The lower-order personality trait Assertiveness (or agentic Extraversion) will predict manic/hypomanic symptomatology, and this relationship will amplified in O-AD.

Aim 2: To examine the relationship between maternal affective diagnoses, offspring cognitive vulnerability, and the emergence of offspring depressive symptomatology both concurrently and prospectively.

- This aim will be examined using an overall attributional bias measure that reflects individual differences in offspring internal, stable, and global attributions.

    <u>Hypothesis 7</u>: O-AD will exhibit more negative overall attributional styles than O-WELL.

    <u>Hypothesis 8</u>: Negative attributional styles will predict heightened affective symptoms in O-BD and O-MD.

Aim 3: To examine how cognitive vulnerability interacts with offspring personality traits to amplify or protect against risk for affective disorders among at risk offspring.

- This aim will be addressed by examining whether the interaction between maternal diagnoses, offspring cognitive vulnerability, and offspring personality traits prospectively predicts offspring affective symptomatology.

    <u>Hypothesis 9</u>: High Withdrawal and low Enthusiasm will predict offspring depressive symptoms concurrently and prospectively. Further, maternal diagnosis and attributional biases will moderate this relationship, such that the association between Withdrawal and Enthusiasm and offspring

symptoms will be amplified in the context of a maternal affective

diagnosis and more negative overall attributional biases.

Hypothesis 10: High Volatility and high Assertiveness will predict

offspring manic symptoms concurrently and prospectively. Further,

maternal diagnosis and attributional biases will moderate this relationship,

such that the association between Volatility and Assertiveness and

offspring symptoms will be amplified in the context of a maternal

affective diagnosis and maladaptive attributional biases.

Preliminary Analysis: Derivation of Lower-Order Aspect Scales

The emergence of the Big Five has provided an organizing framework from

which psychologists can study the influence of individual differences on human

functioning and outcomes. However, the Big Five is not the only level at which

individual differences in personality are manifest. Rather, personality traits are arranged

hierarchically with traits near the top of the hierarchy reflecting covariation among a

broad range of behaviors and experiences, and traits near the bottom of the hierarchy

reflecting more narrow and differentiated behaviors (Costa & McCrae, 1995; Jang,

McCrae, Angleitner, Riemann, & Livesley, 1998; Markon, Krueger, & Watson, 2005; see

Figure 1). Empirical study of lower-order traits below the level of the Big Five has been

shown to provide novel insights into relationships between personality and

psychopathology (Quilty et al., 2013). In this preliminary study, we use a large sample

from the Collaborative Longitudinal Personality Study to derive and validate several

lower-order personality traits, each of which are hypothesized to be differentially related to depressive and manic illness within our main analyses.

Personality Traits below the Big Five. The lowest level of the personality hierarchy is comprised of narrowly-defined traits called facets. Researchers have typically selected facets of interest based on purely conceptual grounds, leading to little consistency or agreement surrounding which of the many facets are most relevant to each domain. As a result, a central question emerging from the Big Five has been how to best parse each domain into lower-order traits that adequately capture distinctions most common in existing research and relevant to the prediction of behavior.

This question can be partially answered by reviewing the personality literature, which reveals an interesting pattern in which independent researchers have repeatedly identified two core subcomponents of each Big Five domain, suggesting that an intermediate level of personality may exist between facets and domains (for a review of this literature, see DeYoung, Quilty, & Peterson, 2007). Empirical evidence also supports the idea that each domain is comprised of two separable subdomains. Jang and colleagues (2002) have used behavioral genetic techniques to demonstrate that two genetic factors are needed to account for the shared covariance among lower-order facets of each Big Five domain. Extending this work, DeYoung and colleagues (2007) have used factor analysis of facet scales to demonstrate that covariation among lower-level traits can indeed be adequately accounted for by two factors per Big Five domain. Taken together, this work points to an intermediate level of personality in which two traits, which DeYoung and colleagues have labeled aspects, comprise each Big Five domain.

Importantly, the aspect level of the personality hierarchy appears to identify subcomponents of Extraversion and Neuroticism that have long been relevant to development and adaptation. For instance, both childhood and adult researchers have distinguished between a more sociable, gregarious, and joyful aspect of Extraversion, and a more dominant or assertive aspect (Shiner & Caspi, 2003; DePue & Collins, 1999). These two components clearly map on to Extraversion's two aspects, labeled Enthusiasm and Assertiveness, respectively. Research supports a similar distinction within the Neuroticism domain. Rothbart and Bates (1998) have described two important components of Neuroticism, labeled anxious distress and irritable distress, with the former reflecting tendencies toward sadness and anxiety and the latter reflecting tendencies toward anger and irritability. These two factors appear to map directly on to Neuroticism's two aspects, Withdrawal and Volatility (Shiner & DeYoung, 2013). Similar Neuroticism factors have been described in Jeffrey Gray's Reinforcement Sensitivity Theory, which articulates the Fight-Flight-Freeze and Behavioral Inhibition Systems (Gray, 1982; Gray & McNaughton, 2000), and in more recent research on the structure of adult psychopathology (Krueger, 1999).

Of relevance to the current study, the lower-order aspects of Neuroticism and Extraversion are differentially related to depression and mania (Naragon-Gainey et al., 2009; Quilty et al., 2013). We seek to extend these findings to an at risk population by examining whether the lower-order aspects of Extraversion and Neuroticism predict affective pathology in offspring of mothers diagnosed with an affective illness. However, research on the aspect-level of the personality hierarchy has only emerged in the last

decade or so, and so there were no measures to assess the aspects at the outset of the

offspring study. Given this dilemma, we used data from the Collaborative Longitudinal

Personality Disorders sample to determine whether a current measure of the Big Five

aspects can be recovered from the Schedule for Nonadaptive and Adaptive Personality,

which we could then use to examine the relationship between aspects and

psychopathology in our offspring sample.

Preliminary Analysis: Method for Scale Derivation

      Participants. Lower-order aspect scales were derived using the Collaborative

Longitudinal Personality Disorders Study (CLPS) sample (see Skodol et al., 2005 for a

detailed description of this study). The present sample includes a subset of the original

participants (715 of the original 733). Participants in the CLPS study were diagnosed

with a primary personality disorder based on the Diagnostic Interview for DSM-IV

Personality Disorders (DIPD-IV; Zanarini, Frankenburg, Sickel, & Yong, 1996) and

corroborating evidence from either the Schedule for Nonadaptive and Adaptive

Personality (Clark, 1993) or the Personality Assessment Form (Shea, Glass, Pilkonis,

Watkins, & Docherty, 1987). A comparison group diagnosed with major depression (N =

95) was also included in the sample. Individuals ranged from 18 to 45 years old (M =

32.50, SD = 8.11) and the majority of the sample was female (63.63%; N=455). Over

half the sample identified as White (69.37%, N = 496), while 14.67% identified as Black,

12.73% identified as Hispanic, and 2.10% identified as Asian.

      Aspect scales derived from the CLPS data were also validated in our primary

sample of offspring of mothers diagnosed with an affective illness. Participants in the

primary sample were drawn from 98 families participating in an ongoing longitudinal study of the offspring of depressed mothers (Radke-Yarrow, 1998). Forty-two of the mothers in the study were diagnosed with unipolar depression, 26 with bipolar disorder I or II, and 30 were healthy controls. Two children were selected as participants from each family (4% of the families had only one child) resulting in a total of 192 child participants, including 48 O-BD, 84 O-MD, and 60 O-WELL. Detailed demographics for the full sample are described in the methodology of our primary analyses, below, and in Table 1. Aspect scales were validated on a subsample of participants for whom SNAP-Y data was available at the fourth wave of data collection. Slightly over half of this subsample was female (54.79%; N = 80). The average age of the younger sibling cohort at Time 4 was 13.42 (SD = 1.73), while the average age of the older cohort was 17.14 (SD = 2.03). The majority of the sample was White (87.67%, N= 128), while 9.59% (N = 14) of individuals identified as Black, 1.37% (N = 2) identified as Asian, and 1.37% (N = 2) identified as Hispanic.

Measures. *The Revised NEO Personality Inventory* (NEO-PI-R; Costa & McCrae, 1992): The NEO-PI-R is the most widely used self-report measure assessing the Big Five personality traits in adults. The measure includes 240 items measured on a 5-point Likert scale. Each domain is comprised of six facets measured by eight items. Facets were developed rationally based on the scale developers' conceptualization of the lower-order traits that comprise each of the five domains. More recently, the NEO-PI-R has been used to derive the ten Big Five aspects (Ross & DeYoung, 2015). The current analysis uses this revised scoring procedure to calculate the NEO-BFAS aspects, which

are then used to guide the construction of analogue scales in the Schedule for

Nonadaptive and Adaptive Personality.

The Schedule for Nonadaptive and Adaptive Personality (SNAP; Clark, 1993).

The original SNAP is a 375-item instrument developed to comprehensively measure a

broad range of normal and abnormal personality traits. The measure has been extensively

validated in community samples, as well as both inpatient and outpatient psychiatric

samples. The SNAP contains 3 higher-order "temperament" trait scales, 12 scales

measuring more specific facets of personality, and 6 validity scales. More recent

psychometric work with the SNAP shows that it can also be used to derive four of the

Big Five—namely, Neuroticism, Extraversion, Conscientiousness, and Agreeableness

(Calabrese, Rudick, Simms, & Clark, 2012).

Validity scales from the SNAP were used to gauge inconsistent or careless

responding. Scores that were greater than three standard deviations from the mean on

either the VRIN (inconsistent/random responding) or TRIN (yay-saying or nay-saying)

scales were deemed invalid. We chose to emphasize VRIN and TRIN over the SNAP's

other validity scales in order to exclude random or inconsistent responding, while still

preserving data for participants reporting high rates of psychopathology. In total, this

procedure resulted in 11 cases being deemed invalid and dropped from further analyses,

resulting in a total CLPS sample size of 704.

The Schedule for Nonadaptive and Adaptive Personality-Youth Version (SNAP-

Y; Linde, Stringer, Simms, & Clark, 2013). The SNAP-Y is an alternative version of the

SNAP developed by altering items to make them more appropriate for youth and

adolescents. Changes to items between the SNAP and SNAP-Y, when present at all, were extremely subtle. For instance, the SNAP item "I used to skip school quite a bit when I was a kid," was changed to "I have skipped school a lot" in the SNAP-Y. The SNAP-Y also maintains the same structure as the SNAP, with 3 higher-order "temperament" trait scales, 12 narrow-band trait scales, and 6 validity scales. Because of extensive item overlap, the same items used by Calabrese and colleagues (2012) to derive the Big Four from that SNAP- 2 can be used to create composite Big Four scales in the SNAP-Y. The SNAP-Y has been used extensively in both community and psychiatric adolescent samples. However, unlike the adult SNAP there are no population norms for the SNAP-Y validity scales. We nonetheless calculated validity scores within our sample and excluded any participants scoring greater than three standard deviations from the means on either the VRIN or TRIN scales. Six individuals were excluded based on this procedure, resulting in a total sample size of 140 offspring.

       Initial Item Selection. To establish an initial item pool, we examined the correlations between all 375 SNAP items and the ten NEO-BFAS aspect scales in the CLPS data. Items were then assigned to the aspect with which they were most highly correlated. In order to improve aspect specificity and to reduce excessive cross-loadings, we excluded any item that had a correlation on a secondary aspect within .11 of the correlation it shared with its primary aspect. For example, if an item's strongest correlation was .51 with the NEO-BFAS Volatility scale, that same item would be excluded if it also had a correlation with an absolute value of .40 with any other NEO-BFAS scale. The .11 threshold was selected to maximize the number of items that were

effective in both the CLPS and validation (offspring) sample while also maintaining adequate reliability.

Preliminary Analysis: Results of Scale Derivation

   Final Item Selection. Initial correlations between the NEO-BFAS and SNAP, excluding items with high cross-loadings, suggested that five aspect scales could be derived from the SNAP: Volatility (18 items), Withdrawal (15 items), Enthusiasm (12 items), Politeness (13 items), and Industriousness (6 items). Aspect scales that included fewer than five items following this procedure were excluded from further analyses, as such few items are likely to increase both Type 1 and Type 2 error rates (Credé, Harms, Niehorster, & Gaye-Valentine, 2012). Notably, this meant that we were unable to recover enough items to form a meaningful Assertiveness scale, rendering our hypotheses for this trait untestable. Exploratory factor analyses were conducted using the five aspect scales derived from our initial correlations between SNAP items and the NEO-BFAS scales. Principal-axis factoring with direct oblimin rotation (delta = 0) was used to extract five factors. In order to reduce collinearity in the final scales, Items were originally included only if their loading on the intended aspect was .10 greater than on any other aspect. Finally, reliability analyses were conducted on the resulting scales to examine estimates of Cronbach's alpha, as well as inter-item and item-total correlations. Items that could be deleted to improve reliability were removed from the scale. Final scales and factor loadings can be seen in Table 2, along with correlations between each item and its corresponding NEO-BFAS scale. Table 3 includes descriptive statistics and reliability

estimates for the SNAP-aspect scales. Finally, correlations between the five aspect scales and domain scores derived from the NEO and SNAP are presented in Table 4.

Scale Validation. Final scales derived from the CLPS data were externally validated using our primary sample that included offspring of mothers diagnosed with an affective disorder. Aspect scales in the offspring sample were subjected to exploratory factor analysis, once again using principal-axis factoring with direct oblimin rotation (delta = 0). To account for the fact that the offspring sample features siblings nested within families, we employed a Huber-White sandwich estimator to correct standard errors and fit statistics and account for non-independence of observations. Results of this factor analysis can be seen in Table 2. Items were inspected to ensure that their primary loading was on the intended aspect. Four items did not load on the intended aspect (three from Politeness, one from Industriousness) in the offspring sample, likely due to our small sample size. These items were not included in the composite scales used in our main analyses. One additional item was excluded from the aspect scales because it loaded on Volatility in both our samples, but was listed as part of the Agreeableness scale previously published by Calabrese and colleagues (2012). Reliability estimates suggested that four of the final aspect scales demonstrated good reliability, with Industriousness demonstrating poorer reliability due to the low number of items that make up that scale. Cronbach's alphas and descriptive statistics for each scale can be seen in Table 3.

Main Analysis

Participants

Participants in the present study were drawn from a sample of 98 families participating in an ongoing longitudinal study of the offspring of depressed mothers (Radke-Yarrow, 1998). Recruitment and ongoing assessments took place between 1979 and 2003. All mothers were biological mothers and primary caregivers for the offspring participants. Offspring and their families were assessed five times through the course of the study. Families were assessed, on average, every three years over the course of the first four assessments, with the last assessment occurring approximately seven years later.

Forty-two of the mothers in the study were diagnosed with unipolar depression, 26 with bipolar disorder I or II, and 30 were healthy controls. Two children were selected as participants from each family (4% of the families had only one child) resulting in a total of 192 child participants, including 48 O-BD, 84 O-MD, and 60 O-WELL (Table 1). The families entered the longitudinal study when the younger child was a toddler and the older sibling was approximately three years older.

Demographic characteristics of offspring, mothers, and participating families are presented in Table 1. The younger sibling cohort was 2.63 (SD = .62), 5.54 (SD= .55), 9.26 (SD= 1.06), 13.87 (SD=1.71), and 20.51 (SD=1.56) years of age at the first (T1), second (T2), third (T3), fourth (T4), and fifth (T5) assessment periods, respectively. The older sibling cohort was 6.36 (SD = 1.06), 9.28 (SD= 1.24), 13.03 (SD= 1.42), 17.68 (SD=1.95), and 24.21 (SD=2.07) years of age at T1, T2, T3, T4, and T5, respectively. Fifty percent of the younger cohort and 42% of the older cohort were males.

The majority of families were middle- to upper middle-class, with a mean score of 51.00 (SD=14.84) on the Hollingshead (1975) Index of Socioeconomic Status, though the

study included a broad range of socioeconomic groups. The sample was 86% Caucasian,

11% African-American, 2% Asian-American, and 1% Latin-American. The majority of

mothers had at least some college level education at T1 (79%). At the initial assessment,

most families (e.g., 94%) consisted of two offspring living with both biological parents.

Measures

      Parental psychopathology. Determination of maternal psychiatric diagnoses at

T1 of the current study was based on the initial interview using the Schedule for

Affective Disorders and Schizophrenia: Lifetime Version (SADS-L; Spitzer & Endicott,

1977). Psychiatric diagnoses were subsequently assessed at a T3 interview using the

Structured Clinical Interview for DSM-III-R (SCID; Spitzer, Williams, Gibbon, & First,

1989) and an interval SADS-L and using criteria almost identical to those in the

Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). The

SADS-L interviews were conducted by professionals (psychiatric nurse, psychiatrist,

clinical psychologist from the National Institute of Mental Health) trained by staff at the

New York Psychiatric Institute. The clinicians administering the interviews had no

previous contact with the parents or offspring, and were blind to earlier diagnoses. Ten

interviews were coded independently by a staff member at the National Institute of

Mental Health and a staff member at the New York Psychiatric Institute, with 100%

agreement on diagnosis for the presence and type of mood disorder. Mothers were

eligible if they met the Research Diagnostic Criteria (Spitzer, Endicott, & Robins, 1978)

for Bipolar Disorder (BD; Bipolar I or II disorder), Major Depressive Disorder (MDD) or

were without past or current psychiatric disorder. There were nine mothers whose

diagnoses were adjusted over the duration of the study. This included mothers that at recruitment were diagnosed with Minor Depression but later developed MDD as well as other changes (e.g., MDD to BD). The adjusted diagnosis was used in this study because the focus was primarily on the later assessment periods.

If the mother met criteria for eligibility, the father was given a SADS-L interview. At recruitment, spouses of MDD or BD mothers either had no diagnosis or were permitted to have a diagnosis of MDD, anxiety, or substance abuse (spouses of BD mothers were also allowed to have BD). Across both ill groups, the most common paternal diagnosis was depression, accounting for 53% of fathers. Additionally, 7.6% of fathers within the ill group met criteria for substance abuse, two fathers (both in the BD group) met criteria for BD, and four fathers met criteria for Generalized Anxiety Disorder. For control families, both parents were interviewed and neither met criteria for current or past psychiatric disorder at the time of the initial sample selection. However, during the T3 interview two spouses of well mothers were diagnosed with depression.

Personality traits. Offspring personality traits were assessed at T4 using the *The Schedule for Nonadaptive and Adaptive Personality-Youth Version (SNAP-Y).* The SNAP-Y (Clark, 1993) has been previously described in detail in our preliminary analysis (see above). For each of the 375 SNAP-Y items, respondents decided how well the item describes them and marked true if the statement was true or mostly true for them and false if it was false or mostly false for them. Scales of interest in the present analysis included the Big Four—Neuroticism, Extraversion, Agreeableness, and Conscientiousness—which we derived using scoring procedures outlined by Calabrese

and colleagues (2012). Lower-order aspect scales for Volatility, Withdrawal, and Enthusiasm, which we derived in our preliminary analysis, were also used. Industriousness and Politeness were evaluated in exploratory analyses. When using lower-order personality traits in statistical analysis, it is essential to control for shared variance between lower-order traits that is best explained by the overarching domain. For example, when evaluating Volatility as a predictor, one must control for Withdrawal effects in order to be confident that any observed effects are specific to Volatility, as opposed to Neuroticism more broadly. Because we were only able to derive Enthusiasm and not its counterpart Assertiveness from the SNAP scales, we created a "residual Extraversion" variable, by subtracting any Enthusiasm-related items from Calabrese and colleagues' (2012) Big Four Extraversion scale (the same procedure was used for Politeness and Industriousness, with their respective domains). Entering this residual Extraversion variable into our Enthusiasm analyses allows us to be more confident that any effects observed are due to the lower-order aspect, and not Extraversion more broadly. Internal reliability statistics for all Big Four and aspect scales are available in Table 3. We once again excluded six individuals with SNAP data due to excessive scores on the VRIN or TRIN scales.

Cognitive Style / Attributional Bias. The *Children Attributional Style Questionnaire (CASQ;* Seligman et al., 1984) was administered to all subjects at the T3 assessment. The CASQ is a 48-item forced-choice measure of explanatory style. Each item presents a hypothetical event and two possible explanations (one positive, and one negative) for why that event occurred. Respondents are instructed to imagine the event

occurring, then to choose which of the two explanations best describes why the event would happen to them. An example of an item from the CASQ is, "A good friend tells you that he hates you," for which the given attributions are, "A. My friend was in a bad mood that day (external); B. I wasn't nice to my friend that day (internal)." Each item varies one causal dimension (internality, stability, globality) while holding the others constant. In the given example, the stability and globality dimensions are held constant, whereas the internal vs. external attributional dimension is varied. There are 16 events that pertain to each of the three explanatory dimensions. There are six subscales on the CASQ: the internality, stability, and globality scales for bad events, and the externality, stability, and specific scales for good events. The CASQ is scored by assigning a 1 to each internal, stable, or global response (when that dimension is varied), and a 0 to each external, unstable, or specific response. A composite explanatory style score for positive events is obtained by adding the child's scores on each of the three subscales for positive events. A composite explanatory style score for negative events can be obtained by summing the scores of the subscales for negative events. An overall explanatory style score, which is the primary variable of interest in our analyses, can be obtained by subtracting the composite negative score from the composite positive score. The lower the overall style score, the more the child explains bad events in terms of internal, stable, and global causes, while explaining good events in terms of external, unstable, and specific causes. Psychometric examinations of the original CASQ report test-retest reliabilities over 12 months of .71 for the positive composite, .66 for the negative composite, and .35 for the overall composite (Seligman et al., 1984). Others report low to

moderate reliability for positive events ($\alpha = .47$-$.73$), negative events ($\alpha = .42$-$.67$), and

the overall composite ($\alpha = .62$; Nolen-Hoeksema, Girgus, & Seligman, 1986, 1992;

Panak & Garber, 1992). Reliability estimates in the present sample were similarly low

(positive composite, $\alpha = .59$; negative composite, $\alpha = .39$; overall composite, $\alpha = .43$),

which is likely to limit our power to detect significant interactive effects involving

children's attributions. On the other hand, Hilsman and Garber (1995, p. 373) note that

low internal consistency may not be as problematic if domain-specific attributions have

additive effects in the same direction, despite not comprising a unitary factor. To some

extent, the current data is limited by the measures available during data collection, as

measures of attributional bias with superior psychometrics have only become available

recently.

Offspring psychopathology. *Diagnostic Interview for Children and Adolescents-*

Revised (DICA-R, version 5). The mood disorder sections of the DICA-R (Reich &

Welner, 1988) were administered to adolescents at T4 to screen for the presence of

current or past mood disorders. Interviews were administered and coded by masters or

doctoral level clinicians who were unaware of the diagnostic status of parents and

children. Reliability evaluation of results in 26 of the cases revealed an interclass

correlation for affective symptoms of 0.91. The DICA-R provides criteria for diagnoses

in the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; *DSM-III-R*;

American Psychiatric Association, 1987), including major depressive disorder and

bipolar affective disorder. To retain more information concerning adolescents' problems,

symptom counts of subclinical and clinical symptoms rather than yes/no diagnoses were used in analyses.

Symptom Checklist—90—Revised (SCL-90-R). Adolescents were administered the SCL-90-R at T4 (Derogatis, 1983). The SCL-90-R is a 90-item self-report survey of symptoms that has shown to be useful as both a screening and diagnostic tool in adolescents and young adults (Todd, Deane, & McKenna, 1997). The participant rates the presence or absence and intensity of each symptom. Each item is rated on a five point scale of distress from 0 (none) to 4 (extreme). Scoring yields three ratings of global emotional functioning and nine symptom clusters including psychoticism, paranoid ideation, phobic anxiety, hostility, anxiety, depression, interpersonal sensitivity, obsessive compulsive, and somatization (Derogatis & Cleary, 1977). The internal consistency coefficient (Cronbach's alpha) for the depression scale, which is the focus of our analysis, has been reported as .94 (Hoffman & Overall, 1978).

Child Behavior Checklist (CBCL). The CBCL (Achenbach & Edelbrock, 1983; Achenbach, 1991) was completed by the mother at T1, T2, T3, and T4. The CBCL is a parent report form that has been normed on large national samples and is widely used to measure childhood psychopathology in both clinical and research settings. CBCL items form eight subscales: anxious/depressed, withdrawn/depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behavior, and aggressive behavior. Two higher-order factors, Externalizing and Internalizing behavior, have also been obtained consistently using principle-components analysis (Achenbach & Edelbrock, 1978). The reliability of the CBCL, assessed through test-retest agreement, is

acceptable (correlations of .74 to .95) and the CBCL manual reports adequate construct and content validity (Achenbach & Edelbrock, 1983).

In recent years, Achenbach and Dumenci (2001) and Achenbach, Dumenci, and Rescorla (2003) have proposed alternative methods of scoring the CBCL (and its associated versions) to derive DSM-oriented scales that reflect current conceptualizations of child symptomatology. The present study utilizes the Affective Problems subscale that is derived from this alternative scoring procedure as an indicator of depressive symptomatology at T4. Reliability of the Affective Problems scale, measured via coefficient alpha, was .76 for the CBCL.

Internal State Scale (ISS). The ISS (Bauer et al., 1991) is a self-report measure of current bipolar disorder symptoms that was administered at T4. The present study used a modified version of this scale, which included 28 items rated on a 3-point Likert scale ("Never," "A little," "A lot"). A modified scoring procedure was used because our version of the scale contained more usable items than the 15 typically present in other reports using the ISS. Two items serve as indicators for irritability (e.g., "I feel irritable and cranky"), elevated mood (e.g., "I feel extremely excitable and happy (elated)"), talkativeness (e.g., "I tend to talk so fast that others can hardly understand me"), decreased need for sleep (e.g., "I don't feel tired even though I haven't been getting much sleep"), grandiosity (e.g., "I am superior to (better than) most people I know,"), risk-taking behavior (e.g., "I feel like taking risks which might get me into trouble"), flight of ideas (e.g., "I feel like my thoughts are racing through my mind"), thought content (e.g, "I feel like my thoughts don't make sense"), insight (e.g., "I don't think about why I do

things"), and appearance (e.g., ("I feel like I look messy and unkempt"), respectively.

Four items indicate heightened goal-directed activity or psychomotor agitation (e.g., "I

feel hyper and "on the go"). The 11 symptoms subscales were combined into an overall

composite mania scale reflecting manic symptoms at T4. Reliability of the composite

mania scale in the present sample, measured by coefficient alpha, was .76.

Young Mania Rating Scale (Y-MRS). The Y-MRS (Young et al., 1978) is an 11-

item scale in which clinicians rate participants' elevated mood, motor activity, sexual

interest, sleep problems, irritability, speech, language/thought content, aggression,

appearance, and insight. Based on item weighting, seven items are rated on a 0 to 4 scale

and four items are rated on a 0-8 scale, with higher scores indicating greater severity. The

Y-MRS has been found to be a reliable and valid scale for assessing mania in adults,

adolescents, and children (Young et al., 1978; Youngstrom, Danielson, Findling,

Gracious, & Calabrese, 2002). Reliability, as measured by coefficient alpha, has been

reported as .91 in child and adolescent samples (Youngstrom et al., 2002), but was more

moderate in our own sample ($\alpha = .65$).

Structured Clinical Interview for DSM-IV AXIS I Disorders. The SCID-I (First,

Gibbon, Spitzer, & Williams, 1996) was administered to offspring at T5. Trained

clinicians, including a psychiatrist and two advanced doctoral clinical psychology

students, administered the SCID-I. Interrater reliability was based on 16 cases, and

kappas for the major affective diagnoses were 1.0. Clinicians were blind to maternal Axis

I and II diagnoses. As with the DICA-R, symptom counts for current manic and

depressive episodes were used rather than yes/no diagnoses in these analyses.

Beck Depression Inventory. The revised BDI (Beck & Steer, 1987) is a 21-item self-report questionnaire administered to young adults at T5. Each item on the BDI consists of four statements indicating different levels of severity of a particular symptom experienced over the past week. For example, one item assesses the symptom of sadness, and participants are asked to choose among the following statements in order to convey how they have been feeling: "0) I do not feel sad; 1) I feel sad much of the time; 2) I am sad all the time; 3) I am so sad or unhappy that I can't stand it." Scores for all 21 items are summed to yield a single depression score. The internal consistency of the BDI, based on a number of clinical samples, is 0.86 (Beck & Steer, 1987), and reliability in the present sample was similarly high ($\alpha = .91$).

Aggregation of Depressive and Manic Symptoms. Composite variables reflecting depressive and manic symptomatology were created from the various indicators of offspring psychopathology at T4 and T5. At T4, symptom counts for major depression from the DICA-R, the depression subscale of the SCL-90-R, and the affective problems subscale of the CBCL were aggregated to yield an offspring depressive symptom composite. Similarly, symptom counts for manic and hypomanic episodes from the DICA-R and the ISS mania score were aggregated to yield an offspring manic symptom composite at T4. At T5, depressive symptoms were measured via the aggregation of the BDI overall score and symptom counts for major depressive episodes from the SCID-I. Finally, T5 manic symptoms were assessed by symptom counts of manic and hypomanic episodes from the SCID-I.

Covariates. *The McCarthy Scale of Children's Abilities (MSCA; McCarthy, 1972) / The Wechsler Scale of Intelligence for Children, Revised (WISC-R: Wechsler, 1974) / The Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955).* The MSCA was administered to the younger cohort at the T2 assessment to assess general cognitive abilities. The WISC-R was administered at the T3 and T4 assessments. Ten subtests were administered in total: Information, Similarities, Arithmetic, Vocabulary, Comprehension, Picture Completion, Picture Arrangement, Block Design, Object Assembly, and Coding. The accompanying estimates of full scale IQ, verbal IQ and perceptual IQ based on these ten subtests have been shown to be both reliable and valid (Wechsler, 1974). Finally, an abbreviated version of the WAIS was administered to participants at T4 if their age had exceeded the norm cut-offs for the WISC-R. Because our analyses span adolescence, we used the average score of these three measures to control for intelligence.

Hollingshead Four-Factor Index of Socioeconomic Status. The Hollingshead Four-Factor Index of Socioeconomic Status (Hollingshead, 1975) was administered to all families at T1. The measure was designed to assess social status based on four domains: marital status, retired/employed status, educational attainment, and occupational prestige. Each domain was rated based on continuous scales with higher scores reflecting greater social status. Education and occupational scores were then weighted to obtain a single score that reflects one of five social strata, ranging from unskilled laborers and service workers to executives and professionals. When multiple caregivers were assessed, scores were averaged to attain an overall family SES score.

Data Analytic Strategy

Missing Data. Rates of missing data at the time points assessed were: 29.69% on the CASQ at T3, 23.96% on the SNAP at T4, between 2.6% and 13.5% on the various psychopathology measures used at T4, and between 27.6% and 30.2% on the psychopathology measures used at T5. There was no missing data at T1. Missing data was modeled under the assumption that missingness is related to observed variables but unrelated to the missing values themselves (i.e., missing at random; Schafer & Graham, 2002). Multiple imputation (MI) using *SPSS Version 23* was used to address missingness. MI has been studied extensively and is generally considered superior to other missing data techniques (e.g., listwise deletion, mean imputation; Graham & Schafer, 1999; Schafer & Graham, 2002). In MI, missing values are estimated based on relationships among variables in the incomplete, pre-existing dataset. This procedure is done several times, resulting in numerous complete datasets. Variability in the imputed values across these new datasets reflects the inherent uncertainty that is involved in predicting missing values, resulting in more accurate parameter estimates once data are analyzed and results are pooled together.

Data Analysis. Standard statistical methods assume that observations are independent of one another, which means that these techniques are biased by any method of data collection that does not reflect true random sampling (Bryk & Raudenbush, 1992). If the assumption of independent observations is violated then the influence of the contextual unit or group may be attributed to the individual, causing the standard errors of the model parameters to be underestimated and increasing the likelihood of Type 1 errors (Heck & Thomas, 1999). In the current study, the assumption of independent

observations is violated due to within family recruitment of participants. We initially planned to account for the clustered nature of our data using multilevel regression analysis, in which both within- and between- group effects are modeled in the context of nested regression equations. Typically, multilevel modeling is needed when the design effects (1 + (average cluster size-1)*Intraclass Correlation Coefficient), which reflect the degree to which standard errors are underestimated due to clustering, approach two (see Monte Carlo simulation, Table 2 in Satorra & Muthén, 1995). Examination of the ICCs for all dependent variables in the current analysis revealed that designed effects are well below two.

Another concern with adopting a multilevel modeling approach is that the analysis assumes that variables will exhibit non-constant error variance. Initial inspection of our data indicated that this assumption was violated, which can lead to severely biased variance components and standard errors (Maas & Hox, 2005). To account for this difficulty, we chose to employ Huber-White cluster robust standard errors (Huber, 1967; White, 1982). Robust standard errors use observed residual variances to account for the clustered structure of the data and to correct biased asymptotic standard errors (Maas & Hox, 2005; Satorra & Muthén, 1995). When using robust standard errors, regression coefficients are interpreted as they would be in typical ordinary least squares regression analysis.

Analytic Plan. Analyses were conducted using *Mplus Version 6* (Muthén & Muthén, 2010). Preliminary questions to be evaluated included the relationship between maternal diagnosis and offspring personality traits and attributional biases. These

questions were evaluated using a series of multiple linear regressions, with offspring personality or attributions serving as the dependent variables of interest. Analyses entered maternal diagnosis, family SES, and offspring age, gender, and IQ as simultaneous predictors of the relevant offspring characteristics. Robust standard errors were employed to correct for the clustered nature of the data.

The current study's primary questions involved the interaction of maternal affective illness and offspring characteristics in predicting offspring psychopathology. To address these questions, we adopted a hierarchical multiple regression approach. In the first step of regression modeling, standard covariates of family SES and offspring age, gender, and IQ were entered. Offspring symptoms at T4 were also included as covariates when predicting symptom levels at T5. In the second step, maternal diagnosis and the relevant offspring individual differences variables (trait scores, attributional scores, or both) were added to the model. Interactions between offspring characteristics and maternal diagnoses were added in the final step.

Commonly used approaches for comparing hierarchically nested regression models are not currently available when using multiply imputed data sets. As a result, the Wald test of parameter constraints was used to determine whether specific predictors, or groups of predictors, warranted inclusion in the final regression models. When the Wald test for a particular step in the regression model was significant, then the predictors in that step were deemed to explain significant variation in the dependent variable. In contrast, a non-significant Wald test signaled that a predictor or group of predictors did

not add to the explanatory power of the model, and that predictor was therefore dropped

from the regression.

Results

Descriptive Statistics and Bivariate Correlations among Major Constructs

Table 1 shows descriptive statistics, including means and standard deviations, for

the maternal risk groups considered in this study. At the outset of the study, considerable

efforts were directed at recruiting comparable groups and as a result, few demographic

differences are evident across risk groups. However, family SES tended to be lower in

families in which either maternal bipolar depression (β= -.185, SE = .099, p = .063) or

major depression was present (β= -.293, SE = .100, p = .004). O-MD were also

significantly older than their O-BD counterparts at T4 (β= .162, SE = .070, p = .020) and

T5 (β= .161, SE = .067, p = .015). Previous research has demonstrated significant

differences in IQ in this sample (Klimes-Dougan, Ronsaville, Wiggs, & Martinez, 2006).

When both risk groups were considered together, O-AD exhibited lower average IQ than

O-HC across childhood and adolescence (β= -.162, SE = .078, p = .037). Given these

differences, subsequent analyses control for age, gender, SES, and offspring IQ.

Descriptive statistics for all major scales used in the present analyses are available

in Table 3. Intraclass correlation coefficients (ICCs) suggested that sibling clustering

explained modest to moderate variation in personality and psychopathology. Bivariate

correlations between maternal diagnosis, offspring attributional style, personality, and

psychopathology are in Table 5. Correlational relationships among study variables were

largely as expected, though maternal diagnosis was only significantly related to T4

depressive symptoms. Depressive symptoms at both time points were positively correlated with Neuroticism and negatively correlated with Extraversion and Agreeableness. Manic symptoms at both time points also tended to be positively related to Neuroticism and negatively related to Agreeableness. Attributional biases were positively related to Agreeableness and negatively related to both Withdrawal and depressive symptoms at both time points.

Relationship between Maternal Affective Illness and Offspring Personality Traits

One of our initial questions was whether offspring of mothers diagnosed with an affective disorder would exhibit reliable differences in T4 Neuroticism when compared to offspring of healthy comparison mothers. To examine this hypothesis, we conducted a multiple linear regression, in which presence or absence of a maternal affective disorder was entered as a predictor of offspring Neuroticism, controlling for family SES as well as offspring age, gender, and IQ. Contrary to our first hypothesis, O-AD did not appear to differ from O-HC on Neuroticism (β= -.012, SE = .093, p = .901). Gender was the only significant predictor in the model, with female offspring scoring more highly on Neuroticism than male offspring (β= .208, SE = .074, p = .005).

We were also interested in whether the specific type of maternal diagnosis, namely major depression or bipolar disorder, differentially predicted offspring personality. To evaluate this question, a linear regression was constructed by entering all relevant covariates along with dummy coded variables to reference each maternal diagnosis. Healthy comparison mothers served as the initial reference group, though comparisons between the two risk groups were also evaluated. Contrary to our hypothesis

(Hypothesis 1), neither maternal major depression (β= -.044, SE = .099, p = .655) nor maternal bipolar depression (β= -.083, SE = .111, p = .458) significantly predicted offspring Extraversion at Time 4.

On an exploratory basis, we also examined whether type of maternal affective diagnosis predicted any of the remaining Big Four personality traits. The only significant effect of maternal diagnosis occurred in the Neuroticism model, as O-BD (M = 1.482, SD = .272) were significantly higher on Neuroticism at T4 than O-MD (M = 1.367, SD = .241; β= .186, SE = .086, p = .031). However, this difference did not extend to models in which O-HC served as the reference group. To determine whether the effect of maternal diagnoses on Neuroticism was driven by either of the two lower-order Neuroticism aspects, we replicated the model using Withdrawal and Volatility as the relevant dependent variables. Results indicated that O-BD were significantly higher than O-MD on Withdrawal (β= .187, SE = .090, p = .038), but this effect did not hold for Volatility (β= .154, SE = .102, p = .131).

Relationship between Maternal Diagnosis and Offspring Attributional Style

We next examined whether the presence of a maternal affective disorder would predict offspring attributional biases in adolescence (Hypothesis 7). A multiple regression was conducted in a similar fashion as when investigating personality traits as outcomes. Results of the regression indicated that the presence of a maternal affective disorder did not significantly predict offspring attributional biases at T4 (β= -.033, SE = .087, p = .701). Likewise, none of the covariates in the model significantly predicted offspring attributions. Follow-up exploratory analyses examined whether there was an effect of

specific type of maternal diagnosis on offspring attributional bias. Results again revealed that neither maternal major depression (β= -.032, SE = .106, p = .763) nor maternal bipolar depression (β= -.037, SE = .104, p = .726) significantly predicted offspring attributions at T3.

Big Four Predicting Offspring Manic and Depressive Symptoms

Having explored the relationship between maternal affective disorder and offspring individual differences, we next examined whether offspring Extraversion and Neuroticism interact with maternal diagnosis to predict offspring affective symptomatology both concurrently and prospectively (Hypothesis 2).

We first examined the relationship between maternal diagnosis, offspring Neuroticism and Extraversion, and affective symptoms at Time 4. Independent hierarchical multiple regressions were conducted with T4 depressive and manic symptoms serving as outcome variables of interest. Relevant covariates were entered in Step 1 of the regression. Maternal diagnosis (coded 0 for O-HC and 1 for O-AD) was entered in Step 2, along with offspring Extraversion and Neuroticism. In the third and final step, the interaction of maternal diagnosis with Extraversion and Neuroticism was added to the model.

Results of the final regression model for T4 depressive symptoms can be viewed in Table 6. The Wald test of parameter constraints indicated that the interaction of maternal diagnosis and offspring personality in Step 3 of the regression did not explain significant variance in our outcome, and so interaction terms were excluded from the analyses. However, we did find several significant main effects in Step 2 of the model, as

both maternal diagnosis and offspring Neuroticism were significantly associated with concurrent depressive symptoms. Contrary to hypotheses, low Extraversion was not related to concurrent depressive symptoms. Examination of the R^2 change due to the presence of each main effect in the model indicated that maternal diagnosis accounted for only 2.8% of the variance in offspring depressive symptoms, whereas offspring Neuroticism accounted for 16.2% of the variance in T4 depressive symptoms.

A second hierarchical regression model was fit to examine the role of offspring personality and maternal diagnosis in predicting T4 mania. As with the model predicting concurrent depressive symptoms, the Wald test of parameter constraints for the regression involving T4 mania indicated that only the direct effects, and not the interactions, between offspring personality and maternal diagnosis explained significant variance in the outcome. As a result, we excluded the interactions from the final model. Consistent with our hypotheses, increased offspring Neuroticism and Extraversion predicted concurrent manic symptoms, though maternal diagnosis did not emerge as a significant predictor (see Table 6). R^2 change coefficients indicated that offspring Neuroticism accounted for 10.6% of the variance in concurrent manic symptoms, whereas the effect of Extraversion explained only 1.8% of the variance.

We next turned to regressions exploring whether maternal diagnosis and offspring personality predict prospective changes in offspring affective symptoms. Hierarchical multiple regressions were fit in a similar fashion as when we examined concurrent symptoms. Time 4 depressive or manic symptoms were added with the relevant covariates in Step 1 in order to assess the influence of our predictors above and beyond

previous symptom levels. In the model predicting depressive symptoms at T5, the Wald

test was non-significant for Step 3, the step in which the relevant interaction terms were

added to the model. However, examination of the interactions revealed a significant

interaction between maternal diagnosis and offspring Neuroticism. As a result, we

examined a model that excluded the interaction between offspring Extraversion and

maternal diagnosis, but retained the interaction involving Neuroticism. The Wald test for

the revised model was indeed significant, indicating that the interaction term between

Neuroticism and maternal diagnosis explained a significant amount of the variance in T5

depressive symptoms. Results of the final model are presented in Table 7.

Examination of the main effects from Step 2 of the model predicting prospective

depressive symptoms indicated that high Neuroticism at T4 significantly predicted

depressive symptoms in offspring at T5, accounting for about 11.8% of the variance. The

main effect of Neuroticism became non-significant with the addition of the Step 3

interaction term, indicating that the effect of Neuroticism is dependent on the presence or

absence of a maternal affective diagnosis. The addition of the interaction term in Step 3

of the model explained an additional 2.7% of the variance in T5 depressive outcomes.

Simple slopes analyses, as outlined by Aiken and West (1991), were used to probe the

interaction between maternal diagnosis and Neuroticism. As expected, the simple slope

of Neuroticism was significant in the presence ($b = .848$, $t(183) = 3.521$, p = .001), but

not the absence ($b = .560$, $t(183) = 1.846$, p = .066) of a maternal affective disorder.

Simple slopes are plotted in Figure 2.

Finally, we examined a similar hierarchical regression model to explore the relationship between personality, maternal diagnosis, and offspring manic symptoms at T5 controlling for symptoms levels at T4 (see Table 7). Addition of the covariates in Step 1 of the model indicated that manic symptoms at T4 significantly predicted symptoms at T5 ($R^2\Delta = .036$). Results of the Wald test of parameter constraints for Step 2 of the model indicated that neither maternal diagnosis nor offspring Extraversion or Neuroticism explained a significant degree of variance in T5 manic symptoms. As a result, both direct effects and interaction effects were excluded from the model predicting T5 manic symptoms.

Lower-order Aspects Predicting Offspring Manic and Depressive Symptoms

Analyses concluded by examining the present study's central questions--namely, whether maternal diagnosis interacts with lower-order offspring personality traits and attributional biases to predict affective symptoms in adolescence and young adulthood. Regression modeling took a similar form as in previous analyses. A hierarchical procedure was adopted, with relevant covariates entered in Step 1. Main effects of maternal diagnosis, offspring lower-order personality traits, and offspring attributional biases were entered in Step 2. In order to ensure that effects of lower-order traits were specific to the aspect level and not reflective of broader domain effects, we included both lower-order traits in each model (or in the case of Enthusiasm, our "nonspecific Extraversion" variable was added). In Step 3, hypothesized two-way interactions between offspring individual difference variables (traits, attributions) and maternal diagnosis were added to the model. Finally, hypothesized three-way interactions were added in Step 4 of

each model. The Wald test of parameter constraints was once again used to determine if

parameters in each step contributed significantly to the model and therefore warranted

inclusion. Models predicting concurrent depressive and manic symptoms were treated

first.

Final regression models predicting depressive and manic symptoms concurrently

are presented in Table 8. Wald tests for both the first and second steps of the model

predicting T4 depression were significant, indicating significant main effects of our

covariates and predictors. In Step 3, we entered several two-way interactions to reflect

our hypotheses that Withdrawal, Enthusiasm, and attributional style would each interact

with maternal diagnosis to predict offspring depression (Hypotheses 3, 5, and 8,

respectively). In Step 4, we entered two additional three-way interactions, reflecting the

hypothesis that interactions between lower-order traits and maternal diagnosis would be

amplified in the presence of more negative offspring attributional styles (Hypothesis 9).

Contrary to expectations however, Wald tests for Steps 3 and 4 indicated no significant

effect of the hypothesized two- or three-way interactions. Given the results of the Wald

tests, all interactions were excluded from the final model predicting T4 depressive

symptoms, and the model including only direct effects was retained. Examination of the

regression coefficients from the final model confirmed several of our hypotheses, as both

Withdrawal ($R^2\Delta = .129$) and the presence of a maternal affective diagnosis ($R^2\Delta = .018$)

significantly predicted increased T4 depressive symptoms. In contrast, neither

Enthusiasm nor offspring attributional bias emerged as a significant predictor in the

model.

We originally hypothesized that the lower-order Extraversion aspect of Assertiveness would be related to manic symptoms (Hypothesis 6). However, since we were unable to derive a marker of Assertiveness from the SNAP-Y, we excluded the lower-order Extraversion aspects from models predicting manic symptoms, either concurrently or prospectively. As was the case with our model predicting concurrent depressive symptoms, Wald tests for the model predicting T4 mania indicated significant main effects in Step 1 and 2 of the regression. Examination of the regression coefficients of our main predictors indicated that Withdrawal significantly predicted increased T4 manic symptoms ($R^2\Delta = .056$). Contrary to expectations, Volatility, attributional bias, and maternal diagnosis did not emerge as significant predictors of T4 manic symptoms. Given the significant main effect of Withdrawal, an interaction between Withdrawal and maternal diagnosis was added in Step 3 of the model along with the two interactions we hypothesized a priori (Volatility x Maternal Diagnosis, Attributional Style x Maternal Diagnosis; Hypotheses 4 and 8, respectively). Step 4 likewise included three-way interactions involving Withdrawal and Volatility (Hypothesis 10) along with maternal diagnosis and offspring attributions. As can be seen in Table 8 however, Wald tests for Steps 3 and 4 were not significant, leading to the conclusion that none of the interaction terms significantly contributed to the prediction of T4 manic symptoms. As a result, the main effects model (Step 2) was retained as the final regression model.

Results of the final regression models predicting depressive and manic symptoms prospectively are presented in Table 9. As with the model predicting concurrent depressive symptoms, Wald tests of the regression predicting prospective depressive

symptoms revealed significant main effects from Step 1 and 2 symptoms. Examination of

Step 2 of the model indicated that Withdrawal significantly predicted T5 depressive

symptoms even when controlling for previous symptom levels ($R^2\Delta = .075$). Somewhat

surprisingly, Volatility also significantly predicted T5 depressive symptoms in the

prospective model ($R^2\Delta = .049$), suggesting that unique variance from both lower-order

aspects of Neuroticism predict the emergence of depressive symptoms in young

adulthood. Contrary to expectations, neither maternal diagnosis nor offspring

attributional biases emerged as significant predictors of T5 depressive symptoms.

Moreover, Wald tests for Steps 3 and 4 of the model were not significant, indicating that

the interaction of offspring traits and attributions with maternal diagnosis did not

significantly contribute to the emergence of depressive symptoms at T5 (Hypotheses 3, 5,

8, and 9). Consequently, the main effects model from Step 2 was again retained as the

final regression model predicting T5 depressive symptoms.

Our last model examined the prospective prediction of offspring manic

symptoms. As can be seen in Table 9, none of the Wald tests from Steps 1-4 of this

regression model were significant, indicating that no covariates, main effects, or

interaction terms significantly predicted T5 manic symptoms (Hypothesis 4, 8, and 10).

However, variation in T5 mania symptoms was low and visual inspection of the data

revealed a pattern of strong positive skew that is not well-corrected for by transformation

or use of a robust estimator, suggesting that the Wald test, which assumes multivariate

normality, may be flawed in this instance. Indeed, inspection of the regression

coefficients for each successive step of the model revealed some significant, though

small, effects. In Step 1, there was a trend for T4 manic symptoms to positively predict

symptoms at T5. Withdrawal emerged as a significant predictor of increased manic

symptoms at T5 in Step 2 of the model, though it accounted for only a small amount of

variance in the outcome ($R^2\Delta$ = .020). Finally, when two-way interactions were entered in

Step 3 of the model, the interaction between Withdrawal and maternal diagnosis trended

toward significance (β= .185, SE = .100, p = .065), suggesting that the effect of

Withdrawal may be partially moderated by the presence or absence of maternal affective

disorder. There were no significant three-way interactions in Step 4 of the model. Given

that the two-way interaction between Withdrawal and maternal diagnosis did not reach

formal significance, the main effects model, which indicated a direct effect of

Withdrawal, was selected as the final regression model.

Discussion

Offspring of mothers diagnosed with unipolar or bipolar depression are at

increased risk for developing a wide range of mental disorders (Cullen et al., 2014;

Klimes-Dougan et al., 2010, 2013; Weissman et al., 2006). Consistent with a

developmental psychopathology perspective (Cicchetti, 1984; Cicchetti, 1993; Sroufe &

Rutter, 1984), studies have focused extensively on identifying moderators that might

explain the vast heterogeneity of outcomes present in O-AD (Goodman, 2007; Radke-

Yarrow & Klimes-Dougan, 1997; 2002). The goal of the present study was to extend the

search for moderators into the domain of offspring characteristics, investigating whether

individual differences in offspring personality and cognitive style interact with maternal

affective diagnoses to predict the development of affective symptomatology both in

adolescence and young adulthood. Specifically, we sought to 1) determine whether maternal affective disorders are associated with reliable differences in offspring personality traits and cognitive style, 2) examine the role of offspring personality and attributional biases in predicting vulnerability to mania and depression, and 3) investigate whether offspring personality, cognitive vulnerability, and maternal mood disorder interact to increase risk for psychopathology in an additive manner.

Results suggested that the presence of a maternal affective diagnosis was largely unrelated to offspring personality and cognitive style, but did predict depressive symptoms in adolescence. Offspring personality, but not cognitive style, was associated with both concurrent and prospective affective symptoms. Limited evidence was attained to suggest that the relationship between offspring personality and psychopathology was moderated by the presence of a maternal affective disorder.

Individual Differences in Personality among O-AD

Children developing in the context of maternal depression are subject to a diverse confluence of risk factors that serve to shape and organize their emerging personalities (Cicchetti & Toth, 1998). A number of studies investigating offspring differences across common personality traits have suggested that a history of maternal affective disorder is linked to increased offspring Neuroticism and negative emotionality (Davis et al., 2007; Doucette et al., 2013, 2014; Duffy et al., 2007; Farchione et al., 2007; Feldman et al., 2009; Melchior et al., 2012; Whiffen & Gotlib, 1989). Results of the present study partially replicated these findings. Contrary to our expectations, the presence of a maternal affective disorder was not related to offspring Neuroticism in middle and late

adolescence. However, when we considered maternal unipolar and bipolar disorders independently from one another, we found that O-BD scored significantly higher on Neuroticism than their O-MD counterparts, despite neither group differing from healthy comparison offspring. This finding is largely consistent with previous research showing elevated Neuroticism in O-BD (Doucette et al., 2013, 2014; Duffy et al., 2007; Farchione et al., 2007), though other studies have failed to find this same effect (e.g., Jones et al., 2006; Nijjar et al., 2014; Rothen et al., 2009). It remains unclear why differences in Neuroticism among O-BD and O-MD did not extend to our comparison group, though one reason might be that our sample size was considerably reduced when unipolar and bipolar mood diagnoses were considered independently. A larger sample size may have been able to detect more robust differences in offspring personality across the three risk groups.

When we examined the effect of maternal diagnosis on offspring Neuroticism more closely, we found that group differences were largely driven by the relationship between maternal bipolar disorder and increased offspring Withdrawal. This stands in contrast to findings by Farchione and colleagues (2007), who reported increased hostility and irritability (constructs more closely related to Volatility) among O-BD, though their study did not examine traits related to Withdrawal. Further research is needed to establish whether the relationship between maternal bipolar disorder and offspring Neuroticism is part of a broader domain effect or is more specific to one of Neuroticism's lower-order aspects.

It was also somewhat notable that we detected no differences in Neuroticism when comparing O-MD to O-HC, given that other studies have repeatedly documented increased Neuroticism and negative emotionality in children of mothers diagnosed with unipolar depression (Davis et al., 2007; Feldman et al., 2009; Melchior et al., 2012; Whiffen & Gotlib, 1989). One reason for this discrepancy may be related to offspring age at the time of personality assessment. Each of the previous studies showing heightened Neuroticism among O-MD assessed offspring temperament or personality in the first year of life. In contrast, studies assessing offspring individual differences any time after infancy have typically failed to find reliable differences between O-MD and O-HC on Neuroticism or negative emotionality. In early work within our own sample, Radke-Yarrow (1998) reported no significant differences between the three risk groups in "problem temperament" (a construct that likely combines high negative emotionality and low positive emotionality) during early childhood. Two other studies by Durbin and colleagues (2005) and Olino and colleagues (2011) likewise reported no significant differences in Neuroticism between O-MD and O-HC when personality was measured after the conclusion of infancy. Research in normative development has shown that children's negative emotionality tends to decrease gradually over the course of early, middle, and late childhood, while self-regulatory processes tend to increase over these same time periods (Murphy, Eisenberg, Fabes, Shephard, & Guthrie, 1999). Taken together, these findings suggest that O-MD may exhibit a developmental pattern characterized by high early negative emotionality in the first year of life, followed by compensatory decreases in negative emotionality over the course of childhood, perhaps

coinciding with the emergence of protective self-regulatory capacities. Future research would do well to consider this hypothesis in prospective longitudinal studies that include repeated measures, with an emphasis on examining early moderators (e.g., parenting, attachment, emergence of self-regulatory processes) that might identify at risk offspring for whom patterns of high negative emotionality are likely to persist.

Previous research investigating the relationship between parental mood disorders and offspring Extraversion has yielded largely mixed findings. In the present study, we found no significant differences in Extraversion, regardless of maternal diagnostic status. Null findings reported here contradict two previous studies, both of which reported decreased Extraversion among O-MD (Durbin et al., 2005; Olino et al., 2011). There are two potential reasons for this discrepancy. First, both of the prior studies showing a significant relationship between maternal depression and offspring Extraversion assessed offspring personality during the early to middle childhood years, whereas our own study did not assess personality traits until adolescence. It is possible that dispositional differences in positive emotionality gradually decline as O-AD age into adolescence and young adulthood, though more research is needed to confirm this hypothesis. Secondly, choice of measurement may have played a role in our inability to detect group differences in Extraversion. Both Durbin and colleagues (2005) and Olino and colleagues (2011) employed measures that highlight the positive emotionality component of Extraversion, whereas the SNAP version of Extraversion emphasizes facets like sociability and expressiveness. Indeed, when Durbin and colleagues examined the relationship between maternal depression and lower-order Extraversion facets, they found that O-MD were

lower than O-HC on engagement and positive affect, but no different on sociability. In light of these findings, our results appear to indicate that the effect of maternal depression on offspring Extraversion is constrained to the lower-order trait of positive emotionality.

Data from the current study likewise did not support our hypothesis that O-BD would score more highly on measures of Extraversion than offspring of mothers with major depression or no depressive diagnosis. This is not altogether surprising, as research on this topic has been inconsistent at best. Several recent studies report no relationship between maternal bipolar mood diagnoses and offspring Extraversion, particularly when more common personality measures are used (Jones et al., 2006; Nijjar et al., 2014; Rothen et al., 2009). Other studies looking at shyness, which can be considered a lower-order component of Extraversion (reversed), likewise show no differences between O-BD and O-HC (Doucette et al., 2013; Duffy et al., 2007). Studies that do report differences between O-BD and O-HC on Extraversion-related traits tend to use the DOTS-R temperament scale (e.g., Chang et al., 2003; Singh et al., 2008), but even these reports seem to disagree on which facets are most closely linked to the presence of a maternal bipolar diagnosis. Research employing comprehensive trait measures comprised of multiple lower-order facet scales would be helpful in clarifying whether or not familial risk for bipolar disorder is related to the various traits that makeup the broader Extraversion domain.

In general, results of the present study yielded few relationships between maternal affective disorders and individual differences in offspring personality traits. It is possible that the presence of a maternal mood disorder alone is not sufficient to affect pervasive

personality change in at risk offspring. This hypothesis is consistent with the findings of

Durbin and colleagues (2005), who noted that only maternal, as opposed to paternal,

depression was related to low positive emotionality among offspring. This suggests that

the effect of parental depression on offspring personality can be attributed to a confluence

of factors beyond heritability. Indeed, maternal negativity and criticism, insecure

attachment relationships, interparental violence, and an array of other environmental

factors all interact with maternal psychopathology to influence individual differences in

offspring behavior (Meyer et al., 2004; Gravener et al., 2011; Cicchetti, Rogosch, &

Toth, 1998; Narayan, Chen, Martinez, Gold, & Klimes-Dougan, 2015). Future research

should examine how these factors and others interact with maternal depressive disorders

in order to shape offspring personality development.

Maternal Affective Disorder and Offspring Cognitive Vulnerability

Investigations into the role of cognitive vulnerabilities in the development of

depressive disorders are increasingly prevalent within the field of developmental

psychopathology (for a review, see Abela & Hankin, 2008). However, very little is

currently known about the etiology and development of cognitive vulnerabilities in at risk

youth. The present study aimed to address this gap in the literature by investigating

whether the presence of a maternal affective disorder is associated with increased risk for

maladaptive attributional biases among offspring. Contrary to our hypotheses, maternal

mood disorder (regardless of whether bipolar and unipolar diagnoses were considered

together or independently) was not related to offspring attributional biases in the current

sample. This finding contradicts several previous studies showing that both O-MD and

O-BD exhibit more negative attributional styles than O-HC (Garber & Flynn, 2001;

Garber & Robinson, 1997; Jaenicke et al., 1987). It remains somewhat unclear why our

results did not replicate extant findings in this area. Both the demographic characteristics

and sample size of past studies were comparable to our own (N = 240 in Garber & Flynn,

2001 and Gaber & Flynn, 1997, N = 84 in Jaenicke et al., 1987). It is tempting to attribute

the discrepancy to measurement problems, given that the CASQ demonstrates low

internal consistency both in our sample and in the previous literature. Some caution is

warranted with this explanation however, as all three of the previous studies reporting

significant associations between a history of maternal affective disorder and offspring

attributions also employed the CASQ. Nonetheless, studies utilizing the CASQ are

underpowered in detecting effects, and the null findings we report here may be

characteristic of a broader pattern of inconsistency obtained when researchers use this

measure (Abela & Hankin, 2008).

      Another possibility is that additional risk factors need to accompany maternal

psychopathology in order to promote the development of negative inferential styles

among offspring (Cicchetti, Rogosch, & Toth, 1994). For example, Abela and Hankin

(2008) recently reviewed the literature on cognitive vulnerability and offered three

potential sources for maladaptive attributional biases in children, including 1) the

internalization of chronic and/or traumatic negative life events (e.g., maltreatment,

interparental conflict), 2) the reinforcement of maladaptive inferences about the world by

harsh or critical parenting practices, and 3) the modeling of parental attributional biases.

It is possible that the effect of maternal depression on offspring attributions is contingent

upon the presence of one or more of these developmental risk factors, and future research would do well to consider whether these variables mediate the relationship between maternal psychopathology and offspring cognitive vulnerability.

Risk for Depression and Mania in O-AD

The primary objective of this study was to examine whether individual differences in offspring personality and cognitive style interact with a history of maternal mood disorder to predict offspring depression and mania, both concurrently and prospectively. Rates of both unipolar and bipolar depression are elevated in offspring of mothers with affective diagnoses (Findling et al., 2005; Lapalme et al., 1997; Tully, Iacono, & McGue, 2008; Weissman et al., 2006). Somewhat unexpectedly, our own results indicated less robust associations between maternal affective diagnoses and offspring affective symptoms. Maternal psychopathology emerged as a significant predictor of concurrent depressive symptoms in adolescence, but was not significantly associated with adolescent symptoms of mania or either form of affective symptoms in young adulthood. One reason that maternal diagnoses may have been related to concurrent, but not prospective, depressive symptoms is that offspring who developed a depressive disorder at T4 may have sought treatment prior to the T5 assessment, leading to treatment-mediated decreases in depressive symptoms. With regards to mania, it is unclear why we found no association between maternal diagnoses and offspring symptoms. Other studies have reported that maternal negativity, but not diagnosis, significantly predicts offspring bipolar disorder (Meyer et al., 2004), suggesting that the effect of maternal diagnosis on manic symptoms may be mediated by parental affect. It is

also possible that our sample size was not sufficiently powered to detect group differences in manic symptoms given the relatively low base rates of bipolar disorder in adolescence and young adulthood (Lewinsohn, Klein, & Seeley, 2000).

Contrary to expectations, the present study found no relationship between childhood attributional biases and offspring affective symptoms in adolescence and young adulthood. Moreover, interactions between attributional biases and maternal diagnoses or offspring personality traits yielded no significant findings. These results were somewhat surprising given the large body of research showing that negative attributional biases are important vulnerability factors for depressive symptoms (see Abela & Hankin, 2008 for a review). Nonetheless, results in this literature are heterogeneous, and several studies report no association between attributional biases and depression (Abela & Sarin, 2002; Bennett & Bates, 1995; Spence Sheffield, & Donovan, 2002). In fact, our study appears to directly replicate the results of Hammen, Adrian, and Hiroto (1988) who likewise found that attributional styles did not predict prospective depressive symptoms in children of mothers diagnosed with either unipolar or bipolar depression. There are several viable explanations for the inconsistency across studies. First, studies reporting null findings typically include samples of younger adolescents (around age 12), as was the case in this study (average age at T3 = 11.11). It is possible that offspring inferential styles are not fully stable or internalized until the later adolescent years (Shiner & Caspi, 2012). Another possibility is that attributional tendencies of offspring changed between the T3 assessment and the T4/T5 assessments, when affective symptoms were assessed. Abela and Hankin's review of extant literature

on attributional biases and depressive symptoms shows that previous studies have typically conducted symptom follow-ups within a few months to a year from when attributional biases are measured, whereas our own follow-up was on average about four years after attributions were initially assessed. Longitudinal studies with repeated measures of offspring attribution are needed to clarify the stability of children's inferential style across developmental periods.

Results of the present study are partially consistent with previous research examining the relationship between personality and the development of affective symptoms. Consistent with our hypotheses, offspring Neuroticism predicted depressive symptoms both concurrently during adolescence and prospectively in young adulthood, even after controlling for previous symptoms levels. These findings are largely consistent with developmental research showing that high Neuroticism during childhood and adolescence predicts the emergence of later depressive symptoms (e.g., Lonigan, Phillips, & Hooe, 2003; Klimstra et al., 2010; Wetter & Hankin, 2009). We also found that the presence of a maternal mood disorder interacts with offspring Neuroticism during adolescence to increase risk for depressive symptoms in young adulthood. Consistent with transactional accounts of development (e.g., Cicchetti & Lynch, 1993; Cicchetti & Toth, 1998), the moderating influence of maternal mood disorder suggests a need to consider dynamic interactions between parental and offspring characteristics in predicting later offspring psychopathology. For instance, it is possible that high Neuroticism shapes the way in which at-risk offspring construe or represent events occurring in their environment, such that maternal parenting or affect may have more deleterious

consequences for the development of these youth. On the other hand, it is also possible that high Neuroticism among at-risk offspring elicits greater maternal negativity or criticism. Future research should extend these findings by considering these and other mechanistic processes by which parental psychopathology and offspring personality interact to influence the emergence of depressive illness among offspring.

Models investigating the relationship between depressive symptoms and the lower-order aspects of both Neuroticism and Extraversion only partially confirmed our hypotheses. As anticipated, high Withdrawal was associated with increased depressive symptoms during both adolescence and young adulthood. This is consistent with previous studies showing that Withdrawal is related to diagnoses of unipolar depression (Johnson, Turner, & Iwata, 2003; Quilty MacKew, & Bagby, 2014; Quilty et al., 2013). Somewhat surprisingly, Volatility also predicted depressive symptoms prospectively, even after accounting for its shared variance with Withdrawal. It is possible that the effect of Volatility on depressive symptoms may be accounted for by irritability, which is central to both constructs. Indeed, a recent study by Stringaris, Cohen, Pine, and Leibenluft (2009) showed that irritability during childhood significantly predicted the emergence of major depression nearly 20 years later.

Contrary to our expectations, neither low Extraversion nor its lower-order aspect, Enthusiasm, were significantly related to depressive symptoms in this sample, though effects were consistently in the anticipated direction. Meta-analytic work by Kotov and colleagues (2010) has shown that the relationship between depressive illness and Extraversion is inconsistent, and if there is a relationship, it is likely to be quite modest in

nature. Interestingly, that same study reported stronger links between low Extraversion

and dysthymia, suggesting that the relationship between Extraversion and depression may

vary based on the persistence or heterogeneity of symptoms. Finally, the preponderance

of evidence linking childhood Extraversion to later depression has employed measures

that tend to emphasize the positive emotionality facet of Extraversion (e.g., Dougherty,

Klein, Durbin, Hayden, & Olino, 2010; Lonigan et al., 2003; Naragon-Gainey et al.,

2009), suggesting that the link between Extraversion and depression may be driven by a

facet-level effect that is obscured when the broad domain or even aspect levels are

examined.

Results of our models predicting manic symptoms in adolescence and adulthood

were similarly mixed. At the level of the Big Five, both Extraversion and Neuroticism

positively and significantly predicted concurrent manic symptoms, which was consistent

with expectations. This aligns with a wide body of literature showing links between

Neuroticism and bipolar disorder in adults (Barnett et al., 2011; Jylhä et al., 2010; Quilty

et al., 2009). Moreover, it seems to provide additional evidence in support of the BAS

model of bipolar disorder (Alloy & Abramson, 2010). However, it is important to note

that only Neuroticism, and not Extraversion, predicted prospective symptoms of mania. It

is unclear why the effect of Extraversion did not persist into young adulthood. One

possibility is that our T5 assessment of mania was insufficient to detect subtle differences

among offspring, as we used only a simple symptom count with low sensitivity to

subclinical symptoms. It may also be the case that the dimensional measures used during

the T4 assessment were more sensitive to aspects of mania that are more closely related to Extraversion than Neuroticism (e.g., euphoria, activity).

Finally, models examining the relationship between affective symptoms and the lower-order aspects of Neuroticism largely diverged from our hypotheses. Based on previous findings, we anticipated that Volatility, as opposed to Withdrawal, would be differentially related to offspring mania (Quilty et al., 2013; Urošević et al., 2008). In contrast, the current study found that Withdrawal, and not Volatility, was associated with offspring manic symptoms. Moreover, there was a trend for the presence of a maternal mood disorder to increase risk for manic symptoms at T5 when offspring were also high on Withdrawal. These results should be interpreted with considerable caution given their divergence from previous research. One possible explanation for these findings may be that our measures were capturing symptoms of mania that are also present in internalizing disorders, such as irritability, difficulties with sleep, psychomotor agitation, and distractibility. Future research may want to consider whether different clusters of manic symptoms show distinct relationships with various personality traits.

Limitations

Several limitations of the current study are worth noting. First, our analysis was limited by our modest sample size. For our main analyses, maternal mood disorders needed to be considered together (as opposed to considering unipolar and bipolar diagnoses independently) in order to retain adequate power to detect interactions between maternal diagnosis and offspring individual differences. Even with this accommodation, the present study was likely somewhat underpowered given that the average effect size in

personality research has been estimated at only .21 (Richard, Bond, & Stokes-Zoota, 2003). As a result, non-significant findings involving offspring individual differences and their interaction with maternal diagnoses should be considered with some caution.

A second important limitation involves the low variability in manic symptoms observed at Time 5, which is consistent with a previous report in this sample showing that only 9 offspring were diagnosed with bipolar disorder at the time of the young adulthood assessment (Meyer et al., 2004). The low incidence of bipolar disorder and mania reported here is consistent with studies showing the base rate of childhood and adolescent bipolar disorder is only about 1% in the general population (Lewinsohn et al., 2000; Moreno et al., 2007). It is unlikely that our sample was large enough to detect group differences in mania given this prevalence rate. Another contributing factor is that our only measure of manic symptoms in young adulthood was a symptom count from the SCID, which is likely to be less sensitive to detecting subclinical manic symptoms than other dimensional measures (e.g., the Y-MRS).

Third, our evaluation of offspring cognitive vulnerability was likely limited by the low internal consistency of the CASQ. Previous research has also reported low reliability of the CASQ (Nolen-Hoeksema, Girgus, & Seligman, 1986, 1992; Panak & Garber, 1992), suggesting that the items comprising both the overall composite scale and its subscales may not be measuring well-defined latent constructs (Tavakol & Dennick, 2011). Readers should therefore treat our null findings with regards to attributional biases with considerable caution. Newer and more psychometrically sound measures of attributional biases are now available in the literature (e.g., the CASI, Conley, Haines,

Hilt, & Metalsky, 2001), and future studies should consider using these measures in lieu of the CASQ.

Finally, exploratory factor analysis in the CLPS sample failed to recover a meaningful Assertiveness subscale from the SNAP, which meant we were unable to evaluate our hypotheses for that trait. While many SNAP items appear to reflect Assertive tendencies, our analyses showed that a number of promising items were confounded by content related to traits like Enthusiasm, Industriousness, or Politeness, resulting in exclusionary cross-loadings. Nonetheless, current research suggests that there may be at least three distinct components of the Behavioral Approach System (Assertiveness, Enthusiasm, Sensation-Seeking; Quilty, DeYoung, Oakman, & Bagby, 2014). Future studies should consider including measures of personality that assess these lower-order approach components in order to increase understanding into how lower-order components of the BAS may be related to the development and course of mania.

Future Directions

The current study adds to the limited literature examining how individual differences in offspring behavior, affect, and cognition influence the transmission of psychopathology from mother to child. However, comprehensive models that seek to understand the influence of maternal mood disorders on offspring outcomes must include additional predictors beyond maternal diagnosis. Cicchetti and Toth (1998) have argued that a developmental psychopathology perspective approach to offspring development in the context of maternal depression requires an ecological transactional model that incorporates dynamic and reciprocal influences among environmental forces, familial

stress and conflict, caregiver characteristics, and offspring individual differences. Incorporating all of these factors would be beyond the scope of any single study. The lack of robust relationships between maternal affective diagnoses and offspring personality reported in this study points to a need for research that examines more distal factors (e.g., parenting behavior, marital conflict, and contextual risk) that may moderate or mediate the relationship between maternal psychopathology and offspring personality development.

Future studies should also consider assessing personality traits and psychopathology at multiple time points and using multiple informants (Shiner & Allen, 2013). Longitudinal personality assessment would be particularly helpful for two reasons. First, personality is relatively stable but also malleable, especially early in life, and early patterns of personality change and development in youth have been linked to a range of adult outcomes (Roberts & DelVecchio, 2000). Second, the current study represents a test of the vulnerability or predisposition model of personality and psychopathology, in which personality may increase risk for the development of later pathology (Krueger & Tackett, 2003). However, repeated measures of both personality and psychopathology would allow for more formal testing of other personality-pathology models including complication/scar models, in which episodes of psychopathology have lasting impacts on personality, and pathoplasty models, in which personality influences the course, duration, and severity of psychopathology.

We chose to focus our hypotheses on the personality traits of Extraversion and Neuroticism (and their lower-order aspects), given the abundance of research tying these

traits to maternal affective disorders and the development of depression and mania. Exploratory analyses evaluating whether maternal diagnoses predicted Agreeableness and Conscientiousness yielded no significant findings here. Nonetheless, studies that assess additional personality traits at the domain, aspect, and facet levels are likely to add to our understanding of offspring development in the context of maternal mood disorder. For example, a recent study by Narayan, Allen, Cullen, and Klimes-Dougan (2013) found that O-BD are more likely to experience disturbances in reality testing (DRTs) than their O-MD and O-HC counterparts, and DRTs have been linked to pathological forms of the personality trait Openness (DeYoung, Grazioplene, & Peterson, 2012). A number of other studies likewise show relationships between affective symptoms and Agreeableness, Openness, and Conscientiousness (especially the Achievement Striving facet; Lozano & Johnson, 2001; Quilty et al., 2009, 2013). Additionally, future studies should consider the possibility that personality effects are multiplicative in nature, meaning that the presence of one trait may interact with the presence of another to further modulate risk for psychopathology (e.g., Vasey et al., 2014).

Finally, future research should examine our hypotheses regarding attributional biases using measures with psychometric qualities that are superior to those of the CASQ. It is also important to note that cognitive vulnerability is a broad concept that extends beyond attributional biases. For instance, Beck's (1967, 1983) theory of depression emphasizes the influence of depressogenic schemas consisting of negative attitudes of the self, the world, and the future. Nolen-Hoeksema's (1991) response styles theory of depression has likewise pointed to maladaptive coping strategies as important markers of

vulnerability to depression, and results from our own sample suggest there may be subtle

differences in coping strategies between O-AD and O-HC (Klimes-Dougan & Bolger,

1998). Other research shows that bipolar disorder is closely associated with cognitive

styles characterized by perfectionism, autonomy, self-criticism, and self-consciousness

(Alloy et al., 2006). Longitudinal studies in which psychopathology and an array of

cognitive biases are assessed at repeated assessments would be a useful way to extend

cognitive theories of depression to at-risk offspring samples.

Closing Remarks

In conclusion, the results of the present study provide evidence that maternal

affective diagnoses interact with offspring characteristics to predict the emergence of

affective pathology during the transition from adolescence to young adulthood.

Specifically, offspring of mothers diagnosed with an affective disorder who also exhibit

high levels of negative emotionality during adolescence may be at greatest risk for

developing adulthood depression. Yet, even in the absence of maternal psychopathology,

individual differences in offspring personality appear to influence risk for the

development of psychopathology. These results extend our understanding of the

relationship between early experience, offspring individual differences, and

developmental outcomes. Future studies should build on these findings by examining

additional risk factors associated with maternal affective disorders that may likewise

shape offspring personality development. Findings reported here may be helpful in

guiding translational efforts to identify offspring at greatest risk, personalize intervention

based on offspring characteristics, and predict which offspring are most likely to respond

to treatment. Links between youth personality and affective symptoms reported in this study may likewise guide research interested in understanding the etiology and developmental trajectory of depressive disorders. Taken together, these results provide a strong foundation to research on personality differences in at risk offspring. Longitudinal studies that include repeated measurements of offspring individual differences across multiple levels of analysis would represent a promising next step to understanding the developmental pathways by which maternal depressive illness confers risk to offspring.

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Table 1

Demographic Characteristics of Control, Bipolar, and Depressed Groups

Child Characteristics	Control	Bipolar Disorder	Major Depression
Number of Participants	60	48	84
Cohort 1 (%)	30 (50%)	26 (54%)	42 (50%)
Female (%)	30 (50%)	29 (60%)	45 (54%)
Race (%)			
Asian	2 (3%)	2 (4%)	0
Black	6 (10%)	4 (8%)	12 (14%)
White	52 (87%)	42 (88%)	70 (83%)
Hispanic	0	0	2 (2%)
Mean Age Time 1 (SD)	4.37 (1.88)	4.50 (2.06)	4.49 (2.19)
Mean Age Time 2 (SD)	7.21 (1.91)	7.23 (2.11)	7.57 (2.22)
Mean Age Time 3 (SD)	11.18 (2.12)	10.76 (2.29)	11.26 (2.34)
Mean Age Time 4 (SD)	15.57 (2.69)	15.01 (2.54)	16.23 (2.59)*
Mean Age Time 5 (SD)	22.07 (2.64)	21.35 (2.41)	22.70 (2.55)*
Maternal Characteristics			
Maternal Age Time 1 (SD)	32.63 (4.43)	32.25 (3.44)	32.71 (4.89)
Mean GAF Time 3 (SD)	79.48 (8.24)	57.02 (13.72)	64.76 (10.54)
Time 1 Highest Education			
Less than High School	0	0	4 (5%)
High School Degree	8 (13%)	10 (21%)	18 (21%)
Partial College	10 (17%)	12 (25%)	16 (19%)
College Graduate	22 (37%)	16 (33%)	28 (33%)
Graduate Degree	20 (33%)	10 (21%)	18 (21%)
Family Characteristics			
Living Arrangement Time 1			
Biological Parents (%)	60 (100%)	43 (90%)	78 (93%)
Mother Only (%)	0	5 (10%)	6 (7%)
Mean SES Time 1 (SD)	56.40 (10.51)	50.08 (14.23) †	47.67 (16.76)†
Range	27 to 66	18 to 66	11 to 66

†Different from control only at *p* <.05, *Different from BD only at *p* < .05, ** Different from both other groups at *p* < .05; Abbreviations: SES = socioeconomic status, GAF = Global Assessment of Functioning (low scores indicate poor functioning).

Table 2

Final SNAP Aspect Scales, Correlations with NEO-BFAS, and Factor Loadings

Aspect Scale	NEO-BFAS r	CLPS Factor Loading	Offspring Factor Loading
Volatility			
17. Rarely get so angry that I lose control. [R]	.482	.652	.788
109. Very hard to make me angry. [R]	.523	.506	.624
141. Have a violent temper.	.454	.559	.855
153. Have no trouble controlling my anger. [R]	.557	.702	.575
165. Temper sometimes gets me into trouble.	.583	.734	.833
194. Become angry more easily than most people.	.579	.717	.908
252. Anger often gets the better of me.	.619	.709	.880
274. Often take my anger out on those around me.	.484	.494	.749
Withdrawal			
65. Sometimes get so upset I feel like hurting myself.	.367	.318	.448
75. Mostly concerned about pleasing others when deciding.	.306	.441	.325
81. Feel a strong need to have others approve of me.	.366	.531	.512
123. Check with others to see if what I'm doing is ok.	.271	.398	.390
248. Often feel nervous and "stressed."	.424	.482	.773
250. Find myself worrying about things.	.414	.588	.776
281. Worry about things I have done or said.	.358	.569	.752
288. Life seems pretty confusing to me.	.341	.395	.616
303. Feel personally or socially inadequate.	.455	.408	.705
304. My feelings are hurt rather easily.	.378	.543	.724
311. Often troubled by guilt feelings.	.403	.548	.686
316. Worry about terrible things that might happen.	.398	.465	.707
325. Life often feels like just a big struggle.	.409	.410	.669
340. Worry if I'll be able to handle things when alone.	.326	.444	.610
350. Too sensitive for my own good.	.381	.504	.683
Enthusiasm			
9. Keep to myself even when others are around.[R]	.455	.525	.856
22. Don't like spending time with other people. [R]	.555	.659	.909
35. Prefer not to have people around me. [R]	.492	.611	.859
61. Would rather be with a friend than alone when unhappy.	.297	.459	.756
74. Get a warm feeling when with a good group of friends.	.461	.541	.631
101. Spend free time with friends/family rather than alone.	.427	.594	.856
140. I am more of a loner than most people. [R]	.462	.605	.765
158. Enjoy working with people more than working alone.	.440	.547	.735
196. Keep a distance between myself and others. [R]	.450	.603	.778
218. Am a "people person."	.518	.535	.676
284. Takes a lot to get me excited. [R]	.393	.396	.496
319. Often playful around other people.	.473	.474	.527
Politeness			
25. Would not use others' weaknesses to my advantage.	.424	.507	.624
76. Fool people into doing what I want them to do. [R]	.509	.537	.603
91. Lying comes easily to me. [R]	.349	.603	.865
102. Willing to "bend the truth" if it will benefit me. [R]	.363	.571	.859
105. It is fun to take advantage of others' weak points. [R]	.328	.354	.660^
117. Done things for which I could have been arrested. [R]	.287	.387	.598
119. I am good at getting others to do my work. [R]	.330	.441	.537
129. Will step on others' toes a little if it helps me out. [R]	.446	.551	.748

200. Would never hurt other people just to get what I want.	.353	.333	.420^
208. Enjoy bending the rules and getting away with it. [R]	.325	.488	.628
232. I have stolen things from time to time. [R]	.269	.489	.593^
353. Tell a lot of lies. [R]	.317	.504	.799
Industriousness			
29. When I start a task, I am determined to finish it.	.486	.536	0.611
247. Often stop one activity to start another. [R]	-.336	.336	0.763
279. Put a lot of energy into everything I do.	.423	.397	0.381
351. Fail to finish things, even when I know I could. [R]	-.520	.708	0.646
367. Put things off and sometimes miss deadlines. [R]	-.519	.448	0.488^

Note: ^Item did not have its highest loading on the intended aspect during external validation, so it was not included in composite scales used in the at risk offspring sample. Abbreviations: CLPS = Collaborative Longitudinal Personality Disorders Study; SNAP = Schedule for Nonadaptive and Adaptive Personality; NEO-BFAS = Big Five Aspect Scales of the NEO-PI-R.

Table 3

Descriptive Statistics for the CASQ, SNAP Trait, and Psychopathology Measures

	Offspring Sample				CLPS Sample		
SNAP Aspect	Mean	SD	α	ICC	Mean	SD	α
Volatility	1.30	.30	.83	.27	1.43	.34	.85
Withdrawal	1.42	.25	.82	.25	1.72	.22	.81
Enthusiasm	1.78	.24	.84	.19	1.45	.29	.84
Politeness	1.71	.26	.78	.22	1.71	.24	.79
Industriousness	1.63	.33	.63	.14	1.50	.31	.63
SNAP Big Four							
Neuroticism	1.40	.27	.84	.27	1.73	.23	.81
Extraversion	1.71	.24	.83	.15	1.40	.27	.84
Conscientiousness	1.64	.22	.81	.15	1.67	.21	.80
Agreeableness	1.77	.22	.85	.25	1.75	.23	.84
CASQ							
Attributional Style	5.59	4.08	.43	.18			
T4 Depression				.30			
CBCL AP	.17	.22					
DICA Count	2.60	2.60					
SCL-90	43.16	9.33					
T4 Mania				.18			
ISS	.44	.27					
Y-MRS	3.25	3.92					
DICA Count	1.00	1.45					
T5 Depression				.33			
BDI	4.15	6.14					
SCID Count	.58	1.76					
T5 Mania				.15			
SCID Count	.14	.79					

Note: Abbreviations: CLPS = Collaborative Longitudinal Personality Disorders Study; SNAP = Schedule for Nonadaptive and Adaptive Personality; SD = Standard Deviation; ICC = Intraclass Correlation Coefficient; T4 = Time 4; T5 = Time 5; CBCL AP = Child Behavior Checklist Affective Problems Subscale; DICA Count = Diagnostic Interview for Children and Adolescents, depression and mania symptom counts, respectively; SCL-90 = Symptom Checklist-90; ISS = Internal State Scale; Y-MRS = Young Mania Rating Scale; BDI = Beck Depression Inventory; SCID Count = Schedule Clinical Interview for DSM, symptom counts for depression and mania, respectively.

Table 4

Correlations among SNAP and NEO Personality Traits Scales in the CLPS

Scale	SNAP Volatility	SNAP With	SNAP Enthus	SNAP Polite	SNAP Indust
SNAP Extraversion	**-.136**	**-.232**	**.901**	.013	**.102**
SNAP Neuroticism	**.371**	**.854**	**-.250**	**-.108**	**-.171**
SNAP Agreeableness	**-.620**	**-.083**	**.188**	**.742**	**.082**
SNAP Conscientiousness	**-.090**	**-.092**	.064	**.263**	**.611**
NEO BFAS Extraversion	**-.090**	**-.237**	**.669**	-.030	**.207**
NEO BFAS Neuroticism	**.600**	**.638**	**-.267**	**-.138**	**-.209**
NEO BFAS Agreeable	**-.307**	**.082**	**.253**	**.577**	.063
NEO BFAS Conscientious	.006	**-.132**	.020	**.136**	**.657**
NEO BFAS Openness	**-.091**	-.020	**.130**	-.013	.016
NEO Volatility	**.769**	**.368**	**-.183**	**-.257**	**-.112**
NEO Withdrawal	**.128**	**.704**	**-.258**	**.071**	**-.241**
NEO Enthusiasm	**-.191**	**-.198**	**.755**	.070	**.086**
NEO Assertiveness	.039	**-.206**	**.383**	**-.122**	**.268**
NEO Politeness	**-.324**	.057	**.084**	**.638**	.059
NEO Compassion	**-.139**	.069	**.311**	**.231**	.036
NEO Industriousness	**-.115**	**-.289**	**.116**	**.183**	**.722**
NEO Orderliness	**.151**	**.111**	**-.105**	.025	**.323**
NEO Openness (Aspect)	-.057	**.087**	**.149**	-.040	**-.100**
NEO Intellect	**-.099**	**-.109**	**.083**	.013	**.115**

Bolded values indicate significance at p < .05.

Note: Abbreviations: CLPS = Collaborative Longitudinal Personality Disorders Study; SNAP = Schedule for Nonadaptive and Adaptive Personality; NEO BFAS = NEO Big Five Aspect Scales; With = Withdrawal; Enthus = Enthusiasm; Polite = Politeness; Indust = Industriousness.

Table 5

Correlations among Maternal Diagnosis, Personality Traits, Attributional Style, and Psychopathology in the Offspring Sample

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Maternal Diagnosis	---														
2. Neuroticism	.041	---													
3. Extraversion	-.077	-.063	---												
4. Conscient	-.083	-.067	**.181**	---											
5. Agreeable	-.096	**-.260**	**.144**	**.288**	---										
6. Volatility	.082	**.386**	**-.201**	**-.314**	**-.643**	---									
7. Withdrawal	.074	**.860**	-.037	.068	**-.225**	**.276**	---								
8. Enthusiasm	-.043	-.078	**.898**	**.145**	**.156**	**-.200**	-.056	---							
9. Politeness	-.070	**-.252**	.082	**.414**	**.680**	**-.385**	**-.188**	.098	---						
10. Industrious	-.106	**-.193**	**.219**	**.655**	**.303**	**-.276**	-.137	**.198**	**.320**	---					
11. Attribution Style	-.073	-.120	.080	.033	**.236**	-.085	**-.148**	.088	**.217**	.030	---				
12. T4 Depress	**.220**	**.429**	-.163	-.119	**-.193**	**.237**	**.439**	**-.146**	**-.229**	**-.170**	**-.167**	---			
13. T4 Mania	.123	**.347**	.096	-.028	**-.167**	**.184**	**.294**	.064	**-.177**	-.028	-.064	**.462**	---		
14. T5 Depress	.120	**.453**	**-.181**	-.068	**-.193**	**.382**	**.430**	**-.157**	-.082	**-.158**	**-.144**	**.348**	**.156**	---	
15. T5 Mania	.076	**.196**	-.048	.074	-.050	.132	**.199**	-.028	.033	.033	-.015	.141	**.198**	**.338**	---

Bolded values are significant at p < .05.

Note: Depression and mania at T4 and T5 are composite variables created from standardized scores on symptom measures, as described in the text. Atribution style is a net overall measure of attributional bias (positive attributions - negative attributions). Maternal diagnosis is coded 0 = healthy comparison mothers, 1 = mothers with a diagnosis of either major depression or bipolar disorder. Abbreviations: T4 = Time 4, T5 = Time 5, Conscient = Conscientiousness, Agreeable = Agreeableness, Industrious = Industriousness, Depress = Depression.

Table 6

Regressions Predicting T4 Symptoms from Extraversion, Neuroticism, Maternal Diagnosis

	T4 Depression				T4 Mania		
	β	SE	B		β	SE	B
Gender	.023	.070	.035		.056	.067	.088
Age	.223**	.060	.066		.240**	.067	.071
IQ	.100	.078	.005		-.030	.076	-.002
Family SES	-.101	.093	-.005		-.060	.079	-.003
Maternal AD	.173**	.069	.288		.084	.073	.141
Extraversion	-.107	.074	-.336		.136*	.066	.432
Neuroticism	.424**	.074	1.225		.342**	.078	.993
R^2 Step 1			.028				.096
R^2 Step 2			.307				.220
R^2 Step 3			.310				.223
Wald Test							
Step 1	$\chi^2 (4) = 18.534, p = .001$				$\chi^2 (4) = 20.909, p = .001$		
Step 2	$\chi^2 (3) = 42.540, p < .001$				$\chi^2 (4) = 19.121, p < .001$		
Step 3	$\chi^2 (2) = .451, p = .798$				$\chi^2 (2) = .251, p < .882$		

† $p < .10$, * $p < .05$, ** $p < .01$

Note: Standard errors are for standardized regression coefficients. Maternal affective diagnosis is coded 0 = Healthy Comparison, 1 = Maternal Affective Disorder. Abbreviations: SE = standard error; T4 = Time 4, Maternal AD = Maternal affective diagnosis.

Table 7

Regressions Predicting T5 Symptoms from Extraversion, Neuroticism, Maternal Diagnosis

	T5 Depression				T5 Mania		
	β	SE	B		β	SE	B
Gender	-.103	.079	-.176		-.109	.070	-.199
Age	.031	.085	.010		.033	.075	.012
IQ	-.005	.087	.000		-.018	.072	-.001
Family SES	-.071	.096	-.004		-.050	.070	-.003
T4 Depression	.144†	.087	.156		---	---	---
T4 Mania	---	---	---		.145	.085	.168
Maternal Diagnosis	.055	.066	.098		.038	.054	.073
Extraversion	-.113	.071	-.391		-.032	.056	-.117
Neuroticism	.177	.110	.560		.159**	.060	.537
Maternal AD x Neuroticism	.273*	.107	.288		---	---	---
Maternal AD x Extraversion	---	---	---		---	---	---
R^2 Step 1			.024				.056
R^2 Step 2			.277				.083
R^2 Step 3			.304				.096
Wald Test							
Step 1	$\chi^2 (5) = 16.603, p = .005$				$\chi^2 (5) = 6.223, p = .285$		
Step 2	$\chi^2 (3) = 23.412, p < .001$				$\chi^2 (3) = 4.856, p = .183$		
Step 3	$\chi^2 (3) = 5.613, p < .018$				$\chi^2 (2) = 1.866, p = .393$		

† $p < .10$, * $p < .05$, ** $p < .01$

Note: Standard errors are for standardized regression coefficients. Maternal affective diagnosis is coded 0 = Healthy Comparison, 1 = Maternal Affective Disorder. Abbreviations: SE = standard error; T4 = Time 4, T5= Time 5, Maternal AD = Maternal affective diagnosis.

Table 8

Final Regression Models Predicting Concurrent Manic and Depressive Symptoms at Time 4

	T4 Depression			T4 Mania		
	β	SE	B	β	SE	B
Gender	.070	.067	.109	.119	.067	.185
Age	.217**	.059	.064	.227**	.067	.067
IQ	.112	.072	.006	-.035	.076	-.002
Family SES	-.100	.096	-.005	-.051	.082	-.003
Maternal AD	.142*	.069	.237	.059	.075	.100
Withdrawal	.384**	.079	1.168	.250**	.074	.764
Volatility	.122	.085	.309	.115	.083	.290
Enthusiasm	-.020	.091	-.062	---	---	---
Nonspecific E	-.075	.088	-.209	---	---	---
Attributional Bias	-.060	.069	-.011	.027	.080	.005
R^2 Step 1			.094			.096
R^2 Step 2			.330			.189
R^2 Step 3			.335			.198
R^2 Step 4			.349			.216
Wald Test						
Step 1	$\chi^2(4) = 18.53$, p = .001			$\chi^2(4) = 20.909$, p < .001		
Step 2	$\chi^2(6) = 68.76$, p < .001			$\chi^2(4) = 34.861$, p < .001		
Step 3	$\chi^2(3) = 1.098$, p = .778			$\chi^2(3) = .917$, p = .821		
Step 4	$\chi^2(2) = .057$, p = .972			$\chi^2(2) = 1.202$, p = .548		

† $p < .10$, * $p < .05$, ** $p < .01$

Note: Standard errors are for standardized regression coefficients. Maternal affective diagnosis is coded 0 = Healthy Comparison, 1 = Maternal Affective Disorder. Abbreviations: SE = standard error, T4 = Time 4, Maternal AD = Maternal affective diagnosis, Nonspecific E = Nonspecific Extraversion (see main text for variable explanation).

Table 9

Final Regression Models Predicting Depressive and Manic Symptoms Prospectively at Time 5

	T5 Depression			T5 Mania		
	β	SE	B	β	SE	B
Gender	-.040	.079	-.069	-.095	.067	-.174
Age	.057	.089	.019	.039	.078	.013
IQ	.018	.085	.001	-.019	.071	-.001
Family SES	-.052	.099	-.003	-.055	.073	-.003
T4 Depression	.114	.090	.123	---	---	---
T4 Mania	---	---	---	.141†	.082	.164
Maternal AD	.023	.067	.041	.035	.053	.066
Withdrawal	.315**	.086	1.042	.153*	.073	.543
Volatility	.241**	.106	.667	.050	.095	.152
Enthusiasm	.040	.097	.135	---	---	---
Nonspecific E	-.161†	.093	-.488	---	---	---
Attributional Bias	-.036	0.075	-.008	.039	.112	.008
R^2 Step 1			.147			.056
R^2 Step 2			.327			.095
R^2 Step 3			.344			.109
R^2 Step 4			.379			.134

Wald Test

	T5 Depression	T5 Mania
Step 1	$\chi^2(5) = 16.603, p = .005$	$\chi^2(5) = 6.223, p = .285$
Step 2	$\chi^2(6) = 31.501, p < .001$	$\chi^2(4) = 5.748, p = .219$
Step 3	$\chi^2(4) = 3.439, p = .487$	$\chi^2(3) = 2.655, p = .448$
Step 4	$\chi^2(3) = .221 \; p = .974$	$\chi^2(2) = .047, p = .977$

† p < .10, * p < .05, ** p < .01
Note: Standard errors are for standardized regression coefficients. Maternal affective diagnosis is coded 0 = Healthy Comparison, 1 = Maternal Affective Disorder. Abbreviations: SE = standard error, T4 = Time 4, T5 = Time Five, Maternal AD = Maternal affective diagnosis, Nonspecific E = Nonspecific Extraversion (see main text for variable explanation).
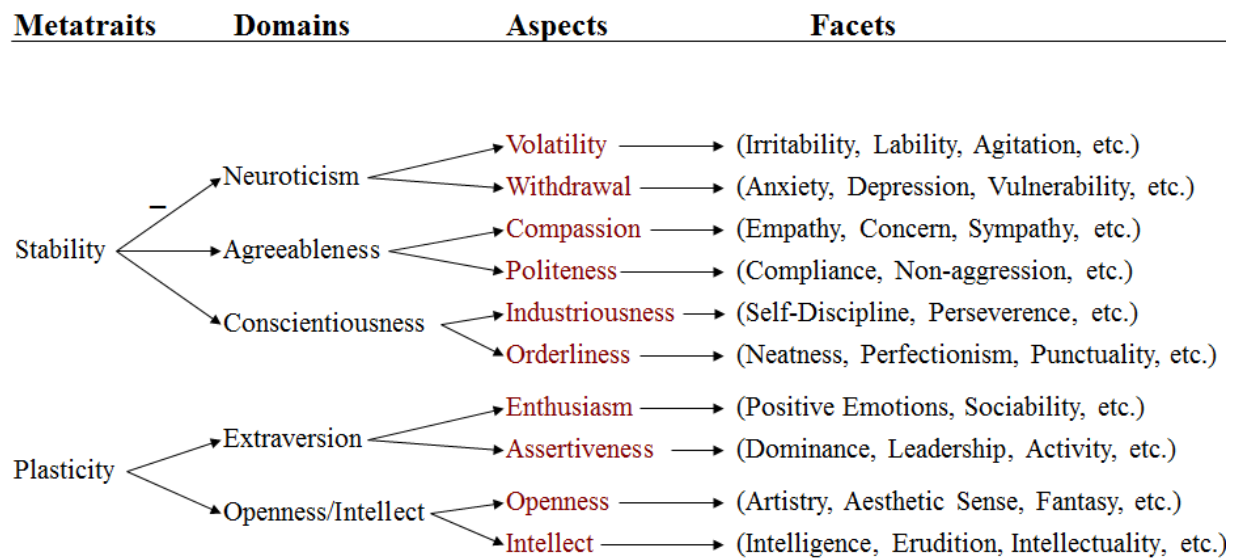
Figure 1

The Big Five Personality Hierarchy

Metatraits	**Domains**	**Aspects**	**Facets**

Stability — Neuroticism
- Volatility → (Irritability, Lability, Agitation, etc.)
- Withdrawal → (Anxiety, Depression, Vulnerability, etc.)

Agreeableness
- Compassion → (Empathy, Concern, Sympathy, etc.)
- Politeness → (Compliance, Non-aggression, etc.)

Conscientiousness
- Industriousness → (Self-Discipline, Perseverence, etc.)
- Orderliness → (Neatness, Perfectionism, Punctuality, etc.)

Plasticity — Extraversion
- Enthusiasm → (Positive Emotions, Sociability, etc.)
- Assertiveness → (Dominance, Leadership, Activity, etc.)

Openness/Intellect
- Openness → (Artistry, Aesthetic Sense, Fantasy, etc.)
- Intellect → (Intelligence, Erudition, Intellectuality, etc.)

Figure 2