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Associations Between Parental Psychopathology and Personality and Offspring Temperament: Implications for the Conceptualization of Temperament

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Early childhood temperament has been associated with both later psychopathology and personality. Temperament may also be the construct that is responsible for transmission of psychopathology or personality across familial generations. The present study examined the possibility that parental psychopathology and personality are related to offspring temperament. Lastly, a number of demographic/contextual factors that may moderate the relationship between parental characteristics and offspring outcomes were examined. These associations were examined in a large (n = 375) community based sample of three-year old children. Children participated in a series of laboratory activities that were designed to elicit emotional displays, including positive affect (PA), fear, sadness, anger, and behavioral inhibition, and other behavioral tendencies, including interest, sociability, and impulsivity.
Each parent completed a diagnostic interview to assess lifetime history of depressive and anxiety disorders. Each parent also completed self-report measures of personality and demographic/contextual factors. No significant main effects of maternal depressive or anxiety disorder on offspring temperament were found. Interactions between maternal depressive disorders and maternal employment and maternal depressive disorders and maternal age was predictive of offspring PA and interest, such that offspring of chronically depressed, non-working mothers had lower levels of PA and interest compared to offspring of non-working mothers without a history of depression. This is suggestive of increased risk for depressive disorders. For offspring of older working mothers, maternal chronic depression was associated with higher levels of offspring PA and impulsivity, suggesting that these children may be at risk for externalizing behavior problems. Few associations between parental personality constructs and offspring temperament were significant. Results suggest that the influence of maternal chronic depression on offspring temperament varies as a function of additional demographic/contextual factors. The results highlight the need to conduct more fine grained studies to identify the processes through which maternal age and employment influence the relationship between maternal depressive disorder and offspring risk.
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Introduction

Familial transmission of mood and anxiety disorders is well-established (Beardslee et al., 1998; Beidel & Turner, 1997; Biederman et al., 2001; Brennan et al., 2002; Downey & Coyne, 1990; Klein et al., 2005; Lieb et al., 2002; Merikangas, Lieb, Wittchen, & Avenevoli, 2003; Turner, Beidel, & Wolff, 1996). Numerous processes have been proposed to account for the transmission of these disorders (Goodman & Gotlib, 1999; 2002), but few have been studied in a fully systematic manner. One important variable that may be involved in the intergenerational transmission of mood and anxiety disorders is temperament (Clark, 2006; Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Klein, Durbin, Shankman, & Santiago, 2002; Nigg, 2006; Rothbart & Bates, 1998). From this perspective, parental psychopathology may be linked to offspring temperament, which may in turn be linked to the subsequent development of psychopathology in offspring. However, relatively few studies have empirically examined the initial parent psychopathology to offspring temperament association.

Temperament

Temperament refers to early emerging, stable, and heritable individual differences in emotional style or emotional reactivity (Buss & Plomin, 1975; Goldsmith, Buss, Plomin, Rothbart, Thomas, Chess, Hinde, & McCall, 1987; Goldsmith, Lemery, Buss, & Campos, 1999; Rothbart & Bates, 1998). Many investigators agree that temperament serves as the underpinnings of later personality (Caspi & Shiner, 2006; Shiner, 1998; Shiner & Caspi, 2003; Tackett, 2006). Two of the most commonly discussed dimensions of temperament and personality are positive emotionality (PE) and negative emotionality (NE), which make up two of the “big three” in personality (with the remaining dimension being constraint vs. disinhibition) (Watson, Clark, & Harness, 1994; Watson, Gamez, & Simms, 2005). These broadband
dimensions are commonly discussed in and share the same meaning in the personality, developmental, and psychopathology literatures (Caspi, Roberts, & Shiner, 2005; Caspi & Shiner, 2006; Nigg, 2006; Rothbart & Bates, 1998; Shiner & Caspi, 2003).

In the adult personality literature, PE is closely linked to extraversion. Support for the overlap between PE and extraversion has been noted in psychological theories of personality (McCrae & Costa, 1987; Tellegen, 1985) and longitudinal studies have found associations between early temperamental style and later personality (Caspi, 2000). PE includes tendencies to demonstrate high levels of positive affect (Tellegen, 1985; Watson, Wiese, Vaidya, & Tellegen, 1999), responsiveness to potential rewards (Klein, 1987; Lucas et al., 2000; Meehl, 1975), and engagement in social interactions (McCrae & Costa, 1987). Several theorists have proposed that each of these components reflects an underlying biobehavioral motivational system that has been referred to as the behavioral approach system (Davidson, 1998), behavioral activation system (Fowles, 1994; Gray, 1970), and behavioral facilitation system (Depue & Collins, 1999).

Negative emotionality (NE) is similar to the adult personality construct of neuroticism. It is a higher order factor that includes the tendency to experience and display sadness, fear, and anger, particularly in response to stress (Eysenck & Eysenck, 1985; Muris, Schmidt, Merckelbach, & Schouten, 2001; Tellegen, 1985). While fear and sadness are often conceptualized as reflecting an underlying biobehavioral motivation system related to avoidance (Fowles, 1994; Gray, 1970) or withdrawal (Davidson, 1998), some have argued that anger is more closely related to an approach system (Carver & Scheier, 1998; Goldsmith, Lemery, & Essex, 2004; Putnam & Stifter, 2005). Indeed, there is emerging evidence that different forms of NE may have different biobehavioral underpinnings (George, Ketter, Parekh, Horowitz,
Another temperamental dimension that has received a substantial amount of research attention is behavioral inhibition (BI) (Garcia-Coll, Kagan, & Reznick, 1984; Kagan, 1989, 1997). BI is a heritable (DiLalla, Kagan, & Resnick, 1994; Matheny, 1989) pattern of behavior characterized by fearfulness, constraint, and low levels of approach in unfamiliar contexts. Thus, BI is typically assessed during laboratory observations in novel situations. In these situations, children high on BI display wariness, reticence, and decreased exploration. However, in non-novel situations, the behavior of children with BI is similar to that of other children (Laptook et al., submitted; Signoretta, Maremmani, Liguori, Perugi, & Akiskal, 2005).

These higher order dimensions have utility in describing a large number of types of behaviors. However, a number of studies have found that not all lower-level components of these dimensions are equally related to the higher-order dimension (Lucas et al., 2000; Olino et al., 2005). Additionally, some studies have found that lower-order facets of personality predict psychopathology better than higher-order domains (Bagby, Costa, Widiger, Ryder, & Marshall, 2005; Clark, 2007). Thus, it appears to be important to examine both higher and lower order child behavior constructs.

*Relationship Between PE, NE, and BI*

There has been greater attention paid to the relationship between PE and NE than either PE and BI, or NE and BI. Watson and colleagues (Watson & Tellegen, 1985; Watson, Wiese, Vaidya, & Tellegen, 1999) have developed a circumplex model of affect that specifies PE and NE as the primary axes. Thus, this model hypothesizes that PE and NE are largely uncorrelated
constructs. This theoretical relationship has been documented in many empirical studies (Durbin et al., 2005; Tellegen, Watson, & Clark, 1999).

Associations between BI and PE and NE are discussed much less in the literature. BI and NE and BI and PE may be more strongly associated with each other than PE and NE due to construct overlap. BI and NE share the component of fear. Indeed, Muris and Dietvorst (2006) found that children identified as BI had higher levels of NE than children who did not demonstrate BI. There is also overlap between BI and PE. Low levels of approach behaviors are common to both high BI and low PE (Durbin et al., 2005) and some authors include indicators of PA (e.g., smiling) in their definitions of inhibition (Gest, 1997; Kagan, Snidman, & Arcus, 1998). Gest (1997) found that BI was modestly positively associated with NE and modestly negatively associated with PE in early adulthood. Despite minimizing construct overlap between BI and PE, Durbin et al. (2005) found a similar pattern of modest associations in preschoolers.

**Methodological Issue Related to the Assessment of Temperament**

Many studies have relied on the use of parent report questionnaires to examine how child temperament is related to developmental outcomes from other domains. However, the use of parent report measures have limitations, as they may be biased by reporter or mood-state bias (Youngstrom, Izard, & Ackerman, 1999). Thus, observational methods have been developed to assess behaviors that are meaningfully reflective of temperament (e.g., Goldsmith et al., 1995).

**Associations Between Temperament and Psychopathology**

The relationship between temperament or, more broadly, personality and psychopathology has received much theoretical (Clark, 2006; Meehl, 1975; Nigg, 2006; Posner, Russell, & Peterson, 2005) and empirical attention (Biederman et al., 2001; Newman, Caspi, Moffitt, & Silva, 1997). Theorists have argued that low PE represents a vulnerability to
depressive disorders (Akiskal, 1989; Akiskal et al., 1983; Clark & Watson, 1991; Clark, Watson, & Mineka, 1994; Meehl, 1975; Nigg, 2006; Rothbart & Bates, 1998). There is also some evidence to suggest that PE may be related to social phobia (Brown, Chorpita, & Barlow, 1998). NE has also been found to serve as a risk factor for many forms of psychopathology, including mood and anxiety disorders (Clark & Watson, 1991). In addition, investigators have proposed that BI is a risk factor for the development of anxiety disorders (Fox et al., 2005; Kagan, 1997; Rapee, 2002). Lastly, specific forms of NE, such as anger/hostility, may be associated with vulnerability to externalizing disorders (Krueger & Markon, 2006).

*Temperament and Psychopathology in Offspring of Parents with Depressive and Anxiety Disorders*

Although familial transmission of mood and anxiety disorders is well-established (Beardslee et al., 1998; Biedel & Turner, 1997), it is unclear exactly what is being transmitted. One possibility is that parents transmit a temperamental vulnerability to develop psychopathology to their offspring through genetic or environmental mechanisms (Silberg & Rutter, 2002). Unfortunately, few studies have investigated the associations between parental history of depressive and anxiety disorders and facets of PE, NE, and/or BI in offspring. Studies using observational measures of temperament would be particularly valuable, as parent-report measures of temperament may be biased by the parents’ mood or history of psychopathology (Youngstrom, Izard, & Ackerman, 1999).

Several studies have reported associations between parental mood disorders and observations of low PE in offspring. Forbes, Cohn, Allen, and Lewinsohn (2004) found that infants of depressed parents exhibited lower levels of positive affect during a still-face task at three, but not six, months compared to infants of non-depressed parents. Kelley and Jennings
(2003) found that mothers with higher levels of depressive symptomatology had children that displayed lower levels of interest and engagement. Neff and Klein (1992) found that toddlers of depressed mothers displayed lower levels of PE than toddlers of non-depressed mothers during a naturalistic home observation. Durbin et al. (2005) found that mothers with a history of depressive disorder, particularly those with chronic/recurrent depressive disorders, had children with lower levels of PE during a laboratory observation than mothers without a history of depression.

Some of these studies also reported higher levels of NE in offspring. Kelley and Jennings (2003) reported that children of mothers with higher levels of depressive symptomatology displayed more sadness than children of controls. Durbin et al. (2005) found that children of depressed mothers displayed more NE in situations where NE was not normative than children of non-depressed mothers. However, overall child NE and specific forms of NE were not associated with maternal history of depression.

Lastly, a number of studies have examined offspring of parents with depressive and anxiety disorders using parent report and behavioral observation measures of BI (see Hirshfeld-Becker et al., 2003 for a concise review). Kochanska (1991) found that children of parents with recent onsets of depressive disorders had higher levels of BI than comparison children. Rosenbaum et al. (1988) found that BI was higher in children of parents with panic disorder with agoraphobia, regardless of the presence of MDD, compared to children of parents with no such history. Rosenbaum et al. (2000) found that the likelihood of being defined as behaviorally inhibited was greater when offspring had parents with major depressive or panic disorder.
Temperament and Psychopathology in Prospective Longitudinal Studies

There have also been only a few prospective longitudinal studies of temperament related variables as risk factors for mood and anxiety disorders. Two interesting studies found associations between dimensions of behavior that were neither clearly PE nor NE and later psychopathology. Caspi et al. (1996) found that three-year old children identified as “inhibited,” exhibited an increased rate of depressive, but not anxiety, disorders at age 21. Goodwin, Fergusson, and Horwood (2004) found that withdrawn behavior at age 8 was associated with increased risk for anxiety and depressive disorders during adolescence and young adulthood. For both of these studies, it is unclear if the “inhibited” and withdrawn behaviors are more strongly influenced by a global tendency (i.e., low PE or high NE) or by context specificity (i.e., high BI).

Few studies have specifically examined the relationship between low PE relevant temperament dimensions and later psychopathology. Messman and Koot (2000a, 2000b) reported that low approach in 2-3 year old children was associated with internalizing problems at ages 10-11, as reported by both parents and teachers. Van Os, Jones, Wadsworth, and Murray (1997) found that physician-rated behavioral apathy in childhood predicted depression in adolescence and chronic depression in adulthood. Additionally, Gjerde (1995) found that descriptors similar to those used to denote high PE, such as gregariousness, cheerfulness, and engagement, were associated with lower levels of depressive symptoms in early adulthood for girls. Lastly, Dougherty et al. (under review) found that age 3 PE was associated with depressive symptoms at age 10 and this association was significant after controlling for age 3 internalizing behavior problems and NE.

Some studies have reported that higher levels of NE are associated with greater risk for internalizing behavior problems or psychopathology. Brendgen, Wanner, Morin, and Vitaro
(2005) examined trajectory classes of depressive symptoms during early adolescence. They found that NE, assessed during kindergarten was associated with membership in both the chronically high depressive symptoms class and the increasing depressive symptoms class compared to the class characterized by consistently low levels of depression.

There have also been several studies that have examined the relationship between BI and later internalizing behavior problems. Schwartz, Snidman, & Kagan (1999) found that BI in early childhood was associated with general social anxiety, but not other forms of anxiety disorders, during adolescence. Additionally, Leve, Kim, and Pears (2005) found that shyness, which overlaps with BI, rated at age 5 was associated with internalizing behavior problems at age 17. Thus, there is evidence from several studies that implicates BI in the development of internalizing behavior problems, particularly anxiety disorders (Hirshfeld et al., 1992; Hirshfeld-Becker et al., 2003; Kagan, 1997; Perez-Edgar & Fox, 2005; Rapee, 2002).

While the emphasis of most studies of BI has focused on the high end of the dimension, some studies have focused on the low end. Individuals who demonstrate very low levels of BI are described as disinhibited, which includes the tendency to seek out novelty and demonstrate heightened approach behaviors in unfamiliar contexts (Hirshfeld-Becker et al., 2003). Hirshfeld et al. (1992) found that children who were uninhibited were at greater risk for oppositional defiant disorder compared to inhibited or non-inhibited children. Lastly, Rubin et al. (2003) found that undercontrolled children, similar to those described as disinhibited, had higher levels of externalizing behavior problems at age 4.

**Heterogeneity of Depressive Disorders**

Depressive disorders vary with respect to a number of dimensions, including severity, recurrence, and chronicity. Of these features, recurrent and chronic forms of depressive
disorders demonstrate higher degrees of familial aggregation than single episode depressive disorders (Kendler et al., 1999; Klein et al., 2002). Chronic depression is also characterized by lower levels of extraversion and higher levels of neuroticism than other types of depressive disorders (Hirschfeld, 1990; Klein et al., 1988). This suggests that temperamental vulnerabilities associated may be more strongly associated with chronic forms of depression. Additionally, previous work in this area (Durbin et al., 2005) suggests that parental chronic/recurrent depressive disorders demonstrate stronger associations with child PE as compared to single, non-chronic depressive disorders. Therefore, I divided depressive disorders into three categories: no history of depressive disorder; a history of a single, non-chronic depressive disorder, and a history of chronic/recurrent depression.

The Present Study

I examine whether dimensions of child behavior, including facets of PE, NE, and BI, are associated with parental depressive, anxiety, and substance use disorders (SUD). Based on the literature reviewed above, I expect to find that parental depressive, but not anxiety disorders, will be associated with low levels of child PE. Durbin et al. (2005) found specificity such that parental chronic or recurrent, rather than single episode, depressive disorders were most highly related to offspring PE behaviors. Within facets of PE, parental chronic or recurrent depressive disorders were more high associated with child PA and interest/engagement than sociability. I expect to replicate these findings.

With respect to NE, I anticipate that facets of NE that are associated with internalizing behaviors (e.g., sadness, fear) may be related to parental mood and/or anxiety disorders (i.e. internalizing disorders). In addition, parental anxiety, but not depressive, disorders may be associated with child BI.
I also explore associations between parental externalizing disorders and child temperament. Facets of NE that are associated with externalizing behaviors (i.e. anger) may be related to parental externalizing disorders (Patrick & Bernat, 2006), indexed in the present study by history of SUD. Based on a limited literature, I speculate that parental externalizing disorders will be associated with low levels of BI (Hirshfeld-Becker, Biederman, Faraone, Violette, Wrightsman, & Rosenbaum, 2002; Nigg, 2000).

*Nature of the Association Between Temperament and Psychopathology*

There have been two major conceptualizations of the relationship between temperament and psychopathology (Frick, 2004; Lahey, 2004; Tackett, 2006). The first suggests that temperament is an early manifestation, or form, of a disorder. Thus, the causal processes associated with temperament are the same as, or overlap with, those that are associated with psychopathology. The second perspective holds that temperament is a risk factor for psychopathology. This implies that temperament and psychopathology are the product of different causal processes, but that temperamental traits increase the likelihood of the onset of psychopathology given other risk or precipitating factors.

These competing conceptualizations lead to different predictions regarding the role of parental personality in the relationship between parental psychopathology and offspring temperament. If temperament is a precursor of psychopathology, then parental psychopathology should be associated with offspring temperament. However, the critical prediction in this model is that even after accounting for parental personality, parental psychopathology should still have a direct effect on offspring temperament (Figure 1).

In contrast, if temperament is a predisposing factor for psychopathology, then parents should transmit temperament traits to their offspring, but the transmission of psychopathology
should be indirect, operating via other mechanisms. This implies that parental personality should be directly associated with offspring temperament, but the association between parental psychopathology and offspring temperament should be accounted for by parental personality (Figure 2). Thus, if the relationship between parental psychopathology and offspring temperament is accounted for by personality, this would suggest that temperament is more strongly related to personality.

Child temperament develops into later personality (Shiner, 1998). Based on behavior genetics studies, dimensions of personality and temperament are heritable (Borkenau, Riemann, Angleitner & Spinath, 2001; Goldsmith & Lemery, 2000), which suggests that genetic and environmental (i.e., familial) factors influence the development of the trait. However, very little published literature exists that examines the associations between parental personality and offspring temperament.

Although these perspectives are often presented as mutually exclusive, it is possible that both parental psychopathology and personality will be associated with domains of child temperament. Twin studies have demonstrated that anxiety and mood disorders have common and specific genetic variance that is associated with NE (Hettema et al., 2006; Middeldorp, Cath, van Dyck, & Boomsma, 2005). This would suggest that each association, from parental psychopathology to offspring temperament and parental personality to offspring temperament, can be operating simultaneously.

I examine parental personality traits that are theoretically related to both offspring temperament and parental psychopathology. I expect specificity, such that parental PE/extraversion will be associated with offspring PE. Similarly, I anticipate that parental
NE/neuroticism will be associated with offspring NE. Lastly, I expect that parental constraint will be inversely associated with offspring impulsivity and, possibly, anger.

Consideration of Moderators and Other Explanatory Variables

Recently a number of papers have suggested (Goodman & Gotlib, 1999) or demonstrated (Bornstein et al., 2006; Essex et al., 2006; Feldman & Masalha, 2007; Hill et al., 2005) that a variety of demographic/contextual factors that may be important in understanding how parental characteristics, including psychopathology and personality, are related to child outcomes. Three such factors are maternal employment, maternal age, and parental education. Hill et al. (2005) examined the influence of maternal employment on cognitive development. In their report, they suggest that the associations that they found may have been moderated by maternal depression. Likewise, Feldman and Masalha (2007) found differential relationships between maternal depressive symptomatology and offspring behavior problems as a function of cultural differences, which included elements of maternal work-family balance. Bornstein et al. (2006) found that maternal age was associated with parenting practices, birth complications, and social support, all of which have been linked to child outcomes. Thus, the authors suggested that in addition to examining the associations between maternal age and parenting related constructs, it is important to examine if maternal age influences socioemotional development in their children. Additionally, Essex et al. (2006) found that parental education, as a proxy measure of socioeconomic status, served as an important moderator in the relationship between a number of risk factors, including family history of psychopathology and family distress, and the development of internalizing behavior problems during childhood. Like Durbin et al. (2005), I examine a number of family and demographic characteristics (e.g., maternal employment, maternal age, education, and marital discord) that may influence child temperament.
Additionally, I examine whether these factors may moderate the relationship between parental psychopathology and personality and offspring temperament.

The present study will attempt to replicate and extend a previous study conducted by my research group (Durbin et al., 2005). First, I examine a larger community sample of preschool aged children. The larger sample may include greater heterogeneity and power to examine moderators of the relationship between parental psychopathology and personality and offspring temperament. Second, I examine associations between child behavior and both maternal and paternal mood and anxiety disorders and SUD. Due to the smaller sample (N = 100) and the lower rates of depressive and anxiety disorders in fathers, Durbin et al. (2005) was limited in fully addressing associations between offspring temperament and paternal internalizing disorders. Lastly, I attempt not only to replicate the association between child low PE and maternal depression, but to go beyond this to explore the processes through which parental psychopathology is related to offspring temperament.
Method

The current study presents data on 375 three-year old children from Long Island, NY. Participants were recruited through a commercial mailing list and were initially contacted by the Stony Brook University Center for Survey Research. Families with a child between three and four years of age who lived with at least one English-speaking biological parent and did not have any significant medical conditions or developmental disabilities were eligible for participation.

Demographic statistics concerning the study sample are displayed in Table 1. A majority of the mothers and fathers in the study were Caucasian and many families had at least one parent who graduated from a four year degree program. Slightly less than half of the children were female (43.2%) and child verbal ability, as assessed by the Peabody Picture Vocabulary Test (PPVT; Dunn & Dunn, 1997) and the Expressive One-Word Picture Vocabulary Test (EOWPVT; Brownell, 2000), was average.

Child Assessment Procedures

Laboratory Assessment. Laboratory visit lasted approximately two hours, during which children participated in a standardized set of twelve laboratory episodes. Eleven were from the Laboratory Temperament Assessment Battery (Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1995) and one (Exploring New Objects) was adapted from an original Lab-TAB episode. The episodes in the Lab-TAB were drawn from previous studies that examine a number of research questions related to child development and emotionality (Kochanska & Knaak, 2003; Pfeifer et al., 2002). Previous work in my laboratory reported moderate-ranged associations between child behaviors observed during laboratory observations and independent home observations (Durbin et al., 2007). Additionally, stability in the domains of interest in the present study was found in a previous study over the course of four years (Durbin et al., 2007).
The selected episodes were designed to elicit different temperament traits or emotional displays. Between each episode, the child took breaks to return to a neutral state before entering a new situation. Each task was videotaped through a one-way mirror and later coded. The episodes are described below in the order that they were presented to the children.

*Risk Room* (behavioral inhibition). This episode was originally designed to assess BI (Kagan, 1997). The episode allows children to explore a set of novel and ambiguous stimuli, including a Halloween mask, balance beam, and a black box.

*Tower of Patience* (inhibitory control; interest). The child and experimenter alternated turns in building a tower together. The experimenter took increasing amounts of time before placing her block on the tower during each of her turns. The child was asked to wait until the experimenter takes her turn to place their block.

*Arc of Toys* (PA; interest; anger). The child played independently with toys for a five-minute period. The experimenter then asked the child to clean up the toys.

*Stranger Approach* (behavioral inhibition). The child was left alone briefly in the empty assessment room while the experimenter left to look for toys. In the experimenter’s absence, a male research accomplice entered the room and spoke to the child while slowly walking closer.

*Make that Car Go* (PA, interest). The child and experimenter raced remote controlled cars.

*Transparent Box* (persistence, interest, anger, sadness). The experimenter locked an attractive toy in a transparent box. The child was then left alone with a set of keys to attempt to open the box. After a few minutes, the experimenter returned to the child and told them that she had left the wrong set of keys. The child was then encouraged to use the new keys to open the box and play with the toy.
Exploring New Objects (behavioral inhibition). This episode allowed the child to explore a set of novel and ambiguous stimuli, including a mechanical spider, a mechanical bird, and sticky water-filled soft gel balls.

Pop-up Snakes (PE). The child and experimenter surprised the child’s mother with a can of potato chips that actually contained coiled snakes.

Impossibly Perfect Green Circles (anger, sadness, persistence). The experimenter repeatedly asked the child to draw a circle on a large piece of paper. Each attempt was mildly criticized. After approximately two minutes, the experimenter praised the child for their efforts.

Popping Bubbles (PA, interest). The child and experimenter played with a bubble-shooting toy.

Snack Delay (inhibitory control). The child was instructed to wait for the experimenter to ring a bell before eating a snack. The experimenter systematically increased the delay before ringing the bell.

Box Empty (anger, sadness). The child was given an elaborately wrapped box to open, under the impression that a toy was inside. After the child discovered that the box was empty, the experimenter returned with several toys for the child to keep.

Tape Coding Procedures. Two different methods of coding the videotape data were employed. One approach involved making ratings at discrete time intervals based on highly specific behavioral codes. Like the vast majority of previous studies that have examined BI, I used this microcoding method to code the three episodes specifically designed to assess BI: Risk Room; Stranger Approach; and Exploring New Objects. The second method was more global and allowed raters to use their knowledge of child behavior and contextual influences to derive ratings to summarize observed behavior across the entire task. This method allows for all
episodes to be coded for the same set of emotions and behaviors. For each participant, different raters completed the two coding methods. Moreover, global ratings for most of the episodes were completed by different raters.

For micro-level coding of episodes eliciting BI, ratings were made for each epoch, which spanned 20-30 seconds depending on the particular episode. Affective codes were based on a system developed for the Lab-TAB by Goldsmith (Goldsmith et al., 1995), which draws from the Affex system of deriving affective meaning from facial muscle movements (Izard, Dougherty, & Hembree, 1980). Within each epoch, a maximum intensity rating of facial, bodily, and vocal fear was coded on a scale of 0 (absent) to 3 (highly present and salient). The definition of BI in the present study was largely based on Durbin et al. (2005), and was computed as the average standardized ratings of latency to fear (reversed); facial, vocal, and bodily fear (Risk Room, Stranger Approach, and Exploring New Objects); latency to touch objects; total number of objects touched (reversed); tentative play; reference parent; proximity to parent; reference experimenter; time spent playing (reverse) (Risk Room and Exploring New Objects); startle (Exploring New Objects); sad facial affect (Exploring New Objects and Stranger Approach); latency to vocalize; approach towards the stranger (reverse); avoidance of the stranger; gaze aversion; and verbal/nonverbal interaction with the stranger (Stranger Approach).

The global coding methodology considered facial, bodily and vocal indicators of PA, fear, sadness, and anger. The major difference between the coding methods was that a single rating was made per episode for global coding, as compared to multiple ratings per episode for the micro-coding methodology. The single global ratings based on all behaviors that were relevant to each dimension during that episode.
Ratings of PA were made with consideration of the qualitative and quantitative aspects of displays of joy and enthusiasm. Overall PA ratings were computed as the average standardized weighted sum of instances of low, moderate, and high displays of facial, vocal, and bodily PA across all episodes. Similarly, overall ratings of sadness, anger, and fear were computed as the average standardized weighted sum of instances of low, moderate, and high displays of facial, vocal, and bodily of the respective form of affect from all episodes. Global interest ratings were based on the child’s comments about the activity and how engaged the child was in play. Global sociability ratings were based on the quality and quantity of the child’s attempts to engage and interact with the experimenter and, to a lesser extent, the parent. Global impulsivity ratings were based on child behaviors that suggest that the child is acting with little consideration for consequences or little inhibitory control (e.g., grabbing toys from the experimenter). Table 2 displays the internal consistency estimates, as measured by alpha, inter-rater reliabilities, as measured by intra-class correlations, descriptive statistics, and intercorrelations of child behavior scales used in the present study. Most of the ratings used in this report demonstrated at least adequate levels of both internal consistency and inter-rater reliability.

*Child verbal ability.* Child receptive and expressive language ability was assessed with the Peabody Picture Vocabulary Test (PPVT; Dunn & Dunn, 1997) and the Expressive One-Word Picture Vocabulary Test (EOWPVT; Brownell, 2000), respectively. Each has high levels of reliability, as indexed by high levels of test-retest stability, validity, as indexed by associations with indices of intelligence, and extensive age norming.

*Parental Assessment Procedures*

*Parental Psychopathology.* Biological mothers and fathers of the children were interviewed using a slightly expanded version of the Structured Clinical Interview for DSM-
Non-patient (SCID-NP; First et al., 1996). The SCID is currently one of the most widely used
diagnostic instruments and has established acceptable levels of reliability and validity (Williams,
Gibbon, First, Spitzer, Davies, et al., 1992). Interviews were conducted by telephone, which
generally yields comparable results to face-to-face interviews (Rohde, Lewinsohn, & Seeley,
1997; Sobin et al., 1993). Two Masters-level raters conducted the diagnostic interviews. All
diagnostic interviews were conducted without knowledge of the temperament ratings from the
laboratory observation; likewise, all coders of the temperament data were unaware of the
diagnostic data on parents. Based on audiotapes of 30 assessments (20 with mothers and 10 with
fathers), inter-rater reliability (measured as kappa) for lifetime chronic/recurrent MDD or
dysthymic disorder; single, non-chronic MDD episode; or no depressive disorder diagnosis was
.94; for presence/absence of a lifetime anxiety disorder diagnosis was .91; and for
presence/absence of a lifetime SUD/externalizing disorder diagnosis was 1.00.

*Parental Personality.* Biological mothers and fathers of the children completed the brief
form of the Multidimensional Personality Questionnaire about themselves (MPQ-BF; Patrick,
Curtin, & Tellegen, 2002). The MPQ includes 155 items that comprise the following scales:
well-being, social potency, achievement, social closeness, stress reaction, alienation, aggression,
control, harm avoidance, traditionalism, and absorption. The brief form is highly correlated with
the full MPQ, and is similar with respect to internal consistency, factor structure, and
relationships with other personality measures (Patrick et al., 2002). The MPQ has a hierarchical
structure with three superfactors: PE, NE, and constraint, which serve as the indices of
personality in the present study. Internal consistency estimates for the first order scales ranged,
across mothers and fathers, from .66 to .87 (M = .76) and were consistent with internal
consistency data from previous uses of the MPQ.
Marital Satisfaction. Individual members of parental dyads that were married or cohabitating were asked to complete the Dyadic Adjustment Scale (DAS; Spanier, 1976). The DAS is one of the most widely used measures of relationship satisfaction and discord. The overall scale score has high levels of reliability and has been used very often to distinguish between couples with and without discord. For mothers and fathers, internal consistency was high ($\alpha = .95$ and .93, respectively)
Results

In order to examine the internal structure of the dimensions of temperament, I conducted a principal components analysis (PCA) with a promax rotation (Table 3). The data suggested that three factors should be retained. Using a cutoff factor loading of .40, the PE factor included PA, interest, and sociability; the fearful inhibition factor included overall fear and BI; and the NE factor included sadness, anger, and impulsivity. In order to examine higher order child behavior constructs, the results of the PCA served as the basis of creating dimensions of behavior. However, as fear did not meaningfully load on the NE factor, it suggested that facet-level analyses were appropriate. The correlations among these three higher order factors were not significant: PE and NE, $r = .08, p = .14$; PE and fear, $r = -.09, p = .09$; NE and fear, $r = .09, p = .06$.

I examined associations between demographic and contextual variables and each of the child behaviors of interest. Child sex differences were found on the broad domains of child behavior of PE $[F (1, 373) = 4.42, p < .05]$, fearful inhibition $[F (1, 373) = 6.85, p < .01]$, and NE $[F (1, 370) = 9.78, p < .01]$, such that girls displayed higher levels of PE and fear, but lower levels of NE, than boys. Analyses at the level of specific facets found differences on child fear, BI, sociability, and impulsivity, such that girls displayed higher levels of fear $[F (1, 370) = 7.71, p < .01]$, BI $[F (1, 370) = 9.59, p < .01]$, and sociability $[F (1, 372) = 4.92, p < .05]$, and lower levels of impulsivity than boys $[F (1, 370) = 17.61, p < .001]$. These gender differences are consistent with past findings (Else-Quest et al., 2006) and are displayed in Table 3. Children who were members of racial minority groups displayed higher levels of impulsivity than Caucasian children, $F (1,357) = 4.28, p < .05$. Children of mothers who worked outside the home displayed higher levels of PE $[F (1, 356) = 7.34, p < .05]$, PA $[F (1, 355) = 6.10, p < .05]$,
and sociability \( F(1, 355) = 10.46, p < .05 \). Of the working mothers, the number of hours worked by mother was associated with child NE \( (r = .19, p < .05) \) and sadness \( (r = .16, p < .05) \). No other significant associations were found between demographic/contextual characteristics and child behavior.

**Maternal Depressive Disorders and Child Behavior**

My first set of analyses examined differences in child behavior as a function of maternal depressive disorders, coded as no depressive history; single, non-chronic, depressive episode; or chronic or recurrent MDD or dysthymic disorder. These analyses did not identify significant differences on any higher- or lower-order dimension of child temperament (Table 5). The next set of analyses examined the possibility that the relationship between maternal depressive disorders and child behavior varied as a function of demographic/contextual variables. I focused on marital satisfaction (Goodman & Gotlib, 1999), maternal age (Bornstein et al., 2006); maternal employment, coded as mothers working outside the home or not working outside the home (Hill et al., 2005); and parental education, coded as neither parent having graduated a four year degree program, one parent graduated a four year degree program, and both parents having graduated a four year degree program (Essex et al., 2006); as potential moderators and examined all possible two-way interactions that included maternal depressive disorder terms (i.e., interactions that only included demographic or contextual factors were not examined).

In these initial moderational analyses, I found that the interaction between maternal depression and maternal employment was associated with child PE, \( F(2, 351) = 5.44, p < .01 \); PA, \( F(2, 351) = 6.49, p < .01 \); and interest, \( F(2, 351) = 5.49, p < .01 \). The interaction between maternal depression and maternal age was significantly associated with child PE, \( F(2, 368) = 3.07, p < .05 \); PA, \( F(2, 368) = 3.09, p < .05 \); fearful inhibition, \( F(2, 368) = 3.37, p < .05 \); and
global fear, $F(2, 368) = 3.23, p < .05$. Interactions between maternal depression and dyadic adjustment and education were not significantly associated with any dimensions of child behavior.

I now focus on examining the specific differences within the interactions involving maternal depressive disorders and demographic moderators. These post-hoc contrasts are also presented in Table 5. For the association between child PE, PA, and interest and the interaction between maternal depression and maternal employment, I examined the differences in those dimensions of child behavior as a function of maternal depression for mothers who were and were not working, separately. For children of mothers who were not working, there was a difference on child PE as a function of maternal depression, $F(2, 180) = 3.27, p < .05$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly lower levels of PE than children of never depressed mothers and children of mothers with a single depressive episode. For children of mothers who were working, there was a trend level difference on child PE as a function of maternal depression, $F(2, 171) = 2.34, p = .07$. The post-hoc LSD test did not identify any significant differences.

For children of mothers who were not working, there was a difference on child PA as a function of maternal depression, $F(2, 180) = 3.94, p < .05$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly lower levels of PA than children of never depressed mothers and children of mothers with a single depressive episode. For children of mothers who were working, there was a trend level difference on child PA as a function of maternal depression, $F(2, 171) = 2.68, p = .07$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly higher levels of PA than
children of never depressed mothers and, at the level of a trend, than children of mothers with a single depressive episode.

For children of mothers who were not working, there was also a difference on child interest as a function of maternal depression, $F(2, 180) = 5.55, p < .01$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly lower levels of interest than children of never depressed mothers. For children of mothers who were working, there was no evidence for differences on child interest as a function of maternal depression, $F(2, 171) = 1.14, p = .32$.

In order to conduct follow-up analyses for the interactions including maternal age in the same manner as the follow-up analyses for maternal employment, maternal age was dichotomized based on a median split. Mothers were classified as younger if their present age was at most 35 and older if their age was at least 36 years. For children of younger mothers, there was a difference on child PE as a function of maternal depression, $F(2, 160) = 2.57, p = .08$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly lower levels of PE than children of never depressed mothers and children of mothers with a single depressive episode. For children of older mothers, there was no significant difference on child PE as a function of maternal depression, $F(2, 212) = 1.01, p = .37$.

For children of younger mothers, there was a difference on child PA as a function of maternal depression, $F(2, 160) = 3.96, p < .05$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly lower levels of PA than children of never depressed mothers and children of mothers with a single depressive episode. For children of older mothers, there was no significant difference on child PA as a function of maternal depression, $F(2, 212) = 1.27, p = .28$. 
For children of younger mothers, there was a difference on child fearful inhibition as a function of maternal depression, $F (2, 160) = 4.78$, $p < .05$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly higher levels of fearful inhibition than children of never depressed mothers. For children of older mothers, there was no significant difference on child fearful inhibition as a function of maternal depression, $F (2, 212) = .08$, $p = .93$.

For children of younger mothers, there was a difference on child fear as a function of maternal depression, $F (2, 160) = 4.19$, $p < .05$. The post-hoc LSD test found that children of mothers with chronic/recurrent depression had significantly higher levels of fear than children of never depressed mothers. For children of older mothers, there was no significant difference on child fear as a function of maternal depression, $F (2, 212) = .05$, $p = .95$.

*Exploratory Post-hoc Analyses*

In order to better understand the interaction between maternal depression and employment, I examined associations between maternal hours spent working and offspring PA and interest as a function of maternal depressive history. For mothers with no history of depression or a single, non-chronic depressive episode, there was no association between the numbers of hours spent working and child PE ($r = .10$, $p = .11$; and $r = .10$, $p = .94$, respectively), PA ($r = .08$, $p = .21$; and $r = .05$, $p = .74$, respectively) or interest ($r = .02$, $p = .78$; and $r = -0.01$, $p = .96$, respectively); however, for mothers with a history of chronic/recurrent depressive disorders, there were significant associations between the numbers of hours spent working and child PE ($r = .41$, $p < .01$), PA ($r = .38$, $p < .001$) and interest ($r = .34$, $p < .01$).

In order to better understand the interaction between maternal depression and maternal age and employment with respect to child PA, I examined the PA scores of children and noticed
that there was a subgroup of children with chronic/recurrent depression who had very high PA and whose mothers were both older and working. Therefore, I conducted an exploratory 3-way interaction that included main effects for maternal depressive disorder, maternal employment, and maternal age, which was dichotomized based on a median split (age 36) in order to allow for easier interpretation of the effect; all two-way interactions; and the three-way interaction. In this analysis, I found a significant association between the three-way interaction of maternal depression, maternal age, and maternal employment on child PA, $F(2, 345) = 4.17, p < .05$. The results of the three-way interaction of maternal depression, age, and employment on child PA are presented graphically in Figure 3, with the left portion of the figure displaying the data for mothers who are not working and the right portion of the figure displaying the data for mothers who are working. For mothers who were not working, the interaction of maternal depression and maternal age was not significantly associated with child PA, $F(2, 177) = .01, p = .99$. As described earlier, for mothers who were not working, offspring PA differed as a function of maternal depressive disorder, $F(2, 180) = 3.94, p < .05$.

For mothers who were working, the interaction of maternal depression and maternal age was significantly associated with child PA, $F(2, 168) = 8.89, p < .001$. I examined this interaction by comparing the means of child PA for working younger and older mothers separately. For younger working mothers, there was no overall significant difference in child PA as a function of maternal depression, $F(2, 75) = 1.40, p = .25$. For older working mothers, there was an overall difference in child PA as a function of maternal depression, $F(2, 93) = 9.64, p < .001$. Post-hoc differences, using the LSD, found that children of chronically depressed mothers displayed significantly higher levels of PA than either mothers of never depressed or mothers with a single, non-chronic, depressive episode.
As it appeared that the children of chronically depressed, older, working mothers had a very high level of PA, I examined the possibility that these children may be at risk for externalizing behavior problems by using child impulsivity as a possible marker of externalizing behavior problems. For mothers who were working, the interaction of maternal depression and maternal age was associated with child impulsivity at a trend level, $F(2, 168) = 2.89, p = .059$. I examined this trend by comparing the means of child impulsivity for working younger and older mothers separately. These results are presented graphically in Figure 4. For younger working mothers, there was no overall significant difference in child impulsivity as a function of maternal depression, $F(2, 75) = .78, p = .46$. For older working mothers, there was a trend towards an overall difference in child impulsivity as a function of maternal depression, $F(2, 93) = 2.43, p = .093$. Post-hoc differences, using the LSD, found that children of chronically depressed mothers displayed significantly higher levels of impulsivity than children of mothers with a single, non-chronic, depressive episode.

The most striking differences were between the younger and older chronically/recurrently depressed working mothers. Hence, I examined the differences between those two sub-groups in order to determine whether any additional factors might help explain the moderational effect. In these analyses, I compared these mothers on number of hours of mother working, depressive characteristics, comorbid psychopathology, self-reported personality, parenting assessed via observation and self-report methods, paternal depressive disorder characteristics, paternal comorbid psychopathology, and paternal personality. Within the chronically depressed working mothers, older mothers had significantly higher global assessment of functioning (GAF) scores in the last four years, $F(1, 31) = 4.59, p < .05$ ($M = 71.44, SD = 9.98$; and $M = 64.43, SD = 8.03$, respectively); higher rates of maternal anxiety disorder, $\chi^2(1) = 4.57, p < 0.05$ [$n = 12$,
(66.7%); and n = 4 (28.6%)); and higher rates of paternal SUD, $\chi^2(1) = 4.44, p < 05 [n = 10, \text{ (66.7%)}; and n = 2 (22.2%)$] than chronically depressed working younger mothers.

Maternal Non-Affective Psychopathology and Child Behavior

Next, I examined differences in child behavior as a function of type of maternal non-affective psychopathology (Table 6). No differences were found based on maternal anxiety disorder. Children of mothers with a history of SUD displayed higher levels of fear, $F(1, 373) = 5.86, p < .05$; global fear, $F(1, 373) = 5.57, p < .05$; and BI, at a trend, $F(1, 373) = 3.46, p = .06$, than children of mothers without a history of SUD. No two-way interactions that involved demographic characteristics and maternal anxiety disorders or SUD were significantly associated with child behavior.

Paternal Psychopathology and Child Behavior

I examined differences in child behavior as a function of paternal depressive and anxiety disorders and SUD (Table 7). No overall significant differences were found as a function of paternal depressive or anxiety disorder. Offspring of fathers with SUD had significantly lower levels of NE than offspring of fathers without SUD, $F(2, 297) = 5.54, p < .05$.

Parallel analyses were conducted to examine the possibility that demographic/contextual factors moderated the relationship between paternal psychopathology and child behaviors. Potential moderators included maternal age, paternal age, maternal employment, parental education, and marital satisfaction, based on paternal report.

There were few significant interactions. The interaction between paternal anxiety disorder and parental education was significantly associated with child fearful inhibition, $F(2, 276) = 4.23, p < .05$, and BI, $F(2, 276) = 4.23, p < .05$. For children, neither of whose parents graduated from a four year degree program, there was no significant difference in fear or BI as a
function of paternal anxiety disorder, $F(1, 70) = .73, p = .39$; and $F(1, 70) = .04, p = .84$, respectively. For children with one parent who graduated from a four year degree program, there was a trend level difference in fear and BI as a function of paternal anxiety disorder, $F(1, 93) = 3.81, p = .054$; and $F(1, 93) = 3.89, p = .052$, respectively. Of these children, those who had fathers with a history of anxiety disorders had higher levels of BI than children of fathers without a history of anxiety disorders. For children with two parents who had graduated from a four year degree program, there was a significant difference in BI as a function of paternal anxiety disorder, $F(1, 109) = 4.33, p < .05$. Of these children, those with fathers with a history of anxiety disorders had lower levels of BI than children of fathers without a history of anxiety disorders. However, there was no significant difference on fear as a function of paternal anxiety disorder among those children with two parents who had graduated from a four year degree program, $F(1, 109) = 2.12, p = .15$.

Additionally, the interaction between paternal SUD and marital satisfaction was significantly associated with offspring sadness, $F(1, 184) = 6.81, p < .05$. Discordant and non-discordant couple dyads were distinguished using the clinical cutoff of 100 for father reports on the DAS. For non-discordant couples, there was no significant difference in offspring sadness as a function of paternal SUD, $F(1, 154) = .04, p = .85$. For discordant couples, there was a significant difference in offspring sadness as a function of paternal SUD, $F(1, 33) = 6.75, p < .05$. Surprisingly, among offspring of martially discordant fathers, offspring of fathers with SUD had lower levels of sadness ($M = -.30, SD = .54$) than offspring of fathers without SUD ($M = .51, SD = 1.25$).

*Maternal Personality and Child Behavior*
Next, I examined the associations between maternal personality and child behavior (Table 8). In the interest of minimizing Type I errors, I focused on the broadband personality constructs of PE, NE, and constraint. Maternal NE was negatively associated with child interest, \( r = -.11, p = .05 \); and positively associated with child BI at the level of a trend, \( r = .10, p = .054 \).

I conducted a parallel set of moderational analyses involving maternal personality and demographic factors. In these analyses, the interaction between maternal NE and maternal employment was associated with child PE, \( F(1, 315) = 11.24, p < .001 \), PA, \( F(1, 315) = 8.87, p < .01 \); interest, \( F(1, 315) = 8.28, p = .32 \); and sociability, \( F(1, 314) = 7.23, p < .01 \). Additionally, the interaction between maternal constraint and maternal age was associated with child PA, \( F(1, 317) = 7.07, p < .01 \), NE, \( F(1, 317) = 6.38, p < .05 \), and anger, \( F(1, 317) = 8.98, p < .01 \).

To examine the follow-up analyses for the interaction between maternal NE and maternal employment, I examined the association between maternal NE and child behavior for mothers who were and were not working, separately (Table 8). For mothers who were not working (\( n = 171 \)), maternal NE was significantly negatively associated with child PE, \( r = -.24, p < .01 \); PA, \( r = -.19, p < .05 \); interest, \( r = -.26, p < .01 \); and sociability, \( r = -.16, p < .05 \). For mothers who were working (\( n = 148 \)), maternal NE was positively associated with child PA at the level of a trend, \( r = .15, p = .08 \); but not associated with child PE, \( r = .13, p = .12 \); interest, \( r = .06, p = .48 \); and positively associated with child sociability at the level of a trend, \( r = .14, p = .09 \).

I conducted the follow-up analyses differently for the interaction between maternal constraint and maternal age due to the fact that both the independent and moderator variables were continuous. Thus, I dichotomized maternal age based on the median split (age 36) to examine the association between constraint and child behaviors for younger and older mothers,
separately. For younger mothers, constraint was significantly associated with child PA, $r = .25$, $p < .01, n = 141$. For older mothers, constraint was not associated with child PA, $r = -.11, p = .15, n = 180$. For younger mothers, constraint was not associated with child NE or anger, $r = .08, p = .33; r = .05, p = .53$, respectively, $n = 141$. For older mothers, constraint was significantly negatively associated with child anger, $r = -.18, p < .05, n = 180$, however, constraint was negatively, but not significantly, associated with child NE, $r = -.11, p = .16$.

**Paternal Personality and Child Behavior**

I examined associations between broadband paternal personality constructs and child behaviors. None of these associations approached significance (Table 8).

I also examined the possibility that demographic/contextual factors may influence the association between paternal personality and child behavior (Table 8). In these analyses, I found that the interaction between paternal NE and maternal age was associated with child interest, $F(1, 262) = 7.43, p < .01$. I probed this interaction by examining the association between paternal NE and child interest in families with younger and older mothers, based on the median split, separately. For families with younger mothers, there was a negative significant association between paternal NE and child interest, $r = -.19, p < .05, n = 115$. However, for families with an older mother, there was no significant association between paternal NE and child interest, $r = .09, p = .26, n = 151$.

**Simultaneous Associations: Maternal Personality and Psychopathology and Child Behavior**

An aim of this study was to examine the simultaneous associations of psychopathology and personality on child behavior in order to understand whether these associations are unique to either maternal personality or psychopathology or whether personality and psychopathology represent overlapping factors. Unfortunately, few significant associations were found between
parental psychopathology and personality and child behaviors in the overall sample and there were no child behavior variables that were significantly associated with both a form of parental psychopathology and a personality construct. Hence, a direct comparison of strength of associations between parental personality and psychopathology are not very informative for the full sample.

Instead, I chose to examine the simultaneous associations between parental psychopathology and personality and offspring temperament within the particular subsamples that exhibited bivariate associations between these sets of variables. Among non-working mothers, offspring PA and interest were both predicted by maternal history of depressive disorder and maternal NE. When maternal history of depressive disorder and maternal NE were entered simultaneously to predict offspring PA in this subsample, maternal history of chronic/recurrent depression and maternal NE were both associated with lower levels of offspring PA at the level of a trend [\( B = -.27, \beta = -.15, t(170) = -1.89, p = .06; B = -.02, \beta = -.15, t(170) = -1.92, p = .06, \) respectively]. When maternal history of depressive disorder and maternal NE were entered simultaneously to predict offspring interest within non-working mothers, maternal history of chronic/recurrent depression and maternal NE were both associated with lower levels of offspring interest [\( B = -.14, \beta = -.17, t(170) = -2.12, p < .05; B = -.02, \beta = -.21, t(170) = -2.70, p < .01, \) respectively].

Additionally, among younger mothers, maternal depression, maternal constraint, and paternal NE Were all associated with offspring PA. Maternal history of depressive disorder, maternal constraint, and paternal NE all were associated with offspring PA in this subsample. However, as associations between maternal and paternal variables may represent different etiological pathways, I did not include maternal and paternal factors simultaneously. I entered
maternal depressive disorder and maternal constraint to predict offspring PA and found that chronic/recurrent depressive disorder was associated with lower levels of PA at the level of a trend \([B = -.28, \beta = -.15, t(140) = -1.75, p = .08]\) and maternal constraint was associated with higher levels of PA \([B = .04, \beta = .21, t(140) = 2.51, p < .05]\).
Discussion

Previous work has found that child temperament is related to both later psychopathology and personality. A number of studies have linked early low PE and high BI to depressive disorders (Caspi et al., 1996; Moffitt et al., 2007) and high BI to anxiety disorders (Kagan, 1997; Schwartz, Kagan, & Snidman, 1999). Other studies have found that early PE is associated with later extraversion and early NE is associated with later neuroticism (Caspi, 2000). Thus, I examined associations between parental psychopathology, namely depressive and anxiety disorders and SUD, and personality and offspring temperament. We expected to find associations between maternal history of chronic/recurrent depressive disorders and low levels of offspring PE relevant behaviors of PA and interest (Durbin et al., 2005). Previous work also suggested that parental anxiety disorders would be associated with higher levels of fear and/or BI (Rosenbaum et al., 2000). Based on recent work, I also examined whether demographic/contextual factors moderated the relationship between parental psychopathology and personality and offspring temperament.

No significant main effects were found on child PE related behaviors as a function of parental history of depressive disorders. Thus, in the whole sample, the major substantive hypothesis was not supported. Only one significant temperament dimension differed as a function of maternal depressive or anxiety disorder or paternal depressive, anxiety, or SUD. This was for child fear as a function of maternal SUD. Offspring of mothers with a history of SUD displayed higher levels of fear during the laboratory observation compared to offspring of mothers without SUD. This may suggest that mothers with a history of SUD may provide more chaotic family environments or engage in more hostile parenting practices with their children. These experiences could foster a temperamental style characterized by fearfulness.
In contrast to the lack of direct effects, I found a number of significant interaction effects between maternal psychopathology and contextual factors on child temperament. Maternal employment interacted with maternal depressive disorder, such that offspring of non-working mothers with chronic/recurrent depression displayed lower levels of PA and interest compared to offspring of non-working mothers with no history of depressive disorders; however, offspring of working mothers with chronic/recurrent depression displayed higher levels of PA compared to offspring of working mothers with no history of depressive disorders. Maternal age also interacted with maternal depressive disorder to predict offspring PA and fear, such that offspring of younger mothers with chronic/recurrent depression displayed lower levels of PA and higher levels of fear compared to offspring of younger mothers with no history of depressive disorders; however, there were no significant differences in offspring PA or fear as a function of maternal depressive disorders for offspring older mothers.

These results suggest that family history of chronic/recurrent depressive disorders are associated with lower levels of offspring PE, however, this is true for subsets of the sample, specifically those offspring of younger and of non-working mothers. Interestingly, the associations between history of depressive disorder and child PE were specific to chronic/recurrent forms of the disorder, rather than also single, non-chronic episode disorders. These data are consistent with studies finding that chronic/recurrent depressive disorders represent more familial forms of the disorder and may be more etiologically related to temperamental vulnerability (Durbin et al., 2005; Kendler et al., 1999; Klein et al., 2002). This study also provides some evidence that maternal chronic/recurrent depression is differentially related to risk of internalizing and externalizing psychopathology based on maternal age and employment. In contrast to offspring of younger and of non-working mothers, who displayed
low levels of PE related behaviors, which may represent vulnerability for internalizing psychopathology, offspring of older and working mothers displayed markedly high levels of PA and impulsivity, which may represent vulnerability for externalizing psychopathology. This suggests that contextual factors influence risk for type of psychopathology. However, the differential relationships between maternal history of depressive disorder and offspring temperament based on maternal employment and age raise important issues concerning the meaning of these contextual factors and the processes through which maternal depressive disorders are related to offspring temperament.

I found that the number of hours of maternal employment was related to child PA and interest. Importantly, however, this was only for mother-child dyads where mothers had a history of chronic/recurrent depression. For offspring of mothers with chronic/recurrent depression, number of hours spent working was associated with higher levels of child PA and interest. This suggests an environmental effect, such that greater exposure to mothers with a history of chronic/recurrent depression is related to lower levels of PE-related behaviors. Mothers with chronic/recurrent depression may not recognize PE displays in their offspring, which may reduce the likelihood of reinforcing those behaviors, or do not model PE behaviors to their children. Although we identified one factor (i.e., numbers of hours spent working by the mother) relevant to explaining the moderational role of maternal employment on the relationship between maternal depressive disorders and offspring temperament, there may be additional factors, including reasons for working (Alvarez, 1985; Barling, Fullagar, & Marchl-Dingle, 1988), positive and negative impacts of work related stress (Feldman & Masalha, 2007), and timing of returning to work (Hynes & Clarkberg, 2005), that need to be examined in the future.
The data suggested that there is a greater effect of maternal psychopathology and personality on child behavior than of paternal behaviors. This is consistent with much of the work in this area (Connell & Goodman, 2002). Although few significant effects of paternal behavior were identified, it is possible that paternal influences on risk for psychopathology may become apparent during subsequent developmental periods (Lewinsohn, Olino, & Klein, 2005). However, as mothers spend greater amounts of time with their young children, the lack of effect of fathers is also consistent with an environmentalist interpretation and pattern of transmission.

Maternal age may also reflect important contextual differences. Perhaps one of the most commonly discussed age related risk factor is teenage pregnancy and childbearing. However, the results of this study are not greatly influenced by teenage motherhood, as nearly all of the mothers in the sample were older than 20 when their child was born.

After the teenage years, additional changes occur that are relevant to childbearing and child rearing. A recent meta-analysis examined the associations between parental psychopathology and offspring behavior problems and found that child age moderated that relationship, such that the association between parental psychopathology and offspring was stronger for younger children than for older children (Connell & Goodman, 2002). However, it is possible that child age served as a proxy for parental age in this study and the finding could be interpreted that the association between parental psychopathology and offspring behavior problems was stronger for younger parents. Additionally, Bornstein et al. (2005) examined a number of factors that may change with respect to mothers, including parenting behaviors, maturity, birth complications, and other factors that may change over the course of childbearing age (Bornstein et al., 2005). In the present cross-sectional study, I examined whether the moderational role of maternal age may be better accounted for by parental education, which can
serve as a proxy of SES (Essex et al., 2006). The data suggested that maternal age, rather than education was meaningful as the moderator. It is important to consider additional potential moderators, many of which were suggested by Bornstein et al. (2005), such as specific parenting behaviors or birth complications, that may also explain the association between maternal depressive disorders and offspring temperament.

In addition to the exploring differences in meaning of maternal employment and age to explain the differential relationships between depressive disorders and offspring temperament, it is important to note that there may be differences in depressive disorders themselves based on employment or age. Thus, in addition to the heterogeneity of depressive disorders with respect to recurrence and chronicity, depressive disorders may also differ on age of onset, years since depressive disorder onset, number of depressive symptoms, comorbidity, and global functioning as a function of maternal age or maternal employment. Alternatively, it is also possible that the results may have been due to assortative mating. Older, working chronically/recurrently depressed mothers had husbands with higher rates of SUD compared to younger, working chronically/recurrently depressed mothers and offspring with higher levels of PA and impulsivity. Perhaps this represents a genetic transmission of risk, which is related to externalizing behavior problems. Alternatively, in the context of older, working chronically depressed mothers, children may exhibit markedly intense behaviors, characterized by high PA and impulsivity, in order to elicit responses from their caregivers. In contrast, younger, working mothers may be responsive to less intense child behaviors.

It is also possible that the interactions found in the present study are the result of Type I error. Many tests were conducted and some significant findings may have been due to chance. However, it is interesting to note that the domains of offspring temperament, PA, interest, and
fear, that were predicted by the interactions were the domains of offspring temperament of most interest to this study. This bolsters support for the claim that these results are not due to Type I error.

Additionally, in order to examine the possibility that the effects were the result of Type I errors, I examined the data from Durbin et al. (2005) that served as the impetus for the present study. I conducted analyses to examine whether the same two-way interactions were significantly associated with offspring PA or that post-hoc comparisons lead to similar conclusions. The interaction between maternal depressive disorders, defined as no history of depression; a single, non-chronic depressive episode; and chronic/recurrent depressive disorder, and maternal age, dichotomized based on a median split, was significantly associated with offspring PA (see Appendix 1). Importantly, the post-hoc interpretation of this interaction with respect to PA was identical to the results of the present study. That is, offspring of younger mothers with chronic/recurrent depression had significantly lower levels of PA compared to younger mothers with no history of depression or a single, non-chronic depressive episode. Consistent with the present study, for offspring of older mothers, there was no significant difference in PA as a function of maternal depressive history.

However, the interaction between maternal depressive disorder and maternal age was not significantly associated with offspring fear in the previous study. This may have been due to a methodological difference in fear-eliciting situations between the previous and present study. The present study included three fear-eliciting episodes, whereas the previous study included two. Thus, the present study may have provided a more robust measure of fear tendencies in the children.
Again, in Durbin et al. (2005), the interactions between maternal depressive disorders and maternal employment were not associated with offspring PA or interest in the previous study (Appendix 1). However, not identifying significant moderation may have been due to lack of power. An exploratory analysis of mothers who were not working found that offspring of mothers with a history of chronic/recurrent depression had significantly lower levels of PA and interest than offspring of mothers without a history of depression. However, no significant differences between PA or interest based on maternal history of depressive disorders for working mothers.

Both the present and previous study had approximately half of the mothers employed (48.5% vs. 57.8%, respectively). However, the present study appeared to include a higher proportion of full-time working mothers (32.5%) than the previous study (9.8%). Hence, differences in number of hours spent working may have accounted for some of the differences between these findings. The replication of results across two independent samples provides evidence for the moderational role of maternal age and, to a more limited extent, employment on the associations between maternal depressive disorders and offspring temperament.

In addition to examining the associations between parental psychopathology and offspring temperament, I also examined associations between parental personality and offspring temperament. As children’s temperament has been found to be predictive of their later personality, parental personality was expected to be associated with offspring temperament (Goldsmith et al., 1994). Overall associations demonstrated few significant associations. Higher maternal NE was associated with lower levels of child interest; however, this association was qualified by a significant interaction between maternal NE and maternal employment. This interaction was also significantly associated with offspring PA and sociability and suggested that
higher maternal NE was associated with lower levels of PA, interest, and sociability when mothers were not currently working. No significant associations were found between maternal NE and offspring PA, interest, or sociability when mothers were working. These associations for non-working mothers suggests that NE may lead to lower offspring PE related constructs due to greater exposure to their mothers, which may be more proximally related to less positive parenting practices. Alternatively, mothers with higher levels of NE may be less likely to work.

Interactions between maternal constraint and maternal age were also significantly associated with child PA. For younger mothers, higher maternal constraint was associated with higher offspring child PA. Additionally, the interaction between paternal NE and maternal age was significantly associated with offspring interest, such that higher paternal NE was associated with lower offspring interest for younger mothers.

Importantly and interestingly, the moderators of the relationship between parental personality and offspring temperament were the same moderators identified in analyses examining the relationship between parental psychopathology and offspring temperament. Additionally, the interactions were associated with the same dimensions of temperament. Thus, it is important to consider that the same mechanisms that may account for the differential relationships between parental personality and offspring temperament in older and younger and working and non-working mothers. Additionally, past study findings may have been due to relative homogeneity with respect to these specific maternal characteristics and led to identifying associations between parental psychopathology and dimensions of child temperament as main effects.

Alternatively, again, the significant interactions between maternal personality and age may be due to chance. Thus, I examined the significant interactions in the present study in
unpublished data from the previous work in my lab. The association between the interaction of maternal constraint and maternal age on offspring PA approached significance in this smaller sample (Appendix 2). Interestingly, the post-hoc associations examining the associations between maternal constraint and offspring PA within older and younger mothers demonstrated similar patterns of associations demonstrated in the present study. The associations between the interactions between maternal personality and maternal employment on offspring temperament in the previous study did not approach significance. However, this may have been due to the relatively small number of mothers in the previous sample who were working full time.

In addition to examining individual effects of parental psychopathology and personality on offspring temperament, I also planned on examining simultaneous effects of psychopathology and personality on offspring temperament. The goal of these analyses was to examine whether offspring temperament was more closely reflective of early psychopathology or personality. However, based on the empirical associations, this aim could not be addressed in the full sample. No target dimension of offspring temperament was significantly associated with a form of psychopathology and a personality construct. For particular subsamples, however, I was able to examine the simultaneous associations between parental psychopathology and personality and offspring temperament. These analyses tended to demonstrate unique associations. That is, despite overlapping variance between maternal psychopathology and personality, psychopathology and personality both remained associated with child temperament. As maternal psychopathology exhibited significant unique effects on offspring temperament after controlling for parental personality, it suggests that the effects of parental psychopathology on offspring are not entirely mediated by parental personality, as the predisposition model suggests. However, the fact that parental personality also had unique effects on offspring after controlling for
parental psychopathology indicates that differences in offspring temperamental emotionality do not entirely reflect precursors of later psychopathology. This suggests that neither the precursor nor predisposition models fully account for familial transmission of risk for psychopathology, as indexed by child temperament. In contrast, these results suggest that both mechanisms may be operating, although longitudinal studies are necessary to identify if these different processes are influential for some children, but not others.

Based on these analyses, in a limited and preliminary manner, I speculate about some of the implications of these results for the conceptualization of temperament. Temperament is intended to reflect biologically based and early emerging emotional and behavioral tendencies (Buss & Plomin, 1975; Goldsmith, Buss, Plomin, Rothbart, Thomas, Chess, Hinde, & McCall, 1987; Rothbart & Bates, 1998). I hypothesized that these emotional and behavioral tendencies would vary as a function of parental psychopathology and personality constructs; however, I found that these relationships were moderated by two different contextual factors, maternal age and employment. I speculated above concerning what differences in maternal age and employment may mean for the mother and developmental context of the child. These differential relationships suggest that temperament may be modifiable by environmental contexts (Rubin, Burgess, & Hastings, 2002; Fox et al., 2001). The findings of the present study may also suggest that assessments of temperament should be conducted before age three. This would allow for the examinations of direct associations between parental psychopathology and personality and offspring temperament before contextual factors mitigate these relationships. Although more tenuous, these data may suggest that alternative labels with fewer causal connotations, such as emotional or affective style, should be used to describe these behaviors in three year old children.
The present study highlights the need to consider multiple developmental pathways to the
development of psychopathology. I found that low PE behaviors in offspring may be preceded
by maternal depressive disorders, particularly those that are chronic or recurrent, when mothers
are younger or employed. It is important to identify processes through which temperamental
vulnerabilities are related to pathological outcomes, including cognitive (Hamburg, 1998;
Hayden et al., 2006) and biological mechanisms (Shankman et al., 2005). Conversely, the
present study also suggests that the same risk factor may play a role in the development of other
problematic behaviors. Offspring of older, working, chronically depressed mothers had very
high levels of PA and impulsivity, which resembled disinhibition. These children may be at risk
for externalizing disorders (Hirschfeld et al., 2003).

The present study had a number of strengths, including a large community based sample
of children at a homogenous developmental level with mothers that had meaningful
heterogeneity with respect to age and employment status. Additionally, direct diagnostic
interviews were conducted with both biological parents and intensive observational measures
were used, rather than parent report questionnaires. The behavioral observation measures
allowed me to examine dimensions of temperament relatively purely, such that measures of BI
did not overlap with measures of PE. However, the study was limited by a number of factors.
First, the assessment of maternal employment was not comprehensive. I assessed only whether
mothers were currently working and how many hours they were working, but did not ask about
when they began working following the birth of the child, whether they were employed
continuously, or reasons for working. These factors may have served as important mediators of
the influence of maternal employment on offspring temperament. Second, although I was able to
identify two meaningful contextual factors that moderated the relationship between maternal
depressive disorder and offspring behavior, it is important to continue to examine contextual and biological factors that may moderate or mediate the relationship between parental psychopathology and personality and offspring behavior. Third, although the sample was large, the prevalence of specific anxiety disorders was quite low. This may have accounted for some differences in findings between the present study and previous work that has focused on panic disorder as being related to offspring BI (Biederman et al., 2001). Likewise, my index of parental externalizing disorder was SUD, which was most indicated by alcohol use disorders. Associations between parental externalizing disorders and offspring temperament may be present for more severe forms of externalizing disorders, including hard drug dependence or antisocial personality disorder. Fourth, it is possible that a number of the significant interactions may have been found due to chance. However, I examined the interactions in an independent sample and found some similarity between the results and interpretations. Fifth, I results came from behavioral observations of the children at a single laboratory visit. The external validity of this assessment may be questionable. However, previous work has demonstrated reasonable associations between home and laboratory observations (Durbin et al., 2007) of child behavior. Additionally, stability of laboratory assessed PE related constructs was moderate from age 3 to 7 (Durbin et al., 2007). Laboratory observations of temperament are also indirect, as observable behaviors which are thought to reflect temperament are assessed, rather than temperament itself. It is important to consider that the behaviors that were used to define temperament are, in fact, appropriate proxies.

The present study found support for the hypothesis that maternal depressive disorders, particularly chronic/recurrent forms, are associated with low PE behaviors, including low PA and interest. However, this association was present only for offspring of non-working mothers and
offspring of younger mothers. Replication of these findings outside of our own research group is especially important. It is important for studies to examine the developmental outcomes of children with low levels of PE to bolster the claim that low PE serves as a risk factor for depressive disorders. Finally, much of the work on maternal employment status and age has utilized cross-sectional designs, which do not allow for examinations of changes in the effects of these contextual factors. Thus, it is important to conduct longitudinal studies that focus on maternal and offspring behavior as a function of changes of maternal employment status and age. Studies of this type would make it possible to shed light on the processes that account for the differential relationships between maternal depressive disorders and based on age and employment.
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Table 1. Demographic and Contextual Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>N (%) / M (SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female Child</td>
<td>163 (43.2)</td>
</tr>
<tr>
<td>Child Race</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>346 (86.9)</td>
</tr>
<tr>
<td>Racial Minorities</td>
<td>52 (13.0)</td>
</tr>
<tr>
<td>Live with biological mother</td>
<td>397 (99.7)</td>
</tr>
<tr>
<td>Live with biological father</td>
<td>379 (95.2)</td>
</tr>
<tr>
<td>Maternal Age</td>
<td>35.9 (4.51)</td>
</tr>
<tr>
<td>Mother work outside the home</td>
<td>192 (48.5)</td>
</tr>
<tr>
<td>Mothers – hours worked</td>
<td>28.2 (12.62)</td>
</tr>
<tr>
<td>Mothers – full time</td>
<td>62 (32.5)</td>
</tr>
<tr>
<td>Paternal Age</td>
<td>38.1 (5.27)</td>
</tr>
<tr>
<td>Father work outside the home</td>
<td>375 (97.2)</td>
</tr>
<tr>
<td>Fathers – hours worked</td>
<td>47.0 (11.34)</td>
</tr>
<tr>
<td>Fathers – full time</td>
<td>323 (93.6)</td>
</tr>
<tr>
<td>Parental Education</td>
<td></td>
</tr>
<tr>
<td>Neither Mother or Father BA/BS</td>
<td>121 (31.6)</td>
</tr>
<tr>
<td>Either Mother or Father BA/BS</td>
<td>133 (34.7)</td>
</tr>
<tr>
<td>Both Mother and Father BA/BS</td>
<td>129 (33.7)</td>
</tr>
<tr>
<td>Family Income</td>
<td></td>
</tr>
<tr>
<td>&lt; $70,000</td>
<td>116 (29.9)</td>
</tr>
<tr>
<td>$70,001 - $100,000</td>
<td>132 (34.0)</td>
</tr>
<tr>
<td>Income Level</td>
<td>Marital Satisfaction</td>
</tr>
<tr>
<td>------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td>&gt; $100,000</td>
<td>140 (36.1)</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a N and percentage is reported.  b Mean and standard deviation are reported. Marital Satisfaction as reported by the DAS. PPVT = Peabody Picture Vocabulary Test; EOWPVT = Expressive One-Word Picture Vocabulary Test.
Table 2. Internal Consistency and Inter-rater Reliability Estimates for Observed Child Behavior Scales

<table>
<thead>
<tr>
<th></th>
<th>PA$^a$</th>
<th>Interest</th>
<th>Sociability</th>
<th>Fear$^a$</th>
<th>Sadness$^a$</th>
<th>Anger$^a$</th>
<th>BI$^a$</th>
<th>Impulsivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA</td>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interest</td>
<td>.62***</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sociability</td>
<td>.49***</td>
<td>.58***</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear</td>
<td>.03</td>
<td>-.14**</td>
<td>.10$^+$</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sadness</td>
<td>-.09</td>
<td>-.35***</td>
<td>-.09</td>
<td>.17**</td>
<td>---</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td>.07</td>
<td>-.04</td>
<td>.11*</td>
<td>.04</td>
<td>.39***</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BI</td>
<td>-.07</td>
<td>-.32***</td>
<td>-.15**</td>
<td>.60***</td>
<td>.31***</td>
<td>.00</td>
<td></td>
<td>---</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>.27***</td>
<td>.16**</td>
<td>.31***</td>
<td>-.01</td>
<td>.10$^+$</td>
<td>.32***</td>
<td>-.10$^*$</td>
<td>---</td>
</tr>
<tr>
<td>PPVT</td>
<td>.07</td>
<td>.19***</td>
<td>.26***</td>
<td>.01</td>
<td>.03</td>
<td>.01</td>
<td>-.08</td>
<td>-.21***</td>
</tr>
<tr>
<td>EOWPVT</td>
<td>-.01</td>
<td>.13*</td>
<td>.17**</td>
<td>-.01</td>
<td>-.03</td>
<td>-.05</td>
<td>-.06</td>
<td>-.16**</td>
</tr>
<tr>
<td>M</td>
<td>.01</td>
<td>1.93</td>
<td>1.61</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.00</td>
<td>.73</td>
</tr>
<tr>
<td>SD</td>
<td>.79</td>
<td>.35</td>
<td>.45</td>
<td>.83</td>
<td>.85</td>
<td>.80</td>
<td>.41</td>
<td>.34</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>.86</td>
<td>.66</td>
<td>.82</td>
<td>.64</td>
<td>.73</td>
<td>.65</td>
<td>.81</td>
<td>.70</td>
</tr>
<tr>
<td>ICC</td>
<td>.93</td>
<td>.82</td>
<td>.91</td>
<td>.86</td>
<td>.83</td>
<td>.56</td>
<td>.87</td>
<td>.82</td>
</tr>
</tbody>
</table>
$^{+}p < .07; \, * \, p < .05; \, ** \, p < .01; \, *** \, p < .001$. \textsuperscript{a} Denotes that the variable was derived based on standardization. ICC = Intraclass Correlation. PPVT = Peabody Picture Vocabulary Test; EOWPVT = Expressive One-word Picture Vocabulary Test. Standard Scored Were used for these associations.
### Table 3. Principal Components Solution of the Dimensions of Child Behavior

<table>
<thead>
<tr>
<th>Behavior</th>
<th>PE</th>
<th>Fearful Inhibition</th>
<th>NE</th>
</tr>
</thead>
<tbody>
<tr>
<td>PA</td>
<td>.74</td>
<td>.12</td>
<td>.07</td>
</tr>
<tr>
<td>Interest</td>
<td>.85</td>
<td>-.08</td>
<td>-.16</td>
</tr>
<tr>
<td>Sociability</td>
<td>.73</td>
<td>.09</td>
<td>.12</td>
</tr>
<tr>
<td>Fear</td>
<td>.16</td>
<td>.72</td>
<td>-.01</td>
</tr>
<tr>
<td>BI</td>
<td>-.03</td>
<td>.91</td>
<td>-.05</td>
</tr>
<tr>
<td>Sadness</td>
<td>-.18</td>
<td>.18</td>
<td>.53</td>
</tr>
<tr>
<td>Anger</td>
<td>.07</td>
<td>-.10</td>
<td>.74</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>.31</td>
<td>-.08</td>
<td>.43</td>
</tr>
</tbody>
</table>

*Note:* Factor loadings in bold represent the manner in which broad domain scores were computed.
Table 4. Sex Differences in Behavioral Observations of Child Behavior

<table>
<thead>
<tr>
<th></th>
<th>Female</th>
<th>Male</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$(n = 163)$</td>
<td>$(n = 214)$</td>
<td></td>
</tr>
<tr>
<td>PE</td>
<td>.10 (.80)</td>
<td>-.07 (.76)</td>
<td>4.42*</td>
</tr>
<tr>
<td>PA</td>
<td>.09 (.85)</td>
<td>-.05 (.73)</td>
<td>3.09</td>
</tr>
<tr>
<td>Interest</td>
<td>1.96 (.33)</td>
<td>1.91 (.36)</td>
<td>1.96</td>
</tr>
<tr>
<td>Sociability</td>
<td>1.67 (.47)</td>
<td>1.57 (.42)</td>
<td>4.92*</td>
</tr>
<tr>
<td>Fearful Inhibition</td>
<td>.10 (.60)</td>
<td>-.08 (.52)</td>
<td>6.85**</td>
</tr>
<tr>
<td>Fear</td>
<td>.13 (.89)</td>
<td>-.11 (.76)</td>
<td>7.63**</td>
</tr>
<tr>
<td>BI</td>
<td>.07 (.45)</td>
<td>-.05 (.38)</td>
<td>8.97**</td>
</tr>
<tr>
<td>NE</td>
<td>-.09 (.61)</td>
<td>.08 (.64)</td>
<td>9.78**</td>
</tr>
<tr>
<td>Sadness</td>
<td>.00 (.98)</td>
<td>-.00 (.75)</td>
<td>.02</td>
</tr>
<tr>
<td>Anger</td>
<td>-.05 (.74)</td>
<td>.05 (.84)</td>
<td>1.55</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>.65 (.29)</td>
<td>.79 (.36)</td>
<td>16.99***</td>
</tr>
</tbody>
</table>

* $p < .05$; ** $p < .01$; *** $p < .001$. 


Table 5. Child Behaviors as a Function of Maternal History of Depressive Disorders and Influence of Moderators

<table>
<thead>
<tr>
<th>Child Behavior</th>
<th>No Depr. Disorder</th>
<th>Single Depressive Episode</th>
<th>Chronic/Recurrent Depressive Dx.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 257)</td>
<td>(n = 47)</td>
<td>(n = 70)</td>
</tr>
<tr>
<td>PE</td>
<td>.01 (.79)</td>
<td>.03 (.67)</td>
<td>-.02 (.83)</td>
</tr>
<tr>
<td>Non-working Mothers</td>
<td>-.04 (.78)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.07 (.69)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>-.38 (.72)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Working Mothers</td>
<td>.08 (.74)</td>
<td>.01 (.69)</td>
<td>.38 (.81)</td>
</tr>
<tr>
<td>Younger Mothers</td>
<td>.11 (.75)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.19 (.68)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.20 (.77)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Older Mothers</td>
<td>-.07 (.81)</td>
<td>-.14 (.64)</td>
<td>.12 (.86)</td>
</tr>
<tr>
<td>PA</td>
<td>.01 (.77)</td>
<td>.07 (.72)</td>
<td>-.01 (.88)</td>
</tr>
<tr>
<td>Non-working Mothers</td>
<td>-.02 (.78)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.14 (.81)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.38 (.56)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Working Mothers</td>
<td>.06 (.74)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.04 (.72)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>.43 (1.04)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Younger Mothers</td>
<td>.05 (.72)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.24 (.69)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>-.27 (.63)&lt;sup&gt;b&lt;/sup&gt;</td>
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<tr>
<td>Older Mothers</td>
<td>-.02 (.81)</td>
<td>-.11 (.73)</td>
<td>.19 (1.00)</td>
</tr>
<tr>
<td>Interest</td>
<td>1.94 (.35)</td>
<td>1.93 (.28)</td>
<td>1.88 (.36)</td>
</tr>
<tr>
<td>Non-working Mothers</td>
<td>1.95 (.33)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.93 (.19)&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>1.75 (.35)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Working Mothers</td>
<td>1.93 (.34)</td>
<td>1.92 (.32)</td>
<td>2.03 (.34)</td>
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<tr>
<td>Sociability</td>
<td>1.60 (.46)</td>
<td>1.62 (.39)</td>
<td>1.64 (.42)</td>
</tr>
<tr>
<td>Fearful Inhibition</td>
<td>-.03 (.56)</td>
<td>.04 (.66)</td>
<td>.07 (.50)</td>
</tr>
<tr>
<td>Younger Mothers</td>
<td>-.14 (.49)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.05 (.53)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>.15 (.58)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Older Mothers</td>
<td>-.06 (.81)</td>
<td>-.14 (.64)</td>
<td>.12 (.86)</td>
</tr>
<tr>
<td></td>
<td>Fear</td>
<td>Younger Mothers</td>
<td>Older Mothers</td>
</tr>
<tr>
<td>----------------------</td>
<td>------------</td>
<td>-----------------</td>
<td>---------------</td>
</tr>
<tr>
<td></td>
<td>-.05 (.81)</td>
<td>-.18 (.71)(^a)</td>
<td>.04 (.86)</td>
</tr>
<tr>
<td></td>
<td>.09 (1.03)</td>
<td>.11 (.77)(^a,b)</td>
<td>.07 (1.27)</td>
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<tr>
<td></td>
<td>.09 (.74)</td>
<td>.22 (.85)(^b)</td>
<td>.00 (.63)</td>
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Different superscripts denote significant differences at \( p < .05 \).
Table 6. Child Behaviors as a Function of Maternal History of Non-Affective Disorders

<table>
<thead>
<tr>
<th>Child Behavior</th>
<th>Maternal Anxiety Disorder</th>
<th>Maternal SUD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>(n = 241)</td>
<td>(n = 133)</td>
</tr>
<tr>
<td><strong>PE</strong></td>
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<td></td>
</tr>
<tr>
<td>PA</td>
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<td>.01 (.81)</td>
</tr>
<tr>
<td>Interest</td>
<td>1.94 (.36)</td>
<td>1.91 (.33)</td>
</tr>
<tr>
<td>Sociability</td>
<td>1.64 (.45)</td>
<td>1.56 (.44)</td>
</tr>
<tr>
<td><strong>Fearful</strong></td>
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<td></td>
</tr>
<tr>
<td>Inhibition</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fear</td>
<td>.02 (.88)</td>
<td>-.04 (.72)</td>
</tr>
<tr>
<td>BI</td>
<td>-.00 (.43)</td>
<td>.01 (.39)</td>
</tr>
<tr>
<td><strong>NE</strong></td>
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<td></td>
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<tr>
<td>Sadness</td>
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<td>.01 (.74)</td>
</tr>
<tr>
<td>Anger</td>
<td>.01 (.79)</td>
<td>-.01 (.82)</td>
</tr>
<tr>
<td>Impulsivity</td>
<td>.72 (.34)</td>
<td>.73 (.34)</td>
</tr>
</tbody>
</table>

Different superscripts denote significant differences at $p < .05$. 
Table 7. Child Behavior and Paternal Psychopathology

<table>
<thead>
<tr>
<th>Paternal Depressive Disorder</th>
<th>No Depr. Disorder</th>
<th>Single Depressive Episode</th>
<th>Chronic/Recurrent Depressive Dx.</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 250)</td>
<td>(n = 20)</td>
<td>(n = 29)</td>
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<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
</tr>
<tr>
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<td>-.05 (.68)</td>
</tr>
<tr>
<td>PA</td>
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<td>.23 (.86)</td>
<td>.06 (.86)</td>
</tr>
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<td>Interest</td>
<td>1.95 (.35)</td>
<td>1.90 (.26)</td>
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<td>Sociability</td>
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<td>1.53 (.53)</td>
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<tr>
<td>Fearful Inhibition</td>
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<td>.15 (.69)</td>
<td>.03 (.49)</td>
</tr>
<tr>
<td>Fear</td>
<td>-.04 (.82)</td>
<td>.17 (1.05)</td>
<td>.02 (.74)</td>
</tr>
<tr>
<td>BI</td>
<td>-.03 (.39)</td>
<td>.14 (.47)</td>
<td>.03 (.33)</td>
</tr>
<tr>
<td>NE</td>
<td>-.01 (.65)</td>
<td>.07 (.52)</td>
<td>-.06 (.52)</td>
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<tr>
<td>Sadness</td>
<td>-.00 (.91)</td>
<td>-.07 (.48)</td>
<td>-.10 (.77)</td>
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<tr>
<td>Anger</td>
<td>-.03 (.82)</td>
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<td>-.15 (.62)</td>
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<td>Impulsivity</td>
<td>.73 (.34)</td>
<td>.78 (.28)</td>
<td>.75 (.38)</td>
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</table>

<table>
<thead>
<tr>
<th>Paternal Anxiety Disorder</th>
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<th>+</th>
<th>Paternal SUD</th>
<th>-</th>
<th>+</th>
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<tr>
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<td>(n = 112)</td>
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<tr>
<td>Child Behavior</td>
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<td>M (SD)</td>
<td>M (SD)</td>
<td>M (SD)</td>
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<td>.04 (.79)</td>
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<td>PA</td>
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<td>.17 (.80)</td>
<td>.03 (.77)</td>
<td>.04 (.82)</td>
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<td>------</td>
<td>------</td>
<td>-------</td>
<td>------</td>
<td>------</td>
</tr>
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<td>Interest</td>
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<td>-.01 (.56)</td>
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<td>-.06 (.50)</td>
<td>.06 (.66)(^a)</td>
<td>-.12 (.56)(^b)</td>
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<tr>
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<tr>
<td>Impulsivity</td>
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<td>.76 (.35)</td>
<td>.76 (.34)</td>
<td>.69 (.35)</td>
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Different superscripts reflect significant differences at \( p < .05 \).
Table 8. Associations Between Child Behavior and Parental Personality

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<th>F.I.</th>
<th>Fear</th>
<th>BI</th>
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<th>Anger</th>
<th>Impul.</th>
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<td>.01</td>
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<td>.05</td>
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<td>-.11</td>
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<td>-.04</td>
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<td>-.02</td>
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<td>-.06</td>
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<td>.06</td>
<td>.06</td>
<td>.04</td>
<td>-.02</td>
<td>.01</td>
<td>.01</td>
<td>-.05</td>
</tr>
</tbody>
</table>

*p < .05; ** p < .01. Int. = interest; Soc. = sociability; F.I. = fearful inhibition; Impul. = Impulsivity.
Figure Captions

Figure 1. Illustration of the precursor model. For the precursor model to be supported, path ‘a’ should remain significant after including the association between parental personality and offspring temperament.

Figure 2. Illustration of the predisposition model. For the predisposition model to be supported, path ‘a’ should become non-significant after including the association between parental personality and offspring temperament and path ‘b’ should be significant.

Figure 3. Child PA as a function of maternal depressive history, employment, and age. No Depr = no history of maternal depressive disorder; SDE = maternal history of a single, non-chronic, depressive episode; Chr/Rec = maternal history of a chronic and/or recurrent depressive disorder; Young = maternal age is less than 36 years; Older = maternal age is greater than or equal to 36 years; Not working = child’s mother is not employed outside the home; Working = child’s mother is employed outside the home.

Figure 4. Child impulsivity as a function of maternal depressive history and age for children of working mothers. No Depr = no history of maternal depressive disorder; SDE = maternal history of a single, non-chronic, depressive episode; Chr/Rec = maternal history of a chronic and/or recurrent depressive disorder; Young = maternal age is less than 36 years; Older = maternal age is greater than or equal to 36 years.
Figure 1.
Figure 2.

Parental Psychopathology

Parental Personality

Offspring Temperament

a

b
Figure 3.
Figure 4.
Appendix 1. Child Behaviors as a Function of Maternal History of Depressive Disorders and Influence of Moderators from Durbin et al. (2005)

<table>
<thead>
<tr>
<th>Child Behavior</th>
<th>No Depr. Disorder</th>
<th>Single Depressive Episode</th>
<th>Chronic/Recurrent Depressive Dx.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 66)</td>
<td>(n = 9)</td>
<td>(n = 25)</td>
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<tr>
<td>PA Non-working Mothers</td>
<td>1.97 (.38)\textsuperscript{a}</td>
<td>1.71 (.39)\textsuperscript{a,b}</td>
<td>1.58 (.29)\textsuperscript{b}</td>
</tr>
<tr>
<td>Working Mothers</td>
<td>1.78 (.39)</td>
<td>1.77 (.59)</td>
<td>1.60 (.43)</td>
</tr>
<tr>
<td>Younger Mothers</td>
<td>1.86 (.39)\textsuperscript{a}</td>
<td>2.19 (.30)\textsuperscript{a}</td>
<td>1.49 (.31)\textsuperscript{b}</td>
</tr>
<tr>
<td>Older Mothers</td>
<td>1.84 (.41)</td>
<td>1.47 (.31)</td>
<td>1.68 (.39)</td>
</tr>
<tr>
<td>Interest Non-working Mothers</td>
<td>2.19 (.31)\textsuperscript{a}</td>
<td>1.90 (.37)\textsuperscript{a,b}</td>
<td>1.95 (.27)\textsuperscript{b}</td>
</tr>
<tr>
<td>Working Mothers</td>
<td>2.08 (.38)</td>
<td>1.98 (.28)</td>
<td>2.04 (.26)</td>
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<tr>
<td>Fear Younger Mothers</td>
<td>1.60 (.19)</td>
<td>1.44 (.17)</td>
<td>1.62 (.22)</td>
</tr>
<tr>
<td>Older Mothers</td>
<td>1.66 (.26)\textsuperscript{a,b}</td>
<td>1.49 (.09)\textsuperscript{a}</td>
<td>1.77 (.31)\textsuperscript{b}</td>
</tr>
</tbody>
</table>

Different superscripts denote significant differences for post-hoc contrasts at $p < .05$. Maternal age is dichotomized based on a median split. Maternal depression X maternal age is associated with offspring PA, $F (2, 99) = 4.03, p < .05$; and not associated with offspring fear, $F (2, 99) = .49, p = .61$. Maternal depression X maternal employment is not associated with offspring PA, $F (2, 99) = .83, p = .43$; and not associated with offspring interest, $F (2, 99) = .91, p = .40$. 


Appendix 2. Associations Between Child Behavior and Maternal Personality as a Function of Maternal Employment and Age From Unpublished Data

<table>
<thead>
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<th></th>
<th>PA</th>
<th>Interest</th>
<th>Sociability</th>
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<tbody>
<tr>
<td>NE</td>
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<tr>
<td>Non-Working</td>
<td>.03</td>
<td>.10</td>
<td>-.04</td>
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<td>Constraint</td>
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<tr>
<td>Younger Mothers</td>
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<td>Older Mothers</td>
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*p < .05; **p < .01. Maternal age is dichotomized based on a