Temperamental Emotionality in Preschoolers and Parental Mood Disorders

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A number of models developed in the adult psychopathology literature (i.e., L. A. Clark & D. Watson, 1991) have asserted that low levels of positive emotionality (PE) are predisposing factors or precursors for depression and represent a form of temperamental risk for depression. Further support for this claim would derive from evidence linking low PE to known indicators of risk for depression. The authors examined the association between temperamental emotionality in young children and parental mood disorders. One hundred unselected preschool-aged children completed a battery of emotion-eliciting tasks tapping aspects of PE, negative emotionality (NE), and behavioral inhibition (BI). Parental psychopathology was assessed with semistructured diagnostic interviews. Low PE in children was associated with maternal, but not paternal, mood disorder. The low PE-maternal depression link was relatively specific, as there were few associations between low PE and other forms of parental psychopathology or between NE and BI and parental mood disorders.

The term temperament is used to refer to patterns of behavioral and emotional reactivity that are relatively stable across time and situation and are rooted in part in early emerging individual differences in biological systems. Temperament traits are described as shaping adaptation (or maladaptation) to environmental circumstances (Clark & Watson, 1999; Rothbart & Bates, 1998), making the temperament paradigm a compelling heuristic for models of psychopathological risk. Understanding risk mechanisms associated with early appearing temperament traits could help clarify some of the key issues in the etiology and classification of psychopathology. Common temperament constellations could explain the pattern of comorbidity among classes of disorder as well as familial coaggregation of disorders and could point toward more etiologically relevant classification schemes (i.e., Clark & Watson, 1991; Krueger, 1999).

There is a long lineage of psychopathologists interested in temperamental differences in mood as expressions of a diathesis for depression (Akiskal, 1989; Kraepelin, 1921). Drawing upon modern temperament models emphasizing the centrality of individual differences in positive and negative emotionality (e.g., Clark, Watson, & Mineka, 1994; Rothbart & Bates, 1998), theorists from both personality and biological paradigms have identified trait low positive emotionality (PE) as a potential substrate of mood disorders. These models have focused on the propensity to experience positive mood states (Clark & Watson, 1999), hedonic capacity (Meehl, 1975), pleasure (D. F. Klein, 1987), and behavioral-motivational approach systems governing incentive behavior (i.e., Gray’s [1990] Behavioral Activation System [BAS]; Depue & Collins, 1999; Davidson, 1992). Although primarily descriptive in nature, these models typically assert that low PE conveys risk through the alteration of mood states, cognition (particularly expectation for rewarding outcomes contingent upon behavior), and behavior (chiefly goal-directed behavior).

The most prominent temperament model of mood disorders was proposed by Clark and Watson (1991), who described two independent temperament superfactors: Positive and Negative Affectivity (later renamed Positive and Negative Emotionality, or PE and NE; Clark & Watson, 1999). PE (which is closely related to extraversion) refers to a propensity for positive affect and cognitions and high levels of perceived stress. On the basis of cross-sectional and longitudinal research on the correlates of these self-reported traits, Clark and Watson (1991) developed a tripartite model of mood and anxiety disorders, including the factors of PE, NE, and physiological overarousal. The tripartite model proposed that high NE characterizes both anxiety and depression, whereas low PE is specific to depression and physiological overarousal is specific to anxiety. Cross-sectional studies of adult clinical and nonclinical samples using self-report measures of positive and negative mood generally support the tripartite model’s claims, including the specificity of low PE to depression (Brown, Chorpita, & Barlow, 1998; Watson, Clark, & Carey, 1998). The smaller child self-report literature, based primarily upon inpatient samples, also supports the tripartite model (Chorpita & Daleiden, 2002; Lonigan, Carey, & Finch, 1994). Most of these studies, however, have focused more on discriminating depressive from anxiety symptoms than on exploring the
origins of individual differences in trait emotionality and its role in the development of psychopathology.

Although the majority of research linking PE and NE to depression has consisted of cross-sectional studies of adult samples, there is a smaller longitudinal literature examining the predictive validity of child temperament for the later development of depression. Caspi, Moffitt, Newman, and Silva (1996) reported that behavioral ratings at age 3 were associated with depression in young adulthood. Specifically, a cluster of children rated as inhibited, socially reticent, and fearful of novelty at age 3 had an elevated rate of mood (but not anxiety) disorders at age 21. In a birth cohort study, van Os, Jones, Lewis, Wadsworth, and Murray (1997) found that childhood behavioral apathy predicted both childhood onset depression and chronic depression in adulthood. Finally, low extraversion in childhood has shown prospective links to subsyndromal depression at age 18 (Block, Gjerde, & Block, 1991). Thus, there is suggestive evidence that temperamental behaviors consistent with low PE and high NE, measured in childhood, are prospectively associated with the development of depression.

The processes by which temperamental emotionality increases risk for mood disorders have not been well delineated. However, there are a number of plausible models (Rothbart, Posner, & Hershey, 1995). First, PE and NE may bias information processing, shaping the development of depressogenic cognitive styles (Davidson et al., 2002; Depue & Collins, 1999). Second, PE and NE may influence reactivity to the environment, which may enhance or attenuate the development of mastery and competence (Frederickson, 2001). For example, individuals with low PE may be less responsive or may be responsive to a smaller range of potentially rewarding stimuli, may demonstrate less effort in the pursuit of reward, and may experience less facilitation of approach behavior in pursuing abstract or delayed goals (Depue & Collins, 1999). Third, the effects of stress may be buffered or potentiated by PE (Meehl, 1975), NE (Rutter et al., 1997), or both. For example, Lengua, Wolchik, Sandler, and West (2000) found that child low PE and a rejecting parenting style interacted to predict child adjustment following parental divorce. Fourth, evocative and niche-picking processes associated with temperament traits may influence the likelihood that individuals will experience various environmental contributors to risk for depression. For example, low PE children may elicit fewer protective factors, such as positive relationships with peers, and high NE children may elicit harsher parenting (Rutter, 1990). Finally, some temperamental extremes may in themselves be precursors, or early forms, of mood disorders (Akiskal, 1989).

The present study provides a further test of the validity of temperamental models of risk for depression by examining the association between temperamental emotionality in preschool aged children and a history of mood disorder in their parents. We chose to focus in particular on PE, rather than other traits, because of the confluence of theoretical perspectives emphasizing its centrality to mood disorders and evidence for its specificity to depression. The study design was based on the rationale that if low PE reflects one pathway or predisposition to the development of mood disorders, then it should be correlated with known risk factors for depression that may be part of the same etiological chain. One of the better-established risk factors for depression is parental mood disorder (Downey & Coyne, 1990; Goodman & Gotlib, 1999). Moreover, many theorists believe that the intergenerational transmission of depression is mediated by the transmission of temperament traits (Costello et al., 2002; Silberg & Rutter, 2002). Thus, we hypothesized that low PE in young children would be associated with a history of depression in their parents. This study was not designed to examine the processes that account for the association between child temperament and parental depression, as we believe that it is important to first establish that such an association exists. Further research will then be necessary to delineate the genetic and environmental pathways through which parental depression influences child temperament, to determine whether children with low levels of PE are indeed at elevated risk for developing mood disorders, and to explore the processes through which temperament increases risk for depression.

This study was designed to extend the existing literature on temperament and depression in three additional respects. First, most studies of psychopathology and emotionality have used school-age children, adolescents, or adults, many of whom have already experienced depressive symptoms or disorders. We chose to focus instead on the preschool age period, an optimal one in which to observe temperamental emotionality without being confounded by concurrent depression, as temperament has begun to stabilize during this period (Caspi, 2000), but depressive disorders are rarely evident prior to middle childhood (Garber & Horowitz, 2002).

Second, most studies of child temperament have assessed traits through the use of self- or parent reports. However, self-reports are of limited utility in early childhood, and parent reports of child temperament and behavior have typically shown poor convergent validity with teacher reports and observational and laboratory measures (Seifer, Sameroff, Barrett, & Krafchuk, 1994), poorer predictive validity for later psychological adjustment (Mesman & Koot, 2000), and evidence of dysphoria-related bias (Youngstrom, Izard, & Ackerman, 1999). Thus, we felt that the literature on temperament models of risk would be bolstered by the use of observational measures, rather than sole reliance upon parent or child report (Chorpita & Daleiden, 2002).

Finally, although low PE has been proposed to be specific to depression (Clark & Watson, 1991), the validity of this claim remains unresolved. Low PE has also been suggested as a diathesis for schizophrenia (Meehl, 1962) and has been linked to social phobia (Brown et al., 1998), whereas high PE has been implicated in substance abuse (Zuckerman, 1994) and disruptive behavior disorders (Cole & Zahn-Waxler, 1994). In addition, other temperament dimensions have been hypothesized to predispose to mood disorders. High NE has been viewed as a risk factor for most forms of psychopathology, including depression (Clark et al., 1994). Moreover, behavioral inhibition (BI), an important construct from the child temperament literature that has not been well integrated into adult temperament models, may also be related to risk for mood disorders.

BI is a lower-order trait defined by wariness, fear, and low exploration in novel situations (Kagan, 1997). It shares low behavioral approach with PE and anxiety with NE (Nigg, 2000) and is also related to constraint, which was defined by Tellegen (1985) as a nonaffective factor including low impulsivity, harm avoidance, and traditionalism. It is unclear if BI is solely a lower order facet or developmental precursor of constraint or if it is a complex mixture of PE, NE, and constraint. For instance, Caspi et al. (2003) found that children rated as inhibited at age 3 reported both low
levels of PE and high levels of constraint at age 26. Several studies have reported associations between parental depression and BI in offspring, although the effects may be due to comorbid anxiety disorders in the parents (Kochanska, 1991; Rosenbaum et al., 2000). Because BI and PE have not been examined simultaneously with regard to children’s risk for depression, we chose to also examine the association between BI and parental history of depression.

In summary, the present study examined the association between laboratory measures of PE, NE, and BI and parental lifetime psychopathology in a community sample of preschool aged children. We addressed three questions: First, is low PE associated with parental mood disorders? Second, is the low PE-parental psychopathology association specific to mood disorders, or is low PE also associated with other forms of psychopathology? Third, is the temperament emotionality-parental mood disorder association specific to low PE, or are NE and BI also associated with parental mood disorder? In addition, we explored, in a preliminary fashion, the role of several factors that have been identified as potential mediators or confounding factors in the child low PE-parental mood disorder relationship (Downey & Coyne, 1990; Cicchetti, Rogosch, & Toth, 1998), including parental behavior, socioeconomic status (SES) and marital discord, and exposure to parental depressive symptoms. These factors were included to provide an initial test of whether low PE in children is associated with parental history of depression because of the effects of environmental risk factors that may be correlated with parental depression.

Method

Participants

Recruitment. The final sample of participants included 100 children between the ages of 3 years, 0 months, and 4 years, 1 month, and their biological parents. An additional six participants were excluded: Two children had significant medical or psychiatric disabilities, and four families completed only the laboratory assessment. Two recruitment strategies were used: contacting families in local zip codes via a commercial mailing list (51.9%) and requesting volunteers for a study of children’s personality through ads in local newspapers and preschools (48.1%). Participants obtained through the two methods did not differ on any of the child temperament or parental psychopathology variables used in this study.

Children were administered the Peabody Picture Vocabulary Test (PPVT; Dunn & Dunn, 1997) to screen for gross cognitive impairment ($M = 103.5, SD = 13.9$). All families were compensated financially.

Demographics. The sample was mostly White (85.0%) and working or middle class, with a mean score of 34.9 ($SD = 9.9$) on Hollingshead’s Four Factor Index of Social Status (Hollingshead, 1975). The mean age of parents was 33.6 years for mothers ($SD = 4.3$) and 36.4 ($SD = 5.2$) for fathers. The mean age of child participants was 3.6 years ($SD = 0.3$), and 45% were female. The vast majority (97.0%) of the children came from two-parent homes, and 58.2% of the mothers worked outside the home part- or full-time. 7 tests comparing boys and girls on temperament traits yielded significant differences on two variables: Boys exhibited higher levels of anger, and girls exhibited higher levels of sociability.

Laboratory Assessment of Child Temperament

The laboratory assessment lasted approximately 2 hr, during which children were videotaped while participating with a female experimenter in 12 standardized tasks selected from the Laboratory Temperament Assess-
advantage of capturing instances of normative behavior that were not specifically predicted by the design of the task as well as increasing the number of ratings, which allowed for more reliable estimates.

Intercorrelations between the micro and global scales measuring the same trait ranged from .51 (fear) to .83 (noncompliance), with a median r of .70. Analyses were conducted using both coding schemes, and the results were the same for each system. Hence, to conserve space, only the results using the global coding system (with one exception noted below) are reported here.

In the global coding system, discrete emotions (happiness-positive affect, anger, sadness, and fear) were assessed by separately coding facial, vocal, and bodily indicators of the emotion in each episode. These indicators were averaged to produce composite variables for each emotion. For example, the positive affect scale reflects the frequency and intensity of smiling, positive verbalizations, and joyful bodily movements. Other temperament traits (e.g., sociability, engagement) were coded on a single scale for each episode. The following scales were used in this report, each consisting of the average of codes across episodes: positive affect (α = .90), sociability (α = .81), engagement (α = .56), sadness (α = .67), anger (α = .75), fear (α = .59), and incongruous NE (a measure of context-inappropriate negative affect; α = .67). Median interrater reliability, indexed by the intraclass correlation coefficient (ICC), assessed on a subsample of 15 cases, was .82 (ranging from .66 for fear to .94 for positive affect).

Although most scales included ratings from all 12 episodes, episodes in which less than 15% of the sample displayed behaviors relevant to the particular trait were not included in the final composite scale for that trait. The engagement scale did not include stranger approach (the variable was not coded) or painting a picture episodes (because of weak intercorrelations with other episodes). The incongruous NE scale was intended to tap negative affective expressions that are unusual or inappropriate to the context in which they occur. It includes all NE variables in episodes designed to be exclusively positive and NE variables that are unusual for a particular negative episode (such as anger during stranger approach). This scale was composed of the following: facial fear in tower of patience, arc of toys, transparent box, snack delay, popping bubbles, and painting a picture. The average intercorrelation between this scale and the other global scales was .27, ranging from r = −.05 with positive affect to .63 with the Anger Scale.

Most previous studies of BI have used a micro coding approach using a small number of episodes specifically designed to elicit BI (e.g., risk room, stranger approach; Kagan, 1997). Although many of these micro coded variables reflect fearful affect, a number tap low approach behavior. Hence, in order to compare our findings to the literature on BI, we included the micro BI scale in addition to the global coding fear scale in the present report. The micro BI scale was an average of z-scored codes (α = .86; ICC = .85) from risk room and stranger approach. These included the following: tentative play, latency to verbalize, latency to touch objects, and total number of objects played with (risk room); gaze aversion and avoidance of the stranger (stranger approach); and fearful facial, vocal, and bodily affect (both episodes).

Data reduction. The eight coding variables (micro coding BI and global coding positive affect, fear, anger, sadness, incongruous NE, sociability, and engagement) were subjected to an exploratory principal components factor analysis with varimax rotation. Consistent with recent temperament conceptualizations and other factor analyses of child temperament components factor analysis with varimax rotation. Rothbart, Ahadi, & Evans, 2000) eigenvalues and a scree test suggested a three-factor solution. The first factor, accounting for 33.5% of the variance, was a PE factor, with high loadings for positive affect (.87), engagement (.85), and sociability (.79). The second factor, interpreted as NE, accounted for 22.8% of the variance and included ratings of anger (.90), incongruous NE (.84), and sadness (.52). The third factor was composed of fear (.83) and micro BI (.82), and it accounted for an additional 15.3% of the variance; we interpreted this third factor as reflecting inhibition. The PE scale correlated .07 with the NE scale and −.28 with the BI factor; the correlation between the NE and Inhibition factors was .26. Composite scores for each temperament factor were derived by averaging the variables that loaded on the factor.

Assessment of Parents

Diagnostic interviews with parents. Semistructured diagnostic interviews, using the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996), were conducted with 100% of the biological mothers and 91% of the biological fathers of the children. The SCID is among the most widely used diagnostic interviews, and its interrater reliability and procedural validity have been well documented (Williams et al., 1992). Information was collected from mothers on those fathers who failed to complete the SCID using the Family History Research Diagnostic Criteria interview guide (FH-RDC; Andreasen, Endicott, Spitzer, & Winokur, 1977). The diagnostic interviews were conducted by two masters-level clinicians who had completed a formal training course on the SCID, had been certified by expert SCID interviewers at other centers based on videotapes of the clinicians’ SCID interviews, had at least several years of experience conducting SCIDs in other studies, and had demonstrated high levels of interrater reliability on SCID and FH-RDC diagnoses in previous studies (D. N. Klein, Crosby-Ouimet, Salisbury-Kelly, Ferro, & Riso, 1994). The interviewers were not involved in collecting, and did not have access to, any of the data on the children.

Rates of Axis I lifetime diagnoses in parents are shown in Table 1; these rates are similar to those from recent epidemiological studies (Kessler et al., 1994). The rates of substance use disorders were somewhat high; however, the majority of these cases represented abuse that onset in late adolescence and remitted in early adulthood (the mean durations of alcohol and substance abuse disorders were < 3 years). With regard to mood disorders, all of the cases of major depressive disorder (MDD) were past diagnoses; no parent met criteria for current major depressive episode. Three mothers, but no fathers, met criteria for current dysthymic disorder (DD).

Home observation ratings of maternal behavior. As part of a separate assessment, each child was observed during two independent home visits by pairs of graduate and undergraduate raters who did not have access to laboratory or interview data. A different pair of raters conducted the second visit, without knowledge of the first home visit data. Among a battery of ratings, observers completed a scale (Goldsmith, 1995) tapping aspects of the mothers’ affective behavior in interaction with her child. For this report, we used ratings of maternal pleasure and criticism, each rated on a 0–5 scale. All four ratings (two raters from each visit) were averaged to create composite scores. Data are available for 99 children, as one family did not participate in the second home visit. For pleasure (M = 3.26, SD = 0.89) and criticism (M = 3.52, SD = 0.69), interrater reliability (ICC) was .66 and .64, respectively, and test-retest stability from the first to second observation (ICC) was .50 and .46.

Parental marital satisfaction and current depressive symptoms. From a self-report battery completed by 99% of the mothers and 80% of the fathers, we selected the Dyadic Adjustment Scale (DAS; Spanier, 1976; α = .91) to measure relationship discord and the Inventory to Diagnose Depression (IDD; Zimmerman, Coryell, Corenthal, & Wilson, 1986; α = .81) to measure current depressive symptoms (M = 6.76, SD = 5.66). The DAS was not available for four children (one due to missing questionnaire data; three because the mother was not currently in a relationship; M = 77.31, SD = 10.34).
Results

Associations Between Child Temperament and Parental Depression

Our primary analyses focused on the association between the PE, NE, and Inhibition factors and maternal and paternal depressive disorders (defined as a lifetime history of meeting diagnostic criteria for either MDD or DD). However, in light of evidence that lower-order factors can contribute unique predictive information (Paunonen, 1998; Reynolds & Clark, 2001), we also conducted exploratory analyses of the specific temperament scales. The correlations between parental depression and child temperament factors are shown in Table 2. As predicted, child low PE was associated with maternal MDD/DD. However, child PE was not significantly correlated with MDD/DD in fathers. Neither of the remaining higher order factors (NE and Inhibition) was associated with MDD/DD in mothers or fathers. In order to examine the relative associations between the three temperament factors and parental depressive disorders, we entered PE, NE, and Inhibition into a simultaneous multiple logistic regression analysis. Only PE was uniquely associated with maternal MDD (odds ratio /H110050.25, 95% confidence interval /H110050.06–0.98, p /H11005.05). None of the three temperament factors was uniquely associated with paternal MDD/DD.

We also conducted exploratory analyses at the lower order trait level (see Table 2). Of the three PE subtraits, maternal MDD/DD was correlated with low levels of positive affect and engagement but was not associated with sociability. None of the PE subtraits

Table 1
Rates (in Percentages, with Means and Standard Deviations) of Lifetime Axis I Psychopathology in Parents (N = 100)

<table>
<thead>
<tr>
<th>Diagnosis/feature</th>
<th>Mothers</th>
<th></th>
<th>Fathers</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>M</td>
<td>SD</td>
<td>%</td>
</tr>
<tr>
<td>Mood disorder</td>
<td>34</td>
<td>12</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Major depressive disorder (MDD)</td>
<td>25</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dysthymic disorder (DD)</td>
<td>16</td>
<td>2</td>
<td></td>
<td>2</td>
</tr>
<tr>
<td>Recurrent MDD</td>
<td>12</td>
<td>3</td>
<td></td>
<td>3</td>
</tr>
<tr>
<td>Chronic major depressive episode</td>
<td>8</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age of onset of MDD (years)</td>
<td>23.2</td>
<td>7.4</td>
<td></td>
<td>25.3</td>
</tr>
<tr>
<td>Age of onset of DD (years)</td>
<td>18.9</td>
<td>10.4</td>
<td></td>
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<tr>
<td>Any anxiety disorder</td>
<td>40</td>
<td>22</td>
<td></td>
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</tr>
<tr>
<td>Specific phobia</td>
<td>17</td>
<td>10</td>
<td></td>
<td>10</td>
</tr>
<tr>
<td>Social phobia</td>
<td>14</td>
<td>12</td>
<td></td>
<td>12</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>9</td>
<td>1</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Generalized anxiety disorder</td>
<td>3</td>
<td></td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Post-traumatic stress disorder</td>
<td>5</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Any substance abuse disorder</td>
<td>47</td>
<td>68</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol abuse</td>
<td>31</td>
<td>41</td>
<td></td>
<td>41</td>
</tr>
<tr>
<td>Alcohol dependence</td>
<td>11</td>
<td>20</td>
<td></td>
<td>20</td>
</tr>
<tr>
<td>Drug abuse</td>
<td>14</td>
<td>17</td>
<td></td>
<td>17</td>
</tr>
<tr>
<td>Drug dependence</td>
<td>11</td>
<td>17</td>
<td></td>
<td>17</td>
</tr>
</tbody>
</table>

Table 2
Correlations Between Familial Risk for Mood Disorder and Laboratory-Assessed Temperament Traits

<table>
<thead>
<tr>
<th>Trait</th>
<th>MDD/DD</th>
<th>PE scale</th>
<th>NE scale</th>
<th>I scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>PE</td>
<td>−.23*</td>
<td>.15</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>NE</td>
<td>.03</td>
<td>−.07</td>
<td>−.07</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>.12</td>
<td>.06</td>
<td>−.28*</td>
<td>.26**</td>
</tr>
<tr>
<td>Pos affect</td>
<td>−.26**</td>
<td>.15</td>
<td>.88***</td>
<td>.03</td>
</tr>
<tr>
<td>Eng</td>
<td>−.23*</td>
<td>.15</td>
<td>.84***</td>
<td>−.21**</td>
</tr>
<tr>
<td>Sociability</td>
<td>−.10</td>
<td>.08</td>
<td>.83***</td>
<td>−.02</td>
</tr>
<tr>
<td>Anger</td>
<td>.02</td>
<td>.00</td>
<td>−.01</td>
<td>.80***</td>
</tr>
<tr>
<td>Sadness</td>
<td>−.12</td>
<td>−.20*</td>
<td>.02</td>
<td>.74***</td>
</tr>
<tr>
<td>IncNE</td>
<td>.22*</td>
<td>.06</td>
<td>−.19</td>
<td>.81***</td>
</tr>
<tr>
<td>Fear</td>
<td>.06</td>
<td>.08</td>
<td>−.26**</td>
<td>.18</td>
</tr>
<tr>
<td>BI</td>
<td>.13</td>
<td>.05</td>
<td>−.24*</td>
<td>.26**</td>
</tr>
</tbody>
</table>

Note. N = 100. MDD/DD = lifetime history of major depressive disorder or dysthmic disorder; PE = Positive Emotionality factor; NE = Negative Emotionality factor; I = Inhibition factor; Pos affect = Positive affect; Eng = Engagement; Soc = Sociability; Ang = Anger; Sad = Sadness; IncNE = Incongruous NE; BI = Behavioral Inhibition.

* p < .05. ** p < .01. *** p < .001.
were significantly correlated with paternal MDD/DD. With regard to NE constructs, maternal MDD/DD was associated only with high incongruous NE. Paternal MDD/DD was correlated only with child sadness, and the direction of this association was opposite to that predicted. Finally, neither of the Inhibition subscales was correlated with maternal or paternal MDD/DD.

The relatives of probands with early onset, recurrent, and chronic forms of depression are at particularly high risk for depression (D. N. Klein, Lewinsohn, Rohde, Seeley, & Durbin, 2002; Sullivan, Neale, & Kendler, 2000). This suggests that low PE may be especially pronounced in the children of parents with early onset and recurrent or chronic forms of depression. To explore this hypothesis, we examined child PE as a function of parental age of onset and course of depression. As previous studies have generally used a cutoff of age 21 to distinguish early from late onset (D. N. Klein et al., 2002), we used this convention to create a three-level early onset depression variable: 0 = no MDD or DD; 1 = late onset (≥ age 21); 2 = early onset (< age 21). We also created a three-level variable for recurrent or chronic depression: 0 = no MDD/DD; 1 = single, nonchronic MDE; 2 = recurrent MDD, chronic MDE, or DD. Two linear contrasts were computed, testing the hypothesis of decreasing PE scores in children as a function of age of onset and course of parental depression. The linear contrast was significant for both early onset, F(1, 97) = 4.79, p = .03, and chronic or recurrent, F(1, 97) = 4.97, p = .03, maternal depression, indicating that low PE is particularly associated with early onset and chronic or recurrent depression in mothers. Neither of the contrasts was significant for paternal depression.

** Associations Between Child PE and Parental Nonmood Disorders**

Child PE was not significantly correlated with lifetime diagnoses of any anxiety disorder or substance abuse or dependence in mothers or fathers. Child PE correlated −.03 with any anxiety disorder in mothers and −.10 with any anxiety disorder in fathers. The correlations between parental substance abuse or dependence and child PE were .13 for mothers and −.01 for fathers. In addition, we examined the specific nonmood disorders with sufficient lifetime prevalence in the sample: social phobia, specific phobia, alcohol abuse or dependence, and drug abuse or dependence. Child PE was not significantly correlated with any of these diagnoses in mothers or fathers.1

** Effects of Potential Confounding and Mediating Variables**

Finally, we conducted subsidiary analyses to examine the effects of several variables that could potentially serve as either confounding or mediating variables in the low PE-maternal depressive disorder relationship: maternal nonmood psychopathology, family SES and marital discord, maternal behavior in interacting with the child, and child exposure to maternal depression.

**Nonmood psychopathology.** Although child PE was not significantly associated with specific maternal nonmood disorders, it is important to explicitly rule out the possibility that the child low PE–maternal depression association was due to the presence of comorbid psychopathology. A hierarchical multiple regression revealed that maternal MDD or DD (entered in the second block) continued to predict child PE after controlling for maternal lifetime anxiety and substance abuse disorders (entered in the first block). As a set, maternal anxiety (B = −.03, SE = .07) and substance abuse (B = .09, SE = .07) did not predict child PE, F(2, 97) = 0.94, p = .39. Maternal MDD or DD (B = −.18, SE = .07) accounted for a significant increment in variance explained, F change (1, 96) = 6.04, p = .02, sr² = .058. When entered in a subsequent 3rd and 4th block, neither the two-way nor the three-way interactions among maternal depression, anxiety, and substance abuse disorders significantly predicted child PE.

**SES and marital discord.** It is conceivable that the association between maternal depression and child low PE is due to contextual factors that are common in families with a depressed parent, such as low SES and marital discord (Cicchetti et al., 1998; Downey & Coyne, 1990). As a preliminary test of this possibility, we conducted a hierarchical multiple regression in which SES and marital discord (assessed by the DAS) were entered on the first step, and maternal MDD/DD was entered on the second. Because of missing data for paternal DAS, analyses were conducted using only maternal scores; however, results were the same when analyses were limited to families in which DAS scores were available from both parents and were averaged across mothers and fathers. Entered as a set on the first step, SES (B = .00, SE = .00) and marital discord (B = .00, SE = .00) were not associated with child PE, F(2, 96) = 0.42, p = .66. However, maternal history of depression (B = −.16, SE = .07), entered on the second step, predicted a significant increment in variance explained, F change (1, 95) = 5.12, p = .03, sr² = .046.

**Maternal behavior.** A potential explanation for the association between maternal MDD or DD and child low PE is that depressed mothers exhibit less positive and more negative behavior in interacting with their children (Downey & Coyne, 1990), and that this leads to lower levels of PE in the child. In order to explore this in a preliminary fashion, we examined whether maternal lifetime MDD or DD predicted child PE after controlling for home observation ratings of maternal pleasure and criticism in interacting with her child. Maternal pleasure and criticism, entered as a set in the first step, significantly predicted child PE, F(2, 96) = 5.60, p = .01. Maternal pleasure (B = .14, SE = .04), but not criticism (B = .05, SE = .05), was significantly associated with child PE (p < .05). However, when maternal lifetime MDD or DD was entered on the second step, it continued to account for significant variance in child PE, F(1, 95) = 5.40, p = .02, sr² = .048. These results suggest that maternal behavior at the time of the observation did not account for the association between maternal depression and child PE.

**Child exposure to maternal depression.** Although many mothers with a history of mood disorder in this sample had not experienced a depressive episode during their child’s lifetime, some children had been exposed to full syndromal or subthreshold depressive episodes in their mothers. In order to determine whether the relationship between maternal depression and child PE was accounted for by child exposure to maternal depressive symptoms, we also conducted correlations between each nonmood disorder and the three PE subtraits as an initial exploration of differential associations. Only one correlation was significant: Low positive affect was associated with maternal social phobia (r = .21, p = .04).
we conducted two analyses. First, we conducted a partial correlation between maternal MDD or DD and child PE, controlling for self-reported depressive symptoms at the time of the assessment, as measured by the IDD. The partial correlation was significant \( (r = -0.22, p = 0.03, n = 94 \text{ due to missing data on the IDD}) \). To provide an even more stringent test, we recomputed the correlation between child PE and maternal history of depression after eliminating all families in which mothers met criteria for a serious mood disorder (MDD or DD) or a subthreshold depressive disorder (Depression Not Otherwise Specified or Minor Depressive Disorder) during the child’s lifetime. After eliminating these participants \((n = 10)\), maternal MDD/DD was still significantly correlated with PE \((r = -0.24, p = 0.02)\). This conservative test suggests that the relationship between maternal depression and low PE in children cannot be attributed to dampening effects of depressive symptoms in mothers.

**Discussion**

As predicted, low PE in preschoolers, as assessed with a comprehensive battery of emotion-eliciting laboratory tasks, was associated with a maternal history of mood disorders. This finding is consistent with a number of theorists’ hypotheses that low PE may be a predisposing factor or precursor for depressive disorders (Clark & Watson, 1999; Davidson, 1992; Depue & Collins, 1999; D. F. Klein, 1987; Meehl, 1975). Moreover, these results add to the literature on maternal transmission of depression (Goodman & Gotlib, 1999) by suggesting that child low PE may be one mediator of this process.

The association between child PE and maternal depression fell in the range of a small-to-medium effect size (Cohen, 1977) and could not be accounted for by comorbid lifetime anxiety and substance abuse disorders in mothers. In addition, the effect was stronger for early onset and chronic or recurrent forms of depression, the two features associated with the highest levels of familial aggregation in family and twin studies (Sullivan et al., 2000).

In contrast, low PE in children was not associated with paternal mood disorders. Very little is known about the relationship between depression in fathers and children’s behavior, temperament, or adjustment. Unfortunately, the current study is unable to address many of these questions. The number of fathers with a history of mood disorder in our community sample was low \((n = 12)\); thus, the somewhat unexpected pattern of findings may not replicate in a larger study. The few studies in the literature that have addressed the psychological adjustment of children of depressed fathers have generally not replicated findings associated with maternal depression (cf. Brennan, Hammen, Katz, & Le Brocque, 2002; Jacob & Johnson, 2001). In a meta-analytic study, Connell and Goodman (2002) concluded that maternal depression was more strongly associated with childhood internalizing problems than was paternal depression. However, they also found that the effect size for parental psychopathology upon child adjustment varied as a function of parental sex and child age, such that effects of paternal psychopathology were stronger in samples using older children, whereas the reverse was true for maternal psychopathology. Thus, the lack of effects for paternal depression in this study may have been due to the young age of our participants, environmental factors that are more closely associated with mothers than fathers (perhaps attributable to mothers’ greater involvement in child rearing), or sex-specific genetic effects (Kendler, Gardner, Neale, & Prescott, 2001).

Consistent with the tripartite model (Clark & Watson, 1991), low PE demonstrated strong specificity to depression. Thus, it was not significantly associated with anxiety or substance use disorders in mothers or fathers. The sole exception was at the subtrait level; child low positive affect was associated with paternal social phobia, which is consistent with previous findings of a link between low PE and social phobia (Brown et al., 1998). Also consistent with the tripartite model and a recent study of PE and NE in middle childhood and adolescence (Lonigan, Hooe, David, & Kistner, 1999), PE and NE were orthogonal in this sample of preschoolers. These findings extend the literature on the tripartite model by using structured, developmentally appropriate laboratory tasks to assess PE and NE, rather than self- or parent-report.

Contrary to the tripartite model, we did not find strong evidence that parental mood disorders were associated with high NE in children. These results differ from the adult literature that supports links between NE and psychopathology (Clark et al., 1994). The lack of findings in this study may be due to the participants’ developmental stage; perhaps high NE is more strongly indicative of risk among older children, whose greater emotion regulation skills should exert a dampening effect upon overt NE expressions. Thus, deficits in the regulation of NE may become increasingly salient with age. Interestingly, high incongruous NE was associated with maternal depression, suggesting that negative emotions that are unusual in their situational context may be more strongly related to parental depression than are individual differences in normative NE expressions. Alternatively, NE might reflect more of a state component of psychopathology than a trait risk factor. Finally, our lack of findings for NE may also stem from the lower reliability we obtained for NE, compared with PE, measures. This lower reliability of NE may be due to the fact that coders were required to differentiate among the three negative emotions (sadness, anger, and fear). Similar to findings for NE, Inhibition was not associated with parental depression, despite being moderately correlated with PE. The high comorbidity between anxiety and mood disorders may have contributed to findings linking BI to depression in other studies (Rosenbaum et al., 2000).

With regard to PE, many researchers have suggested that sociability should be considered distinct from the positive affect and approach motivation-reward sensitivity components that are increasingly seen as central to PE (Depue & Collins, 1999; Lucas, Diener, Grob, Suh, & Shao, 2000; Tellegen, 1985). Consistent with this claim, sociability was not correlated with parental depression in this sample. Future studies of temperamental risk should explore associations unique to more finely parsed temperament traits, in addition to the predictive validity of high-order aggregate constructs, given that trait aggregation may conceal important temperament-risk relationships. However, our power to test whether the associations between particular subtraits and parental psychopathology were significantly different from one another was very low; larger studies with adequate power are necessary to fully address the issue of differential associations.

An important issue concerning these findings is whether the link between maternal depression and low PE in children is due to the direct transmission of a liability for mood disorder or to differences in maternal child-rearing behavior that are the result of psychopathology. Although our ability to assess this was limited,
we did not find evidence in this sample that mothers’ current behavior with their child observed during two home visits mediated the relationship between maternal history of depression and low PE in children, even though maternal behavior was significantly correlated with child PE. These findings are consistent with other studies that have found parental depression and parent–child interactions to have independent effects on risk for psychopathology (Andrews, Brown, & Creasy, 1990; Lizardi & Klein, 2000). Also, lifetime history of mood disorders in mothers remained significantly associated with low PE in children when analyses were limited to mothers who had not experienced any depressive disorders since the birth of the child and after controlling for current depressive symptomatology.

Although the children in this study were well below the age of risk for depression, we cannot rule out the possibility that the low PE we observed is an early manifestation of an affective disorder. Current knowledge of the presentation of mood disorders in preschool aged children is limited (Garber & Horowitz, 2002; Luby et al., 2003). However, if later work reveals that the differences in emotionality observed in this study are early expressions of depression, it could have important implications for defining an early childhood depressive phenotype and designing developmentally appropriate procedures for clinical assessment.

This study has several strengths, including the use of a comprehensive laboratory assessment of child temperament tapping a range of traits relevant to risk for psychopathology and the use of structured clinical interviews to assess lifetime parental psychopathology. An important point is that by using laboratory measures of child temperament rather than parent report, our temperament assessments were completely independent of parental psychopathology. Moreover, we used a community sample, considered both maternal and paternal psychopathology, and studied young children who were well below the age of risk for experiencing depressive disorders.

However, the study also has some limitations that temper the confidence with which certain conclusions can be drawn. First, the amount of variance in maternal depression accounted for by child low PE was small, indicating that other factors must be taken into account in a comprehensive model of risk for depression. The modest sample size precluded a full examination of disorders with low base rates (including paternal depression). It also was not feasible to examine whether the associations found differed between boys and girls; however, there were no gender differences on any of the child traits associated with parental psychopathology. We did not correct for multiple analyses; thus, the findings of this study need to be replicated. We were able to examine parental child-rearing behavior in only a preliminary way, as our assessment was limited to uncontrolled observations of mother–child interactions in the home. Also, our home observer measure of maternal behavior has not been previously validated and had lower reliability than our ratings of child temperament, which may have contributed to its weak associations with parental depression. Therefore, this study cannot rule out the possibility that mother–child interactions mediate the maternal depression–child PE association. It is likely that other environmental variables not assessed in this study may also be important for understanding depression risk. Future studies are needed to build on these findings by including more comprehensive assessments of parental child-rearing behavior and the family environment.

Although low PE in children was associated with maternal depression, longitudinal studies are needed to determine if children with low PE actually do develop mood disorders at an elevated rate later in life. The temporal stability of laboratory measures of emotionality in children is also unknown; therefore, follow-up studies are necessary to determine the stability and continuity of emotionality across development. Finally, if temperamental emotionality mediates the association between maternal depression and child risk, one would also expect an association between maternal personality and child temperament. Thus, future studies should include in-depth assessments of parental personality.

Much work is needed to explore the possible mechanisms by which emotionality may act as a diathesis for the development of mood disorders. Low PE may contribute to risk for mood disorders by its impact on daily mood, via long-term alteration of the biological systems governing emotions and emotion regulation, or through cognitive mechanisms (such as diminished effectance/mastery motivation or expectations for rewards contingent upon active behavior), or interpersonal processes (e.g., PE may be linked to peer acceptance). High levels of incongruous NE may reflect abnormalities in the context regulation of affect, which Davidson, Jackson, and Kalin (2000) have implicated in mood and anxiety disorders. Future studies addressing the developmental trajectory and sequelae of child emotionality are necessary to develop a more complete understanding of the mechanisms that link temperamental emotionality to depression.

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