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Maternal prenatal psychological distress and preschool cognitive functioning: The protective role of positive parental engagement

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Abstract

Considerable animal research and available human studies suggest that psychological distress experienced by mothers during gestation is associated with later neurodevelopmental deficits in offspring; however, little research has examined potential protective factors that might mitigate this risk. The current study examined the impact of maternal prenatal psychological distress during pregnancy on cognitive outcomes in preschoolers (ages 2.5-5 years) and positive parenting as a potential protective factor. Mother-child dyads ($N=162$, mean child age=44 months, 49% female) were recruited from a longitudinal cohort of women who had previously participated in a study of maternal mood disorders during pregnancy. Maternal prenatal distress was assessed with multiple measures collected throughout pregnancy. During a follow-up visit, mothers were interviewed about their psychological symptoms since the birth of the child, parenting behaviors were recorded during a parent-child interaction, and children's cognitive abilities were measured using the Differential Ability Scales, 2nd Edition. Maternal prenatal distress significantly predicted lower general cognitive abilities; however, this relationship was strongest for children whose mothers exhibited low levels of positive engagement and not significant when mothers exhibited high levels of positive engagement. Results suggest that positive parental engagement can protect against the detrimental effects of maternal prenatal distress on preschoolers cognitive abilities.

Keywords

prenatal distress; depression; stress; parenting; protective factors; cognitive development; preschool children; latent factor; structural equation modeling; psychotropic medication; pregnancy

Children of women who experienced psychological distress, such as depression and anxiety, during pregnancy are at higher risk for an array of negative outcomes (Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; Huizink, Robles de Medina, Mulder, Visser, & Buitelaar, 2003; O'Connor, Heron, Glover, & Alspac Study Team, 2002; Sandman, Davis, Buss, & Glynn, 2011). In particular, a number of studies have found that distress experienced during pregnancy is linked to reduced cognitive abilities in offspring (Bergman, Sarkar, Glover, & O'Connor, 2010; Huizink et al., 2003; Laplante, Brunet, Schmitz, Ciampi, & King, 2008). For example, in a recent meta-analysis of 11 studies of young children ranging from birth to 60 months, maternal prenatal stress was found to have a small but significant negative association with child cognitive development (Tarabulsky et al., 2014). Cognitive abilities in early childhood are a critical predictor of functioning in later life: childhood intellectual functioning has been linked to trajectories in academic achievement during the school age and adolescent periods (Campbell, Pungello, Miller-Johnson, Burchinal, & Ramey, 2001; Gutman, Sameroff, & Cole, 2003; McWayne, Fantuzzo, & McDermott, 2004), as well as literacy rates in adulthood (Baydar, Brooks Gunn, & Furstenberg, 1993; Davies, 2011). Given that pregnancy has been identified as a time marked by increased risk for developing a mental disorder or re-experiencing some form of psychopathology (Levey, Ragan, Hower-Hartley, Newport, & Stowe, 2004), elucidating the impact of maternal prenatal distress on child cognitive outcomes is an important area of inquiry.

It makes sense that environmental threats occurring during gestation can have a long-lasting impact on children's development given the vulnerability of the fetal brain as well as the rapid neurodevelopmental changes that occur during gestation (Sandman et al., 2011). The Fetal Programming Hypothesis (Barker et al., 1993) has been posited as a theory to explain how maternal prenatal psychological distress might influence the fetus' adaptations to the intrauterine environment, and how these adaptations may be associated with postnatal development and outcomes (Talge, Neal, & Glover, 2007). For example, it has been suggested that fetal exposure to mothers' excess cortisol in response to distress experienced during pregnancy may impact offspring's cognitive abilities by affecting the development of neural structures crucial to intellectual functioning, such as the hippocampus (Bergman et al., 2010), amygdala (Buss et al., 2012), and prefrontal cortex (Buss, Davis, Muftuler, Head, & Sandman, 2010).

Despite the noted association between maternal prenatal distress and children's cognitive functioning, relatively few studies have attempted to identify factors that might mitigate risk. Given the well-documented associations between sensitive and responsive parenting behaviors and positive child outcomes (NICHD Early Child Care Research Network, 1999), it follows that specific parenting behaviors may serve as potential protective factors. Indeed, studies from the animal literature suggest that a sensitive postnatal environment—specifically, increased maternal care (e.g., licking, crouching over, picking up) shortly after birth—may ameliorate negative effects of prenatal distress on both biological and behavioral outcomes (Maccari et al., 1995; Vallée et al., 1999).

However, there is limited clinical research examining parenting as a protective factor in this developmental risk process. Bergman and colleagues (2008) examined whether mother-child

attachment moderated the effect of prenatal stress on children's cognitive outcomes using retrospective reports of prenatal stress by mothers who were 14-19 months postpartum. Their results suggested that attachment did not affect the relationship between antenatal stress and children's cognitive outcomes. In a later study with the same sample, however, Bergman and colleagues (2010) found a negative relationship between mothers' prenatal cortisol and children's cognitive development at 17 months, as well as a moderating effect of attachment, such that the maternal prenatal cortisol and child cognitive functioning association was stronger for infants with an insecure attachment and nearly nonexistent for youth with a secure attachment. Grant et al. (2010) prospectively examined women's anxiety during pregnancy, as well as maternal sensitivity during a still-face paradigm when children were 7-months-old. Results suggested that maternal sensitivity moderated the relationship between prenatal anxiety and infant mental development, such that maternal prenatal anxiety was associated with lower scores on the Bayley Scales of Infant Development only for women who were rated as low on sensitivity. Further, this interaction between maternal prenatal anxiety and postnatal sensitivity remained significant after controlling for postnatal anxiety and depression.

These mixed findings from the limited extant parenting literature may be explained by study design (retrospective vs. prospective), diverse measures of maternal stress (cortisol vs. self-report of life events vs. diagnosis via a structured interview), and different techniques for assessing caregiving (attachment paradigm vs. still-faced paradigm). Additional prospective research that utilizes standardized measures of parenting is needed to better understand how the postnatal caregiving environment might affect the relationship between prenatal distress and child cognitive outcomes. The current study will use a well-validated and developmentally appropriate measure of parent-child interactions (The Dyadic Parent-Child Interaction Coding System, 3rd Edition; Eyberg, Nelson, Duke, & Boggs, 2004) and explore whether parenting behaviors may act as a protective factor in the relationship between prospectively assessed maternal prenatal distress and child cognitive outcomes in the preschool phase of development. This is an important research endeavor as identifying protective factors, such as parenting behavior, provides a mechanism for early intervention with at-risk youth (Cicchetti, Rogosch, & Toth, 2000).

The current study also uses a latent factor approach to measure maternal prenatal distress. Most studies examining maternal prenatal distress rely upon a sole measure of distress such as self-report of daily hassles (Huizink et al., 2003), negative life events (Bergman et al., 2007), traumatic events (Laplante et al., 2004), or presence of mood disorder symptomatology (Deave, Heron, Evans, & Emond, 2008). In addition, researchers often attempt to statistically control for one measure of distress while exploring the effect of another. While this method allows for the examination of the unique contribution of a specific type of distress on child outcomes, the high correlations among measures of distress (Feldman et al., 2009; Goodman & Tully, 2008) suggest the need for a different, more ecologically valid approach. Specifically, the use of a latent factor allows for the combining of several related measures into a single predictor. Such an approach has been previously used to study the role of stress and anxiety experienced during pregnancy on child outcomes (Lobel et al., 2008; Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999). For example, Rini et al. (1999) used a latent factor to study the differential effects of prenatal psychosocial

factors on birth outcomes, and Lobel and colleagues (2008) used a latent variable to test associations between maternal prenatal stress and infant birth weight. The current study will use a similar latent factor method by taking advantage of the high intercorrelations among prenatal distress measures and combining measures completed by multiple raters (mother, clinician, research interviewer) into a latent factor, thus reducing measurement error and providing a more reliable, comprehensive measure of the overall load of distress experience by women during gestation. Furthermore, the majority of studies have used healthy community samples. The current study includes a sample of women seeking treatment for psychiatric illness and allows us to examine a greater range of prenatal distress while controlling for pharmacological intervention during pregnancy.

The goals of the proposed study are to examine the relationship between maternal prenatal distress—studied as a latent factor composed of prospectively collected measures assessing depression, perceived stress, and overall psychiatric functioning—and offspring cognitive outcomes. We chose to study cognitive outcomes in preschoolers (2.5-5 years old) as this is a developmental period in which cognitive intervention may have the greatest impact on academic trajectories (Campbell et al., 2001). In addition, the potential protective role of positive parenting was explored. We hypothesized that maternal prenatal distress would be inversely associated with cognitive functioning in preschool-aged children and that positive parenting behaviors would attenuate this association, thus acting as a protective factor.

Method

Participants

Participants were women drawn from a cohort prospectively assessed at the Emory Women's Mental Health Program, a tertiary referral center for the treatment of mental disorders in pregnant and postpartum women. Informed consent was obtained from all individual participants included in the study. Participants were enrolled in an observational study of prenatal mood disorders before completion of the 20th week of gestation. As the study was naturalistic, visits were scheduled at 4-6 week intervals as would be typical in routine prenatal care. Only children from singleton births were included in the study. Women were re-contacted to participate in a follow-up study when their child was between 2.5-5 years old. Approximately 77% of the women who were invited to participate in the longitudinal follow-up agreed to do so. Women who participated did not differ from those who refused in terms of education level ($t=-.65$, $p=.52$), age ($t=.17$, $p=.86$), prenatal medication status ($\chi^2=1.98$, $p=.74$) or race ($\chi^2=2.95$, $p=.57$). Their infants did not differ on gestational age at birth ($t=-.96$, $p=.34$). Mother-child dyads were included in the current analyses if they completed a preschool child assessment of cognitive functioning and if data were available from two or more measures of maternal distress at least three times during pregnancy to ensure that an Area Under the Curve (AUC) measure could be computed.

The final sample was comprised of 162 women (mean age=37 years, standard deviation=5 years) and their children (age range=29-67 months, mean age=44 months, standard deviation=11 months, 49% female). The majority of the sample (83%) was married/partnered, 7% divorced, 3% separated, and 6% never married. Thirty five percent of the sample had graduated from a four-year college and 42% had completed a graduate/

professional degree. The sample was 86% White, 8% Black or African American, 3% Hispanic or Latino, 2% Asian, and less than 1% biracial.

Ninety-one percent of the participants met criteria for at least one Axis I diagnosis during their lifetime. Specifically, 77.5% of sample met for anxiety (Panic Disorder, Social Phobia, Specific Phobia, Obsessive Compulsive Disorder, PTSD, Generalized Anxiety Disorder, Anxiety Disorder NOS); 53.8% depression (Major Depressive Disorder, Dysthymia, Depressive Disorder NOS), 23.2% bipolar disorder (Bipolar I, Bipolar II, and Bipolar Disorder NOS), and 6.3% met for a psychotic disorder (Schizophrenia, Schizoaffective, Brief Psychotic Disorder, Psychotic Disorder NOS). During pregnancy, 37.4% met criteria for anxiety, 19% depression, 8.6% bipolar disorder, and 4.2% a psychotic disorder. Eighty two percent of the sample took psychotropic medication at some point during pregnancy. The rate of prematurity was low in this sample (6.8%), and mean infant birthweight was 7.4 pounds (standard deviation=1.2, 0.6% were below 5.5 pounds, which is the World Health Organization cutoff for low birthweight).

Maternal Prenatal Assessment

Maternal prenatal distress—Psychological distress was prospectively and longitudinally evaluated across pregnancy using multiple measures. At study entry, a trained interviewer assessed current and lifetime Axis I diagnoses using the Structured Clinical Interview for DSM-IV (SCID) (First, Spitzer, Gibbon, & Williams, 2002). The SCID has been shown to be a valid and reliable measure of anxiety and mood disorder in research studies (kappas ranging from .70- .80; Zanarini & Frankenburg, 2001). Interrater reliability for the SCID administered prenatally was well established with kappas for each disorder exceeding .80.

On average, women who participated in the preschool follow-up study were evaluated five times during pregnancy, and all participants had prenatal distress data for each trimester. At all prenatal visits, a psychiatrist assigned a Clinical Global Impression (CGI) score, which incorporates a patient's behaviors, psychosocial functioning, psychiatric symptoms, as well as the effect of these symptoms on overall functioning (Guy, 1976; Busner & Targum, 2007). The CGI is a widely used measure that has been shown to correlate well with other symptom scales (Busner & Targum, 2007). In addition, a research interviewer administered the 17-item Hamilton Rating Scale for Depression (HRSD 17; Hamilton, 1960), which has been shown to be a reliable and valid measure of depressive symptoms (Williams, 1988). Participants also completed self-report assessments of stress (Perceived Stress Scale; PSS; Cohen, Kamarck, & Mermelstein, 1983) and depressive symptoms (Beck Depression Inventory; BDI). Both the PSS (Cohen, Kamarck, & Mermelstein, 1983) and BDI (Beck, Steer, & Brown, 1997; Steer, Ball, Ranieri, & Beck, 1997; Whisman, Perez, & Ramel, 2000) have been shown to have good reliability and validity across samples.

The current study used Structural Equation Modeling (SEM) to construct a latent factor of maternal distress composed of different indices (depression, stress, and severity of psychiatric symptoms) obtained from numerous sources. Additionally, AUC indices for each scale collected during pregnancy were computed.

Maternal prenatal psychoactive substance use—Women reported on their daily dose of alcohol (i.e., number of servings) and cigarettes (i.e., number cigarettes smoked) on a week-by-week basis since their previous prenatal visit (or since conception if first visit). Psychotropic medication use (divided into functional classes including antidepressants, mood stabilizers/antiepileptic drugs, antipsychotics, benzodiazepines, and hypnotics) during pregnancy was collected prospectively and verified by treating psychiatrists and research nurses. Prenatal exposure to each substance class was operationalized as drug weeks of exposure (i.e., number of drugs in a class x number of weeks exposed), standardized to 40 gestational weeks. Information regarding marijuana exposure during pregnancy was collected via obstetrician records and dichotomized into present or absent.

Maternal Postnatal Assessment

Maternal symptomatology during the child's lifetime and maternal concurrent distress—To assess the level of symptoms experienced by women since the birth of their child, the SCID was re-administered at the preschool follow-up visit by either a PhD level psychologist or a trained graduate student. Interrater reliability was well established with kappas for each mood disorder diagnosis ranging from .78-.90. Maternal symptomatology since the birth of the child was operationalized as the proportion of the child's postnatal life (in months) that mothers had experienced an Axis I disorder. In addition, maternal concurrent distress was measured during the preschool follow up visit by: (1) the research interviewer rated the patient's current functioning using the Global Assessment of Functioning (GAF) scale and (2) mothers completed the BDI assessing depressive symptom severity during the preceding week.

Child Assessment

Child cognitive outcomes—Children underwent standardized assessment at the preschool follow up visit with a trained researcher masked to the history of maternal psychopathology as well as maternal prenatal and concurrent distress levels. Cognitive abilities were measured using the Differential Ability Scales (DAS), 2nd Edition, Early Years (Elliott, 1990). The DAS is a standardized, age-normed, well-validated measure of cognitive ability (Elliott, 1990). It provides a composite measure of overall cognitive functioning (General Conceptual Ability; GCA), which taps verbal (i.e., expressive and receptive language), nonverbal reasoning, and spatial abilities. Fidelity checks were done approximately every 6-months by a licensed clinical psychologist (KJ).

Parenting Assessment

Parenting—The Dyadic Parent-Child Interaction Coding System, 3rd Edition (DPICS; Eyberg et al., 2004) was used to code a 20-minute interaction between the mother and child in the laboratory. The DPICS is a widely used behavioral observation system with adequate reliability and validity (Eyberg et al., 2004). Half of the interaction consisted of unstructured play where mother-child pairs were provided age-appropriate toys and instructed to “play as you would at home.” Afterwards, dyads participated in structured play where they were asked to clean up the toys and work on a puzzle. Trained researchers, masked to maternal

distress levels and child cognitive abilities, coded the video recorded interaction for parenting behaviors.

Specific parenting behaviors coded using the DPICS scheme included: direct commands, indirect commands, information questions, descriptive questions, neutral talk, reflections, negative talk, unlabeled praise, behavioral descriptions, and labeled praise (Eyberg et al., 2004). Inter-rater reliability ranged from .83-.99 for variables with adequate frequency. A principal component analysis (PCA) was completed using a Promax rotation. Factor scores were exported and used as measures of parenting in analyses testing study hypotheses.

Data Analytic Plan

Preliminary analyses examining confounds—The current study used a clinical sample and psychotropic medication use during pregnancy was common; therefore, prenatal exposure to psychotropic medication was explored as a potential confound. Prenatal exposure to marijuana, tobacco, and alcohol were also examined. Whether the child had experience in a daycare/school (i.e., structured early learning) was explored as a potential confound, given that exposure may vary in this age range. Maternal socioeconomic status (SES) during pregnancy was assessed via the widely used Hollingshead SES scale (Hollingshead, 1975), which takes into account maternal education and occupation. Mothers' education level was assessed by asking women how far they had gone in school at the time of the preschool follow-up visit. Mother BDI score during the preschool study was also examined as a potential confound. Demographic variables were explored as potential covariates including child's birth weight, child age at the time of the preschool visit, child gender, mother age, and number of children living in the home. Estimated gestational age and delivery complications (extracted from medical records) were also examined as potential confounds. Covariates that were significantly associated with both the predictor and dependent variables were included in final models.

Calculating area under the curve scores—The AUC method was chosen to capture the cumulative psychological distress experienced by women across pregnancy. AUC scores were calculated for each individual prenatal distress variable using the linear trapezoid method. The cumulative severity of prenatal distress was then calculated from the AUC scores, adjusted to a standard 40-week pregnancy by dividing the raw AUC score by the duration of the participant's pregnancy and multiplying by 40.

Data reduction of parenting variables—PCA of the parenting behaviors exhibited during the parent-child interaction identified three components: (1) Positive Engagement, (2) Negative Engagement, and (3) Positive Reinforcement. Component 1 was composed of behaviors that are considered Positive Engagement, including indirect commands, informational questions, descriptive questions, neutral talk, and reflections. Component 2 was composed of behaviors considered Negative Engagement, including negative talk and direct commands. Component 3 was composed of behaviors considered Positive Reinforcement, including labeled praise, unlabeled praise, and behavioral descriptions. Independent variables (prenatal distress and each of the parenting factor scores) were centered and distress x parenting interaction terms were computed, controlling for the total

amount of talking by mothers during the play session and covariates identified in preliminary analyses.

Hypothesis testing—SEM with the AMOS 18.0 program (Arbuckle, 2008) was used to test the relationship between the latent factor of prenatal distress and child cognitive abilities. Fit statistics and conventions recommended by Hu and Bentler (Kline, 2011) were used for all SEM analyses. Maximum likelihood estimation was used, and adequate model fit was confirmed with the Bollen Stine Bootstrap test, which accounts for nonnormality in data. Multiple regression analyses were completed in SPSS to assess for the potential protective role of parenting behaviors. Independent variables (i.e., latent prenatal distress factor composed of AUC variables and each of the parenting factor scores) were centered and distress x parenting interaction terms were computed, controlling for the total amount of talking by mothers during the play session, covariates identified in preliminary analyses, and the main effects of maternal distress and parenting factor scores.

Results

Preliminary analyses

Descriptive statistics for the prenatal distress measures are presented in Table 1. Each of the four measures of maternal prenatal distress significantly predicted cognitive abilities individually ($p < .05$; data not shown) and were correlated with each other (Table 2). The four measures were combined into a latent factor of distress.

Measurement model

Prior to the inclusion of dependent variables, AMOS was used to examine the fit of the measurement model. The structural model fit the data adequately, $\chi^2(df=2)=3.47$, $p=.18$, CFI=.99, RMSEA=.06, 90% CI [.00,.18], further supporting the use of a latent model of maternal prenatal distress in the current analyses.

Covariates

Prenatal anxiolytic exposure, SES during pregnancy, prenatal tobacco exposure, marijuana use during pregnancy, and daycare/preschool experience were significantly correlated with the DAS GCA and prenatal distress (Table 3), and were included as controls in SEM models predicating the DAS GCA. Prenatal maternal distress, maternal psychopathology since the birth of the child, and maternal concurrent distress measures were highly correlated (Table 2). To isolate the role of prenatal distress, the proportion of time that mothers had experienced an Axis I disorder since the child's birth, as well as current GAF and current BDI scores (i.e., maternal concurrent distress) were included in secondary analyses.

Prenatal distress and child cognitive abilities

The structural model predicting the DAS GCA (Figure 1) demonstrated good fit, $\chi^2(df=20)=23.05$, $p=.29$, CFI=.99, RMSEA=.03, 90% CI [.00, .08]. Results indicated that greater levels of maternal prenatal distress significantly predicted lower scores on the DAS, $\beta=-.17$, $p=.04$. In addition, when maternal psychopathology since the birth of the child, concurrent GAF, and concurrent BDI were entered into the model separately and

simultaneously as covariates, model fit remained strong and prenatal distress continued to significantly predict to lower DAS scores (Table 4).

Parenting as protective factor

Maternal prenatal distress and mothers' Positive Engagement interacted to predict DAS GCA, $\beta = .19$, $F = 4.10$, $p = .045$, even after controlling for maternal psychopathology since the birth of the child and maternal concurrent distress (Table 5). Plotting simple slopes of the regression at different levels of the moderating variable (Aiken & West, 1991) revealed a significant, negative association between maternal prenatal distress and child performance on the DAS when mothers engaged in less Positive Engagement, $b = -.23$, $t(114) = -2.68$, $p = .009$. No relationship was found between prenatal distress and DAS GCA at mean levels of Positive Engagement, $b = -.09$, $t(114) = -1.54$, $p = .13$, or one standard deviation above the mean, $b = .05$, $t(114) = 0.50$, $p = .62$. Controlling for maternal psychopathology since the birth of the child and concurrent distress did not change these results (data not shown).

A trend level interaction was observed between maternal prenatal distress and Positive Reinforcement in the prediction of DAS GCA, $\beta = .15$, $F = 2.78$, $p = .10$, and the strength of this interaction increased when maternal postnatal psychopathology and concurrent distress were included in the model, $\beta = .18$, $F = 3.75$, $p = .056$. Using the Aiken and West (1991) method, prenatal distress predicted significantly lower DAS scores one standard deviation below the mean of Positive Reinforcement, $b = -.19$, $t(114) = -2.43$, $p = .02$. Prenatal distress did not predict DAS scores at the mean, $b = -.10$, $t(114) = -1.61$, $p = .11$, or one standard deviation above the mean, $b = -.002$, $t(114) = -0.03$, $p = .98$. When controlling for maternal postnatal psychopathology and concurrent distress, maternal prenatal distress significantly predicted lower scores on the DAS at the mean, $b = -.18$, $t(112) = -2.61$, $p = .01$, and one standard deviation below the mean of Positive Reinforcement, $b = -.29$, $t(112) = -3.33$, $p = .001$, while results remained nonsignificant for high levels of Positive Reinforcement, $b = -.07$, $t(112) = -0.74$, $p = .46$.

Discussion

Current results are consistent with findings from previous studies showing associations between maternal prenatal distress and cognitive or developmental outcomes (Bergman, Sarkar, Glover, & O'Connor, 2010; Bergman, Sarkar, O'Connor, Modi, & Glover, 2007; Davis & Sandman, 2010; Deave et al., 2008; Huizink et al., 2003; Laplante et al., 2004; Talge et al., 2007) in a preschool sample. Further, these results indicate that the postnatal environment can act as a protective factor. Specifically, higher levels of maternal Positive Engagement mitigated the negative effect of prenatal distress on cognitive outcomes. The current study is consistent with previous research (Grant et al., 2010) and extends findings by using a larger sample (162 vs. 77 mother-child dyads), a measure that captures parenting behavior in the context of a more naturalistic interaction between mother and child (DPICS vs. still-face paradigm), and an older age range (preschool vs. infant).

Maternal prenatal distress did not predict offspring general cognitive abilities when mothers exhibited high levels of Positive Engagement. Positive Engagement was composed of verbal behaviors that reflect how parents converse with their children, including asking questions

and reflecting back information. These behaviors align with features of “maternal sensitivity,” which has been found to affect the quality of parent-child interactions (Feldman et al., 2009; Grant et al., 2010; NICHD Early Child Care Research Network, 1999) as well as predict verbal comprehension, expressive language, and school readiness in preschoolers (Murray, Hipwell, Hooper, Stein, & Cooper, 1996; NICHD Early Child Care Research Network, 1999; Nulman et al., 2002). It is possible that mothers high on sensitivity talk more with their children and encourage their children to speak (NICHD Early Child Care Research Network, 1999), resulting in increased exposure to language (Feldman et al., 2009; NICHD Early Child Care Research Network, 1999; Sohr-Preston & Scaramella, 2006) and providing an environment with greater opportunity to advance cognitive abilities. Importantly, however, the current findings were significant even when controlling for the amount of talking done by mothers during the interaction. Thus, these results suggest that it is not the *amount* of talking, but rather the *quality* of what is being said during interactions that may help to compensate for the vulnerabilities associated with exposure to maternal prenatal distress.

Parents' Positive Engagement may also protect against negative cognitive outcomes via indirect pathways. For example, certain parenting practices have been linked to children's emotion regulation skills (Cole, Martin, & Dennis, 2004; Morris, Silk, Steinberg, Myers, & Robinson, 2007). Specifically, findings suggest that parents facilitate emotion regulatory abilities in their children by engaging in “emotion-coaching” techniques, including being aware of children's emotions and responding appropriately, as well as helping children label their emotions (Gottman, Katz, & Hooven, 1997; Morris et al., 2007). These behaviors are similar to those measured by the Positive Engagement component. Increased emotion regulation is positively associated with academic success and performance (Graziano, Reavis, Keane, & Calkins, 2007), and may reflect an indirect pathway by which positive parenting mitigates risk.

Negative Engagement did not influence the association between maternal prenatal distress and child cognitive abilities. The role of negative parenting has received less attention than positive parenting, and the few prenatal distress studies that have examined negative parenting failed to identify it as an additional risk factor for negative cognitive outcomes (Bergman et al., 2007; Grant et al., 2010). Extreme negative parenting, such as abuse, has been related to poor cognitive abilities in children (DePrince, Weinzierl, & Combs, 2009); thus, it may be that negative verbal behavior alone has little impact on the prenatal distress-child cognitive ability relationship. Notably, Negative Engagement was associated with both prenatal and postnatal distress measures, and pre- and postnatal distress were significantly correlated, a common finding in the perinatal psychopathology literature (Dipietro, Costigan, & Sipsma, 2008; Pesonen, Raikkonen, Strandberg, & Jarvenpaa, 2005). However, the current study suggests that certain parenting behaviors may help to mitigate negative outcomes, even for high-risk children whose mothers experienced distress during pregnancy and after birth.

Study Limitations

The current study has several limitations. First, no measure of maternal IQ was collected. Given the heritable nature of IQ, statistically controlling for maternal IQ would have strengthened our ability to draw conclusions about the unique effect of prenatal distress on child cognitive outcomes. Socioeconomic status was related to child IQ in our sample and included as a statistical control; however, it is at best a weak proxy for parent IQ. The relevance of maternal IQ also highlights a broader issue of our inability to fully separate genetic from environmental influences, given the observational nature of the study and the lack of a genetically-sensitive design. Parenting behaviors were also coded during a laboratory visit. Though parenting behaviors coded using the DPICS in laboratory settings have also been found to occur in home environments (Eyberg et al., 2004), mothers may have been more or less likely to engage in certain behaviors while being video recorded. Our reported parenting and child cognitive measures were assessed concurrently, and it is important to consider the potential bidirectional relationship between parenting and children's cognitive functioning. Child factors have been found to influence parenting behaviors (Clark, Kochanska, & Ready, 2000; van Bakel & Riksen-Walraven, 2002), and it is likely that a cognitively advanced child elicits different parenting behaviors than a child with lower cognitive functioning. In addition, in the current study, being male was associated with lower scores on the DAS (Table 3) and gender has previously been shown to moderate associations between prenatal distress and child outcomes (De Bruijn, Van Bakel, Wijnen, Pop, & Van Baar, 2009; Rodriguez & Bohlin, 2005); however, given sample size limitations, we were unable to examine three way interactions with gender.

The majority of the sample was composed of moderate-to-high SES families. Low SES has been consistently linked to reductions in the quality of parent-child interactions (Dodge, Pettit, & Bates, 1994; Kotchick & Forehand, 2002); thus, the current study may underestimate the effect of negative parenting behaviors on the prenatal distress-child cognitive relationship. Lastly, though overall psychiatric functioning was assessed via the CGI, the latent model of distress did not contain specific measures of anxiety or negative/traumatic life events, both of which have been previously related to child outcomes (Bergman et al., 2007; Grant et al., 2010; Huizink et al., 2008; Laplante et al., 2004; O'Connor et al., 2002; Tarabulsy et al., 2014). In addition, a few studies have found a positive association between maternal prenatal distress and child cognitive outcomes in high-functioning, non-clinical samples (DiPietro, Novak, Costigan, Atella, & Reusing, 2006; Laplante et al., 2008). Thus, the results may not generalize to a non-clinical sample of women.

Study Strengths

To our knowledge, the current study was one of the first to examine the relationship between prenatal distress and child cognitive abilities in a psychiatric sample, an important area of inquiry as many women experience clinical levels of distress throughout their lifetime, and pregnancy is not a protective factor against mental illness. Furthermore, a large proportion of women in the study took psychotropic medication during pregnancy to manage their psychiatric symptoms; however, in line with previous findings (Nulman et al., 2002), psychological distress predicted children's cognitive functioning while medication exposure

did not. In addition, many women in the current sample experienced psychological distress since the birth of their child; yet, prenatal distress predicted cognitive outcomes above and beyond the degree of psychological distress experienced by women during the child's lifetime. Thus, using a psychiatric sample provided a rich opportunity to parse the effects of medication and timing of maternal distress on child outcomes, and these findings have important clinical implications as described below.

In addition, the current study took a novel approach in operationalizing maternal prenatal psychological distress. The measure of distress incorporated serial prospective self-report and clinician administered measures throughout pregnancy. Further, instead of relying upon an average or single cross-sectional score, the AUC was calculated for each measure, and these AUC scores were then used to construct a latent variable of distress in SEM. While the AUC approach has some drawbacks, such as potentially optimizing effects (Fitzmaurice, Laird, & Ware, 2012), this method also offers a comprehensive, dimensional measure of prenatal distress.

Clinical Implications

These findings underscore the importance of patient education and early intervention. Physicians and professionals working with psychiatrically ill pregnant women may want to discuss this risk with their patients as they weigh treatment options. Patients might be encouraged to seek early intervention for their children should they have concerns regarding their cognitive development, given the additional risk asserted by prenatal distress and the association that early cognitive abilities have on later functioning.

Engaging positively with children appears to reduce the likelihood of children's negative outcomes following exposure to prenatal distress. Furthermore, positive engagement was a protective factor even in the context of mothers experiencing distress during the child's lifetime and concurrently. The current findings indicate that if mothers can engage in certain parenting behaviors, even when experiencing current symptomatology, the risk to their children's cognitive functioning may be significantly reduced.

This suggests some specific intervention targets for psychiatrically ill mothers. Toddler-Parent Psychotherapy (TPP) is one intervention designed to enhance the parent-child interaction through improving communication between the mother and her child. Cicchetti and colleagues (2000) randomly assigned depressed women to receive TPP when their children were toddlers (mean age=20 months). At age three, children whose mothers had received TPP exhibited cognitive abilities that did not differ from those of healthy controls. Children of depressed mothers who did not receive TPP displayed lower cognitive functioning. No studies that we are aware of have targeted prenatally distressed women. Given the current findings, it may be beneficial to implement TPP or similar parenting interventions even earlier than the toddler years. For example, psychiatrists and obstetricians may want to recommend TPP to their pregnant patients who are experiencing psychological distress to curb potential negative cognitive outcomes before they develop.

Future Directions

While the current research adds to the maternal prenatal psychological distress literature in several ways, future research is needed to better understand the mechanisms underlying the association between distress and child cognitive outcomes. For example, using voxel-based morphometry techniques, Buss and colleagues (2010) found that maternal prenatal anxiety was associated with reduced gray matter volume in brain areas associated with executive functioning, including the prefrontal cortex. In a separate study, the researchers found that higher levels of maternal cortisol during gestation were linked to a significant increase in right amygdala volume in 7-year-old girls, and amygdala size partially mediated the association between maternal prenatal cortisol and girls' affective problems (Buss et al., 2012). Future studies would benefit from employing magnetic resonance imaging and voxel-based morphometry procedures to help elucidate the potential neural mechanisms underlying the prenatal distress-child cognitive ability relationship.

In addition, several animal and human studies have indicated that the timing of distress during gestation can differentially impact child outcomes. However, results from the human literature have been rather inconsistent (Davis et al., 2007; O'Connor et al., 2002; Rodriguez & Bohlin, 2005) and some studies have found no effect of gestational timing (DiPietro et al., 2006; Laplante et al., 2008). The current study focused on chronicity and severity of maternal prenatal stress; future studies might focus on more fine-grained timing analyses to assess whether fetuses at particular stages of gestation may be more vulnerable to prenatal maternal distress.

Conclusion

Existing studies suggest that maternal psychological distress during pregnancy can have a negative impact on offspring cognitive development. This study extended the current literature by focusing on a clinical sample of women and their preschool-aged children, using prospective data to examine whether positive parenting mitigates the negative influence of prenatal distress on child outcomes. Maternal prenatal distress significantly predicted children's overall cognitive functioning, and these findings remained significant when controlling for psychiatric symptoms experienced by women during the child's lifetime and maternal concurrent distress, suggesting that the observed cognitive deficits were uniquely associated with prenatal distress. Furthermore, specific types of positive parenting behavior acted as a protective factor in this relationship: maternal prenatal distress did not predict to cognitive abilities when mothers displayed greater levels of positive engagement with their child. Findings from the current study suggest that parenting interventions should be targeted to women experiencing high levels of prenatal distress.

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Informed consent was obtained from all individual participants included in the study. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

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Figure 1.

Maternal prenatal distress predicting to DAS GCA. Model fit was adequate, $\chi^2(df=20)=23.05$, $p=.29$, CFI=.99, RMSEA=.03, 90% CI [.00, .08]. Prenatal distress predicted lower DAS GCA scores ($\beta=-.17$, $p=.04$). Model fit was also adequate when controlling for mothers' Axis I diagnoses across her lifetime $\chi^2(df=23)=24.67$, $p=.37$, CFI=.99, RMSEA=.02, 90% CI [.00, .07] and distress continued to predict to lower DAS GCA scores ($\beta=-.10$, $p=.03$). CGI=Clinical Global Impression. BDI=Beck Depression Inventory. HRSD=17-item Hamilton Rating Scale for Depression. PSS=Perceived Stress Scale. DAS GCA=Differential Ability Scales, General Conceptual Ability. SEL=Structured Early Learning. * $p<.05$, ** $p<.01$, *** $p<.001$

Table 1
Means and Standard Deviations of Study Measures, Retained Covariates, and Parenting Measures

Measure	<i>N</i>	Mean (<i>SD</i>)
Prenatal CGI	162	1.79 (.72)
Prenatal BDI	162	10.02 (7.80)
Prenatal HRSD17	162	10.08 (4.32)
Prenatal PSS	162	24.30 (8.61)
Proportion of Child's Life Mother Experienced an Axis I Disorder	160	.35 (.41)
Concurrent Psychiatric Functioning (GAF)	160	70 (12)
Concurrent BDI	162	7 (8.50)
DAS GCA	161	105.04 (13.83)
Retained Covariates	<i>N</i>	Mean (<i>SD</i>) or %
SES during pregnancy (Hollingshead)	139	53.09 (8.65)
Prenatal Medication Exposure: Anxiolytic	162	16.7%
Prenatal Tobacco Exposure	159	8.2%
Marijuana Use During Pregnancy	159	1.2%
Structured Early Learning Experience	136	72.8%
Parenting Measures	<i>N</i>	Mean (<i>SD</i>)
Negative Talk	156	2.54 (1.77)
Direct Commands	156	11.79 (4.16)
Indirect Commands	156	9.69 (3.62)
Information Questions	156	11.46 (3.57)
Descriptive Questions	156	10.25 (2.29)
Neutral Talk	156	15.84 (2.38)
Reflections	156	3.21 (1.53)
Unlabeled Praise	156	3.42 (1.95)
Behavioral Descriptions	156	.84 (.92)
Labeled Praise	156	.46 (.73)

Note. CGI=Clinical Global Impression. BDI=Beck Depression Inventory. HRSD17=17-item Hamilton Rating Scale for Depression. PSS=Perceived Stress Scale. GAF=Global Assessment of Functioning. DAS GCA=Differential Ability Scales, General Conceptual Ability.

Table 2
Intercorrelations Among Measures of Maternal Distress, Parenting Behaviors, and DAS GCA

	1	2	3	4	5	6	7	8	9	10
1. Prenatal CGI	---									
2. Prenatal BDI	.77***	---								
3. Prenatal HRSD	.69***	.81***	---							
4. Prenatal PSS	.65***	.78***	.76***	---						
5. Psychopathology since Birth	.33***	.32***	.34***	.32***	---					
6. Concurrent GAF	-.37***	-.38***	-.39***	-.34***	-.50***	---				
7. Concurrent BDI	.37***	.52***	.47***	.47***	.36***	-.48***	---			
8. Positive Engagement	-.26**	-.23**	-.19*	-.17*	-.18*	.18*	-.18*	---		
9. Negative Engagement	.31***	.28***	.24**	.24**	.21*	-.17*	.21*	-.85***	---	
10. Positive Reinforcement	-.11	-.12	-.10	-.14	-.07	-.01	-.09	-.16	-.32***	
11. DAS GCA	-.30***	-.27**	-.16*	-.22**	-.00	.04	-.02	-.09	-.35***	-.10

Note. Total amount of mothers' talking controlled in all analyses with parenting behaviors. CGI=Clinical Global Impression. BDI=Beck Depression Inventory. HRSD17=17-item Hamilton Rating Scale for Depression. PSS=Perceived Stress Scale. GAF=Global Assessment of Functioning. DAS GCA=Differential Ability Scales, General Conceptual Ability

* $p < .05$,

** $p < .01$,

*** $p < .001$

Table 3
Correlations Between Potential Covariates and Child Neurocognitive Outcomes and Prenatal Distress

	DAS GCA	Prenatal Distress
Prenatal Medication Exposure: Hypnotic	-.04	.18 *
Prenatal Medication Exposure: Anxiolytic	-.19 *	.20 **
Prenatal Medication Exposure: Antipsychotic	-.09	.04
Prenatal Medication Exposure: Antiepileptic	.07	-.03
Prenatal Medication Exposure: Serotonin Reuptake Inhibitor	.06	.07
Child Birth Weight	.14	.05
SES during pregnancy	.30 ***	-.23 **
Child Age	.28 ***	.03
Mother's Age	.33 ***	-.15
Mother Highest Level of Education	.12	-.19 *
Mother's Current BDI	-.02	.52 **
Prenatal Alcohol Exposure	-.10	.09
Prenatal Tobacco Exposure	-.20 *	.26 **
Marijuana Use During Pregnancy ^a	-.17 *	.16 *
Child Gender ^a	-.21 **	.01
Structured Early Learning Experience ^a	.18 *	-.18 *
Number of Obstetric Complications	.05	.00
Estimated Gestational Age at Delivery	.19 *	-.08

Note. Pearson correlations computed for all analyses except those with marijuana use, child gender, and structured early learning experience.

^a Spearman's rho reported.

* $p < .05$,

** $p < .01$,

*** $p < .001$

Table 4
SEM Fit Indices For Models Predicting Maternal Prenatal Distress to DAS GCA

Dependent Variable	Controlled Postnatal Variables	χ^2	df	p	CFI	RMSEA (90% CI)	β
DAS GCA	None	23.05	20	.29	.99	.02 (.00-.08)	-.17*
	Psychopathology Post Birth	25.50	23	.33	.99	.03 (.00-.07)	-.22*
	Concurrent GAF	24.94	23	.35	.99	.02 (.00-.07)	-.21*
	Concurrent BDI	26.87	23	.26	.99	.03 (.00-.08)	-.27**
	Psychopathology Post Birth & Concurrent GAF & BDI	33.38	30	.27	.99	.03 (.00-.07)	-.29**

Note. β represent the effect of interest (prenatal distress on neurocognitive outcomes) in each model. Prenatal anxiolytic medication, tobacco exposure during pregnancy, marijuana use during pregnancy, SES, and structured early learning controlled for in all models. DAS GCA=Differential Ability Scales, General Conceptual Ability. BDI=Beck Depression Inventory. GAF=Global Assessment of Functioning.

* $p<.05$,

** $p<.01$

*** $p<.001$

Table 5
Exploring Positive Engagement as a Protective Factor

Interaction	Controlled Postnatal Variables	R^2 change	F change	β
Prenatal Distress X Positive Engagement				
	None	.03	4.10	.19 *
	Psychopathology Since Birth	.03	4.11	.19 *
	Concurrent GAF	.03	4.10	.19 *
	Concurrent BDI	.03	4.76	.20 *
	Psychopathology Since Birth & Concurrent Distress	.03	4.30	.19 *

Note. All analyses controlled for total occurrences of mother talking, prenatal anxiolytic medication, SES, structured early learning, tobacco exposure during pregnancy, and marijuana use during pregnancy.

*
 $p < .05$