Preschoolers' Executive Control and Diurnal Cortisol as Mediators of the Relations of Prenatal Stress to Child Adjustment

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Abstract

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Prenatal stress is thought to confer unique and enduring impairments in postnatal functioning in children (Van den Bergh, Mulder, Mennes, & Glover, 2005). Although prenatal stress is thought to have pervasive and lasting effects on children, few human studies have formally tested putative mechanisms or mediators conferring such effects. The existing literature on prenatal stress is clouded by interchanging definitions of prenatal stress between maternal psychological symptoms experienced in pregnancy and maternal stressful experiences during pregnancy.

This study examined the effects of maternal prenatal depression and stressful events on children's adjustment in 306 preschool-age children and their mothers assessed across 4 time points. Multiple assessment methods were implemented, including neuroendocrine indicators of

HPA activity, neuropsychological assessment of executive control, as well as the utilization of maternal and teacher-report questionnaire data. Using longitudinal modeling and multi-method assessments, the current study explored the unique effects of prenatal and postnatal psychological symptoms and the prenatal and postnatal experience of stress on preschooler's social competence and total problems. Putative mediators, including children's diurnal cortisol and executive functioning, were tested as potentially conferring the effect of prenatal stress. Sex differences in these relations were examined.

Prenatal and postnatal maternal mood and stressful experiences were not related to children's diurnal cortisol (morning level or slope). There were trends of higher prenatal mood symptoms and prenatal stressful experiences predicting lower initial levels of preschooler's executive control. There was a trend for higher prenatal alcohol exposure predicting greater growth in executive control. Postnatal maternal mood and prenatal and postnatal stressful experiences did not predict executive control.

Higher postnatal mood symptoms predicted lower social competence and higher mother-report total problems. There was a trend for higher prenatal mood symptoms to predict lower mother-report social competence. Greater prenatal exposure to medications predicted higher mother-report total problems. Greater prenatal exposure to alcohol predicted higher teacher-report total problems. Greater postnatal stressful experiences predicted higher mother-report total problems and lower mother-report social competence. Higher initial levels of executive control predicted higher social competence and lower total problems per teacher report. Greater growth in executive control predicted higher social competence and lower total problems by teacher report. There was no support for mediated relations. There was no evidence of child sex differences in these relations or evidence of differences in pathways predicted by prenatal maternal mood symptoms versus prenatal stressful experiences.

The sample and study design afford the unique opportunity to evaluate two forms of prenatal stress, prenatal mood symptoms and prenatal stressful experiences, in a longitudinal framework. Prenatal medication and alcohol exposure were related to behavioral outcomes in the preschool period. Postnatal mood and stressful experiences related to mother report of adjustment. Study findings highlight potential targets for intervention for mothers in pregnancy that may promote long-term regulation and adjustment in children.

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The prenatal period has been put forth as a sensitive window in development during which maternal stress can confer lasting impairments to the psychological/health functioning of the children (Seckl & Holmes, 2007). Animal studies have consistently demonstrated that stress experienced in gestation alters offspring's organs, tissues, and systems and that these alterations result in lifelong observable changes to physiology, cognition, and behaviors (Weinstock, 2001). Published reviews of the prenatal stress literature conclude that prenatal stress consistently relates to psychological maladjustment (Talge, Neal, & Glover, 2007; Van den Bergh et al., 2005). Further, prenatally stressed children tend to show dysregulation in their neuroendocrine systems (Harris & Seckl, 2011) and behavioral states (Van den Bergh et al., 2005). Disruptions to these systems and their development might represent pathways of the effect of prenatal stress on children's adjustment and account for the marked and enduring implication of prenatal experiences of stress. The current study seeks to elucidate mechanisms explaining the relation between prenatal stress exposure and child maladjustment, testing children's cortisol, executive functioning, and the development of these systems as putative mediators of the relation of prenatal stress and child maladjustment.

1. Prenatal stress

Frequently used measures of prenatal stress include measures of daily hassles, perceived strain, or the experience of a stressful event such as a natural or man-made disaster or bereavement (Beydoun & Saftlas, 2008; Nast, Bolten, Meinlschmidt, & Hellhammer, 2013). A recent review of the literature identified a total of seven constructs used under the umbrella of prenatal stress; In order of their frequency of use, these categories are: anxiety, depression, daily hassles, symptoms related to anxiety or depression, life events, specific socio-environmental stressors, and pregnancy-specific anxiety (including worry about pregnancy, delivery, and parenting). Authors relying on measures of anxiety and depression have argued that anxiety and depression reflect emotional responses to stressful events (Lazarus, 1984;

Mulder et al., 2002; Ruiz & Fullerton, 1999). However, no study was found that adequately established that psychopathology is a proxy for stress. Pregnancy-specific anxiety, such as fear of bearing a handicapped child, has also been used as an indicator of prenatal stress (Davis & Sandman, 2012; Gutteling, et al., 2004; Gutteling et al. 2005; Tollenaar, Beijer, Jansen, Riksen-Walraven & de Weerth, 2011). In a recent review of measurement in the prenatal stress literature (Nast et al., 2013), the authors made a recommendation to incorporate all categories of stress in studies of prenatal stress. The authors cited the notion that stress is a multidimensional concept (Beydoun & Saftlas, 2008) in making this recommendation.

Notably, no research has tested the equivalence of stress categories in their relation to putative mediators of prenatal stress, nor to child adjustment. This is surprising given the extensive literature on different dimensions of stress, and their relation to a variety of outcomes. For example, a recent meta-analysis showed that much of the variability or seeming contradiction in the relation of stress to activity of the hypothalamic-pituitary-adrenal axis (increased activation versus the opposite) is explained by qualities of the stressor (Miller, Chen, & Zhou, 2007). Low income and cumulative risk are associated with lower or blunted cortisol levels, whereas acute stress is associated with elevated diurnal levels (Dowd, Simanek, & Aiello, 2009; Miller, Chen, & Zhou, 2007). One might therefore predict that exposure to chronic stress during pregnancy would have a different physiological impact on the developing fetus than exposure to acute stress. No studies to date have tested for potential differences in the influence of stress categories.

Psychopathology may reflect individual differences in stress experiences. In the case of anxiety, worry is often a basic feature (Davey & Tallis, 1994) and results in anticipatory stress and related physiological activation prior to a stressful experience. Rumination, most often implicated in depression (Nolen-Hoeksema, 1991), influences stress sequelae. Both cognitive processes are thought to prolong the effect of the stressor and have been shown to contribute to long-term health consequences including cardiovascular, endocrinological, and immunological disease (Brosschot, Gerin, & Thayer, 2006 for review). As such, it has been hypothesized that individuals with psychopathology may experience more chronic stress (Brosschot, et al., 2006). Notably, woman with psychopathology may additionally play a role in stress generation (Hammen, 1991). For example, women with unipolar depression have been shown to evoke stress, particularly interpersonal stress. This phenomenon is attributed to the symptoms, behaviors, characteristics, and social context of the women. Therefore, it is plausible that woman with psychopathology experience more stress than normal woman (Hammen, 1991), at that the stressor is experienced in a protracted way given cognitive processes associated with psychopathology (Brosschot et al., 2006). Acute stress has been shown to increase activity in immune parameters, while more chronic stress is thought to be immunosuppressive (Segerstrom & Miller, 2004). In sum, the dimensions of stress are likely to have different biological consequences, particularly among the acute versus chronic distinction. Psychopathology may facilitate chronic experiencing of stress, but has not been shown to be a proxy for stress. The current study includes two domains of prenatal stress, prenatal mood (depression) as well as prenatal stressful experiences (life events). Further, equivalence tests will be conducted to determine if these categories relate differentially to proposed mediators and adjustment outcomes.

The few rigorous studies of prenatal stress in the preschool period provide mixed support for the enduring effects of prenatal stress. Three studies provide evidence in support of enduring effects (de Bruijn, van Bakel, & van Baar, 2009a; Loomans et al., 2011; O'Connor, Heron, Glover, & the ALSPAC Study Team, 2002). The Avon Longitudinal Study of Parents and Children (ALSPAC), a prospective, longitudinal study that collected multiple antenatal and postnatal assessments of maternal anxiety, depression, antenatal risks, psychosocial risks, and measures of children's development and adjustment in a sample of over 7,400 dyads, found that prenatal psychological symptoms correlated positively with child adjustment problems. Independent of timing within pregnancy, higher prenatal anxiety predicted behavioral/emotional problems in girls and boys at age 4 (O'Connor et al., 2002). Prenatal anxiety was associated with hyperactivity/attention problems in boys. This study also found evidence for an association between prenatal anxiety, and not prenatal depression, with mixed handedness in the preschool period (Glover, O'Connor, Heron, Golding, & ALSPAC, 2004). Mixed handedness is thought to reflect reduced brain asymmetry, to be an indicator of disturbance to normal brain development (Satz & Green, 1999), and is seen more often in individuals with psychopathology (Reid & Norvilitis, 2000; Sommer et al. 2001). A second study replicated associations between prenatal stress and preschool adjustment problems (de Bruijn et al., 2009a). First trimester prenatal psychological symptoms were associated with internalizing and total problems in boys at 14-54 months, whereas third trimester emotional complains were associated with internalizing, externalizing, and total problems in girls. Notably, the study incorporated both mother and father report of adjustment. Recently, the association between prenatal anxiety and preschoolers' adjustment was observed in the Amsterdam Born Children and their Development (ABCD) study, another large, population based birth cohort study (Loomans et al., 2011). This study modeled postnatal stress and utilized both maternal and teacher report of adjustment. Per mother report, children of mothers who reported more prenatal anxiety had more overall problem behaviors, hyperactivity/inattention problems, emotional symptoms, peer relationship problems, conduct problems and showed less pro-social behavior. Per teacher report, these children had more overall problem behaviors and less pro-social behavior.

In contrast, there is research to suggest that postnatal stress better accounts for adjustment in preschoolers. In a large, prospective longitudinal study, prenatal anxiety and depression were unrelated to children's physical aggression and crying behavior at 36 months, whereas postnatal maternal anxiety predicted these child adjustment outcomes (Bekkus, Rutter, Barker, & Borge, 2011). Similarly, again in a population based cohort study, postnatal parental hostility accounted for the observed relation between prenatal depressive symptoms and children's internalizing and externalizing symptoms (Velders et al., 2011). Additionally, one study reported no observed association between prenatal psychological symptoms and mother report of attention problems in their 3-year-old children (Van Batenburg-Eddes et al., 2013), whereas attention problems were associated with maternal postnatal mood symptoms.

The prediction of early childhood adjustment by prenatal experience is equally mixed in the domain of prenatal stressful experiences. In a prospective longitudinal study utilizing the Western Australia Pregnancy Cohort, a broadband measure of common stressors predicted maternal report of ADHD behaviors in their children at 2 years of age (Ronald, Pennell, & Whitehouse, 2005). Alternatively, in a study of prenatal psychosocial stressors among women living in an urban South African setting, prenatal stress was not associated with mother reported child behaviors problems at age 2 but was associated with behavior problems at age 4 (Ramchandani, Richter, Norris, Stein, 2010). Notably, this study did not account for postnatal stress. Similarly, in a separate study, stressful events in pregnancy increased the risk of internalizing problems at age 5 but not age 2, and externalizing problems when combining observations at ages 2 and 5, but not either time point independently (Robinson et al., 2008). Unfortunately this study also did not control for postnatal stressful experiences. By failing to account for postnatal stressors when examining the effects of prenatal stress, these studies do not establish the unique and independent effects of prenatal stress. Moreover, even in designs that control for postnatal stress, other explanations are possible (e.g., stress exposure across both pregnancy and childhood account for findings). The current study considers not only the effects of prenatal stress above postnatal stress, but also paths of stress from the prenatal through the postnatal period in predicting outcomes.

Stressed mothers, as well as mothers with psychopathology, are thought to be subject to greater response bias when reporting on their child's adjustment than mothers without psychopathology (De Los Reyes & Kazdin, 2005; Najman et al., 2001; Youngstrom, Izard, & Ackerman, 1999). There is evidence to suggest that the maternal response bias is a function of a mother's level of stress (De Los Reyes & Kazdin, 2001) and her level of psychological impairment (De Los Reyes & Kazdin, 2001; Najman et al., 2001), with increases in stress/psychopathology relating to increased discrepancy between the mother's ratings and the ratings of other reporters. Notably, of the studies of prenatal stress and preschool psychological adjustment reviewed above, two studies supporting a relation of prenatal stress with preschool adjustment avoided reporter bias (de Bruijn et al., 2009a; Loomans et al., 2011). These studies incorporated father (de Bruijn et al., 2009a) and teacher (Loomans et al., 2011) reports of child adjustment. More studies that minimize reporter bias are needed. This study utilizes multiple respondents to address the potential bias introduced by reliance on a single reporter.

The potential for sex differences in the effects of prenatal stress has not been adequately addressed by the current literature. Animal studies of prenatal stress suggest sexually dimorphic effects of prenatal stress. Prenatal stress has been associated with sexually dimorphic alterations to hormonal responses (of corticosterone in response to stress), to cognition in spatial memory tasks, and to anxious behavior (Bowman et al., 2004). Few studies have tested for sex differences in relation to prenatal stress and human adjustment, with existing studies reporting contradictory findings. With regard to the preschool period, the relation of prenatal psychological symptoms, child adjustment, and sex differences was evident in the ALSPAC sample (O'Connor et al, 2002). In this study, prenatal anxiety was most robustly associated with increased ADHD in boys and total emotional/behavioral problems in both boys and girls at 4 years. Notably, when the children in this sample were 7, prenatal anxiety was associated comparably with increased odds of emotional problems, ADHD and conduct disorder in both sexes (O'Connor et al., 2003). In the ABCD population study in Amsterdam, exposure to prenatal anxiety was associated with a stronger increase in overall problem behavior in boys compared to girls (Loomans et al., 2011). Furthermore, prenatal anxiety was significantly related to an increase in hyperactivity/inattention problems in boys but not girls. De Bruijn and colleagues (2009) found early pregnancy prenatal psychological symptoms to be associated with internalizing and total problems in boys, whereas third trimester prenatal psychological symptoms were associated with internalizing, externalizing, and total problems among girls.

With regard to prenatal stressful experiences, one study found the association of prenatal stressful experiences and ADHD among 7-year-olds to be most pronounced among males (Rodriguez & Bohlin, 2005). In contrast, prenatal stress was found to predict ADHD behaviors in both sexes in a separate study (Ronald et al., 2005). This study reported sex differences in the association of prenatal stressful experiences and autism in among 2-year-olds, with the association between prenatal stress and autism observed among males but not females. These findings raise the possibility for sex differences in the relations of prenatal stress and child adjustment. The current study examines potential sex differences in these relations.

2. Cortisol

One candidate mechanism explaining the link between prenatal stress and later child adjustment is disruption to the hypothalamic pituitary adrenal (HPA) axis (Van den Bergh et al., 2005; Van den Bergh et al., 2008). In response to stress, the hypothalamus secretes corticotropin releasing hormone (CRH), which stimulates the production of adrenocorticotropin hormone (ACTH) from the pituitary gland, and this in turn stimulates the secretion of cortisol by the adrenal cortex (Gunnar & Quevedo, 2007). Salivary cortisol is a common, noninvasive index of HPA functioning used in research (Schwartz, Granger, Susman, Gunnar, & Laird, 1998). The HPA axis displays a diurnal rhythm, with cortisol levels reaching their peak about 30 minutes after awakening and decreasing throughout the day to close to zero at bedtime (Kirschbaum et al., 1990).

Prenatal stressors including daily hassles (Gutteling et al., 2004; Gutteling et al., 2005), exposure to Chernobyl (Huizink et al., 2008), significant life events in pregnancy (Entringer, Kumsta, Hellhammer, Wadhwa, & Wüst, 2009), prenatal anxiety and/or depression (de Bruijn et al., 2009b; Grant et al., 2009; Laurent et al., 2013; O'Connor et al., 2005; Van den Bergh et al., 2008), and pregnancy related anxieties (Gutteling et al., 2004 & 2005; Tollenaar et al., 2011) have been linked to altered cortisol function in children. These associations are observed in infancy (Grant et al., 2009; Tollenaar et al., 2011; de Bruijn et al., 2009b), early childhood (Laurent et al., 2013; Gutteling et al., 2004 & 2005), middle childhood (O'Connor et al., 2005), adolescence (Huizink et al., 2008; Van den Bergh et al., 2008) and adulthood (Entringer et al., 2009). The most robust association between cortisol and prenatal stress is increased basal levels in children exposed to prenatal stress (de Bruijn et al., 2009b; Gutteling et al. 2004, 2005; Huizink et al., 2008; O'Conner et al., 2005; Van den Bergh, et al., 2008). With regard to diurnal cortisol patterns, one study found support for a relation between stress exposure and a flattened diurnal profile (Van den Bergh et al., 2008), one study reported a steeper diurnal slope among prenatally stressed children on school days compared to a weekend day (Gutteling et al., 2005); and two studies found no differences in diurnal pattern among the prenatally stressed (Entrigner et al., 2009b; O'Conner, et al., 2005). In further contradiction to these findings, one study found prenatal stress to predict higher salivary cortisol at five weeks, but then lower salivary cortisol reactivity at five months and one year (Tollenaar et al., 2011). One study additionally reported an independent effect of prenatal stress on low cortisol levels in preschoolers (Laurent et al., 2013). In a review of conceptual issues related to the use of salivary cortisol as an outcome measure in the study of stress in pregnancy and HPA axis function, Egliston and colleagues highlighted that that the level at which salivary cortisol reaches "sub-optimal" levels in children is presently unknown (Egliston, McMahon, & Austin, 2007). Additionally, these researchers highlighted that studies point to the importance of a variety of diurnal cortisol indicators, including morning level, evening level, diurnal pattern, and night-time time secretions. In the absence of a clear, best indicator of developmental risk, the authors advised that the research question, population characteristics, and resource constraints determine which measure is used (Eliston et al., 2007). Based on the literature implicating both morning levels and diurnal patterning as subject to the influence of prenatal stress, the present study uses children's cortisol morning level and diurnal slope as measures of regulation of the HPA-axis.

Harris & Seckl (2011) propose that exposure to excess glucocorticoids in pregnancy influence HPA functioning in children, but that the direction of effect (heightened versus muted diurnal patterns) may vary. The authors hypothesized that the direction of change is moderated by additional vulnerability factors. In support of this argument, one study found that children of mothers directly exposed to the 9/11 World Trade Center attacks showed lower salivary cortisol, but only if the mother developed PTSD (Yehuda, Engel, Brand, Seckl, Marcus, & Berkowitz, 2005). This suggests that an additional factor, perhaps implicated in determining if a woman will develop PTSD in response to a traumatic event, plays a role in the prenatal stress and children HPA axis relation.

Both high and low levels of HPA activity have been associated with child psychopathology (Alink et al., 2008; Ashman, Dawson, Panagiotides, Yamada, & Wilkson, 2002; Gunnar, Sebanc, Tout, Donzella & van Dulmen, 2003). High levels of cortisol were observed among children with internalizing and externalizing symptoms (Essex et al., 2002; Klimes-Dougan, Hastings, Granger, Usher, & Zahn-Waxler, 2001; Gunnar, Sebanc, Tout, Donzella, & van Dulmen, 2003). In contrast, low levels of cortisol were observed among children with greater adjustment problems (Granger, Serbin, Schwartzman, Lehoux, Cooperman, & Ikeda, 1998). Much of the literature on children's cortisol and adjustment has focused on cortisol reactivity. In this domain, high cortisol reactivity has been associated with lower social competence (Granger, Weisz, McCraken, Ikeda & Douglas, 1998; Gunnar, Tout, de Haan, Pierce, & Stansbury, 1997; Hart, Gunnar, & Cicchetti, 1995), higher internalizing symptoms (Ashman, Dawson, Panagiotides, Yamada, & Wilkson, 2002), with mixed findings on the relation of greater externalizing symptoms and low cortisol reactivity (Alink, van Ijzendoorn, Bakersmans-Kranenburg, Mesman, Juffer & Koot, 2008). Taken together, these findings may suggest that any variation in typical diurnal cortisol patterns reflect a dysregulated stress response system implicated in children's adjustment problems.

HPA axis disruption is known to have long-term effects including learning deficits, reduced attention, and altered immune function in animals (e.g. Gué et al., 2004; Vallee et al., 1997) and humans (e.g. O'Conner et al., 2003). In animal models, prenatal stress has been shown to alter the offspring's HPA axis (Harris & Sekl, 2011; Tegethoff et al., 2009) and to confer HPA axis feedback insensitivity (Welberg, Seckl, & Holmes, 2000). Critically, animal studies have been able to document that offspring's altered HPA axis mediates behavioral disturbances (Egliston et al., 2007). Only one study (Van Den Bergh et al., 2008) was found that investigated the potential mediating effect of disruptions in the HPA axis to the relation between prenatal stress and adjustment. Van den Bergh and colleagues (2008) found disruptions in diurnal cortisol served as the mechanism linking prenatal stress and depressive symptoms in adolescent girls. The current study seeks to build upon this work to examine if disruptions to the HPA axis mediate the relations of prenatal stress and child adjustment observed among preschool-age children.

Prenatal stress and children cortisol activity have not been studied longitudinally. Even outside the prenatal stress literature, most studies of children's cortisol have utilized a cross-sectional design and few studies have examined children's cortisol over time. Extant longitudinal studies point to relatively consistent profiles of cortisol functioning over time within the same developmental period (Van Ryzin, Chatham, Kryzer, Kertes, & Gunnar, 2009; Zalewski, Lengua, Thompson, & Kiff, under review). One study found that children's morning and evening cortisol levels at age 6 were predicted by levels at age 4.5 and showed significant stability. At the same time, there were significant individual differences in the degree of stability attributed to home chaos, and postnatal parental stress and psychopathology (Laurent, Neiderhiser, Natsuaki, Shaw, Fisher, Reiss, et al., 2013). This study identified the need for repeated measurements to yield a better picture of HPA axis stability and the contributions of environmental and parental risk to the stability of the system. The present study has the potential to examine the stability of preschooler's cortisol, and whether the trajectory of children's cortisol relates to prenatal and postnatal mood and stressful experiences.

3. Executive control

Research also points to the potential for stress exposure to have effects on children's behavioral self-regulatory abilities (Van den Bergh et al., 2005). Executive control is a behavioral measure of volitional regulation via the capacity to inhibit a dominant response in favor of a more adaptive, subdominant response (Murray & Kochanska, 2002). Executive control facilitates self-monitoring, flexibility, response inhibition, and resistance to interference (Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996). Executive control processes are centered in the prefrontal cortex (Blair & Razza, 2007; Bush, Luu, & Posner, 2000; MacDonald, 2008), with original mentions to this construct referring to an "anterior attention network" (Davis, Bruce, & Gunnar, 2001). This neural underpinning is evidenced by lesion research (e.g. prefrontal lesions result in failures to inhibit prepotent responses of wall-climbing in rats; Kolb, 1984 & 1990) as well as imaging research (e.g. activation of the prefrontal cortex is observed in go-no-go tasks tested developmentally using functional MRI; Casey, et al., 1997).

The prefrontal cortex has been identified as a brain region extremely sensitive to the effect of stress exposure (Arnsten, 2009). While acute stress results in structural changes to the hippocampus after several weeks of stress exposure, dendrites in the prefrontal cortex demonstrate structural changes more rapidly. Existing evidence suggests structural changes to the prefrontal cortex dendrites are evident after week of stress exposure (Brown, Henning, & Wellman, 2005) or even after a single exposure to stress (Izquierdo, Wellman, & Holmes, 2006). Neural effects of exposure to stress have been documented not only for postnatal stress experiences, but also prenatal stress experiences. Prenatal stress is associated with a reduction in dendritic spines density (Murmu et al., 2006), dendritic spine alterations (Michelsen et al., 2007), dendritic spine arborborization and synaptic loss (Barros, Duhalde-Vega, Caltana, Brusco, & Antonelli, 2006) in the prefrontal cortex in rodent studies.

Few human studies have examined executive control in relation to prenatal stress, though the extant literature supports this association (Buss, Davis, Muftuler, Head, & Sandman, 2010; Mennes, Van den Bergh, Lagae, & Stiers, 2009; Van den Berg et al, 2005; Van den Berg et al., 2006). One study assessed visual attention by requiring children to memorize target letters and then quickly report their presence or absence in subsequent trials. Maternal prenatal anxiety related to an impulsive cognitive style, as operationalized by a fast responding, high error style on an encoding task (Van den Bergh et al., 2005). A second study using the same sample found that boys of mothers with prenatal anxiety demonstrated exaggerated decreases in performance on a continuous performance task. This was noted by a more pronounced decrease in processing speed and increase in variability in reaction times compared to control subjects (Van den Bergh et al., 2006). There were no observed differences on the overall task performance. Finally, adolescent males of mothers with high prenatal anxiety were found to perform comparably to non-stressed adolescents on a Go/No Go task (Mennes et al., 2009), which is a behavioral measure of response inhibition.

An independent, prospective study that accounted for postnatal maternal mood symptoms examined the relation between maternal anxiety during pregnancy and aspects of cognitive function (Loomans et al., 2012). Prenatal stress predicted intra-individual variability on one reaction time task. In a subsample of children born to mothers whose prenatal anxiety scores were above the 90th percentile, prenatal psychological symptoms were associated with reaction time and intra-individual variability in reaction time in incompatible trials of a second reaction time task. Additionally in this subsample, the increased intra-individual variability associated with prenatal psychological symptoms in the first reaction time task was observed in boys but not girls. This study did not find an association between prenatal psychological symptoms and children's processing speed.

In contrast, an independent study of adult women exposed to significant prenatal stressors found an association between prenatal stress and working memory (Entringer, Buss, Kumsta, Hellhammer, & Wadhwa et al., 2009). Although the two groups did not differ on a task of working memory in a placebo condition, prenatally stressed women showed longer reaction times after hydrocortisone administration compared to the comparison group. These findings suggest that there are long-term impairments in working memory, though these disturbances may be modulated by other factors. That is, the disturbances to executive control may emerge in the context of stress and stress-responding.

Executive control is consistently and robustly related to children's social competence and adjustment problems (Kochanska et al., 1996; Lengua 2003). Higher executive attention has been correlated with greater social competence in children (Ciairano, Visu-Petra, & Settanni, 2007; NICHD Early Child Care Research Network, 2003; Rudasill & Konold, 2008) and has also been show to prospectively predict greater social competence (Gewirtz, Stanton-Chapman, & Reeve, 2009; Mintz, Hamre, & Hatfield, 2011; Nigg, Quamma, Greenberg, & Kusche, 1999). Self-monitoring, an aspect of executive control, may foster self-monitoring of one's impact on peers and others, and alert children to social norms. Lower executive function correlates with greater externalizing problems (Gusdorf et al., 2011; Hughes, White, Sharpen, &

Dunn, 2000; Nigg et al., 1999; Riggs, Greenberg, Kusche, & Pentz, 2006), ADHD behaviors (Gewirtz et al., 2009; Gusdorf et al., 2011), and to predict greater internalizing problems (Eisenberg, Cumberland, et al., 2001; Eisenberg, Gershoff, et al., 2001). Executive attention may afford the capacity to direct one's attention to attenuate distress, inhibit antisocial behaviors, and modulate anger and acting out behaviors.

Emerging research indicates the potential utility of studying not only static levels of executive control, but also its developmental trajectory. This stems from recent findings that children for whom self-regulation improves more slowly are at higher risk for poor adjustment during later developmental periods (King, Fleming, Monahan & Catalano, 2011; King, Lengua & Monahan, 2011; Monahan et al., 2009). In these studies, the rate of development of self-regulatory capacity predicted adjustment above the effect of self-regulation at a given time point. Executive attention processes are present in the first six months of life (Sheese, Rothbart Posner, White, & Fraundorf, 2008) and increase modestly in the toddler years (Kochanska, Murray & Harlan, 2000). These abilities show marked growth in children between the ages of 3 and 6 and more moderate growth in middle childhood (Carlson, 2005; Diamond & Taylor, 1996; Jones, Rothbart, & Posner, 2003; King, Lengua & Monahan, 2010; Kochanska et al., 1996; Murphy et al., 1999; Reed, Pien, & Rothbart, 1984) but at a more moderate rate. As such, the preschool period reflects a unique window to examine the development of executive control. The current study examines both children's executive function and its development as predicted by prenatal and postnatal stress and as predictors of children's adjustment.

Taken together, these bodies of literature show a relation of prenatal stress to impaired executive function and, in turn, the prediction of adjustment problems by impairments in executive function. These relations raise the possibility that executive control may mediate the relation between prenatal stress and psychopathology. However, no such tests of mediation were found. The current study seeks to further the study of prenatal stress by examining if enduring disruptions to the children's executive control or its development mediate the relations of prenatal stress and child adjustment observed among preschool-age children.

This study

The current study has the potential to inform developmental science in several regards. First, while enduring effects of prenatal stress have been substantiated by animal models and preliminary evidence replicates these findings in humans, the preponderance of human research on prenatal stress does not extend beyond infancy (Talge et al., 2007). Of the studies that examine outcomes beyond infancy, many fail to account for postnatal stressors. As such, the studies do not establish the unique and independent effects of prenatal stress, or the potential role of chronic stress exposure. The current study considers not only the effects of prenatal stress above postnatal stress, but also paths of stress from the prenatal through the postnatal period in predicting outcomes in the preschool period. The preschool period reflects not only an understudied period of development in the prenatal stress literature, but a time in which self-regulatory abilities are rapidly developing (e.g. Lengua, 2012). By examining the preschool period in a longitudinal design, this study is uniquely poised to explore the role of prenatal stress on development. In combination with the potential for continuity between behavior problems, later psychopathology and adult psychiatric disorders (Mesman & Koot, 2001), this is an aptly studied age-group, and a group for whom early intervention could be particularly meaningful.

Second, the operationalization of prenatal stress in the current literature is broad, spanning constructs of prenatal maternal mood or psychopathology (particularly depression and anxiety), prenatal maternal stressful experiences, and pregnancy specific anxieties. The field has failed to show that maternal psychopathology is a proxy for traditional definitions of stress. Further, no studies have tested for differences (specificity) in relations of stress constructs to varied mediators and child adjustment outcomes. The current study aims to tests specificity in the relation of prenatal stress constructs to mediators and outcomes. To account for the possibility that maternal characteristics (stress, psychopathology) influence reports of child adjustment, both maternal and teacher reports of adjustment will be utilized.

The primary aim of the current study is to empirically test mediation pathways from prenatal stress to child adjustment. Extant research has implicated the HPA axis and children's executive control as being altered by prenatal stress. These systems are additionally known to relate to child adjustment, and therefore might represent mechanisms of the effects of prenatal stress on child adjustment. This study is uniquely poised to these two candidate mechanisms. The present study examines children's diurnal cortisol and executive functioning with the hypotheses that: (1) **Prenatal stress exposure will predict disruptions in diurnal cortisol and executive control.** Hypothesis 1A: Stressful experiences, reflecting acute exposure to stress, will predict high morning levels and diurnal slope of cortisol. Maternal mood, perhaps implicated in more prolonged experiences of stressors and their generation, will relate to low morning levels and blunted diurnal slopes of cortisol. Hypothesis 1B: Prenatal stress will predict lower initial levels and slower developmental growth of executive function across the preschool period. Hypothesis 1C: Prenatal mood and stressful experiences will predict putative mediators above the effect of postnatal mood and stressful experiences. (2) **Disruptions to the diurnal cortisol and executive control will mediate the relations of prenatal stress exposure to children's preschool social competence and behavior problems.** Hypothesis 2A: The initial levels and developmental trajectories of morning level and diurnal slope of cortisol and executive control will predict mother and teacher reports of behavior problems and social competence of preschoolers. Hypothesis 2B: Higher levels of prenatal stress will predict mother and teacher reports of more behavior problems and less social competence. Hypothesis 2C: The effect of prenatal stress exposure on child adjustment outcomes will be explained through the indirect paths of disrupted HPA axis functioning and impairments in executive control.

Hypothesis 2D: Prenatal mood and stressful experiences will predict children's adjustment above the effect of postnatal mood and stressful experiences. Exploratory Aim (1) **The relations of prenatal stress, putative mediators (diurnal cortisol and executive control), and preschoolers' adjustment may vary as a function of stressor type or child sex.** Exploratory Hypothesis 1A: There may be differences in the relations of prenatal psychological symptoms versus prenatal stressful experience in their prediction of children's cortisol, executive control, and social-emotional functioning. Exploratory Hypothesis 1B: The relations of prenatal stress to children's cortisol, executive control, and social-emotional functioning may be different for males and female preschoolers.

Method

Participants

Participants were a community-based sample of 306 children and their mothers assessed across 4 waves of data. Time 1 assessments began when children were 3 years old (*M* = 36.75 mos., *SD* = 1.31) with following assessments occurring 9 months later. Families were recruited from the university hospital subject pool, preschools, co-ops, and daycares and were selected to over-represent poverty and low-income. The sample represents the demographic characteristics of the urban area surrounding the university in the Pacific Northwest. Only one child in the target age range per family was permitted to participate. Children with developmental disabilities and families not fluent in English were excluded from the study to ensure adequate comprehension of the procedures. A female primary caregiver was required to participate. At Time 1 the sample consisted of 50% girls, 9% African Americans, 3% Asian Americans, 2% Native Americans, 10% Latino or Hispanic, 64% European Americans, and 12% children with other or multiple ethnic or racial backgrounds. The sample over-represented families in poverty and low income, with 29% of the sample at or near poverty (at or below 150% of the federal poverty threshold), 28% low income (below the local median income of $58K), 25% middle income (above the median income to $100K), and 18% upper income (above $100K). Attrition was low with 95% of participants remaining at Time 2-4.

Procedures

Families were assessed in offices on a university campus. They were assessed at 4 time points each separated by 9 months when children were 36-40, 45-49, 54-58, and 63-67 months. With approval by the Human Subjects Institutional Review Board, both active parental consent and child assent were secured prior to data collection. Children completed neuropsychological measures of executive control while mothers completed questionnaire measures of symptoms in a separate room. Mothers provided contact information for their child's teachers, who were sent brief adjustment questionnaires. Families collected cortisol samples in the morning and evening for three consecutive days after the laboratory session. Families received $70 for their first assessment and compensation increased by $20 for each of the 3 subsequent assessments. All study procedures were approved by the university's Institutional Review Board (IRB).

Measures

Prenatal stressful experiences. At the third assessment (when children were 63 postnatal months), mothers were asked to retrospectively report on stressful events that occurred during their pregnancy. To assess stressors, the 15-item General Life Events Scale (Sandler, Ramirez & Reynolds, 1986) was used. This measure has been previously shown to have significant associations with child adjustment (Lengua & Long, 2002). Mothers are asked to report whether certain stressful events (e.g. medical problems, financial difficulties, conflict with loved one) occurred during her pregnancy with the study child to assess prenatal stressful experiences.

Prenatal psychological symptoms. At the third assessment, mothers were also asked to retrospectively report on their mood in pregnancy. To assess prenatal psychological symptoms, the Edinburgh Postnatal Depression Scale (EPDS; Cox, Holden, & Sagovski, 1987) was utilized. This scale was developed to assist primary care health professionals to detect mothers suffering from postnatal depression. In this study, it was used to assess for prenatal depression by having mothers retrospectively report on the items. The EPDS was developed at health centers in Livingston and Edinburgh. It consists of ten short statements. The mother selects which of the four possible responses is closest to how she was feeling during her pregnancy with the study child. The validation study showed that mothers who scored above threshold 92.3% were likely to be suffering from a depressive illness of varying severity. However, no cutoff score will be used in this study to capture the full range of mood symptoms present in sample mothers during their pregnancy.

At the first time point, mothers retrospectively reported on the following pregnancy and birth variables:

Prenatal alcohol exposure. Mothers reported on their use of beer or wine during pregnancy (0 = never used, 1 = used). Mothers reported on their use of hard liquor during pregnancy (0 = never used in pregnancy, 1 = used in pregnancy). A prenatal alcohol exposure composite was the sum of these two variables.

Prenatal medication exposure. Mothers reported on their use of (1) steroids, (2) valium, (3) tranquilizers, (4) anti-seizure medication (5) diabetes medication (6) antibiotics (7) sleeping pills (8) prescription medication and (9) over-the-counter medication during pregnancy (0 = no use in pregnancy, 1 = use in pregnancy). A prenatal medication exposure composite was the sum of these nine variables.

Prenatal health risk. Mothers reported on their health in pregnancy (0 = good, 1 = fair/poor). Mothers reported on pregnancy complications (0 = no complications, 1 = complications). A prenatal health composite was the sum of these two variables.

Delivery risk. Mothers reported on whether their child was born term or preterm (0 = term, 1 = preterm). Mothers reported on whether their child experienced fetal distress (0 = did not experience, 1 = experienced). A delivery risk composite was the sum of these two variables.

Prenatal cigarette use. Mothers reported on their use of cigarettes in pregnancy (0 = never, 1 = 1-2 times, 2 = 3-9 times, 3 = 10-19 times, 4 = 20-39 times, 5 = 40+ times).

Birth weight. Mothers reported on their child's birth weight (1 = 2-3 pounds, 2 = 4-5 pounds, 3 = 6-7 pounds, 4 = 8-9 pounds, 5 = 10-11 pounds, 6 = other).

Postnatal mood. At T1-T4, mothers reported on their depressive symptoms over the previous month using the 20-item Center for Epidemiological Studies–Depression Scale (CES-D; Radloff, 1977). This questionnaire was designed to measure depressive symptoms in the general population. Participants indicated whether each symptom was present on a scale of 0 (rarely/never) to 3 (most of the time), and the items were summed for a total score at each time point. Internal consistency was .88. Total scores were averaged across time points for a single measure of postnatal mood symptoms.

Postnatal stressful experience. Postnatal stressful experiences were assessed with mother report on the General Life Events Schedule for Children (Sandler, Ramirez & Reynolds, 1986). The 29 events include moderate to major negative events including changing schools, death of a family member or friend, parental arrest, and loss of friends. Mothers reported whether events occurred in the previous 9 months at each time point, and the total score was the number of events averaged across the four postnatal timepoints.

Cortisol. Cortisol data are available at each of the T1-T4 assessments. At the end of each assessment session at the laboratory, mothers were trained in the collection of cortisol and were given a home collection kit, including instructions on how to collect the saliva samples at home. Specifically, mothers were instructed to collect their child's saliva 30 minutes after the child woke in the morning and 30 minutes prior to bedtime for three consecutive days. Mothers were to place a sorbette (Salimetrics, LLC State College, PA) under the child's tongue for 1 minute and then place the sorbette into a prelabeled swab storage tube. Mothers repeated this process with another sorbette to ensure adequate saliva volume. Mothers completed a questionnaire at each time of sampling, indicating the time at which the sample was taken, the occurrence of an event previously known to alter cortisol (e.g. food consumption, use of inhaler, and illness). A staff member called families on the first night to ensure proper collection and answer questions. Mothers were reminded to avoid sampling when their children were using steroid based medications or were ill. Mothers were mailed additional materials if they accidently sampled when the child was ill. A reminder call was placed on the third evening to prompt mothers to return the packets via the mail. These methods have been successfully used in childhood samples (Bruce, Davis, & Gunnar, 2002) and resulted in an 89% return rate at T1, and an 86% return rate at T2 and T3, and 82% at T4.

Samples were sent to the university's Biobehavioral Behavioral and Nursing Systems laboratory for processing, where they were stored at -70 degrees C. until extraction. For processing, all sample tubes were thawed to room temperature and centrifuged at 1000 rpm for 10 minutes in order to separate the saliva from the collection swab. The cleared eluant was then transferred to a 1.6 ml Eppendorf tube and stored at minus 70 degrees Celsius until testing for cortisol. Prior to assay, each sample was subjected to another centrifugation step of 5,000 rpm for 5 minutes in order to separate out small particulates and residual mucin. In order to test for the presence of salivary cortisol, 25 μL of saliva from each sample was transferred into each of two wells, producing duplicate samples for each assay; sample values were then averaged. The concentration of cortisol in each sample was extrapolated from a standard curve generated in each test plate and the results were averaged in order to give an adjusted result. Samples were assayed using the High-Sensitivity Cortisol Salivary Enzyme Immunoassay Kit provided by Salimetrics LLC (State College, PA). The sensitivity of this kit ranges from .005 ug/dL to 2.5

ug/dL. All samples from the same subject for each set of saliva were included in the same assay batch to minimize inter-assay within-subject variability. Intra-assay reliabilities were obtained using the high and low cortisol controls provided by Salimetrics.

A diurnal value was computed by subtracting the average evening from the average morning value. Cortisol data is commonly positively skewed, and our data followed this distribution pattern. In these instances, it is common practice to log transform cortisol values. Log transformations were applied to average morning and the average evening variables and, using the log-transformed morning and evening values, a diurnal pattern was recalculated.

The following cleaning steps were employed for the cortisol data. Assay results over 2.0 ug/dl were deemed biologically implausible and not utilized as part of the study sample, consistent with methods used in other studies (Ashman et al., 2002). Mothers completed a daily questionnaire regarding sampling times and their children's health, medication use and eating times on sampling days. The questionnaires were reviewed to ensure compliance. Samples collected 90 minutes or more from wake or before bed were excluded from analyses. Use of steroid medications, an inhaler, health, and food intake has been shown to affect cortisol levels. Correlations were conducted to examine potential associations between medication use, steroid use, illness, and food intake with cortisol values and other study variables. Overall, there were fewer significant correlations than would be expected by chance, with no systematic pattern and modest magnitudes. Therefore, no cortisol collection compliance variables were included as covariates. Wake time, bed time, and latencies between these times and collection (e.g. the time between the child awakening and the time of the morning sample) were examined as potential covariates. Composites of average wake time/bed time/latency were computed based on the average value for these variables across the 3 days of sampling. These composites were correlated with cortisol values. There were few significant associations. Time 1 morning cortisol levels were modestly associated with time 1 later AM collection time, r =-.17, p =.005 and longer

AM latency to collect, *r* -.14, *p* =.039. Also, time 3 diurnal slope was correlated with time 3 PM latency to collect, *r* =-.18, *p* =.005. Given the few and modest associations among collection times and cortisol levels, bias introduced due to collection times was deemed to be minimal, and therefore these time variables were not included as covariates.

Executive Control. Multiple components of executive control, including attention regulation and cognitive and behavioral inhibitory control were assessed using neurocognitive measures. Attention regulation (focusing and shifting) was assessed using the NEPSY-II auditory and visual attention subscales. The Auditory Attention subtest is a continuous performance test assessing the ability to be vigilant and to maintain and shift a selective auditory set. Cognitive inhibitory control was assessed using the Inhibition task on the NEPSY-II and two Stroop-like tasks, Day/Night (Gerstadt, Hong, & Diamond, 1994) and Dimensional Change Card Sort (Zelazo, Muller, Frye, & Marcovitch, 2003). Inhibition requires the child to inhibit a prepotent response by naming a shape (e.g., circle) while pointing to a different shape (e.g., square). Day/Night requires the child to respond by saying "day" when shown a picture of the moon and stars and "night" when shown a picture of the sun. Scores are the proportion of correct responses. The Card Sort task assesses cognitive inhibitory control, as well as attention focusing and set shifting. Children were instructed to sort cards according to either the shape or color properties on the target cards. Scores are the proportion of correct trials out of the 36 total possible trials. Behavioral inhibitory control was measured using Bear-Dragon (Kochanska, Murray, Jacques, Koenig, & Vandegeest, 1996), a simplified version of the game Simon Says. Children are asked to perform actions when a bear puppet but not a dragon puppet gives the direction. Scores are the proportion of scores across both bear and dragon items to the total possible score. Finally, Head, Toes, Knees, Shoulders (HTKS) integrates attention and inhibitory control (Ponitz et al., 2008). Children are asked to enact the opposite of what the experimenter directs (e.g. touch toes when asked to touch head). Total scores are the proportion of the score across items to the total possible score. From these measures, an index of executive control was created by computing the mean of the proportion scores of the individual tasks, consistent with methods used in previous research (Carlson & Moses. 2001; Kochanska et al., 1996; Lengua, Honorado, & Bush, 2007).

Maternal report of child adjustment. Children's adjustment problems were assessed using parent report on the Child Behavior Checklist (CBCL; Achenbach, 1991). The CBCL has a strong empirical history and has been shown to be both a valid and reliable measure of children's psychopathology (Achenbach, 1991). Internalizing symptoms were assessed by summing maternal responses on the Anxiety (12 items) and Depression (12 items) scales and externalizing symptoms included the Aggression and Delinquency scales (21 items). Together, a total problems score, which combined the internalizing and externalizing symptoms scales, was used. Raw scores were be analyzed.

Mothers additionally reported on children's social competence using the Social Skills Rating Scale (SSRS: Gresham & Elliot, 1990). Mothers rated their child's cooperation (9 items), assertiveness (9 items), responsibility (10 items), and self-control (10 items) for a social competence score. The alphas for mother report of social competence and total problems were .84 and .88, respectively.

Teacher report of chid adjustment. Teachers rated children's social competence and adjustment problems using the preschool teacher form of the SSRS (Gresham & Elliot, 1990). Teachers rated children's cooperation (12 items), assertiveness (8 items) and self-control (10 items) for a social competence score (30 items). Teachers rated children's externalizing problems (7 items), internalizing problems (6 items) and hyperactivity (6 items) for a total adjustment problems score (19 items). The alphas for teacher report of social competence and total problems were .93 and .90, respectively.

Results

Analytic Plan

Before testing the study hypotheses, the correlations of prenatal and postnatal maternal mood and stressful experiences with child cortisol, executive control, and adjustment were examined to assess the feasibility of the proposed hypotheses. As reported below, initial correlations showed children's cortisol to be unrelated to prenatal and postnatal maternal measures of mood and stressful experiences, as well as mother and teacher report of child adjustment. Typically, analyses with these variables would not proceed. Nonetheless, the analyses with cortisol variables were conducted to thoroughly address the hypotheses of this study, and those results are reported in Appendix A.

Unconditioned latent growth curve models of cortisol (one model for morning level, one for diurnal pattern) and executive control with linear growth factors across the 27 months (4 observations at 9 month intervals) were specified. These models indicated if children varied significantly in their initial levels of cortisol/executive control as well as their rate of change in these trajectories. For constructs with significant variability in growth (executive control only, see below), intercept and slope factors were included in further models. Because children's diurnal cortisol (morning level and diurnal slope) did not show significant variability in growth and the intercept of cortisol was unrelated to the study variables, cortisol analyses are not reported in the main text. Results can be found in Appendix A. Because we were interested in obtaining temporal precedence in the prediction of adjustment from levels and growth in putative mediators, the intercept will be set to T1.

In addition, the associations of potential covariates with the predicted variables (executive control, social competence, and total problems) were examined. Potential covariates of relevance to the study of prenatal stress, child regulatory processes and adjustment included: income, child sex, prenatal alcohol exposure, prenatal medication exposure, prenatal health risk, delivery risk, prenatal cigarette use, and birth weight. Covariates were identified in a regression framework. Nonsigificant covariates were trimmed from the full, SEM model.

To test the feasibility of combining teacher and mother reports of each adjustment outcomes, the pattern of results from preliminary regressions, correlations across reporter, and confirmatory factor analysis (CFA) were examined. A well-fitting CFA with significant factor loadings would support the combination of mother and teacher reports of adjustment, using standardized and then averaged mother and teacher reports to create two measures of child adjustment. An ill-fitting CFA would necessitate the assessment of mother and teacher reports separately, doubling the number of statistical tests and potentially suggesting that maternal characteristics bias the reporting of child adjustment.

All tests of the full analytic model used Full Information Maximum Likelihood Estimation (FIML). Participants with and without missing data were compared to assess for potential bias. FIML requires estimation of means, intercepts, covariance, and path coefficients, and uses all data available simultaneously to calculate parameter estimates. FIML has been found to be less biased and more efficient than other techniques for missing data (Arbuckle, 1996). Analyses were conducted using Mplus 6.0 software (Muthén & Muthén, 2010).

To test Aim 1, prenatal stress exposure was tested as a predictor of the intercept and slope of executive control (Aim 1b), where relations with lower initial executive control and slower rates of development were hypothesized. It was further predicted (Aim 1c) that prenatal mood and stressful experiences would predict executive control above the effect of postnatal mood and stressful experiences. Again, it was reasonable to test only feasible hypotheses, where the preliminary examination of correlations suggested the hypothesized relations were plausible. However, to be complete, model results examining the hypothesized associations with cortisol variables are reported in Appendix A. Tested models included previously identified covariates.

Next, to test the relation of executive control and child adjustment (Aim 2a and 2b), intercept and slope executive control were modeled as predictors of child adjustment (adjustment problems and social competence, as reported by mother and teachers). Finally, to test if executive control accounted for the effect of prenatal stress on child adjustment (Aim 2c) an indirect path from prenatal stress, through executive control, to each adjustment outcome was specified. Using the bootstrapping method as outlined in MacKinnon & Lockwood, 2002, bias corrected confidence intervals were computed. A significant indirect effect would reflect evidence of mediation. Models tested hypothesis 2D, that prenatal mood and stressful experiences would predict outcomes above the effect of postnatal mood and experiences, by including both prenatal and postnatal variables in the model. In addition, indirect models were included that examined if the effect of prenatal stress operated through postnatal stress, perhaps reflecting a chronic course of exposure to risk.

Finally, to address the exploratory study aim (Exploratory Aim 1a) exploring whether prenatal psychological symptoms and prenatal stressful experiences differ in their relation to children's executive control and preschool adjustment, two models, with both predictors (prenatal psychological symptoms and prenatal stressful experiences) in each model were specified. In the first, paths were set to be equal across stress domain, in the second, the paths were free to differ across domain. A significant χ^2-difference test would indicate there are significant differences in the structural paths or covariances across the models, in other words, that there were differences in the magnitudes of the two predictors.

To address the exploratory study aim (Aim 3b) exploring whether there is evidence for sex differences in the relations of prenatal stress to disruptions in executive control and adjustment outcomes, sex effects were evaluated. A cross-group path model in which all parameters were free to differ across gender was compared to a model in which all paths and covariances were constrained to be equal across gender. As above, a significant χ^2-difference test would indicate there are significant differences in the structural paths or covariances across the models, in other words, that there is evidence for sex differences in these relations.

Structural equation modeling, and in particular LGC modeling is a longitudinal analysis technique that affords the ability to model growth over time. LGC models are more powerful than traditional fixed effects repeated measures ANOVA for detecting change and correlates of change (Curran & Muthén, 1999). SEM models test not only the magnitude and significance of each proposed relation (both predictors of intercept and change, as well as variables predicted by intercept and change) but also the overall fit of the observed covariance matrix to the expected associations. That is, the implied covariance matrix is compared to the covariance matrix observed in the data. Thus the null hypothesis is that the proposed model is a close approximation to the relations that exist in the data, while the alternative hypothesis is that the proposed model does not fit (MacCallum, Browne, & Sugawara, 1996). Power, therefore, is the ability to detect when the alternative hypothesis is false. This approach uses the root-mean-square error of approximation (RMSEA; Steiger & Lind, 1980) as an estimate of fit. RMSEA is a preferred estimator of model fit as it considers degrees of freedom and the number of parameter estimates, meaning parsimony is favored. RMSEA values between .00 and .05 are thought to indicate good fit, and RMSEA > .08 indicates poor fit.

Missing data

Analyses were conducted to assess the degree of bias introduced by missing data and sample attrition. All participants had complete data on income. Information on the number of cases missing data on each variable, along with other descriptive statistics, is found in Table 1. Levels of prenatal and postnatal mood, prenatal and postnatal stressful experiences, prenatal alcohol exposure, prenatal medication exposure, cortisol morning level across time points, diurnal slope across time points, executive control across time points, mother report of adjustment, and teacher report of adjustment were compared across participants missing no data and those missing any data. Participants missing any data (N = 116) on study variables were compared with those missing no data (N = 190) to assess the extent of bias introduced by missing data. Participants missing data differed from those not missing data in that they had lower T3 executive control (M missing = 0.49, M no missing = 0.50, $t[286] = 0.78$, $p = .01$), lower prenatal alcohol exposure (M missing = 0.12, M no missing = 0.21, $t[236] = 1.70$, $p = .001$), and higher prenatal mood symptoms (M missing = 15.14, M no missing = 13.92, $t[284] = -2.11$, $p = .01$). However, the effect sizes of the associations of missingness to T3 executive control ($r = -.05$), prenatal alcohol exposure ($r = -.11$), and prenatal mood ($r = .12$) were modest and did not reach previously cited thresholds for introducing substantial bias (i.e., $r > .40$, see Collins et al., 2001). Thus, it appears that little bias was introduced due to missing data.

Correlations

To examine if prenatal and postnatal maternal mood and stressful experiences were associated, correlations were conducted. All prenatal and postnatal variables were positively correlated, with medium to large effect sizes. For details of these correlations see Table 2.

Next, to examine the feasibility of the proposed hypotheses, the correlations of prenatal and postnatal maternal mood and stressful experiences with child cortisol, executive control, and adjustment were examined (see Table 3). The prenatal and postnatal maternal variables were unrelated to children's cortisol morning level and diurnal slope. The prenatal maternal variables were negatively correlated to children's executive control, with a small magnitude of correlations. Postnatal maternal depression was modestly negatively correlated with children's executive control. Postnatal stressful experiences were not correlated with children's executive control. The correlations of maternal mood, stressful experiences, and executive control are reported in Table 3.

The correlations of maternal mood and stressful experiences with mother report of child adjustment are reported in Table 3. Prenatal mood and prenatal stressful experiences were positively correlated with mother report of total problems but not social competence. Postnatal maternal mood and postnatal stressful experiences were positively correlated with mother report of total problems and negatively correlated with social competence.

The correlations of maternal mood and stressful experiences with teacher report of child adjustment are also reported in Table 3. Prenatal mood was positively correlated with teacher report of total problems and negatively correlated with social competence. Prenatal stressful experiences were positively correlated with teacher report of total problems but not social competence. Postnatal maternal mood was positively correlated with teacher report of total problems but not social competence. Postnatal stressful experiences were uncorrelated with teacher report of adjustment.

These correlations supported the feasibility of some but not all of the proposed hypotheses. Although there were significant associations of prenatal and postnatal maternal mood and stressful experiences with child executive control and adjustment, prenatal and postnatal maternal mood and stressful experiences were not related to children's diurnal cortisol (morning level or slope).

Growth models of diurnal cortisol and executive control

As a preliminary analysis exploring the possibility that prenatal and postnatal maternal mood and stressful experiences related not to children's cortisol level but to cortisol slope, unconditional models of diurnal morning level and slope were examined. Unconditional growth models diurnal cortisol (morning level and slope) were specified with the intercept reflecting T1 levels, and linear and quadratic growth factors indicated by the T1-T4 measures. Models did not show adequate fit to the data for diurnal morning level (χ^2 (1) = 7.59, RMSEA = 0.15, CFI = 0.85) or slope (χ^2 (1) = 9.81, RMSEA = 0.18, CFI = 0.78).

Both cortisol morning level (M = -0.62, p<.001) and diurnal slope (M = 0.47, p<.001) demonstrated intercepts significantly different than 0 but with nonsignificant variability in initial levels. Cortisol diurnal slope (M = .17, p<.001) demonstrated significant linear growth and quadratic growth. However, the variances of the growth factors were non-significant. This indicates that growth in diurnal slope included a curvilinear pattern, that is, the rate of change decelerated, but that the pattern of deceleration was essentially invariant across children. Morning level did not demonstrate significant linear or quadratic growth.

Given the absence of correlation between diurnal cortisol (morning level and slope) and the prenatal and postnatal maternal variables, in conjunction with the absence of systematic growth in cortisol over time, further tests of hypotheses involving cortisol are not reported in the main text. To thoroughly examine the cortisol data, two additional possibilities were examined. (1) Cortisol was tested in models with covariates to show no associations emerged with these variables in the model. And (2), given that additional degrees of freedom provide additional power to detect variability, models predicting intercept of diurnal cortisol were examined. These results, in which no significant associations of prenatal stress and cortisol variables emerged, and / or which showed poor model fit, are reported in Appendix A.

An unconditional growth model of executive control was specified with the intercept reflecting T1 levels, and linear and quadratic growth factors indicated by T1-T4 measures. Executive control (*M* = .28, *p*<.05) demonstrated an intercept significantly different than 0 with significant variability in initial level. In addition, executive control (*M* = .25, *p*<.05) demonstrated significant linear growth and significant variance in the linear growth factor, indicating individual differences in children's rates of linear growth. Finally, executive control (*M* = -.03, p<.05) demonstrated significant quadratic growth. However, the variance of the quadratic factor was non-significant, suggesting growth in executive control included a rate of change that decelerated, but that the pattern of deceleration was invariant across children. Models estimating the quadratic growth factor were compared to those excluding the quadratic growth factor. The models including the quadratic growth factor fit the data better than the model excluding the quadratic growth factor. As such, SEM analyses included both linear and quadratic growth factors for effortful control. The variances for the quadratic factors were set to 0, and as such, quadratic growth was not examined in relation to prenatal and postnatal mood, prenatal and postnatal stressful experiences, or children's adjustment.

Covariate analyses

Next, a series of regressions were conducted with all potential covariates entered in the model (income, child sex, prenatal alcohol exposure, prenatal medication exposure, prenatal health risk, delivery risk, prenatal cigarette use, and birth weight), as well as prenatal and postnatal exposure to maternal mood symptoms or stressful life experiences. These regressions were conducted to identify significant covariates for retention in the final SEM models and nonsignificant covariates for trimming from the final model.

To examine covariates in the prediction of children's executive control by prenatal depression above postnatal depression, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal maternal mood in the third step, and prenatal mood in the fourth step. Income and child sex predicted children's executive control in the first step, ($\beta = 0.20$, $p < .001$ and $\beta = -0.15$, $p = .03$, respectively). The prenatal risk covariates did not account for significant variance in children's T1 executive control in the second step. In the third step, postnatal mood ($\beta = -0.22$, $p < .01$) predicted significant variance in children's executive control and in the final step prenatal mood ($\beta = -0.26$, $p < .01$) predicted variance in children's executive control, whereas postnatal depression did not ($\beta = -0.08$, $p = 0.36$). See Table 4 for full results of this preliminary regression.

To examine covariates in the prediction of children's executive control by prenatal stressful experiences above postnatal stressful experiences, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal stressful experiences in the third step, and prenatal stressful experiences in the fourth step.

Income and child sex predicted children's executive control in the first step, (β = 0.15, *p* = .03 and β = -0.18, *p* = .02, respectively). The prenatal risk covariates did not account for significant variance in children's T1 executive control in the second step. In the third step, postnatal stressful experiences were not a significant predictor of executive control (β = 0.09, *p* = .23) above income (β = 0.18, *p* = .02), child sex (β = -0.16, *p* = .03. In the final step, prenatal stressful experiences predicted variance in children's executive control (β = -0.17, *p* = 0.04), with postnatal stressful experiences remaining a nonsignificant predictor (β = 0.13, *p* = 0.11). See Table 5 for full results of this preliminary regression.

To examine covariates in the prediction of teacher report of social competence by prenatal depression above postnatal depression, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal maternal mood in the third step, and prenatal mood in the fourth step. Income and child sex predicted teacher report of social competence in the first step, (β = 0.20, *p* = .01 and β = -0.16, *p* = .04, respectively). The prenatal risk covariates did not account for significant variance in teacher report of social competence in the second step. Postnatal mood did not predict significant variance in the third step (β = -0.01 *p* = .06). In the final step prenatal mood (β = -0.21 *p* = .04) along with income (β = 0.18 *p* = .05) and child sex (β = -0.20, *p* = .02) predicted variance in teacher reported social competence, whereas postnatal depression remained a nonsignificant predictor (β = 0.10, *p* = 0.32). See Table 6 for full results of this preliminary regression.

To examine covariates in the prediction of teacher report of social competence by prenatal stressful experiences above postnatal stressful experiences, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal stressful experiences in the third step, and prenatal stressful experiences in the fourth step. Income and child sex predicted teacher report of social competence in the first step, (β = 0.16, *p* = .05 and β = -0.19, *p* = .02, respectively). None of the prenatal risk covariates accounted for significant variance in teacher report of social competence in the second step. Postnatal stressful experiences did not predict significant variance in the third step (β = 0.11 p = .20). In the final step neither prenatal stressful experiences (β = 0.06, p = .56) nor postnatal stressful experiences (β = 0.06, p = .56) predicted teacher reports of child social competence. See Table 7 for full results of this preliminary regression.

To examine covariates in the prediction of teacher report of total problems by prenatal mood above postnatal depression, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal maternal mood in the third step, and prenatal mood in the fourth step. Income and child sex predicted teacher report of total problems in the first step, (β = -0.36, p > .001 and β = 0.17, p = .02, respectively). Of the prenatal risk covariates, prenatal alcohol exposure (β = 0.16, p = .03) accounted for significant variance in teacher report of total problems in the second step. Postnatal mood did not predict significant variance in the third step (β = 0.01 p = .95). In the final step neither prenatal mood (β = 0.08, p = .43) nor postnatal mood (β = -0.04, p = .71) predicted teacher reports of child total problems. See Table 8 for full results of this preliminary regression.

To examine covariates in the prediction of teacher report of total problems by prenatal stressful experiences above postnatal stressful experiences, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal stressful experiences in the third step, and prenatal stressful experiences in the fourth step. Income and child sex predicted teacher report of total problems in the first step, (β = -0.33, p > .001 and β = 0.19, p = .01, respectively). Of the prenatal risk covariates, prenatal alcohol exposure (β = 0.18, p = .02) and prenatal medication exposure accounted (β = 0.17, p = .04) for significant variance in teacher report of total problems in the second step. Postnatal stressful experiences did not predict significant variance in the third step (β = -0.09, p = .26). In the final step neither prenatal stressful experiences (β = -0.02, p = .80) nor postnatal stressful experiences (β = -0.02, *p* = .80) predicted teacher reports of child total problems. See Table 9 for full results of this preliminary regression.

To examine covariates in the prediction of mother report of social competence by prenatal mood above postnatal depression, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal maternal mood in the third step, and prenatal mood in the fourth step. Income predicted mother report of social competence in the first step, (β = 0.14, *p* = .05). Of the prenatal risk covariates, prenatal medication exposure (β = -0.17, *p* = .03) accounted for significant variance mother report of social competence in the second step. Postnatal mood predicted significant variance in the third step (β = -0.26 *p* = .00). In the final step postnatal mood (β =- 0.35, *p* = .43) and prenatal mood (β = 0.18, *p* = .05) predicted mother report of social competence. See Table 10 for full results of this preliminary regression.

To examine covariates in the prediction of mother report of social competence by prenatal stressful experiences above postnatal stressful experiences, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal stressful experiences in the third step, and prenatal stressful experiences in the fourth step. Income and child sex did not predict mother report of social competence in the first step, (β = 0.13, *p* = .09 and β = 0.03, *p* = .71, respectively). None of the prenatal risk covariates accounted for significant variance in mother report of social competence in the second step. Postnatal stressful experiences did not predict significant variance in the third step (β = -0.11, *p* = .18). In the final step neither prenatal stressful experiences (β = -0.01, *p* = .91) nor postnatal stressful experiences (β = -0.11, *p* = .20) predicted teacher reports of child total problems. See Table 11 for full results of this preliminary regression.

To examine covariates in the prediction of mother report of total problems of prenatal mood above postnatal depression, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal maternal mood in the third step, and prenatal mood in the fourth step. Income predicted mother report of total problems in the first step, (β = -0.19, p = .01). Of the prenatal risk covariates, prenatal medication exposure (β = 0.24, p = .01) accounted for significant variance in mother report of total problems in the second step. Postnatal mood predicted significant variance in the third step (β = 0.37 p = .00). In the final step postnatal mood (β = 0.39, p = .00) but not prenatal mood (β = -0.04, p = .60) predicted mother report of child total problems. See Table 12 for full results of this preliminary regression.

To examine covariates in the prediction of mother report of total problems by prenatal stressful experiences above postnatal stressful experiences, a regression was conducted with income and child sex in the first step, the prenatal risk covariates in the second step, postnatal stressful experiences in the third step, and prenatal stressful experiences in the fourth step. Income predicted mother report of total problems in the first step, (β = -0.20, p = .01). Of the prenatal risk covariates, prenatal medication exposure (β = 0.25, p = .01) accounted for significant variance in mother report of total problems in the second step. Postnatal stressful experiences predicted significant variance in the third step (β = 0.33, p > .001). In the final step postnatal stressful experiences (β = 0.32, p > .001) but not prenatal stressful experiences (β = 0.04, p = .61) predicted mother reports of child total problems. See Table 13 for full results of this preliminary regression.

Taken together, income and child sex were significant predictors in all models. These covariates were retained. In addition to examining collinearity diagnostics, correlations were conducted to examine if the prenatal covariates co-occurred with (were a function of) low income. The results of these correlations, presented in Table 14, indicate that the prenatal covariates did not uniformly vary as a function of low income. Prenatal medication exposure was a significant covariate in the prediction of social competence and total problems. Prenatal alcohol exposure was relevant to the prediction of total problems only. These covariates were included in the SEM models. Prenatal health risk, delivery risk, prenatal cigarette use, and birth weight were non-significant in all models and were trimmed from tests of the full models.

Cross-reporter measures

To examine the feasibility of creating a cross-reporter measure of adjustment, we first considered whether the pattern and magnitude of results in the preliminary regressions differed across reporter (teacher or mother). Postnatal mood predicted social competence by maternal but not teacher report. Prenatal mood was a significant predictor of social competence by both respondents, but with opposite signs. Prenatal and postnatal stressful experiences operated similarly, being non-significant predictors of social competence by mother and teacher report. In contrast, postnatal mood predicted total problems by mother but not teacher report. Prenatal mood was a non-significant predictor of mother and teacher report of adjustment. Mirroring the mood findings, postnatal stressful experiences predicted mother report of but not teacher report. Prenatal stressful experiences were non-significant predictors of mother and teacher report of child total problems.

Next, the correlations between mother and teacher report of adjustment were examined. There were significant, modest to moderate correlations across reporter. These correlations are reported in Table 15. Finally, confirmatory factor analyses (CFA) were conducted to assess the feasibility of creating cross-reporter measures of adjustment. Multiple reporters of adjustment were sought to partially address the effects of shared method variance and examine reporter bias.

The CFAs were specified such that the total problems factor loaded on teacher and mother of total problems and the social competence factor loaded on teacher and mother report of social competence. Error variances were set to equal to identify the model. The CFAs, using maximum likelihood estimation, did not demonstrated adequate fit to the data: Total problems:

$\chi^2(1) = 65.14$, $p = 0.00$, Root Mean Square Error of Approximation (RMSEA) = 0.47, comparative fit index (CFI) = 0.77, normalized fit index (NFI) = -0.38. All estimated factor loading were significant. The factor loadings for teacher and mother report of total problems were 0.60 and 0.55, respectively. The factor loadings for teacher and mother report of social competence were 0.35 and 0.45, respectively. Given the poor fit of the models, mother and teacher reports of child adjustment were tested separately.

Prenatal stress, executive control and child adjustment

Figure 1 illustrates the model of the relations of prenatal stress, children's executive control, and children's adjustment tested in the current study. Direct and indirect effects were examined. Postnatal stress was regressed on prenatal stress to examine if postnatal stress mediated the effects of prenatal stress. The intercept and slope of executive control were conditioned on income, child sex, prenatal covariates, postnatal mood, and prenatal mood. A single respondent's (mother or teacher) report of social competence and adjustment problems were regressed on income, child sex, prenatal covariates, prenatal mood, postnatal mood, as well as the intercept and slope of executive control. Indirect effects reflected mediated pathways. Bootstrapping was used to obtain bias-corrected confidence intervals for indirect effects. Models were repeated for prenatal / postnatal stressful experiences in place of maternal mood. This resulted in four total models. All models demonstrated adequate fit to the data. Fit statistics are reported in Table 16.

Prenatal stress and executive control

Maternal mood and executive control. In the full model, income, child sex, prenatal medication exposure, prenatal alcohol exposure, postnatal mood, and prenatal mood were tested as predictors of the intercept and slope of children's executive control. With regard to intercept, income ($\beta = 0.16$, $p = .02$), child sex ($\beta = -0.13$, $p = .05$) explained significant variance. There was a trend toward an effect of prenatal mood on executive control ($\beta = -0.14$, $p = .06$).

There were no significant predictors of slope of executive control. Prenatal alcohol exposure demonstrated a trend toward an effect on the slope of executive control (β = 0.17, *p* = .08). Model results are presented in Table 17.

Stressful experiences and executive control. In the full model, income, child sex, prenatal medication exposure, prenatal alcohol exposure, postnatal stressful experiences, and prenatal stressful experiences were tested as predictors of the intercept and slope of children's executive control. With regard to intercept, income (β = 0.22, *p* = .002) explained significant variance. There were trends toward effects of child sex (β = -0.11, *p* < .10) and prenatal stressful experiences on the intercept of executive control (β = -0.14, *p* < .10). None of the predictors accounted for significant variance in the slope of children's executive control, though there was a trend toward an effect of prenatal alcohol exposure on the slope of executive control (β = 0.14, *p* = .10). Model results are presented in Table 18.

Maternal mood, executive control and child adjustment

Income, child sex, prenatal risk covariates, executive control (intercept and slope), postnatal mood and prenatal mood were tested as predictors of children's social competence and total problems. Standardized betas reported in text. Complete model results for the models are found in Tables 19-22.

Maternal mood and mother report of social competence. Using maternal report of adjustment, maternal postnatal mood (β = -0.32, *p* = .003) and, at a trend level, prenatal mood (β = 0.16, *p* = .050) accounted for significant variance in social competence. The intercept (β = 0.05, *p* = .56) and slope (β = 0.10, *p* = .27) of executive control were not significant predictors of social competence. There was trend toward an indirect effect of prenatal mood on mother-report social competence, through postnatal mood (β = 0.10, *p* = .053, CI_{95} = -0.00 – 0.21).

Maternal mood and mother report of total problems. Using maternal report of adjustment, prenatal medication exposure (β = 0.15, *p* = .04), and maternal postnatal mood (β =

0.42, $p > .001$) accounted for significant variance in total problems. There was a trend toward an effect of child sex (β = 0.11 $p = .06$) on mother-report total problems The intercept (β = 0.11, $p = .14$) and slope (β = -0.09, $p = .37$) of executive control were nonsignificant in the prediction of social competence .

Maternal mood and teacher report of social competence. Using teacher report of adjustment, child sex (β = -0.17, $p = .01$) accounted for significant variance in social competence. The intercept (β = 0.28, $p = .002$) and slope (β = 0.25, $p = .02$) of executive control were significantly related to teacher-report social competence. Maternal postnatal mood (β = 0.13, $p = .13$) and prenatal mood (β = -0.13, $p = .12$) did not account for significant variance in social competence.

Maternal mood and teacher report of total problems. Income (β = -0.25 $p = .003$), child sex (β = 0.14, $p = .03$), and prenatal alcohol exposure (β = 0.17, $p = .04$), accounted for significant variance in total problems. Postnatal maternal mood and prenatal maternal mood were nonsignifcant predictors (β = -0.01, $p = .62$ and β = 0.05, $p = .91$, respectively). The intercept (β = -0.17, $p = .04$) and slope (β = -0.25, $p = .02$) of executive control predicted teacher-report total problems.

Maternal stressful experiences, executive control and child adjustment

Income, child sex, prenatal risk covariates, executive control (intercept and slope), postnatal stressful experiences and prenatal stressful experiences were tested as predictors of children's social competence and total problems. Standardized betas reported in text.

Stressful experiences and mother report of social competence. Using mother report of adjustment, maternal postnatal stressful experiences (β = -0.13, $p = .04$) accounted for significant variance in mother-report social competence. This was the only predictor that approached significance.

Stressful experiences and mother report of total problems. Postnatal stressful experiences (β = 0.35, p > .001), accounted for significant variance in mother-report total problems. There were trends toward effects of child sex (β = 0.11, p = .06), prenatal medication exposure (β = 0.13, p = .09), and prenatal alcohol exposure (β = 0.10, p = .06) on mother-report total problems. Prenatal stressful experiences were a nonsignifcant predictor (β = 0.01, p = .92). The intercept and slope of executive control were nonsignificant predictors of mother-report total problems (β = 0.02, p = .76 and β = -0.12, p = .25, respectively).

Stressful experiences and teacher report of social competence. Using teacher report of adjustment, child sex (β = -0.16, p = .02) the intercept (β = 0.28, p =.001) and the slope of executive control (β = 0.26, p = .03) predicted teacher-report social competence.

Stressful experiences and teacher report of total problems. Income (β = -0.25, p = .003), child sex (β = 0.14, p = .04) and prenatal alcohol exposure (β = 0.18, p = .03) explained variance in teacher-report total problems. The intercept (β = -0.17, p = .04) and slope (β = -0.26, p = .02) of executive control accounted for significant variance in teacher-report total problems.

Testing differences in effects of prenatal mood and stressful experiences

To assess for potential differences in the relation of prenatal stress constructs (maternal mood and maternal stressful experiences) to children's executive control and preschool adjustment, models with both predictors (prenatal psychological symptoms and prenatal stressful experiences) with paths set to be equal were compared to a models with both predictors in which the paths are free to differ. Chi-square difference tests, reported in Table 23, indicated that there were no significant differences in the structural paths or covariances across the models, in other words, there was no evidence that there were differences in the magnitudes of the two predictors. This finding applied to models using both maternal report and teacher report of adjustment.

Testing gender differences

To assess for potential sex differences in the relations reported above, a cross-group path model was conducted for each of the four SEMs above. The cross-group model compared the model in which all parameters were free to differ across child sex with a model in which all paths and covariances were constrained to be equal across child sex. Chi-square difference tests, reported in Table 24, indicated that there were no significant differences in the structural paths or covariances across child sex. In other words, there was no support for sex differences in the patterns of relations reported above.

Discussion

This study sought to add to our understanding of the relation of prenatal stress to children's diurnal cortisol, executive control, and the role it plays in preschoolers' adjustment. Findings demonstrated that prenatal stress was unrelated to children's diurnal cortisol in the preschool period. There were trends toward effects of prenatal mood and prenatal stressful experiences on initial levels of children's executive control but not its growth. There was also a trend toward an effect of prenatal mood on preschoolers' social competence, but only for mother report of social competence. Neither prenatal mood nor prenatal stressful experiences predicted preschooler's total problems for either maternal or teacher report. Disruptions to children's cortisol and executive control have been posited to account for the effects of prenatal stress on adjustment, but the results of this study do not support that premise.

Contrary to hypotheses and previous studies linking prenatal stressful experiences (Entringer, Kumsta, Hellhammer, Wadhwa, & Wüst, 2009) and prenatal psychopathology (de Bruijn et al., 2009b; Grant et al., 2009; Laurent et al., 2013; O'Connor et al., 2005; Van den Bergh et al., 2008) with postnatal cortisol activity in children, the current study found preschooler's morning cortisol level and diurnal slope to be unrelated to prenatal and postnatal stressful experiences and maternal mood. While two other published studies found no differences in diurnal pattern among the prenatally stressed (Entrigner et al., 2009b; O'Conner, et al., 2005), several explanations, beyond a lack of relation, may account for the failure of this study to detect a relation between prenatal stress and children cortisol. Prenatal experiences may exert influence on the HPA axis, but the direction of these effects may vary, perhaps as a function of stressor type or context. That is, there is evidence to suggest that protracted stressors such as low income are associated with lower or blunted cortisol levels, whereas acute stress is associated with elevated diurnal levels (Dowd, Simanek, & Aiello, 2009; Miller, Chen, & Zhou, 2007). Perhaps both forms of stressors operated on this at-risk sample and obscure tests founded on linear regression. Studies of "purer" exposures to acute stress versus prolonged stress may be beneficial to further the field. Further, there is some evidence to suggest that stress responding, rather than daily patterning of stress systems, may be altered consequent to prenatal stress. For example, one study found steeper diurnal slopes among prenatally stressed children compared to non-stress children on school days but not weekend days (Gutteling et al., 2005). A second study, involving adult women, found an association between prenatal stress and reaction times on a working memory task (Entringer, Buss, Kumsta, Hellhammer, & Wadhwa et al., 2009) in the hydrocortisone administration condition but not in the placebo condition when compared to a non-prenatally stressed comparison group. As such, perhaps prenatal stress does alter stress reactivity, but not daily patterning of cortisol, and is therefore not detected by the current study. It is also possible that prenatal stress interacts with postnatal experiences in determining regulation of the HPA-axis. If prenatal stress exposure sensitizes the child's neuroendocrine stress response system, postnatal stress exposure might then have a greater impact on regulation of the HPA-axis system when there was also prenatal exposure. A complex model including this moderated relation of prenatal and postnatal stress, as recently proposed by Heidemaire Laurent (2014), might be required to understand the role of prenatal stress on HPA-axis functioning.

As a further exploration of the data, this writer examined the relation of prenatal stress, postnatal stress and parenting behaviors (see Appendix B). Prenatal mood related to less warmth (trend level), greater negativity, and less maternal scaffolding. Prenatal stressful experiences related to all dimensions of parenting assessed as part of the larger study: less warmth, more negativity, less scaffolding, less responsiveness, and less limit setting. Parenting behaviors have been shown in the context of the current sample (Lengua, Kiff, Moran, Zalewski, Thompson, Cortes… et al., 2014) and in the field in general to relate to children's cortisol. As such, perhaps prenatal stress does exert influence on children's cortisol, but through parenting, which was not formally modeled in the current study. Future research should incorporate measures of parenting to further understand these findings.

A secondary aim of this study was to examine cortisol activity longitudinally using growth modeling. Children's diurnal slopes included a curvilinear pattern with nonsignificant variability, suggesting that children's diurnal slopes decreased over time, but that this pattern of change was invariant across children. In the current literature, there is little information beyond day-to-day variations of the developmental course and stability of cortisol in children in early childhood (Rotenberg, McGrath, Roy-Gagnon, & Tu, 2012). Few studies have obtained repeated assessments of cortisol, and those that have included a variety of measures (i.e., Bush, Obradović, Adler, & Boyce, 2011) and assessment intervals (i.e., Rotenberg et al., 2012), which makes it difficult to compare across studies. While the current study suggests a uniform change in diurnal slope but not morning level of cortisol, perhaps reflective of a normative developmental change in diurnal patterning, prior studies point to a relatively stable patterning of diurnal cortisol, with significant individual variability (attributed to factors such as postnatal parental stress, parenting, and psychopathology (Van Ryzin, et al., 2009; Laurent, et al., 2013). Additional investigation and replication of the developmental course and stability of cortisol in children is indicated. Notably, this study utilized latent growth curve modeling, which assumes that the phenomenon of interest develops or follows meaningful trajectory across the sample. It is possible that a number of atypical or distinct dysregulated trajectories are contained within this sample and as such, the use of a single growth curve may obscure the true nature of change in the population or the aggregation across subgroups may be misleading (Van Ryzin, et al., 2009). Particularly given the nuanced hypotheses about stressor types uniquely influencing the direction of effect on cortisol, further study may benefit from the use of additional statistical approaches such as group based trajectory modeling, which would also permit examination of predictors of variability over time in a heterogeneous population.

There were trends toward effects of prenatal factors on both the intercept and slope of children's executive control. Higher initial levels of executive control were predicted by higher income, female sex, and at the trend level, lower prenatal maternal mood symptoms and prenatal stressful experiences. These findings are consistent with the results of other studies linking lower levels of executive control with low income (Raver, Blackburn, Bancroft, & Trop, 1999) and exposure to maternal prenatal psychopathology (Buss et al., 2010; Mennes, et al., 2009; Van den Berg et al, 2005; Van den Berg et al., 2006). Notably, these prior studies utilized measures of prenatal anxiety, whereas this study utilized a measure of maternal mood. Further, these factors predict intercept but not slope of executive control, suggesting that income and prenatal mood exert their influence on the development of executive control earlier in childhood or on other factors that in turn predict the development of executive control. Given the research suggesting that disturbances to executive control may emerge only in the context of stress and stress-responding (Entringer et al., 2009), it is possible that prenatal mood and stressful experience do exert robust influences on children's executive control, but these differences would be better captured outside the setting of the current study, which was a relatively non-stressed, laboratory context.

There was a trend toward an effect of prenatal alcohol exposure to greater increases in executive control. This finding is in contrast to the findings of studies finding deficits in attentional measures (on continues performance tasks, freedom from distractibility tasks, etc.) in children of “social drinkers” in pregnancy (Landesman-Dwyer, Rogazin, & Little, 1981, Streissguth, Martin, Barr, Sandman, Kirchner, & Darby, 1984, Brown, Coles, Smith, Platzman, Silverstein, Erickson, & Falck, 1991). One study finding a negative relation between prenatal alcohol exposure and continuous performance demonstrated differences in children at age 4 and enduring to age 11 and 14. (Streissguth et al., 1984; Streissguth, Barr, Sampson, Parrish-Johnson, Kirchner, & Martin, 1986; Streissguth et al., 1994). However, the finding of this study, that prenatal alcohol exposure is related to more favorable executive control, is not without precedent. Fried and colleagues (1992) and Boyd and colleagues (1991) found no relation between alcohol exposure and attentional measures of children in early childhood. Fried and colleagues (1991) instead found exposure to be related to lower levels of impulsive responding while Boyd and colleagues (1991) found no difference is sustained attention among prenatally exposed versus unexposed children. It is important to note that in these two studies, as in the current study, alcohol exposure levels were relatively low.

Contrary to study hypotheses, prenatal mood and prenatal stressful experiences were not significantly related to preschoolers’ adjustment when the other factors in the models were accounted. The prenatal variables were related to adjustment at the zero-order level, but largely nonsignificant in models that accounted for covariates and postnatal factors. Mother and teacher reports of child adjustment were examined separately, given that the results of the CFA did not support combining responses across reporters. Results varied to some degree contingent upon reporter. Prenatal mood predicted preschoolers’ social competence at the trend level, but only by mother report. Neither prenatal mood nor prenatal stressful experiences predicted preschooler’s total problems by maternal or teacher report. Instead, postnatal mood and stressful experiences predicted adjustment, but only by maternal report. Higher postnatal mood symptoms as well as greater postnatal stressful experiences predicted lower mother-reported social competence and higher mother-reported total problems. These findings align with the substantial literature linking maternal postnatal depression (Cummings & Davis, 1994; Downey & Coyne, 1990) and postnatal negative life events (Attar, Guerra, & Tolan, 1994; Kim, Conger, Elder, & Lorenz, 2003) with child behavior problems. As prenatal and postnatal mood were significant predictors for mother report only, these findings should be interpreted with caution. The findings may reflect a response bias among mothers (with and without psychopathology) reporting on their children's adjustment (De Los Reyes & Kazdin, 2005; Najman et al., 2001; Youngstrom, Izard, & Ackerman, 1999) or may simply be a function of the differences in available norms of reference available to teachers versus mothers.

The failure for prenatal stress to predict early childhood adjustment is precedented in studies of both prenatal mood (Bekkus et al., 2011; Van Battenburg-Eddes et al., 2013; & Velders et al., 2011) and prenatal stressful experiences (Ramachandani et al., 2010; Ronald et al., 2005). These findings may highlight the importance of postnatal experiences, such as socialization and attachment, on children. Velders and colleagues (2011) examined maternal prenatal and postnatal psychological symptoms in conjunction with family variables (family functioning, parental hostility). These authors found that postnatal parental hostility accounted for the observed relation between prenatal depressive symptoms and children's internalizing and externalizing symptoms. This finding highlights the possibility that unmeasured, postnatal processes or experiences might account for the enduring effects of prenatal stress on child adjustment observed in some studies and suggests the need for tests of more complex, bioecological models accounting for family characteristics and processes. These more proximal variables, perhaps shaped by prenatal experience, may better account for child adjustment outcomes and explain the mixed findings of the current prenatal stress literature.

Study variables other than the prenatal stress variables were more consistent predictors of children's adjustment. Children's initial levels of executive control and the growth of their executive control predicted teacher-reported social competence and total problems in both the mood model and in the stressful experiences model. Higher initial levels and faster development of executive control predicted more favorable adjustment. These findings are in line with theory suggesting executive control is at the core of self-regulation (Rothbart & Bates, 2006) and studies affirming that executive control is a predictor of social-emotional competence (e.g. Eisenberg et al., 2003) and total problems (e.g. Hopkins, Lavigne, Gouze, LeBaily, & Bryant, 2013; Lengua, 2003). Findings affirm that children's rate of developing these self-regulatory abilities are important predictors of adjustment (King, Fleming, et al., 2011; King, Lengua et al., 2011; Monahan et al., 2009). The trend finding that prenatal alcohol exposure predicts the development of executive control suggests that the development of this self-regulatory ability is mutable by environmental factors. Therefore, further reasearch on modifiable rsk factors (e.g. parenting) with the potential to promote the rate of development of exectuive control, may have important implicaitons for children's adjustment. Additionally, children's sex predicted social competence and total problems in both the mood models and stressful experiences models. Consistent with previous studies (Rose & Rudolph, 2006), boys demonstrated lower social competence and greater total problems than girls. Prenatal alcohol exposure predicted higher teacher-report total problems. This study finding replicates the relation of alcohol exposure to greater adjustment problems (Sood et al., 2001). Prenatal medication exposure predicted mother-reported total problems, with exposure relating to greater adjustment problems. While the most recent meta-analyses on the use of medications such as selective serotonin reuptake inhibitors suggest preliminary evidence for long-term effects of medication on neurobehavior (Lattimore, Donn, Kaciroti, Kemper, Neal & Vazques, 2005; Tuccori et al., 2009), additional research on this topic is needed. Notably, in the current study, there was no comparison condition of women with medical conditions who did not utilize medications during pregnancy. As such, the effects observed in the current study may be better attributable to medical conditions rather than the medication used to treat the condition. Finally, income predicted teacher-reported problems, conforming to the large body of research evidence relating low income to poor psychological adjustment in preschool-age children (e.g. Mistry, Biesanz, Taylor, Burchinal, & Cox, 2004). Of note, the accumulation of multiple risks associated with low income (e.g. violence, chaotic, crowded, and noisy living environment, less responsive parents, etc.), rather than a singular factor is thought to explain these relations (Evans, 2004). Prenatal stress may contribute to this risk, or to family processes shown to mediate the effects of income on preschoolers' social behavior (Mistry et al., 2004).

A central aim of the current study was to test the hypothesis that executive control would account for the effects of prenatal mood and stressful experiences on children's adjustment. There was no evidence to support this hypothesis. Prenatal mood and prenatal stressful experiences predicted children's executive control at the trend level, and children's executive control, in turn, predicted adjustment. However, there was no evidence for an indirect effect of prenatal mood through executive control in predicting adjustment. Similar considerations raised with regard to the lack of cortisol results may apply here. Again, there is some preliminary evidence suggesting that measures of self-regulatory ability in a stressed context (rather than the current study's laboratory context) shows a greater relation with prenatal stress (Entringer et al., 2009; Gutteling et al., 2005), and perhaps impairments in stress responding mediate the relations of prenatal stress to adjustment. As an alternate explanation, prenatal stress was related to postnatal parenting behaviors, which have, in turn, been shown in the current sample to predict children's adjustment (Klein et al., *in preparation*). Postnatal parenting behaviors have not been tested as mediators of prenatal stress, and merit further study.

There were no significant indirect effects in the models tested in the current study. There was a trend toward an effect of prenatal mood on mother-report social competence through postnatal mood symptoms. This path raises the possibility that a chronic course of maternal mood symptoms is particularly detrimental do children's adjustment. This possibility is supported by studies finding the chronicity of depression (albeit across postnatal years) to predict children's adjustment (e.g. Brennan, Hammen, Anderson, Bor, Najman, & Williams, 2000; Hammen & Brennan, 2003), and one finding of elevations in cortisol among 4.5-year-olds whose mothers were concurrently depressed only if maternal depression had also been present in the first 12 months postpartum (Essex et al., 2002).

There was no evidence that prenatal stress constructs (prenatal maternal mood, prenatal stressful experiences) differed in their prediction of children's executive control and adjustment. Notably, we hypothesized that differential relations of prenatal mood and prenatal stressful experiences would emerge in relation to children's cortisol activity. We hypothesized that stressful experiences might reflect more acute exposure to stress whereas maternal mood, implicated in more prolonged experiences of stressors and their generation, might capture chronic exposure to stress. Cortisol has been shown to differentially relate to acute versus chronic stress (Dowd, Simanek, & Aiello, 2009; Miller, Chen, & Zhou, 2007). Future studies, perhaps examining cortisol reactivity, where the effects of postnatal stress may be more prominent (Entringer, et al., 2009; Gutteling et al., 2005), can continue to clarify the potential specificity in the sequela of prenatal mood and prenatal stressful experiences.

Cross-group path models indicated that there was no evidence for sex differences in the current study. That is, the fit of models was not significantly improved when all parameters were free to differ across child sex versus when all paths and covariances were constrained to be equal across child sex, suggesting that the pattern of study findings were identical for boys and girls. These findings of equivalence contradict some studies of prenatal stress supporting sex differences in attention problems (Loomans et al., 2011; O'Conner et al., 2002), behavior problems (De Bruijn et al., 2009; Loomans et al., 2011), and internalizing symptoms (De Bruijn et al., 2009) related to maternal prenatal mood symptoms and studies supporting sex differences in attention problems related to maternal prenatal stressful experiences (Rodriguez & Bohlin, 2005). However, follow-up studies have found these differences did not endure into later postnatal years (O'Connor et al., 2003) and some studies failed to find sex differences in the influence of prenatal stress this developmental period (Ronald et al., 2005). De Bruijn and colleagues (2009) found early pregnancy prenatal psychological symptoms to be associated with internalizing and total problems in boys, whereas third trimester prenatal psychological symptoms were associated with internalizing, externalizing, and total problems among girls, raising the possibility that sex effects are obscured by the lack of consideration for trimester of prenatal exposure in the present study.

Several limitations of the study are noted. First, the measure of prenatal stress is brief, retrospective, and global. As such, questions about the timing of stress within pregnancy could not be examined, and responses are likely less accurate than those afforded by measures administered more proximally to pregnancy. Although the sample represents the ethnic and racial make-up of the population from which it was drawn, the small number of participants in each ethnic and racial group prevents the examination of possible differences across groups. This is a considerable limitation of the study, given the potential for ethnic and cultural variables to influence the occurrence of stress (e.g. exposure to unique stressors related to acculturation, discrimination, etc.) and to influence access to social and personal resources (e.g. institutional barriers to services, discrimination, lower-quality prenatal care) to cope with stress or pregnancy in general. Few studies have explicitly modeled ethnic and racial factors in studies of prenatal stress. The research of Dunkel-Schetter and colleagues on prenatal stress, ethnicity, and preterm birth / low birth weight offers preliminary evidence for the importance of this factor in comprehensive models of prenatal stressors and subsequent child adjustment (Glynn, Dunkel-Schetter, Hobel, & Sandman, 2008; Lobel, Dunkel-Schetter, & Scrimshaw, 1992; Rini, Dunkel-Schetter, Wadhwa, & Sandman, 1999). The use of a community rather than clinical sample may limit the rates at which problem behaviors are exhibited, but will allow for the examination of both normative levels of behaviors and the emergence of problems. Additionally, the sample was recruited to be diverse across family income, resulting in a high-risk sample in which higher levels of psychopathology can be expected. The current study does not account for the potential role of shared genetic influences. Thus, mothers and children share genetic information that may contribute to their HPA-axis functioning (cortisol), biologically based systems of self-regulation (executive control), as well as their development of adjustment problems. Finally, measures of children's cortisol and executive control were done in normative, non-stressed laboratory and environmental settings. This affords the ability to examine typical diurnal cortisol patterning and ideal executive control performance. However, this study design may obscure differences in cortisol reactivity to stress and executive control performance in stressed contexts, which may be particularly relevant to the study of prenatal stress (Entringer et al., 2009; Gutteling et al., 2005).

The study is strengthened by the employment of multiple assessment methods, including physiological, neurocognitive, and questionnaire data with multiple respondents, which helps minimize effects of reporter bias and shared method variance. The longitudinal study design is a further strength, as it allows for tests of prenatal stress effects above postnatal effects, the examination of lability versus stability of the systems, and analysis of rates of development in addition to static levels. The measurement of prenatal stress and both prenatal stressful experiences and prenatal psychological symptoms allows for a more comprehensive characterization of the prenatal stress experienced in utero, and importantly, facilitates a comparison of effects across the domains of stress.

This study aimed to clarify the relation of prenatal mood and prenatal stressful experiences to children's diurnal cortisol, executive control, and adjustment, testing and failing to find support for cortisol and executive control in the preschool period mediating the effects of prenatal stress. Findings highlighted the importance of prenatal exposures to medication and alcohol, as well as the independent effect of executive control on children's adjustment. The current study highlights the need for further explicit study of mediators, perhaps including postnatal family variables. Solidifying prenatal stress' mechanism of effect would serve to identify potential targets for intervention and inform clinical practice. The current study suggests that executive control, which has been shown to be promotable in the preschool period (Bierman, Nix, Greenberg, Blair, & Domitrovich, 2008; Diamond & Lee, 2011) may be an important target for interventions aimed at the promotion of social competence and the reduction of behavior problems in early childhood.

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Table 1. Descriptive statistics of study variables.

	N	Mean	SD	Min.	Max.
Income	306	8.75	3.93	0.50	14.00
Child Sex	306	0.49	0.51	0.00	1.00
Child Age (months)	302	30.25	5.79	17.00	46.00
T1 Morning Level Cortisol	270	-0.63	0.27	-1.72	0.21
T2 Morning Level Cortisol	257	-0.58	0.24	-1.52	0.21
T3 Morning Level Cortisol	261	-0.66	0.26	-1.43	0.24
T4 Morning Level Cortisol	251	-0.69	0.31	-2.22	0.03
T1 Diurnal Slope Cortisol	270	0.46	0.36	-0.58	1.24
T2 Diurnal Slope Cortisol	257	0.66	0.37	-0.55	1.36
T3 Diurnal Slope Cortisol	258	0.68	0.41	-0.67	2.20
T4 Diurnal Slope Cortisol	246	0.80	0.51	-0.62	1.94
T1 Executive Control	306	0.29	0.15	0.00	0.77
T2 Executive Control	290	0.49	0.20	0.00	0.91
T3 Executive Control	288	0.68	0.17	0.18	0.95
T4 Executive Control	287	0.78	0.15	0.18	1.00
Prenatal Alcohol Exposure	238	0.16	0.38	0.00	2.00
Prenatal Medication Exposure	280	1.27	1.05	0.00	4.00
Prenatal Health Risk	303	0.34	0.57	0.00	2.00
Delivery Risk	298	0.39	0.57	0.00	2.00
Prenatal Cigarette Exposure	302	0.26	1.01	0.00	5.00
Birth Weight	303	3.33	0.73	1.00	5.00
Prenatal Mood	286	14.64	4.80	9.00	32.00
Prenatal Stressful Experiences	275	2.33	2.25	0.00	12.00
Postnatal Mood	294	10.72	7.33	0.00	41.33
Postnatal Stressful Experiences	294	5.47	2.09	0.25	12.50
Social Competence (maternal report)	292	51.09	7.99	16.0	73.00
Total Problems (maternal report)	293	10.37	7.63	0.00	51.00
Social Competence (teacher report)	234	43.07	10.15	5.00	60.00
Total Problems (teacher report)	237	9.05	7.06	0.00	30.00

Table 2. Correlations between prenatal and postnatal mood and stressful experiences.

	Prenatal Stressful Experiences	Postnatal Mood	Postnatal Stressful Experiences
Prenatal Mood	0.52***	0.57***	0.37***
Prenatal Stressful Experiences	-	0.46***	0.40***
Postnatal Mood		-	0.49***

Note. *p < .05, **p < .01, *** p < .001.

Table 3. Correlations of prenatal and postnatal maternal mood and stressful experiences with study variables.

	Prenatal Mood	Prenatal Stressful Experiences	Postnatal Mood	Postnatal Stressful Experiences
Diurnal Cortisol Morning Level				
T1 Morning Level	0.03	-0.07	-0.08	-0.08
T2 Morning Level	0.11^{t}	0.04	0.05	0.03
T3 Morning Level	-0.01	-0.05	-0.07	0.01
T4 Morning Level	-0.06	-0.09	-0.12^{t}	-0.09
Diurnal Cortisol Slope				
T1 Diurnal Slope	0.00	-0.06	-0.07	-0.04
T2 Diurnal Slope	0.09	0.13*	0.05	0.04
T3 Diurnal Slope	-0.03	-0.08	-0.10	0.00
T4 Diurnal Slope	-0.03	0.01	-0.07	-0.02
Executive Control				
T1 Executive Control	-0.21***	-0.16**	-0.23***	-0.05
T2 Executive Control	-0.19**	-0.17**	-0.17**	-0.05
T3 Executive Control	-0.21***	-0.15*	-0.26***	-0.11^{t}
T4 Executive Control	-0.21***	-0.09	-0.22***	-0.05
Mother Report of Adjustment				
T4 Social Competence	-0.03	-0.04	-0.23***	-0.14*
T4 Total Problems	0.23***	0.19**	0.42***	0.40***
Teacher Report of Adjustment				
T4 Social Competence	-0.16*	-0.03	-0.08	-0.02
T4 Total Problems	0.16*	0.14*	0.15*	0.08

Note. ^{t} <.10, *p < .05, **p < .01, *** p < .001.

Table 4. Preliminary regression examining potential covariates of executive control in mood model.

T1 Executive Control		β	SE	Standard. β	*p*
Step 1					
	Income	**0.01**	**0.00**	**0.20**	**0.00**
	Child Sex	**-0.04**	**0.02**	**-0.15**	**0.03**
Step 2					
	Income	**0.01**	**0.00**	**0.20**	**0.01**
	Child Sex	**-0.04**	**0.02**	**-0.15**	**0.04**
	Prenatal Alcohol Exposure	-0.02	0.03	-0.04	0.57
	Prenatal Medication Exposure	-0.01	0.01	-0.04	0.63
	Prenatal Health Risk	-0.00	0.02	-0.02	0.82
	Delivery Risk	-0.03	0.02	-0.10	0.17
	Cigarette Use	-0.00	0.01	-0.02	0.82
	Birth Weight	0.01	0.02	0.05	0.55
Step 3					
	Income	0.00	0.00	0.13	0.11
	Child Sex	**-0.04**	**0.02**	**-0.16**	**0.03**
	Prenatal Alcohol Exposure	-0.01	0.03	-0.03	0.63
	Prenatal Medication Exposure	0.00	0.01	0.00	0.97
	Prenatal Health Risk	0.00	0.02	0.01	0.92
	Delivery Risk	-0.03	0.02	-0.10	0.17
	Cigarette Use	0.00	0.01	0.02	0.83
	Birth Weight	0.01	0.02	0.05	0.54
	Postnatal Depression	**-0.00**	**0.00**	**-0.22**	**0.00**
Step 4					
	Income	0.00	0.00	0.12	0.12
	Child Sex	**-0.05**	**0.02**	**-0.17**	**0.01**
	Prenatal Alcohol Exposure	0.01	0.03	0.03	0.71
	Prenatal Medication Exposure	0.00	0.01	0.01	0.85
	Prenatal Health Risk	0.01	0.02	0.04	0.57
	Delivery Risk	-0.03	0.02	-0.10	0.17
	Cigarette Use	0.01	0.01	0.04	0.60
	Birth Weight	0.01	0.02	0.05	0.55
	Postnatal Depression	-0.00	0.00	-0.08	0.36
	Prenatal Mood	**-0.01**	**0.00**	**-0.26**	**0.00**

Note. Bolded text reflects significant predictors, $p < .05$.

Table 5. Preliminary regression examining potential covariates of executive control in stressful experiences model.

T1 Executive Control		β	SE	Standard. β	*p*
Step 1					
	Income	**0.01**	**0.00**	**0.16**	**0.03**
	Child Sex	**-0.05**	**0.02**	**-0.18**	**0.02**
Step 2					
	Income	**0.01**	**0.00**	**0.16**	**0.03**
	Child Sex	**-0.05**	**0.02**	**-0.17**	**0.02**
	Prenatal Alcohol Exposure	-0.03	0.03	-0.07	0.32
	Prenatal Medication Exposure	-0.01	0.01	-0.04	0.56
	Prenatal Health Risk	0.00	0.02	0.00	0.99
	Delivery Risk	-0.03	0.02	-0.12	0.11
	Cigarette Use	-0.01	0.01	-0.03	0.69
	Birth Weight	0.00	0.02	0.01	0.86
Step 3					
	Income	**0.01**	**0.00**	**0.18**	**0.02**
	Child Sex	**-0.05**	**0.02**	**-0.16**	**0.03**
	Prenatal Alcohol Exposure	-0.03	0.03	-0.08	0.28
	Prenatal Medication Exposure	-0.01	0.01	-0.07	0.39
	Prenatal Health Risk	0.01	0.02	0.01	0.94
	Delivery Risk	-0.03	0.02	-0.13	0.10
	Cigarette Use	-0.01	0.01	-0.05	0.54
	Birth Weight	0.00	0.02	0.01	0.92
	Postnatal Stressful Experiences	0.01	0.01	0.09	0.23
Step 4					
	Income	**0.01**	**0.00**	**0.14**	**0.09**
	Child Sex	**-0.04**	**0.02**	**-0.15**	**0.05**
	Prenatal Alcohol Exposure	-0.02	0.03	-0.06	0.46
	Prenatal Medication Exposure	-0.01	0.01	-0.05	0.55
	Prenatal Health Risk	0.01	0.02	0.03	0.74
	Delivery Risk	-0.03	0.02	-0.12	0.12
	Cigarette Use	-0.00	0.01	-0.02	0.85
	Birth Weight	-0.00	0.02	-0.01	0.92
	Postnatal Stressful Experiences	0.01	0.01	0.13	0.11
	Prenatal Stressful Experiences	**-0.01**	**0.01**	**-0.17**	**0.04**

Note. Bolded text reflects significant predictors, $p < .05$.

Table 6. Preliminary regression examining potential covariates of teacher report of social competence in mood model.

Teacher Report-Social Comp.		β	SE	Standard. β	*p*
Step 1					
	Income	**0.52**	**0.21**	**0.16**	**0.03**
	Child Sex	**-3.38**	**1.61**	**-0.16**	**0.04**
Step 2					
	Income	**0.42**	**0.21**	**0.18**	**0.03**
	Child Sex	**-3.73**	**1.68**	**-0.18**	**0.03**
	Prenatal Alcohol Exposure	0.50	2.12	0.02	0.81
	Prenatal Medication Exposure	-1.18	0.83	-0.12	0.16
	Prenatal Health Risk	-0.30	1.62	-0.02	0.85
	Delivery Risk	0.43	1.75	0.02	0.81
	Cigarette Use	-0.89	0.88	-0.09	0.31
	Birth Weight	-0.07	1.24	-0.01	0.95
Step 3					
	Income	0.47	0.24	0.18	0.05
	Child Sex	**-3.73**	**1.69**	**-0.18**	**0.03**
	Prenatal Alcohol Exposure	0.51	2.13	0.02	0.81
	Prenatal Medication Exposure	-1.17	0.64	-0.12	0.16
	Prenatal Health Risk	-0.28	1.65	-0.02	0.87
	Delivery Risk	0.43	1.76	0.02	0.81
	Cigarette Use	-0.07	1.24	-0.01	0.54
	Birth Weight	0.00	0.02	0.01	0.97
	Postnatal Mood	-0.01	0.12	-0.01	0.94
Step 4					
	Income	**0.46**	**0.23**	**0.18**	**0.05**
	Child Sex	**-4.11**	**1.68**	**-0.20**	**0.02**
	Prenatal Alcohol Exposure	1.02	0.83	-0.11	0.22
	Prenatal Medication Exposure	-1.02	0.83	-0.11	0.22
	Prenatal Health Risk	0.34	1.66	0.02	0.84
	Delivery Risk	0.24	1.74	0.01	0.89
	Cigarette Use	-0.54	0.90	-0.05	0.52
	Birth Weight	0.02	1.23	0.00	0.99
	Postnatal Mood	0.14	0.14	0.10	0.31

Note. Bolded text reflects significant predictors, $p < .05$.

Table 7. Preliminary regression examining potential covariates of teacher report of social competence in stressful experiences model.

Teacher Report-Social Comp.		β	SE	Standard. β	*p*
Step 1					
	Income	**0.41**	**0.21**	**0.16**	**0.05**
	Child Sex	**-3.87**	**1.62**	**-0.19**	**0.02**
Step 2					
	Income	0.34	0.22	0.13	0.13
	Child Sex	**-4.21**	**1.70**	**-0.21**	**0.01**
	Prenatal Alcohol Exposure	0.30	2.09	0.01	0.89
	Prenatal Medication Exposure	-1.29	0.82	-0.14	0.12
	Prenatal Health Risk	0.37	1.62	0.02	0.82
	Delivery Risk	0.08	1.73	0.00	0.97
	Cigarette Use	-1.12	0.87	-0.11	0.20
	Birth Weight	-0.06	1.25	-0.00	0.97
Step 3					
	Income	0.40	0.23	0.15	0.08
	Child Sex	**-3.97**	**1.70**	**-0.20**	**0.02**
	Prenatal Alcohol Exposure	0.14	2.09	0.01	0.95
	Prenatal Medication Exposure	-1.57	0.85	-0.17	0.07
	Prenatal Health Risk	0.53	1.62	0.03	0.74
	Delivery Risk	-0.17	1.74	-0.01	0.92
	Cigarette Use	-1.35	0.89	-0.13	0.13
	Birth Weight	-0.17	1.25	-0.01	0.89
	Postnatal Stressful Experiences	0.54	0.42	0.11	0.20
Step 4					
	Income	0.43	0.24	0.17	0.07
	Child Sex	**-4.07**	**1.72**	**-0.20**	**0.02**
	Prenatal Alcohol Exposure	-0.04	2.12	-0.00	0.99
	Prenatal Medication Exposure	-1.64	0.86	-0.18	0.06
	Prenatal Health Risk	0.37	1.65	0.02	0.82
	Delivery Risk	-0.17	1.74	-0.01	0.92
	Cigarette Use	-1.48	0.92	-0.15	0.11
	Birth Weight	-0.11	1.26	-0.01	0.93
	Postnatal Stressful Experiences	0.50	0.43	0.10	0.25
	Prenatal Stressful Experiences	0.25	0.43	0.06	0.56

Note. Bolded text reflects significant predictors, $p < .05$.

Table 8. Preliminary regression examining potential covariates of teacher report of total problems in mood model.

Teacher Report- Total Problems		β	SE	Standard. β	*p*
Step 1					
	Income	**-0.67**	**0.13**	**-0.36**	**0.00**
	Child Sex	**2.44**	**1.06**	**0.17**	**0.02**
Step 2					
	Income	**-0.68**	**0.14**	**-0.37**	**0.00**
	Child Sex	**2.70**	**1.09**	**0.19**	**0.01**
	Prenatal Alcohol Exposure	**3.02**	**1.38**	**0.16**	**0.03**
	Prenatal Medication Exposure	0.96	0.53	0.14	0.07
	Prenatal Health Risk	-0.56	1.05	-0.04	0.60
	Delivery Risk	-0.27	1.31	-0.02	0.81
	Cigarette Use	0.61	0.57	0.08	0.29
	Birth Weight	-0.63	0.80	-0.06	0.43
Step 3					
	Income	**-0.67**	**0.15**	**-0.37**	**0.00**
	Child Sex	**2.70**	**1.09**	**0.19**	**0.01**
	Prenatal Alcohol Exposure	**3.02**	**1.39**	**0.14**	**0.08**
	Prenatal Medication Exposure	-0.57	1.07	-0.05	0.60
	Prenatal Health Risk	-0.27	1.14	-0.02	0.82
	Delivery Risk	0.57	1.07	-0.05	0.60
	Cigarette Use	0.61	0.58	0.08	0.30
	Birth Weight	-0.63	0.81	-0.06	0.44
	Postnatal Mood	0.01	0.08	0.01	0.95
Step 4					
	Income	**-0.67**	**0.15**	**-0.37**	**0.00**
	Child Sex	**2.80**	**1.10**	**0.19**	**0.01**
	Prenatal Alcohol Exposure	**2.76**	**1.43**	**0.15**	**0.06**
	Prenatal Medication Exposure	0.91	0.54	0.14	0.10
	Prenatal Health Risk	-0.74	1.09	-0.06	0.50
	Delivery Risk	-0.21	1.14	-0.01	0.85
	Cigarette Use	0.53	0.59	0.07	0.38
	Birth Weight	-0.66	0.81	-0.06	0.42
	Postnatal Mood	-0.04	0.09	-0.04	0.71
	Prenatal Mood	0.12	0.15	0.08	0.43

Note. Bolded text reflects significant predictors, $p < .05$.

Table 9. Preliminary regression examining potential covariates of teacher report of total problems in stressful experiences model.

Teacher Report-Total Problems		β	SE	Standardized β	*p*
Step 1					
	Income	**-0.60**	**0.14**	**0-0.33**	**0.00**
	Child Sex	**2.70**	**1.08**	**0.19**	**0.01**
Step 2					
	Income	**-0.59**	**0.15**	**-0.32**	**0.00**
	Child Sex	**3.04**	**1.10**	**0.21**	**0.01**
	Prenatal Alcohol Exposure	**3.32**	**1.38**	**0.18**	**0.02**
	Prenatal Medication Exposure	**1.08**	**0.53**	**0.17**	**0.04**
	Prenatal Health Risk	-0.85	1.06	-0.07	0.42
	Delivery Risk	-.20	1.13	-0.01	0.86
	Cigarette Use	0.73	0.57	0.10	0.21
	Birth Weight	-0.76	0.82	-0.07	0.35
Step 3					
	Income	**-0.63**	**0.15**	**-0.34**	**0.00**
	Child Sex	**2.92**	**1.11**	**0.20**	**0.01**
	Prenatal Alcohol Exposure	**3.42**	**1.38**	**0.19**	**0.01**
	Prenatal Medication Exposure	**1.23**	**0.55**	**0.19**	**0.03**
	Prenatal Health Risk	-0.92	1.06	-0.07	0.39
	Delivery Risk	-0.09	1.13	-0.01	0.94
	Cigarette Use	0.86	0.59	0.12	0.14
	Birth Weight	-0.69	0.82	-0.07	0.40
	Postnatal Stressful Experiences	-0.31	0.27	-0.09	0.26
Step 4					
	Income	**-0.64**	**0.15**	**-0.35**	**0.00**
	Child Sex	**2.95**	**1.12**	**0.21**	**0.01**
	Prenatal Alcohol Exposure	**3.47**	**1.40**	**0.19**	**0.01**
	Prenatal Medication Exposure	**1.25**	**0.55**	**0.19**	**0.03**
	Prenatal Health Risk	-0.88	1.08	-0.07	0.42
	Delivery Risk	-0.09	1.13	-0.01	0.94
	Cigarette Use	0.90	0.60	0.12	0.14
	Birth Weight	-0.71	0.82	-0.07	0.39
	Postnatal Stressful Experiences	-0.30	0.28	-0.09	0.29
	Prenatal Stressful Experiences	-0.07	0.29	-0.02	0.80

Note. Bolded text reflects significant predictors, $p < .05$.

Table 10. Preliminary regression examining potential covariates of mother report of social competence in mood model.

Mother Report-Social Comp.		β	SE	Standard. β	*p*
Step 1					
	Income	**0.29**	**0.15**	**0.14**	**0.05**
	Child Sex	0.75	1.14	0.05	0.51
Step 2					
	Income	**0.31**	**0.16**	**0.15**	**0.04**
	Child Sex	0.17	1.19	0.01	0.88
	Prenatal Alcohol Exposure	0.68	1.51	0.03	0.65
	Prenatal Medication Exposure	**-1.25**	**0.58**	**-0.17**	**0.03**
	Prenatal Health Risk	1.17	1.12	0.08	0.30
	Delivery Risk	1.36	1.14	0.09	0.24
	Cigarette Use	0.27	0.68	0.03	0.69
	Birth Weight	-0.07	0.87	-0.01	0.94
Step 3					
	Income	0.13	0.16	0.06	0.44
	Child Sex	0.01	1.16	0.00	0.99
	Prenatal Alcohol Exposure	0.85	1.46	0.04	0.56
	Prenatal Medication Exposure	-0.92	0.58	-0.12	0.11
	Prenatal Health Risk	1.58	1.10	0.11	0.15
	Delivery Risk	1.32	1.12	0.09	0.24
	Cigarette Use	0.60	0.67	0.07	0.38
	Birth Weight	-0.07	0.84	-0.01	0.93
	Postnatal Mood	**-0.28**	**0.09**	**-0.26**	**0.00**
Step 4					
	Income	0.14	0.16	0.07	0.40
	Child Sex	0.19	1.15	0.01	0.87
	Prenatal Alcohol Exposure	-0.06	1.53	-0.00	0.97
	Prenatal Medication Exposure	-0.80	0.58	-0.13	0.09
	Prenatal Health Risk	1.12	1.10	0.09	0.27
	Delivery Risk	1.25	1.11	0.08	0.26
	Cigarette Use	0.45	0.67	0.05	0.50
	Birth Weight	-0.09	0.84	-0.01	0.91
	Postnatal Mood	**-0.39**	**0.10**	**-0.35**	**0.00**
	Prenatal Mood	**0.30**	**0.15**	**0.18**	**0.05**

Note. Bolded text reflects significant predictors, $p < .05$.

Table 11. Preliminary regression examining potential covariates of mother report of social competence in the stressful experiences model.

Mother Report-Social Comp.		β	SE	Standard. β	*p*
Step 1					
	Income	0.26	0.15	0.13	0.09
	Child Sex	0.44	1.17	0.03	0.71
Step 2					
	Income	0.29	0.16	0.14	0.08
	Child Sex	-0.11	1.23	-0.01	0.93
	Prenatal Alcohol Exposure	0.17	1.55	0.01	0.91
	Prenatal Medication Exposure	-1.24	0.59	-0.17	0.04
	Prenatal Health Risk	1.27	1.14	0.09	0.27
	Delivery Risk	1.15	1.16	0.08	0.32
	Cigarette Use	0.26	0.69	0.03	0.71
	Birth Weight	0.05	0.89	0.01	0.95
Step 3					
	Income	0.24	0.16	0.12	0.14
	Child Sex	-0.21	1.23	-0.01	0.87
	Prenatal Alcohol Exposure	0.29	1.55	0.01	0.85
	Prenatal Medication Exposure	-1.01	0.62	-0.14	0.10
	Prenatal Health Risk	1.18	1.14	0.09	0.30
	Delivery Risk	1.21	1.15	0.08	0.30
	Cigarette Use	0.44	0.70	0.05	0.53
	Birth Weight	0.13	0.89	0.01	0.88
	Postnatal Stressful Experiences	-0.41	0.31	-0.11	0.18
Step 4					
	Income	0.24	0.17	0.11	0.17
	Child Sex	-0.19	1.23	-0.01	0.88
	Prenatal Alcohol Exposure	0.32	1.58	0.02	0.84
	Prenatal Medication Exposure	-1.00	0.62	0.14	0.11
	Prenatal Health Risk	1.19	1.15	0.09	0.30
	Delivery Risk	1.22	1.16	0.08	0.29
	Cigarette Use	0.46	0.72	0.05	0.52
	Birth Weight	0.12	0.90	0.01	0.89
	Postnatal Stressful Experiences	-0.41	0.31	-0.11	0.20
	Prenatal Stressful Experiences	-0.04	0.32	-0.01	0.91

Note. Bolded text reflects significant predictors, $p < .05$.

Table 12. Preliminary regression examining potential covariates of mother report of problems in mood model.

Mother Report- Total Problems		β	SE	Standard. β	*p*
Step 1					
	Income	**-0.40**	**0.14**	**-0.19**	**0.01**
	Child Sex	0.42	1.12	0.03	0.71
Step 2					
	Income	**-0.45**	**0.15**	**-0.22**	**0.00**
	Child Sex	0.63	1.13	0.04	0.58
	Prenatal Alcohol Exposure	2.11	1.45	0.10	0.15
	Prenatal Medication Exposure	**1.83**	**0.56**	**0.24**	**0.01**
	Prenatal Health Risk	0.39	1.07	0.03	0.72
	Delivery Risk	0.13	1.10	0.01	0.91
	Cigarette Use	0.74	0.65	0.08	0.26
	Birth Weight	1.08	0.83	0.10	0.19
Step 3					
	Income	-0.18	0.15	-0.09	0.23
	Child Sex	0.82	1.07	0.05	0.44
	Prenatal Alcohol Exposure	1.91	1.36	0.09	0.16
	Prenatal Medication Exposure	**1.33**	**0.53**	**0.18**	**0.01**
	Prenatal Health Risk	-0.18	1.01	-0.13	0.86
	Delivery Risk	0.19	1.03	0.01	0.85
	Cigarette Use	0.26	0.62	0.03	0.68
	Birth Weight	1.08	0.78	0.10	0.17
	Postnatal Mood	**0.40**	**0.08**	**0.37**	**0.00**
Step 4					
	Income	-0.18	0.15	-0.09	0.22
	Child Sex	0.78	1.07	0.05	0.47
	Prenatal Alcohol Exposure	2.13	1.43	0.10	0.14
	Prenatal Medication Exposure	**1.34**	**0.54**	**0.18**	**0.01**
	Prenatal Health Risk	-0.10	1.03	-0.01	0.93
	Delivery Risk	0.21	1.03	0.01	0.84
	Cigarette Use	0.30	0.63	0.03	0.64
	Birth Weight	1.09	0.78	0.10	0.17
	Postnatal Mood	**0.43**	**0.09**	**0.39**	**0.00**
	Prenatal Mood	-0.07	0.14	-0.04	0.60

Note. Bolded text reflects significant predictors, $p < .05$.

Table 13. Preliminary regression examining potential covariates of mother report of total problems in stressful experiences model.

Mother Report-Total Problems		β	SE	Standard. β	*p*
Step 1					
	Income	**-0.42**	**0.15**	**-0.20**	**0.01**
	Child Sex	0.32	1.17	0.02	0.78
Step 2					
	Income	**-0.45**	**0.16**	**-0.22**	**0.00**
	Child Sex	0.48	1.18	0.03	0.68
	Prenatal Alcohol Exposure	2.24	1.49	0.11	0.14
	Prenatal Medication Exposure	**1.86**	**0.57**	**0.25**	**0.01**
	Prenatal Health Risk	0.34	1.10	0.03	0.76
	Delivery Risk	-0.05	1.11	-0.00	0.96
	Cigarette Use	0.72	0.66	0.08	0.28
	Birth Weight	1.18	0.86	0.10	0.17
Step 3					
	Income	**-0.32**	**0.15**	**-0.15**	**0.04**
	Child Sex	0.80	1.12	0.05	0.48
	Prenatal Alcohol Exposure	1.87	1.42	0.09	0.19
	Prenatal Medication Exposure	**1.16**	**0.56**	**0.16**	**0.04**
	Prenatal Health Risk	0.63	1.04	0.05	0.55
	Delivery Risk	-0.26	1.06	-0.17	0.81
	Cigarette Use	0.15	0.64	0.02	0.82
	Birth Weight	0.95	0.82	0.08	0.25
	Postnatal Stressful Experiences	**1.28**	**0.28**	**0.33**	**0.00**
Step 4					
	Income	-0.30	0.16	-0.14	0.06
	Child Sex	0.74	1.13	0.05	0.51
	Prenatal Alcohol Exposure	1.75	1.44	0.08	0.23
	Prenatal Medication Exposure	**1.12**	**0.57**	**0.15**	**0.05**
	Prenatal Health Risk	0.56	1.05	0.04	0.60
	Delivery Risk	-0.29	1.06	-0.02	0.79
	Cigarette Use	0.08	0.66	0.01	0.91
	Birth Weight	0.98	0.82	0.09	0.23
	Postnatal Stressful Experiences	**1.25**	**0.29**	**0.32**	**0.00**
	Prenatal Stressful Experiences	0.15	0.29	0.04	0.61

Note. Bolded text reflects significant predictors, $p < .05$.

Table 14. Correlations of mood, stress, pregnancy and birth variables with income.

	Income
Prenatal Mood	-0.22***
Prenatal Stressful Experiences	-0.37***
Postnatal Mood	-0.38***
Postnatal Stressful Experiences	-0.28***
Prenatal Alcohol Exposure	0.18**
Prenatal Medication Exposure	0.07
Prenatal Health Risk	-0.06
Delivery Risk	-0.06
Cigarette Use	-0.30***
Birth Weight	0.08

Note. *p < .05, **p < .01, *** p < .001.

Table 15. Correlations between mother and teacher report of adjustment.

	Teacher Total Problems	Mother Social Competence	Mother Total Problems
Teacher Social Competence	-0.72***	0.16*	-0.17**
Teacher Total Problems		-0.26***	0.34***
Mother Social Competence			-0.49***

Note. *p < .05, **p < .01, *** p < .001.

Table 16. Fit statistics for structural equation models.

	Chi-square	df	*p*-value	RMSEA	CFI
Stressful experiences (teacher report)	94.24	24	0.00	0.08	0.90
Stressful experiences (mother report)	93.33	24	0.00	0.08	0.90
Mood (teacher report)	67.13	24	0.00	0.08	0.95
Mood (mother report)	65.27	24	0.00	0.08	0.94

Table 17. Standardized coefficients and standard errors of the effects of prenatal and postnatal mood on the intercept and slope of children's executive control.

	Estimate	95% CI	Standardized Beta
Intercept Executive Control			
Income	0.01*	0.00 – 0.01	0.16*
Child Sex	-0.03*	-0.07 –0.00	-0.13*
Prenatal Medication Exposure	-0.01	-0.02 – 0.01	-0.06
Prenatal Alcohol Exposure	0.00	-0.05 – 0.06	0.01
Postnatal Mood	-0.00	-0.01 – 0.00	-0.12
Prenatal Mood	-0.00^{t}	-0.01 – 0.00	-0.14^{t}
Slope Executive Control			
Income	0.00	-0.00 – 0.00	-0.01
Child Sex	-0.00	-0.01 – 0.01	-0.01
Prenatal Medication Exposure	0.00	-0.00 – 0.01	0.08
Prenatal Alcohol Exposure	0.02^{t}	-0.00 – 0.04	0.17^{t}
Postnatal Mood	0.00	-0.00 – 0.00	0.00
Prenatal Mood	0.00	-0.00 – 0.00	-0.06

Note. ^{t} <.10, $^{*}p$ < .05, **p < .01, *** p < .001.

Table 18. Unstandardized coefficients, confidence intervals of model results, and standardized coefficients of the effects of prenatal and postnatal stressful experiences on the intercept and slope of children's executive control.

	Estimate	95% CI	Standardized Beta
Intercept Executive Control			
Income	0.01**	0.00 – 0.01	0.22**
Child Sex	-0.03[t]	-0.06 – 0.01	-0.11[t]
Prenatal Medication Exposure	-0.01	-0.03 – 0.01	-0.10
Prenatal Alcohol Exposure	-0.01	-0.06 – 0.04	-0.02
Postnatal Stressful Experiences	0.00	-0.02 – 0.00	0.07
Prenatal Stressful Experiences	-0.01[t]	-0.00 – 0.01	-0.14[t]
Slope Executive Control			
Income	0.00	-0.00 – 0.00	0.03
Child Sex	-0.00	-0.01 – 0.01	-0.02
Prenatal Medication Exposure	0.00	-0.01 – 0.01	0.04
Prenatal Alcohol Exposure	0.02[t]	-0.00 – 0.03	0.14[t]
Postnatal Stressful Experiences	-0.00	-0.00 – 0.00	0.11
Prenatal Stressful Experiences	0.00	-0.01 – 0.01	-0.06

Note. [t] <.10, *p < .05, **p < .01, *** p < .001.

Table 19. Unstandardized coefficients, confidence intervals of model results, and standardized coefficients of the effect of executive control, postnatal mood, and prenatal mood on mother report of children's adjustment.

	Mother Report		
	Estimate	95 CI	Standardized Beta
Social Competence			
Income	0.07	-0.20 – 0.35	0.03
Child Sex	-0.62	-2.66 – 1.31	-0.04
Prenatal Medication Exposure	-0.24	-1.17 – 0.74	-0.03
Prenatal Alcohol Exposure	-0.51	-2.94 – 2.05	-0.02
Postnatal Mood	-0.32**	-0.55 – -0.13	-0.30**
Prenatal Mood	0.27^{t}	-0.01 – 0.53	0.16^{t}
Intercept Executive Control	2.86	-6.92 – 12.42	0.05
Slope Executive Control	19.34	-10.57 – 57.99	0.10
Total Problems			
Income	-0.12	-0.43 – 0.14	-0.06
Child Sex	1. 61^{t}	0.05 – 3.29	0.11^{t}
Prenatal Medication Exposure	1.06*	0.07 – 2.06	0.15*
Prenatal Alcohol Exposure	2.14^{t}	-0.45 – 4.51	0.11^{t}
Postnatal Mood	0.43***	0.24 – 0.66	0.42***
Prenatal Mood	-0.04	-0.31 – 0.22	-0.03
Intercept Executive Control	6.46	1.64 – 15.51	0.11
Slope Executive Control	-17.31	-60.86 – 11.79	-0.09

Note. ^{t} <.10, *p < .05, **p < .01, *** p < .001.

Table 20. Unstandardized coefficients, confidence intervals of model results, and standardized coefficients of the effect of executive control, postnatal mood, and prenatal mood on teacher report of children's adjustment.

	Teacher Report		
	Estimate	95 CI	Standardized Beta
Social Competence			
Income	0.28	-0.09 – 0.65	0.11
Child Sex	-3.41**	-6.01 – -0.94	-0.17*
Prenatal Medication Exposure	-0.86	-2.19 – 0.48	-0.09
Prenatal Alcohol Exposure	-0.15	-4.09 – 3.55	-0.01
Postnatal Mood	0.17	-0.05 – 0.38	0.13
Prenatal Mood	-0.28	-0.66 – 0.07	-0.13
Intercept Executive Control	21.85**	8.02 – 35.54	0.28**
Slope Executive Control	62.23*	15.87 – 125.40	0.25*
Total Problems			
Income	-0.45**	-0.73 – -0.15	-0.25**
Child Sex	2.02*	0.30 – 3.90	0.14*
Prenatal Medication Exposure	0.41	-0.54 – 1.32	0.06
Prenatal Alcohol Exposure	3.07*	0.19 – 6.03	0.17*
Postnatal Mood	-0.02	-0.17 – 0.14	-0.02
Prenatal Mood	0.07	-0.18 – 0.34	0.05
Intercept Executive Control	-9.26*	-18.25 – -0.47	-0.17*
Slope Executive Control	-43.94*	-87.10 – -14.45	-0.25*

Note. ^{t}<.10, *p < .05, **p < .01, *** p < .001.

Table 21. Unstandardized coefficients, confidence intervals of model results, and standardized coefficients of the effect of executive control, postnatal stressful experiences, and prenatal stressful experiences on mother report of children's adjustment.

	Mother Report		
	Estimate	95 CI	Standardized Beta
Social Competence			
Income	0.15	-0.14 – 0.45	0.08
Child Sex	-0.78	-2.78 – 1.18	-0.05
Prenatal Medication Exposure	-0.33	-1.29 – 0.67	-0.04
Prenatal Alcohol Exposure	0.09	-2.30 – 2.57	0.00
Postnatal Stressful Experiences	-0.49*	-0.97 – -0.03	-0.13*
Prenatal Stressful Experiences	0.18	-0.36 – 0.71	0.05
Intercept Executive Control	4.61	-5.83 – 14.47	0.07
Slope Executive Control	18.81	-13.74 – 057.15	0.09
Total Problems			
Income	-0.17	-0.54 – 0.12	-0.09
Child Sex	1.64^t	0.02 – 3.36	0.11^t
Prenatal Medication Exposure	0.96^t	-0.09 – 2.10	0.13^t
Prenatal Alcohol Exposure	1.97^t	-0.26 – 3.92	0.10^t
Postnatal Stressful Experiences	1.28***	0.85 – 1.71	0.35***
Prenatal Stressful Experiences	0.03	-0.53 – 0.56	0.01
Intercept Executive Control	1.37	-6.64 – 11.07	0.02
Slope Executive Control	-22.83	-68.66 – 6.95	-0.12

Note. ^{t} <.10, *p < .05, **p < .01, *** p < .001.

Table 22. Unstandardized coefficients, confidence intervals of model results, and standardized coefficients of the effect of executive control, postnatal stressful experiences, and prenatal stressful experiences on teacher report of children's adjustment.

Teacher Report			
	Estimate	95 CI	Standardized Beta
Social Competence			
Income	0.31	-0.08 – 0.72	0.12
Child Sex	-3.18*	-5.84 – -0.56	-0.16*
Prenatal Medication Exposure	-1.13	-2.49 – 0.27	-0.12
Prenatal Alcohol Exposure	-1.05	-4.93 – 2.75	-0.04
Postnatal Stressful Experiences	0.10	-0.57 – 0.78	0.02
Prenatal Stressful Experiences	0.25	-0.36 – 0.84	0.05
Intercept Executive Control	22.23**	8.63 – 36.03	0.28**
Slope Executive Control	65.21*	15.30 – 128.97	0.26*
Total Problems			
Income	-0.45**	-0.79 – -0.14	-0.25**
Child Sex	1.97*	0.06 – 4.18	0.14*
Prenatal Medication Exposure	0.52	-0.45 – 1.56	0.08
Prenatal Alcohol Exposure	3.26*	0.45 – 7.19	0.18*
Postnatal Stressful Experiences	-0.04	-0.63 – 0.40	-0.01
Prenatal Stressful Experiences	0.07	-0.31 – 0.69	0.02
Intercept Executive Control	-9.29*	-18.44 – 0.87	-0.17*
Slope Executive Control	-45.17*	-90.25 – -16.48	-0.26*

Note. ^{t} <.10, **p* < .05, ***p* < .01, *** *p* < .001.

Table 23. Chi-square difference test of differences in model by stressor type

Model	Chi-square difference	df	*p*-value
Mother report model	75.46	25	n.s.
Teacher report model	66.72	25	n.s.

Table 24. Chi-square difference test of sex differences

Model	Chi-square difference	df	*p*-value
Stressful experiences model (teacher report)	73.80	51	n.s.
Stressful experiences model (mother report)	77.10	51	n.s.
Mood model (teacher report)	75.35	51	n.s.
Mood model (mother report)	88.05	51	n.s.

Appendix A

Table A1. Preliminary regression examining potential covariates of diurnal cortisol morning level in mood model.

T1 Diurnal Morning Level		β	SE	Standard. β	*P*
Step 1					
	Income	0.01	0.01	0.10	0.17
	Child Sex	-0.04	0.04	-0.01	0.92
Step 2					
	Income	0.01	0.01	0.12	0.16
	Child Sex	0.00	0.04	0.01	0.94
	Prenatal Alcohol Exposure	-0.05	0.05	-0.08	0.31
	Prenatal Medication Exposure	0.03	0.02	0.10	0.23
	Prenatal Health Risk	0.06	0.04	0.13	0.14
	Delivery Risk	-0.00	0.04	-0.01	0.92
	Cigarette Use	0.01	0.03	0.03	0.75
	Birth Weight	0.03	0.03	0.07	0.42
Step 3					
	Income	0.01	0.01	0.10	0.25
	Child Sex	0.00	0.04	0.01	0.94
	Prenatal Alcohol Exposure	-0.05	0.05	-0.08	0.32
	Prenatal Medication Exposure	0.03	0.02	0.11	0.21
	Prenatal Health Risk	0.06	0.04	0.13	0.13
	Delivery Risk	-0.01	0.04	-0.01	0.91
	Cigarette Use	0.01	0.03	0.03	0.74
	Birth Weight	0.03	0.03	0.07	0.41
	Postnatal Depression	-0.00	0.00	-0.03	0.72
Step 4					
	Income	0.00	0.00	0.12	0.22
	Child Sex	-0.05	0.02	-0.17	0.82
	Prenatal Alcohol Exposure	0.01	0.03	0.03	0.12
	Prenatal Medication Exposure	0.00	0.01	0.01	0.29
	Prenatal Health Risk	0.01	0.02	0.04	0.24
	Delivery Risk	-0.03	0.02	-0.10	0.90
	Cigarette Use	0.01	0.01	0.04	0.79
	Birth Weight	0.01	0.02	0.05	0.45
	Postnatal Depression	-0.00	0.00	-0.08	0.20
	Prenatal Mood	-0.01	0.00	-0.26	0.054

Table A2. Preliminary regression examining potential covariates of diurnal cortisol slope in mood model.

T1 Diurnal Slope		β	SE	Standard. β	*p*
Step 1					
	Income	0.01	0.01	0.06	0.41
	Child Sex	0.04	0.05	0.06	0.44
Step 2					
	Income	0.01	0.01	0.05	0.51
	Child Sex	0.04	0.06	0.06	0.44
	Prenatal Alcohol Exposure	0.01	0.07	0.01	0.89
	Prenatal Medication Exposure	0.04	0.03	0.11	0.21
	Prenatal Health Risk	0.03	0.05	0.05	0.58
	Delivery Risk	-0.03	0.05	0.04	0.63
	Cigarette Use	0.01	0.04	0.02	0.80
	Birth Weight	0.06	0.04	0.12	0.16
Step 3					
	Income	0.00	0.01	0.03	0.71
	Child Sex	0.04	0.01	0.06	0.45
	Prenatal Alcohol Exposure	0.01	0.06	0.01	0.87
	Prenatal Medication Exposure	0.04	0.07	0.11	0.18
	Prenatal Health Risk	0.03	0.03	0.05	0.55
	Delivery Risk	-0.03	0.05	-0.04	0.61
	Cigarette Use	0.01	0.05	0.02	0.78
	Birth Weight	0.06	0.04	0.12	0.15
	Postnatal Depression	-0.00	0.00	-0.05	0.57
Step 4					
	Income	0.00	0.01	0.04	0.67
	Child Sex	0.05	0.06	0.07	0.41
	Prenatal Alcohol Exposure	-0.01	0.07	-0.01	0.94
	Prenatal Medication Exposure	0.04	0.03	0.11	0.21
	Prenatal Health Risk	0.03	0.05	0.04	0.64
	Delivery Risk	-0.03	0.05	-0.04	0.61
	Cigarette Use	0.01	0.04	0.02	0.80
	Birth Weight	0.06	0.04	0.12	0.16
	Postnatal Depression	-0.00	0.01	-0.09	0.39
	Prenatal Mood	0.01	0.01	0.07	0.46

Table A3. Preliminary regression examining potential covariates of diurnal cortisol morning level in stressful experiences model.

T1 Diurnal Morning Level		β	SE	Standard. β	*p*
Step 1					
	Income	0.01	0.01	0.10	0.21
	Child Sex	0.00	0.04	0.00	0.99
Step 2					
	Income	0.01	0.01	0.11	0.17
	Child Sex	0.01	0.04	0.01	0.91
	Prenatal Alcohol Exposure	-0.06	0.05	-0.10	0.23
	Prenatal Medication Exposure	0.02	0.02	0.10	0.27
	Prenatal Health Risk	0.07	0.04	0.14	0.10
	Delivery Risk	-0.01	0.04	-0.03	0.73
	Cigarette Use	0.01	0.03	0.03	0.70
	Birth Weight	0.03	0.03	0.08	0.33
Step 3					
	Income	0.01	0.01	0.10	0.26
	Child Sex	0.00	0.04	0.01	0.95
	Prenatal Alcohol Exposure	-0.06	0.05	-0.09	0.26
	Prenatal Medication Exposure	0.03	0.02	0.12	0.17
	Prenatal Health Risk	0.06	0.04	0.13	0.13
	Delivery Risk	-0.01	0.04	-0.03	0.78
	Cigarette Use	0.02	0.03	0.05	0.58
	Birth Weight	0.03	0.03	0.09	0.30
	Postnatal Stressful Experiences	-0.01	0.01	-0.09	0.30
Step 4					
	Income	0.01	0.01	0.09	0.35
	Child Sex	0.01	0.04	0.01	0.92
	Prenatal Alcohol Exposure	0.01	0.03	0.03	0.12
	Prenatal Medication Exposure	-0.06	0.05	-0.08	0.31
	Prenatal Health Risk	0.03	0.02	0.12	0.16
	Delivery Risk	0.06	0.04	0.01	0.12
	Cigarette Use	-0.01	0.04	0.05	0.53
	Birth Weight	0.03	0.03	0.08	0.31
	Postnatal Depression	-0.01	0.01	-0.08	0.35
	Prenatal Stressful Experiences	-0.01	0.01	-0.04	0.68

Table A4. Preliminary regression examining potential covariates of diurnal cortisol morning level in mood model.

T1 Diurnal Slope		β	SE	Standard. β	*p*
Step 1					
	Income	0.01	0.01	0.06	0.47
	Child Sex	0.04	0.05	0.06	0.42
Step 2					
	Income	0.01	0.01	0.06	0.47
	Child Sex	0.05	0.06	0.07	0.40
	Prenatal Alcohol Exposure	-0.01	0.07	-0.01	0.93
	Prenatal Medication Exposure	0.04	0.03	0.11	0.19
	Prenatal Health Risk	0.03	0.05	0.05	0.56
	Delivery Risk	-0.03	0.06	-0.04	0.64
	Cigarette Use	0.01	0.04	0.03	0.72
	Birth Weight	0.05	0.04	0.11	0.19
Step 3					
	Income	0.01	0.01	0.08	0.36
	Child Sex	0.05	0.06	0.07	0.37
	Prenatal Alcohol Exposure	-0.01	0.07	-0.01	0.87
	Prenatal Medication Exposure	0.03	0.03	0.09	0.32
	Prenatal Health Risk	0.04	0.06	0.06	0.59
	Delivery Risk	0.01	0.04	0.10	0.22
	Cigarette Use	0.01	0.04	0.02	0.86
	Birth Weight	0.05	0.04	0.10	0.55
	Postnatal Stressful Experiences	0.02	0.01	0.09	0.29
Step 4					
	Income	0.01	0.01	0.06	0.51
	Child Sex	0.06	0.06	0.08	0.34
	Prenatal Alcohol Exposure	-0.00	0.07	-0.00	0.98
	Prenatal Medication Exposure	0.04	0.03	0.11	0.21
	Prenatal Health Risk	0.03	0.03	0.09	0.29
	Delivery Risk	0.04	0.06	0.07	0.44
	Cigarette Use	-0.03	0.06	-0.04	0.60
	Birth Weight	0.05	0.04	0.10	0.24
	Postnatal Depression	0.02	0.02	0.10	0.25
	Prenatal Stressful Experiences	-0.01	0.02	-0.06	0.50

Table A5. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (morning level), postnatal mood and prenatal mood on mother report of children's adjustment.

	Mother Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.05	0.07
Child Sex	-0.05	0.06
Prenatal Medication Exposure	-0.04	0.06
Prenatal Alcohol Exposure	-0.01	0.07
Postnatal Mood	0.16***	0.08
Prenatal Mood	-0.30*	0.08
Intercept Cortisol Morning Level	-0.01	0.10
Total Problems		
Income	-0.07	0.06
Child Sex	0.10^{t}	0.06
Prenatal Medication Exposure	0.14*	0.06
Prenatal Alcohol Exposure	0.11^{t}	0.06
Postnatal Mood	0.42***	0.07
Prenatal Mood	-0.05	0.07
Intercept Cortisol Morning Level	0.05	0.10
χ^2 (40) = 144.93, RMSEA = 0.09, CFI = 0.67		

Note. ^{t}<.10, **p* < .05, ***p* < .01, *** *p* < .001.

Table A6. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (morning level), postnatal mood and prenatal mood on teacher report of children's adjustment.

	Teacher Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.12	0.07
Child Sex	-0.18**	0.07
Prenatal Medication Exposure	-0.09	0.07
Prenatal Alcohol Exposure	0.05	0.07
Postnatal Mood	0.12	0.08
Prenatal Mood	-0.22*	0.09
Intercept Cortisol Morning Level	0.16	0.11
Total Problems		
Income	-0.24**	0.07
Child Sex	0.15*	0.07
Prenatal Medication Exposure	0.06	0.07
Prenatal Alcohol Exposure	0.12	0.07
Postnatal Mood	-0.03	0.08
Prenatal Mood	0.12	0.09
Intercept Cortisol Morning Level	-0.13	0.10
χ^2 (40) = 149.40, RMSEA = 0.10, CFI = 0.72		

Note. ^{t} <.10, *p < .05, **p < .01, *** p < .001.

Table A7. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (morning level), postnatal stressful experiences and prenatal stressful experiences on mother report of children's adjustment.

	Mother Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.10	0.07
Child Sex	-0.05	0.07
Prenatal Medication Exposure	-0.05	0.07
Prenatal Alcohol Exposure	0.01	0.07
Postnatal Stressful Experiences	-0.13*	0.08
Prenatal Stressful Experiences	0.06	0.09
Intercept Cortisol Morning Level	0.01	0.11
Total Problems		
Income	-0.11^t	0.07
Child Sex	0.11^t	0.06
Prenatal Medication Exposure	0.13*	0.06
Prenatal Alcohol Exposure	0.10	0.07
Postnatal Stressful Experiences	0.37***	0.07
Prenatal Stressful Experiences	-0.02	0.07
Intercept Cortisol Morning Level	0.04	0.09

χ^2 (40) = 154.49, RMSEA = 0.10, CFI = 0.57

Note. ^{t} <.10, *p < .05, **p < .01, *** p < .001.

Table A8. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (morning level), postnatal stressful experiences and prenatal stressful experiences on teacher report of children's adjustment.

	Teacher Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.16*	0.07
Child Sex	-0.17*	0.07
Prenatal Medication Exposure	-0.13[t]	0.07
Prenatal Alcohol Exposure	0.00	0.07
Postnatal Stressful Experiences	0.02	0.07
Prenatal Stressful Experiences	0.05	0.08
Intercept Cortisol Morning Level	0.12	0.11
Total Problems		
Income	-0.26***	0.07
Child Sex	0.14*	0.07
Prenatal Medication Exposure	0.08	0.07
Prenatal Alcohol Exposure	0.14[t]	0.07
Postnatal Stressful Experiences	0.00	0.07
Prenatal Stressful Experiences	0.02	0.07
Intercept Cortisol Morning Level	-0.12	0.10

χ^2 (40) = 158.87, RMSEA = 0.10, CFI = 0.65

Table A9. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (diurnal slope), postnatal mood and prenatal mood on mother report of children's adjustment.

	Mother Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.05	0.07
Child Sex	-0.05	0.06
Prenatal Medication Exposure	-0.04	0.06
Prenatal Alcohol Exposure	-0.01	0.07
Postnatal Mood	-0.31***	0.08
Prenatal Mood	0.16*	0.08
Intercept Diurnal Cortisol Slope	-0.04	0.10
Total Problems		
Income	-0.05	0.06
Child Sex	0.08	0.05
Prenatal Medication Exposure	0.14*	0.06
Prenatal Alcohol Exposure	0.11^{t}	0.06
Postnatal Mood	0.41***	0.07
Prenatal Mood	-0.04	0.07
Intercept Diurnal Cortisol Slope	-0.03	0.09
χ^2 (40) = 215.02, RMSEA = 0.12, CFI = 0.45		

Note. ^{t}<.10, *p < .05, **p < .01, *** p < .001.

Table A10. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (diurnal slope), postnatal mood and prenatal mood on teacher report of children's adjustment.

	Teacher Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.11	0.07
Child Sex	-0.20**	0.07
Prenatal Medication Exposure	-0.08	0.07
Prenatal Alcohol Exposure	0.04	0.07
Postnatal Mood	0.13	0.08
Prenatal Mood	-0.24**	0.09
Intercept Diurnal Cortisol Slope	0.13	0.11
Total Problems		
Income	-0.24**	0.07
Child Sex	0.16*	0.06
Prenatal Medication Exposure	0.05	0.07
Prenatal Alcohol Exposure	0.13^t	0.07
Postnatal Mood	-0.03	0.08
Prenatal Mood	0.13*	0.09
Intercept Diurnal Cortisol Slope	-0.19^t	0.10
χ^2 (40) = 214.21, RMSEA = 0.12, CFI = 0.54		

Note. ^{t} <.10, **p* < .05, ***p* < .01, *** *p* < .001.

Table A11. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (diurnal slope), postnatal stressful experiences and prenatal stressful experiences on mother report of children's adjustment.

	Mother Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.11	0.07
Child Sex	-0.06	0.06
Prenatal Medication Exposure	-0.05	0.06
Prenatal Alcohol Exposure	0.01	0.07
Postnatal Stressful Experiences	-0.13*	0.07
Prenatal Stressful Experiences	0.06	0.07
Intercept Diurnal Cortisol Slope	-0.02	0.10
Total Problems		
Income	-0.09	0.07
Child Sex	0.10^t	0.05
Prenatal Medication Exposure	0.13*	0.06
Prenatal Alcohol Exposure	0.10^t	0.06
Postnatal Stressful Experiences	0.37***	0.06
Prenatal Stressful Experiences	-0.01	0.07
Intercept Diurnal Cortisol Slope	-0.06	0.09
χ^2 (40) = 235.21, RMSEA = 0.13, CFI = 0.30		

Note. ^{t} <.10, **p* < .05, ***p* < .01, *** *p* < .001.

Table A12. Model fit, standardized coefficients, and standard errors of the structural equation model of the effect of the intercept of diurnal cortisol (diurnal slope), postnatal stressful experiences and prenatal stressful experiences on teacher report of children's adjustment.

	Teacher Report	
	Standardized Beta	Standard Error
Social Competence		
Income	0.14[t]	0.07
Child Sex	-0.18**	0.07
Prenatal Medication Exposure	-0.11	0.07
Prenatal Alcohol Exposure	-0.01	0.07
Postnatal Stressful Experiences	0.03	0.07
Prenatal Stressful Experiences	0.01	0.08
Intercept Diurnal Cortisol Slope	0.20[t]	0.10
Total Problems		
Income	-0.25***	0.07
Child Sex	0.15*	0.06
Prenatal Medication Exposure	0.06	0.07
Prenatal Alcohol Exposure	0.15	0.07
Postnatal Stressful Experiences	0.01	0.07
Prenatal Stressful Experiences	0.04	0.08
Intercept Diurnal Cortisol Slope	-0.18[t]	0.10
χ^2 (40) = 231.32, RMSEA = 0.13, CFI = 0.43		

Note. [t] <.10, *p < .05, **p < .01, *** p < .001.

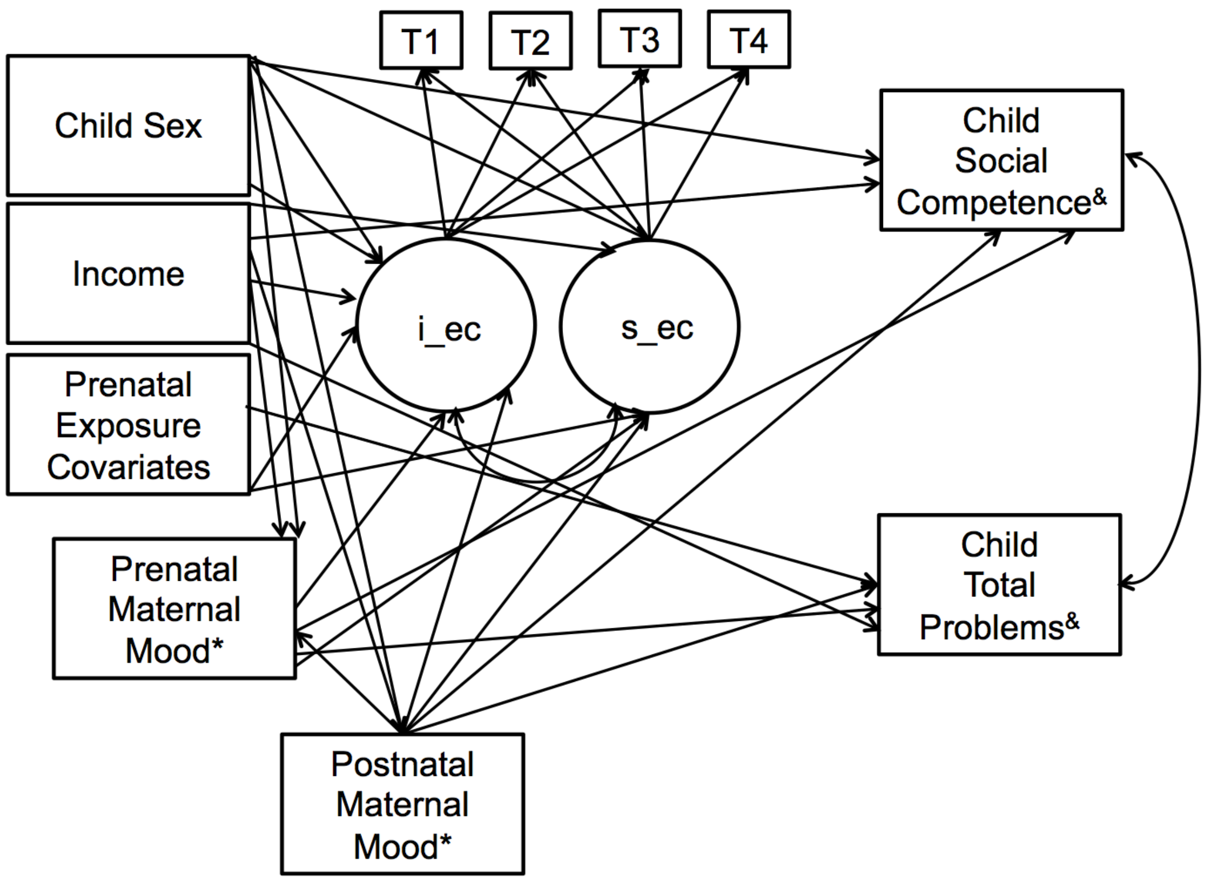
Appendix B

Table B1. Correlations between prenatal and postnatal maternal mood and stressful experiences with maternal parenting.

	Prenatal Mood	Prenatal Stressful Experiences	Postnatal Mood	Postnatal Stressful Experiences
Warmth	-0.11[t]	-0.19**	-0.17**	-0.09
Negativity	0.23***	0.23***	0.21***	0.18**
Scaffolding	-0.18**	-0.29***	-0.25***	-0.16**
Responsiveness	-0.05	-0.14*	-0.01	-0.12*
Limit Setting	-0.03	-0.16*	-0.15*	-0.14*

Note. [t] <.10, *p < .05, **p < .01, *** p < .001.

Figure 1. Model of the relations between prenatal stress, putative mediators and child adjustment.



Note: *Or maternal stressful experiences. [&]Separate models were tested for mother and teacher report of child adjustment outcomes. T1 = Time 1 executive control, T2 = Time 2 executive control, T3 = Time 3 executive control, T4 = Time 4 executive control, i_ec = intercept of executive control, s_ec = slope of executive control.

VITA

Stephanie Fengler Thompson completed her Bachelor of the Arts in Psychology at Emory University. She graduated Summa cum Laude under the mentorship of Sherryl H. Goodman, Ph.D. Her honors thesis examined the role of depression characteristics, infant negative affectivity, stress and social support in maternal sensitive responsiveness among women at risk of depression.

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