A GENETICALLY INFORMED STUDY OF THE INTERACTION BETWEEN PRENATAL ALCOHOL EXPOSURE AND PARENTAL DEPRESSION IN RISK FOR CHILDREN'S EMERGING EXTERNALIZING PROBLEMS

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Prenatal Alcohol Exposure (PAE) has been associated with children's externalizing problems (e.g., D'Onofrio et al.,2007); however, it remains unclear whether low/moderate PAE has a meaningful effect on children's outcomes. Perhaps low/moderate PAE increases children's vulnerability to externalizing but only in the context of postnatal adversity, such as parental depression. An adoption study is advantageous for addressing questions about the independent influence of PAE, as genetic and postnatal contextual risk can be disentangled from one another and their interactive associations may be assessed. Primary aims of the current study were to examine independent and interactive associations between PAE and postnatal exposure to parental depressive symptoms in relation to child externalizing problems at early school-age, after accounting for inherited risk. The role of inhibitory control (IC) as a possible mediator of the relationship between PAE and externalizing was also examined. Study data came from the Early Growth and Development Study, a multi-site prospective longitudinal adoption study. Reported alcohol consumption was lower than expected. There was no evidence for an association between PAE and children's externalizing, independently or in interaction with adoptive parent depression. There was also no effect of PAE on children's IC. Adoptive mother and father depressive symptoms were independently associated with children's externalizing. IC at 27 months was negatively related to child externalizing. Findings did not support the hypothesis that low/moderate PAE would be associated with children's externalizing, regardless

of the presence of postnatal contextual adversity. Study findings are novel because of the adoption design, in which the parents providing the postnatal environment were genetically unrelated to the child and did not provide the prenatal environment. However, adoptive families were relatively low-risk, thus findings may not generalize to families facing higher levels of postnatal contextual adversity.

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1.0 INTRODUCTION

It has largely been established that prenatal alcohol exposure (PAE), particularly at high levels, is associated with detrimental outcomes for children's behavioral and emotional health, specifically conduct problems (Disney, Iacono, McGue, Tully, & Legrand, 2008). Although higher levels of PAE have been associated with worse outcomes, evidence of deficits in children have been documented even at relatively low levels (Sood et al., 2001), albeit not in all studies (Kelly et al., 2009). Alcohol is a known teratogen that is likely to cause harm by disturbing central nervous system development and thereby causing deficits in executive functioning (Kodituwakku, Kalberg, & May, 2001). As PAE may not affect all children equally, further research on moderators of the effect of PAE on child outcomes is critical.

The ability to delineate pathways between low to moderate PAE and poor child outcomes is limited by the fact that pregnant women who choose to consume alcohol tend to differ in important ways from those who abstain, including higher rates of other forms of substance use (Harrison & Sidebottom, 2009; Skagerström, Chang, & Nilsen, 2011) and higher levels of depression (Zuckerman, Amaro, Bauchner, & Cabral, 1989). It is also possible that the link between PAE and children's problem behavior could be due, at least in part, to genetic influences rather than through the potential teratogenic effects of PAE, as it is likely that those with a genetic disposition of psychopathology are more likely to consume alcohol during the prenatal period. Moreover, Hill, Lowers, Locke-Wellman, and Shen (2000) have found that

familial risk for alcoholism and PAE are significantly associated with one another. Another possible "third variable" explanation for the association between PAE and child problem behavior is that mothers who drink while pregnant may be more likely to expose their children to riskier postnatal environmental factors, thereby placing offspring at additional risk for problem behavior. Findings from a high-risk sample suggest that mothers who use alcohol prentally are not only likely to continue, but also to increase their alcohol use postpartum (O'Connor & Paley, 2006). Moreover, maternal alcohol use in early childhood has been associated with child maltreatment and harsh parenting (Kim, Pears, Fisher, Connelly, & Landsverk, 2010). Researchers have attempted to account for these confounds by statistically accounting for genetic and parenting variables, such as parental antisocial behavior and harsh parenting. However, this strategy does not allow for testing the possible interactive effects of PAE and contextual stress, which may be important in determining children's sensitivity to PAE and accounting for the inconsistent pattern of direct associations found between PAE and children's conduct problems. That is, perhaps low or moderate PAE increases children's vulnerability to conduct problems, a risk that could be exacerbated by high levels of postnatal contextual adversity.

One contextual stressor that has consistently been associated with negative child outcomes is parental depression, particularly in early childhood (Shaw & Shelleby, 2014). Although parents' depressive symptoms have been associated with many negative child outcomes, including deficits in social cognitive skills (Jensen et al., 2014), infant negative emotionality (Melchior et al., 2012), and internalizing problems (Pilowsky et al., 2006), they have been most consistently linked to conduct problems (Kim-Cohen et al., 2005). Unfortunately, in the vast majority of studies examining associations between PAE and child problem behavior, biological parents also rear their offspring, making it impossible to

disentangle genetic from postnatal environmental risk. The genetically informed design of the Early Growth and Development Study (EGDS) is advantageous for addressing questions about the independent influence of PAE, as its parent-offspring adoption design eliminates the confound of having biological parents also raise their own offspring.

The primary aims of the current study are to examine the independent and interactive associations between PAE and postnatal exposure to depressive symptoms in relation to child externalizing problems in the early school-age period, after accounting for genetic (i.e., inherited) risk. It is expected that a weakly significant direct effect between PAE exposure and later child problem behavior will be observed because levels of contextual stress in adoptive families are low. However, it is also anticipated that those children with both prenatal *and* postnatal risk will have higher rates of externalizing problems relative to those with low levels of both or only one risk factor (i.e., high PAE but low parental depression). The study will also examine the role of inhibitory control as a possible mediator of the relationship between PAE and children's externalizing problems.

1.1 PRENATAL SUBSTANCE EXPOSURE

Exposure to substances in utero, including alcohol, tobacco, and others (e.g., cocaine, marijuana, and methamphetamine), has long been known to have robust, adverse effects on a range of children's psychological outcomes (Bailey et al., 2004; Disney et al., 2008; Richardson, Ryan, Willford, Day, & Goldschmidt, 2002). The effects of PAE have been particularly well studied, with fetal alcohol syndrome (FAS) being a well-known disorder for which infants are routinely screened. FAS is considered the most serious in a spectrum of PAE-related disorders,

with the presence of facial abnormalities, growth deficits, and central nervous system abnormalities all required to meet criteria for the diagnosis. The most likely mechanism by which PAE compromises the developing fetus is by disturbing the development of the central nervous system (Kodituwakku et al., 2001). Research indicates that PAE has specific effects on several brain structures, including the frontal cortex (Sowell et al., 2002), which may be related to the conduct problems that are frequently observed in affected children.

Prevalence of alcohol use while pregnant varies substantially depending on the sample and also the way in which women are asked to report on their drinking behavior while pregnant. Based on data collected on 4,088 mothers of infants born between 1997 and 2002, Ethen and colleagues (2009) found that 30.3% of women used any alcohol during their pregnancy, and 8.3% of women also reported binge drinking during their pregnancy, the latter of which is thought to be particularly problematic for the developing fetus (Bailey et al., 2004). These rates of prenatal alcohol use are considerably higher than those reported in studies that used national data, such as the National Survey on Drug Use and Health, which found that 9.4% of pregnant women used alcohol in 2012-2013 and 2.3% reported binge drinking (SAMHSA, 2014).

Although much of the literature has focused on children with a diagnosis of FAS, there is evidence that children exposed to alcohol prenatally who do not meet full criteria for diagnosis experience marked deficits in some areas of functioning (Mattson et al., 1998), leading researchers to posit that the range of physical, behavioral, and emotional problems associated with PAE may be more accurately conceptualized as Fetal Alcohol Spectrum Disorders (FASD). FASD, which includes FAS and partial FAS, as well as alcohol-related neurodevelopmental disorders and alcohol-related birth defects, has been estimated to affect two to five percent of young children in the United States (May et al., 2009). Although more adverse postnatal child

outcomes have been associated with higher levels of prenatal alcohol use, evidence of deficits in children have been documented even at relatively low levels of exposure (Sood et al., 2001). Without meeting criteria for a diagnosis of FAS, children with more mild prenatal exposure to alcohol may not be eligible for services that could benefit them (Bertrand, Floyd, & Weber, 2005). If mild PAE is indeed related to negative child outcomes, this would have wide-ranging implications, as some alcohol use during pregnancy is relatively common. Thus, although PAE is a significant public health concern, less research has been conducted on the potential adverse effects on child outcomes at light and moderate levels of exposure.

1.2 ASSOCIATIONS BETWEEN PAE AND THE DEVELOPING BRAIN

The primary reason that PAE is implicated in such a broad range of outcomes for exposed children is thought to be due to the teratogenic effects of alcohol on the development of brain structures important in regulating behavior, most notably the frontal cortex. Researchers have posited that alcohol may disrupt early brain development by interfering with molecules that guide and regulate neuronal growth in the fetus (Goodlett, Horn, & Zhou, 2005) and altering brain metabolism (Fagerlund et al., 2006). Children with PAE have been found to have reductions in white matter across multiple areas in the brain (Fryer et al., 2009). Researchers have found associations between PAE and abnormalities in several other brain structures, including but not limited to the cerebellum (O'Hare et al., 2005), the inferior parietal lobe (Sowell et al., 2002), and the corpus callosum (Riley et al., 1995), with damage to the frontal cortex and closely related areas such as the striatum having the most direct effects on executive functioning. In particular, researchers have found that during tasks requiring response inhibition,

children with PAE exhibit greater activation in the prefrontal cortex but decreased activation in the caudate nucleus compared to non-exposed children (Fryer et al., 2007), indicating that children with PAE may exert greater effort in exercising inhibitory control. This differential pattern of activity may be attributable to the reduced caudate volume (Mattson et al., 1996), frontal cortical shape abnormalities (Sowell et al., 2002), and size reductions (Wass, Persutte, & Hobbins, 2001) that have been demonstrated in children prenatally exposed to alcohol. PAE-related damage to the frontal cortex and associated areas may be linked to children's postnatal functioning in important ways (e.g., deficits in overall intellectual functioning, difficulties with verbal learning), perhaps most notably by putting children at increased risk of executive functioning deficits.

1.3 PAE AND DEFICITS IN EXECUTIVE FUNCTIONING: PATHWAY TO CONDUCT PROBLEMS

There is reason to believe that at least some of the problem behaviors that are more common in children with PAE (D'Onofrio et al., 2007; Disney et al., 2008; Paley, O'Connor, Kogan, & Findlay, 2005) may be attributable to executive functioning deficits (Rasmussen, 2005) that are related to PAE-related damage to the frontal cortex and related areas (Kodituwakku et al., 2001). Executive functioning deficits are among the most well-documented problems that children with PAE face, and may include problems with planning and goal-directed behaviors, shifting attention and working memory, and inhibiting impulsive or inappropriate responses, also known as inhibitory control. Although individuals with PAE often have lower IQs than their non-exposed counterparts, researchers have found that these

generalized differences in intellectual ability do not fully explain executive functioning deficits (Connor, Sampson, Bookstein, Barr, & Streissguth, 2000). These deficits may contribute to learning and memory problems, as diminished performance in these areas has been documented in children with PAE (Streissguth et al., 1994; Willford, Richardson, Leech, & Day, 2004).

Negative effects of PAE on executive functioning have also been confirmed using animal models, in which PAE is more easily manipulated. In studies using primates, researchers have established a link between PAE and a reduction in frontal lobe neurons (Burke, Palmour, Ervin, & Ptito, 2009). PAE has also been linked with a generalized lack of behavioral regulation that inhibits performance on a task requiring attention shifting in monkeys (Schneider, Moore, & Kraemer, 2001); primates with PAE have also been found to exhibit greater irritability and have heightened stress reactivity (Kraemer, Moore, Newman, Barr, & Schneider, 2008).

Inhibitory control (IC) is an important aspect of executive functioning, as deficits in this area in particular may contribute to children's conduct problems. Because IC reflects the ability to inhibit a prepotent response, low levels of IC are related to impulsivity (Logan, Schachar, & Tannock, 1997) and low self-control, which are risk factors for behavior problems (Krueger, Caspi, Moffitt, White, & Stouthamer-Loeber, 1996). PAE's association with low IC may explain why children with PAE are more likely to exhibit disruptive behavior problems, including oppositional and aggressive behavior (Disney et al., 2008; Larkby, Goldschmidt, Hanusa, & Day, 2011), as well as attention-deficit hyperactivity disorder (ADHD) (Mick, Biederman, Faraone, Sayer, & Kleinman, 2002) and learning and memory deficits (Richardson et al., 2002). Specifically, there is evidence to indicate that moderate to heavy drinking in the prenatal period is associated with child conduct problems (Kelly et al., 2009; Colleen M. O'Leary et al., 2010).

1.4 PAE AND GENETIC RISK

In spite of plentiful evidence suggesting that prenatal alcohol consumption is harmful to children, the ability to determine a causal pathway between PAE and poor child outcomes is limited because alcohol use during pregnancy is unlikely to occur independently of other salient risk factors (Day, Cottreau, & Richardson, 1993). That is, because most women are aware that PAE may put their developing child at risk because of widespread public health campaigns since 1973, pregnant women who choose to consume alcohol differ in several important ways from women who abstain, which could potentially confound associations between prenatal alcohol use and child outcomes. Prior researchers have identified predictors of prenatal alcohol use, including higher rates of pre-pregnancy alcohol and substance use (Harrison & Sidebottom, 2009; Skagerström et al., 2011), as well as higher levels of depression (Zuckerman et al., 1989). For this reason, it is important to account for other "third variable" explanations before attributing associations between PAE and child conduct problems entirely to PAE.

One such domain of risk is genetics. Genetically linked tendencies toward addiction and/or depression have been postulated to underlie an expectant mother's propensity to consume alcohol, through which risks associated with PAE might be transmitted to offspring. In support of this theory, in a sample of children at high risk for developing alcoholism based on family history, Hill and colleagues (2000) did not find evidence for a direct relationship between PAE and oppositional or conduct disorders *after* familial risk for alcoholism and prenatal cigarette smoking were taken into account. Other studies suggest that alcohol use includes a heritable component. Swan and colleagues (1990), who conducted a study of adult twin pairs, estimate that the heritability of alcohol use is moderately high, with approximately 60% of the variance in alcohol consumption attributed to genetic effects, although this value decreased to 43% when

adjusted for covariates such as other substance use and psychological characteristics. Although findings from Knopik et al.'s (2005) genetically informed twin study also suggest an association between parental alcoholism and children's behavior problems (ADHD symptoms), much of the genetic risk (i.e., the difference in the similarity of monozygotic versus dizygotic twins) was left unaccounted for by parental history of alcohol abuse or dependence. Another study, albeit one that was not genetically informed, found that PAE had a stronger association with adolescent alcohol problems than did adolescents' family history of alcohol problems (Baer, Barr, Bookstein, Sampson, & Streissguth, 1998), suggesting that PAE contributes unique variance to child outcomes above and beyond family/genetic influence.

Based on the premises of social push theory, which suggest that biological factors should have a greater impact on child problem behavior in more advantaged contexts (Bronfenbrenner & Ceci, 1994; Raine & Venables, 1984; Schonberg & Shaw, 2007), and that those mothers using alcohol prenatally at low or moderate levels might be expected to have fewer contextual stressors than those drinking at high levels prenatally, genetic factors may partially or fully account for associations between PAE and children's problem behavior.

1.5 MODERATING EFFECTS OF CONTEXTUAL RISK ON CHILDREN WITH PAE

Although genetic, prenatal, and postnatal environmental risks in isolation have each been linked to adverse outcomes in children, including child disruptive behavior, several theoretical models have postulated that the presence of cumulative risk across multiple domains is associated with even greater risk (Rutter et al., 1997; Sameroff, Seifer, Baldwin, & Baldwin,

1993). For example, according to diathesis stress and differential susceptibility theory (Belsky & Pluess, 2009; Caspi et al., 2003), environmental stress may potentiate genetic risk to increase the probability of multiple types of child problem behavior. Indeed, much recent work has focused on gene by environment interactions, identifying "risk alleles" such as the low-MAOA activity genotype (Caspi et al., 2002) that, in the presence of environmental adversity, increase children's risk of developing disruptive problem behavior. Less research has focused on the interplay between pre- or perinatal risk and postnatal environment, although there is reason to believe that similar interactions may take place. For example, Beck and Shaw (2005) found that high levels of perinatal complications (e.g., premature birth, maternal conditions such as preeclampsia), combined with family adversity, predicted boys' antisocial behavior at age 10.

Moderate exposure to alcohol in utero may be a similar risk factor that, in combination with environmental risk, may lead to deficits in executive functioning and disruptive problem behavior. Whereas modest direct associations between low and moderate levels of PAE and child conduct problems have been found (D'Onofrio et al., 2007; Sood et al., 2001), associations between PAE and child conduct problems might be amplified in the context of genetic and/or contextual risk. For example, there is evidence that early environmental factors, including a stable home environment, may mitigate some of the potentially deleterious effects of PAE (Jacobson, Jacobson, Sokol, Chiodo, & Corobana, 2004; Streissguth et al., 2004), but also that negative aspects of the early family environment, such as physical or sexual abuse, may exacerbate the effects (Streissguth et al., 2004).

1.6 EFFECTS OF PARENTAL DEPRESSION ON CHILD CONDUCT PROBLEMS AND INHIBITORY CONTROL

Family process variables, including parenting, martial quality, and social support, have all been linked to emerging conduct problems in early childhood (Cummings & Davies, 2002; Dadds & McHugh, 1992; Snyder, Cramer, Afrank, & Patterson, 2005). Parental depression is another critical family process variable and environmental stressor that has consistently been linked to a host of adverse child outcomes, particularly in early childhood (Shaw & Shelleby, 2014). Although parents' depressive symptoms have been associated with many negative outcomes in children, including deficits in social cognitive skills (Jensen, Dumontheil, & Barker, 2014), and negative emotionality in infancy (Melchior et al., 2012), they have most consistently been linked to conduct problems in early childhood, more serious forms of antisocial behavior in later childhood and adolescence (Kim-Cohen, Moffitt, Taylor, Pawlby, & Caspi, 2005; Shaw, Hyde, & Brennan, 2012), and multiple forms of internalizing problems from early childhood through adolescence (Downey & Coyne, 1990; Hammen & Brennan, 2003; Pilowsky et al., 2006). Although the majority of the literature has focused on maternal depression, paternal depression may also have a substantial effect on children's wellbeing, either on its own or by affecting or interacting with maternal depression (Brennan, Hammen, Katz, & Le Brocque, 2002; Lieb, Isensee, Höfler, Pfister, & Wittchen, 2002; Marmorstein & Iacono, 2004; Pemberton et al., 2010). For example, researchers have found that paternal depressive symptoms are independently associated with infant problem behavior (Ramchandani et al., 2013) and toddlers' behavioral difficulties (Kvalevaag et al., 2013), after accounting for maternal depression. There is some evidence to indicate that biased reporting by depressed parents about their children's behavior (Fergusson, Lynskey, & Horwood, 1993) may artificially inflate the relationship

between parental depression and children's conduct problems, so the inclusion of alternative outside informants (e.g., teachers) is crucial.

There is also some evidence that parental depression is associated with suboptimal development of children's self-regulation skills (Silk, Shaw, Skuban, Oland, & Kovacs, 2006); compounding this effect is the possibility that low IC increases children's vulnerability to the effects of parental depression (Gartstein & Fagot, 2003).

Although some of the risk parental depression poses to children may be transmitted genetically (Kim-Cohen et al., 2005), there is evidence that environmental mechanisms are also important (Natsuaki et al., 2014). There is robust evidence suggesting that parental depression has a host of direct and indirect effects on children's well-being, with possible pathways including exposure to the acute and chronic stressors that often accompany depression (e.g., marital conflict), harming parenting quality, compromising decision-making, and modeling maladaptive emotion regulation strategies (Goodman & Gotlib, 1999). Although most studies examining links between parental depression and child problem behavior are confounded methodologically by having biologically related parents raise their children, studies using genetically informed designs also have identified a potent environmental effect of having a parent with depression. For example, a Children of Twins design allows for the detection of environmental effects by comparing the children of monozygotic and dizygotic twins, who share approximately 25% and 12.5% of their genes, respectively, and were raised in different environments. Using this design, Silberg and colleagues (2010) found that having a depressed parent was related to children's conduct problems. Research on the effects of parental depression in adoptive families, where parents raise genetically unrelated children, also indicates

that parental depression is associated with children's disruptive behavior disorders (Tully, Iacono, & McGue, 2008) and externalizing symptoms in toddlers (Pemberton et al., 2010).

1.7 INTERACTIONS BETWEEN PAE AND PARENTAL DEPRESSION ON CHILD CONDUCT PROBLEMS

Although there is reason to believe that parental depression may present significant additional risk for children with PAE, there is scant research that has addressed this issue. One study found evidence consistent with a diathesis-stress model, in which high levels of both PAE and maternal depression were found to increase the likelihood of infant irritability at five months compared to having only one risk factor present (Lemola, Stadlmayr, & Grob, 2009). The work of O'Connor and Kasari (2000) found a similar pattern examining the interaction between PAE and maternal depression in relation to child depressive symptoms among 5- and 6-year-olds (examined concurrently). However, to our knowledge, researchers have not yet studied the interaction between PAE and parental depression in relation to indices of children's self-regulation, including IC and children's conduct problems. This omission exists despite theoretical reasons to suspect that risk for IC deficits and emerging conduct problems associated with PAE would be moderated by the presence of postnatal maternal and/or paternal depression.

2.0 CURRENT STUDY

The current study seeks to enrich our understanding of the association between PAE and risk for early externalizing problems by examining both the independent association and factors that might mediate or moderate it in the early school-age period. Specifically, the current study seeks to test whether the association between PAE and children's externalizing behavior is *moderated* by postnatal parents' depressive symptoms, and/or *mediated* by children's IC, accounting for possible inherited risk for psychopathology (see Figure 1). In addition, we plan to examine how parental depression may amplify associations between PAE and children's IC. As the vast majority of studies examining associations between PAE and children's externalizing problems have utilized biological parents who are also rearing their offspring, making it impossible to disentangle genetic from postnatal environmental influences, the current study will provide a unique perspective by utilizing the genetically-informed Early Growth and Development Study (EGDS). The adoption design of EGDS will permit examination of the independent influence of PAE because biological parents do not raise their own offspring. In addition, birth parent's prenatal use of other substances and birth parent risk factors related to antisocial behavior will also be accounted for in analyses before attributing children's externalizing problems to PAE.

Strengths of the current study include its longitudinal and genetically informed design, which allows hypotheses regarding the independent effects of genetic, prenatal, and postnatal

environmental influence, as well as their interactions, to be evaluated. Other strengths include the inclusion of data from adoptive fathers, an area which has received scant attention in the literature, and data from multiple reporters on children's externalizing symptoms, which helps address the issue of reporter bias.

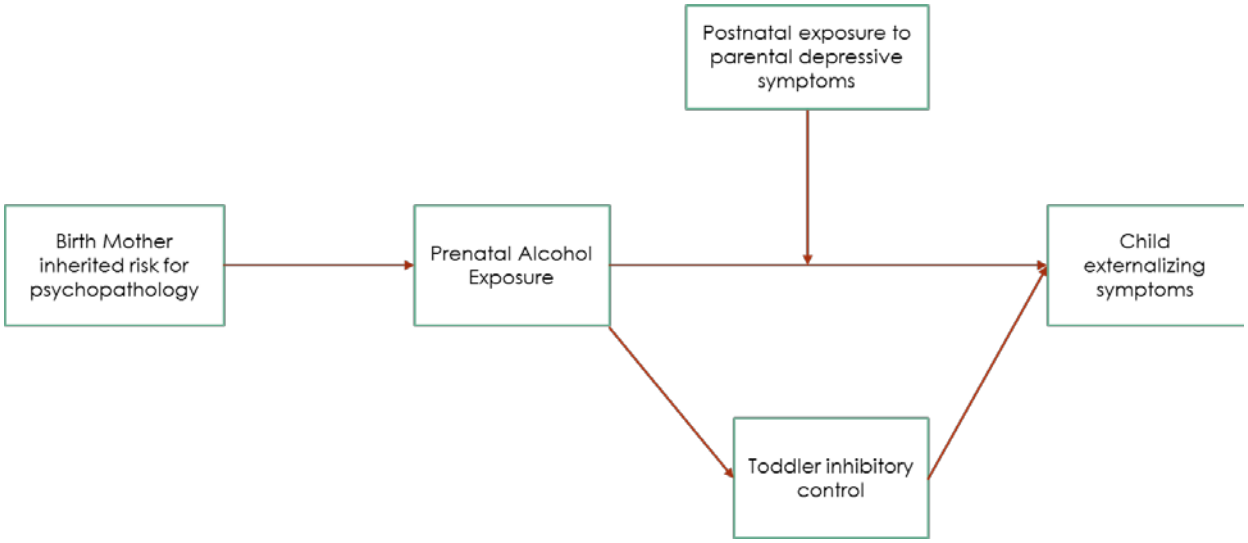

Figure 1. Conceptual model

3.0 HYPOTHESES

The following hypotheses, based on prior research and theory, will be tested:

Hypothesis 1: 1a. Based on theory suggesting that PAE places children in a more vulnerable state for compromised executive functioning (Jacobson & Jacobson, 1994a; Sood et al., 2001), it is expected that the direct association between PAE and children's externalizing problems will be significant at early school-age (age 6).

1b. Based on theory suggesting that parental depression compromises a number of caregiving skills (Goodman & Gotlib, 1999) and past studies suggesting that exposure to elevated levels of parental depressive symptoms is associated with child conduct problems after accounting for genetic risk (Natsuaki et al., 2014), significant associations are expected between adoptive parents' depression (measured at child age 9 and 18 months) and child externalizing behavior (age 6).

1c. Based on prior research suggesting that PAE increases children's vulnerability to suboptimal rearing environments (Jacobson et al., 2004) , it is anticipated that adoptive parents' depressive symptoms will moderate the relationship between PAE and children's externalizing problems, such that associations between PAE and children's externalizing problems are expected to be amplified in the context of high levels of adoptive parents' depressive symptoms (see Figure 2).

*Hypothesis 2:*2a. Based on research suggesting that externalizing disorders are highly heritable (Hicks, Krueger, Iacono, McGue, & Patrick, 2004) and that not only family history of externalizing but also internalizing problems predict externalizing symptoms in childhood (Kim-Cohen et al., 2005), it is predicted that there will be a significant association between birth mother (BM) inherited risk for psychopathology and child externalizing problems.

2b. It is hypothesized that BM inherited risk for psychopathology will moderate the relationship between PAE and child externalizing problems, based on theory suggesting that the combination of these two vulnerabilities will increase the probability of children's externalizing problems above and beyond the independent risk conferred by each on its own (Brookes et al., 2006). Specifically, it is expected that associations between PAE and children's externalizing problems will be amplified in the context of BM history of psychopathology (see Figure 3).

Hypothesis 3: 3a. Based on theory suggesting that PAE places children at greater vulnerability to compromised executive functioning (Kodituwakku et al., 2001), it is predicted that there will be a significant association between PAE and low IC in early childhood (age 27 months).

3b. As both theory (Nigg, 2000) and research (Raaijmakers et al., 2008) suggest that low IC in early childhood is linked to a number of disruptive child behaviors (e.g., reactive aggression, low impulse control), it is expected that low IC in the toddler period (age 27 months) will be related to higher levels of child conduct problems during the early school-age period (age 6 years).

3c. It is expected that the association between PAE and children's externalizing problems at school-age will be mediated by children's IC during the toddler period.

3d. Based on research suggesting that adoptive parents' depression, even at subclinical levels, will amplify biological and environmental risk, it is expected that higher levels of adoptive parents' depression will moderate both of the aforementioned pathways in the model, strengthening the associations between PAE and IC, and between IC and child externalizing (see Figure 4). It is anticipated that this pattern will hold even after accounting for BM inherited risk for psychopathology.

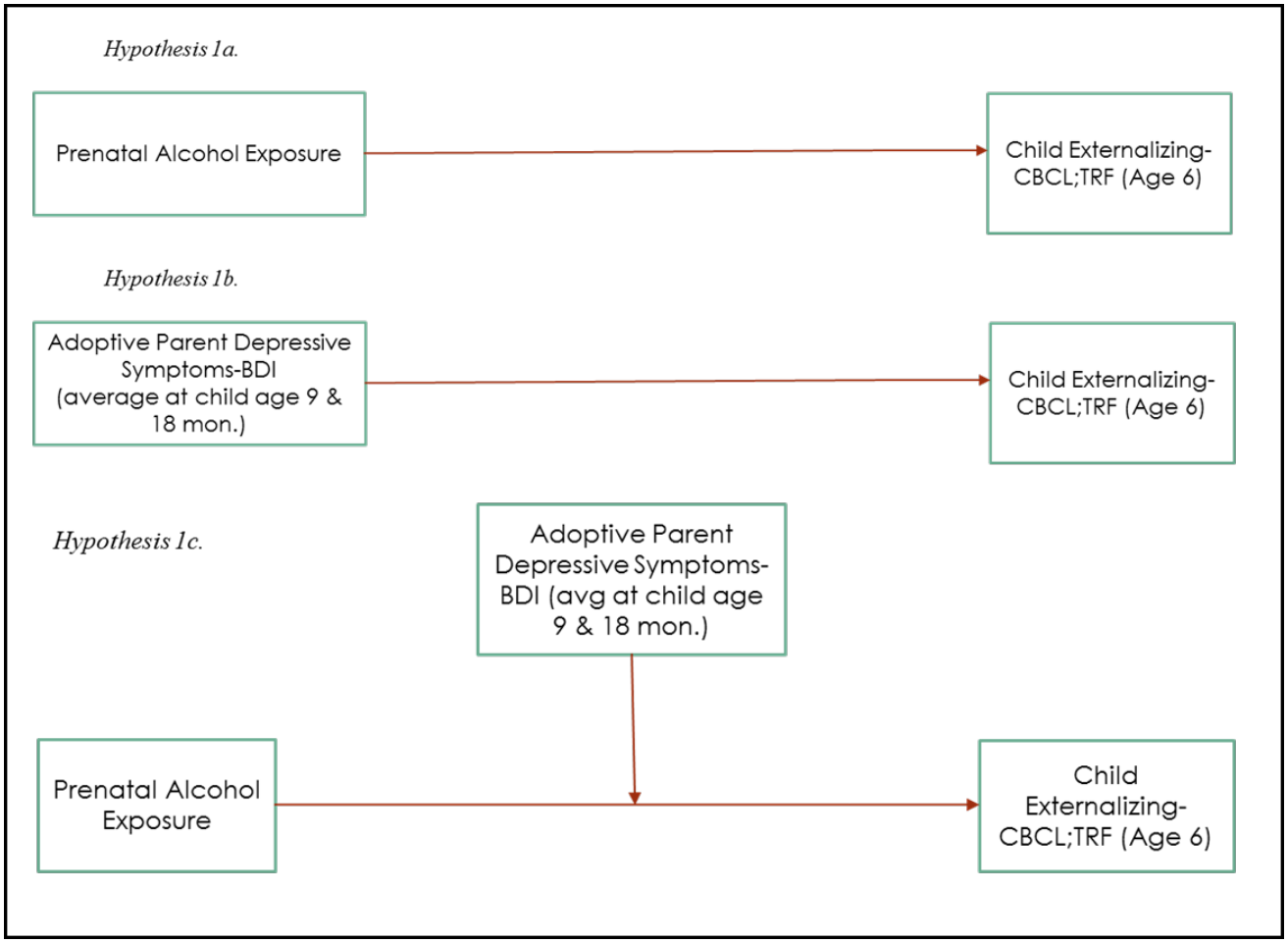

Figure 2. Hypothesis 1

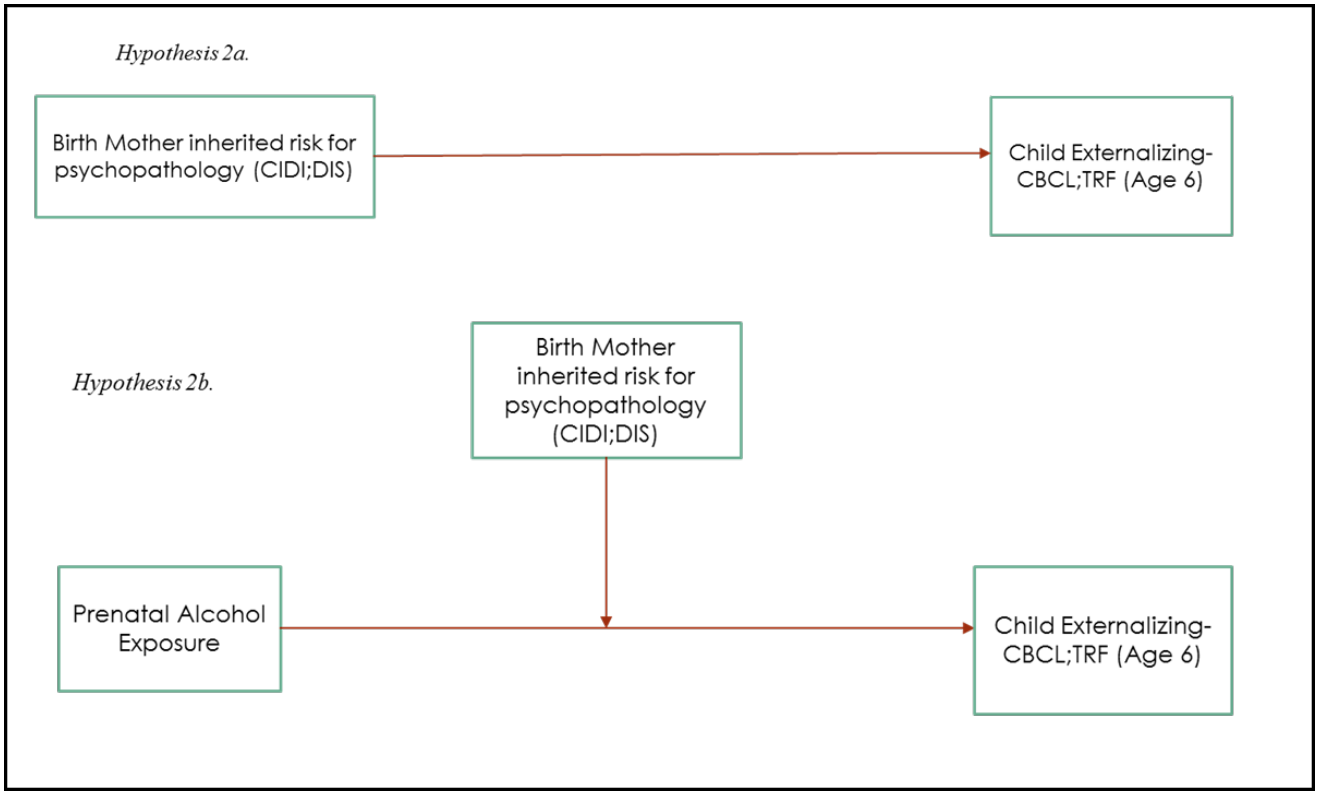

Figure 3. Hypothesis 2

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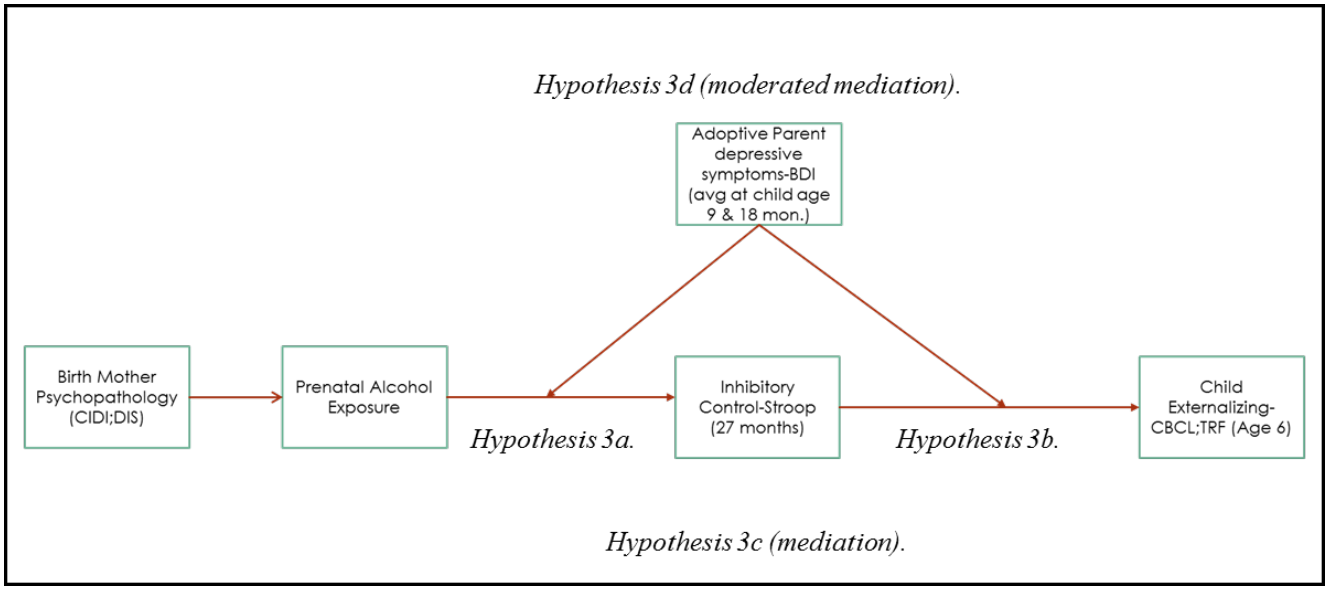

Figure 4. Hypothesis 3

4.0 METHOD

4.1 PARTICIPANTS

The EGDS, comprised of 561 adoptive families, is an ongoing, multisite, longitudinal sample of adopted children, adoptive parents, and birth parents (Leve et al., 2013). Using a rolling recruitment procedure, families were enrolled into the study from 2003 to 2010 by 45 adoption agencies in 15 states located in the Mid-Atlantic, West-Southwest, and Pacific Northwest regions of the United States. The participating adoption agencies were representative of the many adoption philosophies available in the U.S., with public, private, religious, and secular agencies that offered both open and closed adoptions. Families were eligible for study inclusion if they met the following criteria: a) the adoption was domestic, b) the infant was placed within 3 months postpartum, c) the infant was placed with a family that was biologically unrelated to the adopted child, d) there were no known major medical conditions, and e) the adoptive parents could comprehend English at an 8th grade reading level. There were minor differences between families who participated in the study and those who did not: participating birth mothers and fathers were slightly younger than non-participating birth parents; participating adoptive mothers were also slightly younger and more educated than nonparticipants. Families were recruited in two cohorts, with 361 families in the first cohort (recruited between 2003 and

2006), and 200 families in the second cohort (recruited between 2008 and 2010). There were some differences in the data collection process for each cohort that are described where relevant.

In terms of sample demographics, the majority of birth mothers were Caucasian (70.1%; African American = 13.3%; Hispanic/Latino = 6.7%; Multi-ethnic = 4.9%; Other = 5.0%). Their average age at the time of delivery was 24.4 years (*SD* = 6.0), and their median annual household income was less than $15,000. Although 27.3% of birth mothers did not complete high school, the majority (52.2%) reported that high school or a high school equivalency degree was the highest level of education they had completed. Less than one third (30.6%) of birth mothers reported that they were married at the time of the adopted child's birth. Most birth mothers received adequate prenatal care, with a mean number of prenatal care visits of 11.17 (*SD* = 7.95). The Office on Women's Health in the U.S. Department of Health and Human Services recommends a total of 14 routine prenatal care visits during pregnancy, with adequate prenatal care defined as attending at least 80% of the expected 14 prenatal visits.

Although the majority of adoptive parents were heterosexual couples, there were 41 same-sex parent families. The majority of adoptive mothers were Caucasian (91.8%; African American = 3.9%; Hispanic/Latino = 2.0%; Multi-ethnic = 0.9%; Other = 1.4%). Adoptive fathers were demographically similar, with the majority identifying as Caucasian (90.4%; African American = 4.9%; Hispanic/Latino = 1.6%; Multi-ethnic = 1.1%; Other = 2.0%). Adoptive mothers' average age at the child's birth was 37.4 years (*SD* = 5.6), and adoptive fathers' average age was 38.3 (*SD* = 5.8). Their median annual household income was greater than $100,000. Most mothers (78.8%) and fathers (72.5%) had at least a 4-year college degree, and 91.1% were married at the time of the adopted child's birth.

Just over half of the adopted children were male (57.2%). A slight majority of adopted children were Caucasian (55.6%, Multi-racial = 19.3%; African American = 13%; Latino = 10.9%; Other = 1.2%), and their median age at adoption was 2 days (M = 6.2, SD = 12.45; range = 0-91 days). Approximately 10% of infants were born premature (n = 59). Please refer to Leve and colleagues (2013) for additional details regarding the sample and study design.

4.2 PROCEDURE

In-person assessments were conducted with adoptive parents when children were 9, 18, 27 months and 6 years old, and with biological mothers at approximately 4- and 18-months postpartum. For both biological and adoptive parents, visits were conducted at a location convenient for the interviewees, usually in their homes. Families lived in 46 states in the U.S. and Washington, D.C., as well as in seven countries outside of the U.S. Retention rates for adoptive families were as follows: 98.8% at 9 months, 97.5% at 18 months, 94.7% at 27 months, and 82.5% at age 6 years.

4.3 MEASURES

4.3.1 Prenatal substance exposure.

At the 4-month postpartum assessment, the Life History Calendar (LHC) method (Caspi et al., 1996) was used to assess birth mothers' use of substances, including alcohol, cigarettes,

and illicit drugs, during their pregnancy. The LHC is a well-validated method for obtaining retrospective data, which were collected four months following the child's birth. To assist birth mothers in recalling their perinatal substance use, interviewers worked with each participant to create a timeline of events that occurred during the past year and during their pregnancy so that they could refer to this timeline as they completed the LHC. Cohort 1 birth mothers (n = 348) were asked about their alcohol use across their entire pregnancy, generating one total score of drinking throughout pregnancy and average number of drinks per week *over the nine months*, whereas Cohort 2 birth mothers (n = 192) were asked the same questions about their alcohol use separated by trimester, with scores derived by trimester and across the nine months. Because the frequency of reported drinking during pregnancy was relatively low for both cohorts (see Tables 1 and 2), particularly during the latter two trimesters for Cohort 2, PAE was dichotomized based on whether any drinking during any trimester of pregnancy occurred.

Table 1. Alcohol use during pregnancy in cohort 1 biological mothers

Frequency	N (% of cohort)
Any use during pregnancy	84 (24.1%)
Rarely	62 (17.8%)
Infrequently	14 (4.0%)
Somewhat regularly	5 (1.4%)
Regularly	2 (0.6%)
Average drinks/week	
0	23 (6.6%)
1-2	47 (13.5%)
3-6	15 (4.3%)
7+	2 (0.6%)

Table 2. Alcohol use during pregnancy in cohort 2 biological mothers, by trimester

	1st trimester N (% of cohort)	2nd trimester N (%)	3rd trimester N (%)	Entire pregnancy N (%)
Frequency				--
Any use	40 (20.8%)	11 (5.7%)	5 (2.6%)	43 (22.4%)
Rarely	21 (10.9%)	7 (3.6%)	4 (2.1%)	--
Infrequently	5 (2.6%)	3 (1.6%)	1 (0.5%)	--
Somewhat regularly	9 (4.7%)	1 (0.5%)	0	--
Regularly	4 (2.1%)	0	0	--
Average drinks/week				--
0	2	1	1	--
1-2	18	6	3	--
3-6	11	3	1	--
7+	5	1	0	--

4.3.2 Birth mother inherited risk for psychopathology.

The BM inherited risk for psychopathology variable was used to account for BMs' history of both externalizing and internalizing problems. First, the antisocial personality disorder (ASPD) and conduct disorder (CD) portions of the computerized-Diagnostic Interview Schedule (DIS; Blouin, Perez, & Blouin, 1988) were used to capture BMs' externalizing problems. The DIS is a structured interview that is used to diagnose interviewees using criteria from the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV*, 2000), assessing symptoms in the past 12 months and also lifetime diagnoses. The computerized-DIS was administered to BMs at 18 months postpartum. The test-retest ratings in the literature have been found to be acceptable; $\kappa = .49$ (Horton, Compton, & Cottler, 1998). Second, the Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004), also administered 18 months postpartum, was used to assess BMs' history of internalizing problems, including agoraphobia, separation anxiety, dysthymia, generalized anxiety disorder, major depression, panic disorder, recurrent brief depression, and social phobia. The CIDI is a standardized interview that uses DSM-IV

criteria to assess for the presence of psychiatric disorders. Scores on both the DIS (externalizing symptoms) and on the CIDI (internalizing symptoms) were first separately converted to z-scores to give internalizing and externalizing symptoms equal weight, as the CIDI assessed a greater number of possible internalizing symptoms than the DIS did for externalizing symptoms. After the scales were converted to z-scores, the externalizing and internalizing symptom counts were combined into a single composite score.

4.3.3 Adoptive parent depressive symptoms.

The Beck Depression Inventory (BDI; Beck, Steer, & Brown, 1996) was used to measure adoptive parent depressive symptoms in both adoptive mothers (AM) and fathers (AF). The BDI is a well-established and widely used self-report measure of depressive symptoms. Each item asks participants to rate, on a scale from 0 to 3, the extent to which they had experienced specific depressive symptoms in the past week. Although the original version consists of 21 items, the version used for the current study contained 20 items, as the item assessing suicidal ideation was not included. The AM and AF depressive symptoms variables were an average of the BDI scores of each parent from the assessments at 9 and 18 months, as depressive symptoms at these two time points are relatively stable in the current sample. If scores were missing at one time point (n = 60 for AMs and n = 92 for AFs), the BDI score from the one assessment with valid data was used in analyses. Internal consistencies ranged from α =.71-.79 for AMs and α =.75-.81 for AFs across the 9- and 18-month assessments.

4.3.4 Child inhibitory control.

The Shape Stroop task (Kochanska, Murray, & Harlan, 2000), a modified version of Gerstadt, Hong, and Diamond's Stroop-like Day-Night Test (1994), was used as a measure of child IC at 27 months. The Shape Stroop task, which was revised to be appropriate for two-year-olds, required children to inhibit a prepotent response to measure executive functioning and specifically, IC. During the task, an interviewer presented the child with three large and three small pictures of the same fruits. After reviewing the names of the fruits and the meaning of "big" and "little," the examiner then showed the child three new pictures, each showing a small fruit embedded in a different large fruit (e.g., a small banana pasted on a large apple). The examiner then requested the child point to each of the little fruits (e.g., "show me the little banana"), as the prepotent response for young children is to point to the larger item. The activity was subsequently repeated in a trial in which the interview presented the child with pictures of big and little animals instead of fruits (e.g., bunny, dog, teddy bear). Trials were each scored on a scale from 1 to 3, where a score of 1 was given for an ambiguous or incorrect response on the item *and* size of the fruit or animal, a score of 2 was given for a correct item response but incorrect size, and a score of 3 was given for a correct response on both item and size. Items were averaged to compute the scale score (Cronbach's $\alpha = .86$). The intraclass correlation between raters was .83.

4.3.5 Child externalizing symptoms.

Externalizing factor of the parent-report version of the Child Behavior Checklist (CBCL) was used to assess child behavior problems at age 6. The preschool version (CBCL/1½-5;

Achenbach & Rescorla, 2000) was used for cohort 1 and the school-age version (CBCL/6-18; Achenbach & Rescorla, 2001) was administered to parents in cohort 2. The Externalizing factor from the CBCL/1½-5 has 24 items and demonstrated adequate internal consistency in the current sample across informant (AM $\alpha = .91$, AF $\alpha = .93$ in current sample). The Externalizing factor from the CBCL/6-18 contains 35 items and also showed satisfactory internal consistency across informant (AM $\alpha = .90$, AF $\alpha = .86$ in current sample). In the current sample, CBCL parent data are available from 70.6% ($n = 396$) adoptive mothers and 64.0% ($n = 359$) of adoptive fathers.

The Externalizing factor from the Teacher Report Form (TRF; Achenbach & Rescorla, 2001) was also used. The 35-item TRF is a well-validated measure of child disruptive behavior in the school setting; each child's primary teacher was asked to complete the form at child age 6. The broad-band Externalizing factor also showed satisfactory internal consistency in the current sample ($\alpha = .95$ and $\alpha = 97$, for cohorts 1 and 2 respectively). Data from teachers are available for 48.3% of the full sample ($n = 271$). The most commonly cited reasons for not having teacher data were as follows: declined or nonresponsive ($n = 108$), parent refused to grant permission to collect teacher data ($n = 47$), teacher consent not provided ($n = 11$), and other (e.g., child was homeschooled, teacher did not speak English) ($n = 24$).

Raw scores of the CBCL Externalizing factor (parent report) were converted into Z scores for all analyses to create comparable across the different versions of the CBCL used for cohorts 1 and 2. As the same version of the TRF was used for both cohorts, raw scores for teacher-reported child externalizing were used in analyses to maximize variability.

4.3.6 Covariates.

Several covariates were included in testing hypotheses to account for "third variable" explanations of associations between independent variables of interest and child IC and externalizing problems. First, openness of adoption was included as a covariate to account for the possibility that openness in the adoption process could lead to greater similarities between birth and adoptive parents. Birth mothers and adoptive parents reported on their individual perceptions of the openness of the adoption on a scale from 1 (very closed) to 7 (very open) (Ge et al., 2008). The mean of the standardized scores for birth mother, adoptive mother, and adoptive father on this scale was used. Second, exposure to tobacco and exposure to other illicit substances were included as covariates to rule out the possibility that significant associations between PAE and child outcomes are not accounted for by exposure to these other substances. Similar to the alcohol exposure variable, both the tobacco and other substance exposure variables were coded dichotomously. Exposure to other substances included the following: amphetamines, prescription painkillers used illegally, inhalants, marijuana, cocaine, hallucinogens, heroin, and methadone. Exposure to other substances was considered to have occurred if birth mothers endorsed use of *any* of the above substances at any level during pregnancy. Obstetric complications was also included as a covariate because of its possible associations with PAE, and both child IC and externalizing problems. The obstetric complications variable was calculated using a sum of the risk scores that were calculated for each of the following: pregnancy complications (e.g., weight loss, preeclampsia), exposure to toxins (e.g., lead), and neonatal complications (e.g., premature birth, low Apgar score). Scores on these variables were based on decisions made using the McNeil-Sjostrom scale for obstetric complications (McNeil, Cantor-Graae, & Sjöström, 1994); please refer to Marceau and

colleagues (2013) for a more detailed description of how these calculations were made in the current study. Information included in the obstetric complications variable was primarily derived from birth mothers' medical records. Adoptive family income was also included as a covariate.

5.0 DATA ANALYTIC STRATEGY

For analyses that included the child externalizing variable (all hypotheses except 3a), both AM and AF reports were used and aggregated for the adoptive parent report variable based on their moderately high correlation across AMs and AFs (r = .57, p < .001 for cohort 1; r = .62, p < .001 for cohort 2). Although teacher and parent reports were moderately correlated, in each analysis separate models were computed for teacher and parent reports of child externalizing because of our interest in examining child behavior across context (AM and AF composite and teacher report of externalizing are correlated at r = .43, p < .001 for cohort 1; r = .48. p < .001 for cohort 2). For all analyses (hypotheses 1b, 1c, 3d) that included parental depressive symptoms as a variable, analyses were repeated substituting AF depressive symptoms for AM depressive symptoms.

Overall, hypothesis testing proceeded from examining univariate associations between independent and moderating variables (i.e., PAE, adoptive parent depressive symptoms) and child externalizing problems to examining moderating analyses using hierarchical multiple regression analyses, both in SPSS. Following moderation analyses, it was planned that SEM (in MPlus) would be used to examine moderated mediation, specifically whether AP depressive symptoms moderates the potential mediating function of IC between PAE and child externalizing.

6.0 RESULTS

6.1 DESCRIPTIVE ANALYSES

Means, standard deviations, and ranges for all study variables are presented in Table 3. T scores of the CBCL and TRF Externalizing factor are presented in Table 3 to facilitate interpretation and comparisons with other samples. The mean child externalizing scores for both parent and teacher report were slightly lower than averages of 50 found in national normative samples, with 4.4% and 7.2% of the sample reported by parents and teachers, respectively, to be above the clinical cutoff of T = 65 for externalizing problems (Achenbach, 1991; Achenbach & Edelbrock, 1986). Low rates of depressive symptoms were reported on the BDI by both adoptive parents (*M* = 2.98 and 1.81 for adoptive mothers and fathers, respectively), as scores of 9 or greater are indicative of "modest depression" (Beck, Steer, & Carbin, 1988). Although a total symptom count was used in all analyses, percentages of birth mothers meeting diagnostic criteria for psychiatric disorders is presented in Table 3 for comparison with other samples. Almost half of birth mothers qualified for a diagnosis of antisocial personality disorder and/or conduct disorder (i.e., 44%), and nearly a third of birth mothers had current or a history of depression.

Analyses were performed to determine whether children or families who did not complete the assessment at age 6 were different on earlier-collected study variables from those

who did. Children in families who completed the age 6 assessment had significantly lower levels of IC as measured by the Stroop task, t (504) = -2.39, $p < .05$. There were no other statistically significant differences between the two groups on any of the other variables in the current study. Children for whom teacher report data were available did not differ from those without teacher report data on the following variables: sex, family income, IC, or parent report of externalizing behavior. Analyses were also performed to evaluate whether there were any differences between the two cohorts on any study variables. There were two statistically significant differences between cohorts 1 and 2: adoptive mothers in cohort 2 reported higher incomes than those in cohort 1, t (489) = -2.09, $p < .05$, and children in cohort 2 were reported by their teachers to have greater levels of externalizing problems than those in cohort 2 at age 6, t (269) = -2.37, $p < .05$.

Correlations among study variables are displayed in Table 4. BM psychopathology was significantly correlated with tobacco and other substance use during pregnancy; the correlation between BM psychopathology and prenatal alcohol use approached significance (r = .09, p = .06). Adoptive family income and prenatal exposure to tobacco were both significantly correlated with parent but not teacher reports of child externalizing behavior. PAE, but not exposure to tobacco or other substances, was correlated with obstetric complications.

Table 3. Descriptives of Study Variables

Variable	Mean (SD) or % positive/endorsed	Range
Adoptive family annual income (n = 491)	$126,912 (104,959)	7000-1,500,000
Prenatal tobacco exposure (n = 539)	41.6%	0-1

Prenatal exposure to other substances (*n* = 539)	26.7%	0-1
Obstetric complications (*n* = 561)	6.79 (5.3)	0-28
Prenatal Alcohol Exposure (*n* = 539)	23.3%	0-1
Birth Mother (BM) ASPD/CD lifetime diagnosis (DIS) (*n* = 487)	44.3%	0-1
BM MDD lifetime diagnosis (CIDI) (*n* = 512)	28.7%	0-1
BM GAD lifetime diagnosis (CIDI) (*n* = 512)	7.2%	0-1
Adoptive Mother depressive symptoms, child age 9 & 18 mon. (BDI) (*n* = 536)	3.64 (3.3)	0-17.5
Adoptive Father depressive symptoms, child age 9 & 18 mon. (BDI) (*n* = 537)	2.98 (3.1)	0-24
Child Inhibitory Control, age 27 months (Stroop task) (*n* = 506)	1.81 (0.6)	0.5-3.0
Child externalizing T score, age 6 (CBCL; AM/AF average report) (*n* = 411)	48.44 (9.3)	28-84.5
Child externalizing T score, age 6 (TRF; teacher report) (*n* = 271)	49.05 (10.1)	36-84

Table 4. Correlation Matrix

Variable	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Adoptive family income	--											
2. Prenatal tobacco exposure	-.02	--										
3. Prenatal exposure to other substances	-.04	.32*	--									
4. Adoption openness	.02	.00	.05	--								
5. Obstetric complications	.06	.05	.07†	.12*	--							
6. Prenatal Alcohol Exposure	-.07	.23*	.22*	.12*	.09*	--						
7. Birth mother (BM) psychopathology symptoms	-.03	.32*	.29*	-.03	.04	.09†	--					
8. Adoptive mother (AM) depressive symptoms	-.08	.02	-.02	.03	-.05	-.01	-.03	--				
9. Adoptive father (AF) depressive symptoms	-.08†	-.04	.02	-.03	.02	.03	-.02	.25*	--			
10. Child Inhibitory Control (Stroop task)	.03	.03	.01	.16*	-.03	.06	-.06	-.04	-.01	--		
11. Child externalizing Z-score (parent report)	-.12*	.12*	.02	-.03	.01	.01	.11*	.13*	.12*	-.12*	--	
12. Child externalizing raw score (teacher report)	-.01	.01	.00	-.03	.02	.03	.00	.13*	.02	-.20*	.44*	--

*Denotes significance at $p < .05$

†Denotes significance at p <. 10

6.2 DIRECT EFFECTS OF PAE AND AP DEPRESSIVE SYMPTOMS ON EXTERNALIZING PROBLEMS

To test hypothesis 1a that PAE would be directly related to child externalizing problems at age 6, two hierarchical multiple regressions were conducted in which the covariates of prenatal tobacco exposure, other substance exposure, adoptive family income, adoption openness, and obstetric complications were entered first, followed by PAE, in predicting AP and teacher reports of externalizing problems. There was no evidence for a significant association between PAE and externalizing problems ($\beta = .00$, *ns* and $\beta = .02$, *ns* for parent and teacher report, respectively). Similarly, to test hypothesis 1b that AM and AF depressive symptoms would each have a direct association with child externalizing, a total of four multiple regressions were conducted in which the same covariates as used for testing hypothesis 1a were entered before entering AM or AF depressive symptoms. For the four regression equations, alternating the use of AM or AF depressive symptoms and parent or teacher report of child externalizing, both AM and AF depressive symptoms were independently associated with children's externalizing problems using parent reports ($\beta = .12$, $p < .05$ and $\beta = .14$, $p < .05$ for AM and AF depressive symptoms, respectively). Using teacher report, the association between AM depressive symptoms and child externalizing approached significance ($\beta = .13$, $p = .053$), but the association between AF depressive symptoms and externalizing was not significant ($\beta = .04$, *ns*).

Because we found in separate regression equations that both AM and AF depressive symptoms contributed to child externalizing problems, two additional exploratory regressions were conducted to examine their independent contributions and potential for their interaction to account for additional variance with respect to externalizing problems. Thus, in separate equations using parent and teacher report of child externalizing problems, both AM and AF

depressive symptoms were included in the same equation, followed by their interaction term. Using parent report, both AM and AF depressive symptoms were found to have independent effects on children's externalizing (see Table 5). The interaction between AM and AF depressive symptoms was not significant.

Table 5. Regression results: Influence of adoptive mother (AM) depressive symptoms on child externalizing (parent/teacher report), as moderated by AF depressive symptoms

	CBCL Externalizing Parent Report			**TRF Externalizing Teacher Report**		
	B(SE)	**β**	**R^2 Change**	**B(SE)**	**β**	**R^2 Change**
Prenatal alcohol exposure	.06(.12)	.03		1.48(1.99)	.05	
Prenatal tobacco exposure	.22(.11)	.12*		-.30(1.80)	-.01	
Other substance exposure	-.07(.12)	-.03		-.97(2.05)	-.04	
Adoptive family income	.00(.00)	-.09†		.00(.01)	.01	
Openness	.00(.06)	.00		-.20(.90)	-.02	
Obstetric complications	.01(.01)	.03	.03	.08(.15)	.04	.00
Adoptive mother (AM) BDI	.05(.02)	.19*	.02*	.40(.25)	.12	.02
Adoptive father (AF) BDI	.07(.03)	.22*	.01*	.06(.26)	.02	.00
AM BDI x AF BDI	-.01(.01)	-.17	.01	.10(.07)	.11	.01
F	2.47*			F	.81	
R	.25			R	.19	
R^2	.06			R^2	.03	

*Denotes significance at $p < .05$
†Denotes significance at $p <. 10$

6.3 INTERACTIVE EFFECTS OF PAE AND AP DEPRESSIVE SYMPTOMS

To test hypothesis 1c that AP depressive symptoms would moderate the risk of externalizing problems in children with PAE, a series of hierarchical multiple regressions were conducted in which the same previously described covariates were entered first, then PAE and AM (or AF) depressive symptoms, followed by their interaction term. As shown in Table 6, neither AM or AF depressive symptoms was found to moderate the strength of the association between PAE and children's externalizing problems according to parent or teacher reports (values in Table 5 reflect the results of the regression equations that included interaction terms; the beta weights presented in the sections above are from the equations with only the direct, independent effects of PAE and AP depressive symptoms, respectively). In contrast to the model testing hypothesis 1b, in which the direct effect of maternal depressive symptoms on teacher-reported child externalizing was marginally significant, in this model, the main effect of AM depressive symptoms on teacher-reported externalizing was significant ($\beta = .18$, $p < .05$).

Table 6. Regression results; hypothesis 1: Influence of prenatal alcohol exposure (PAE) on child externalizing (parent/teacher report), as moderated by maternal/paternal depressive symptoms

	Maternal Depressive Symptoms						Paternal Depressive Symptoms					
	CBCL Externalizing Parent Report			TRF Externalizing Teacher Report				CBCL Externalizing Parent Report			TRF Externalizing Teacher Report	
	B(SE)	B	R^2 Change	B(SE)	β	R^2 Change		B(SE)	β	R^2 Change	B(SE)	β	R^2 Change
Prenatal tobacco exposure	.21(.11)	.11†		-.08(1.79)	.00		Prenatal tobacco exposure	.21(.11)	.11†		-.23(1.82)	-.01	
Other substance exposure	-.06(.12)	-.03		-1.01(2.05)	-.04		Other substance exposure	-.06(.12)	-.03		-.95(2.06)	-.04	
Adoptive family income	.00(.00)	-.11*		-.00(.01)	.00		Adoptive family income	.00(.00)	-.10†		.00(.01)	-.01	
Openness	-.02(.06)	-.02		.06(.90)	.01		Openness	.00(.06)	.00		.08(.15)	.04	
Obstetric complications	.01(.01)	.03	.03†	.11(.15)	.05	.00	Obstetric complications	.01(.01)	.03	.03†	.08(.16)	.04	.01
Prenatal alcohol exposure (PAE)	.19(.18)	.09	.00	3.81 (2.82)	.14	.00	Prenatal alcohol exposure (PAE)	.22 (.18)	.10	.00	3.55(2.81)	.13	.00
Adoptive mother (AM) BDI	.04(.02)	.15*	.02*	.61(.26)	.18*	.02*	Adoptive father (AF) BDI	.06(.02)	.17*	.02*	.31(.28)	.08	.00
PAE x AM BDI	-.04(.04)	-.08	.00	-.79(.67)	-.13	.01	PAE x AF BDI	-.06(.04)	-.11	.01	-.81(.62)	-.14	.01
F	2.00*			F	.77		F	2.26*			F	.95	
R	.21			R	.17		R	.23			R	.11	
R^2	.05			R^2	.03		R^2	.05			R^2	.01	

*Denotes significance at $p < .05$

†Denotes significance at p <. 10

6.4 DIRECT EFFECT OF BM PSYCHOPATHOLOGY

To test hypothesis 2a that BM risk for psychopathology would be directly related to children's externalizing problems, a series of hierarchical multiple regressions were again conducted with the same aforementioned covariates. Although the bivariate correlation between BM psychopathology and parent report of children's externalizing problems was significant (r = .11, p < .05), this significant association became nonsignificant within the context of the multivariate regression equation (β = .10, p = .11). The association between BM psychopathology and teacher report of child externalizing problems was not significant when examined within a univariate or multivariate framework (r = .01, *ns*).

6.5 INTERACTIVE EFFECTS OF PAE AND BM PSYCHOPATHOLOGY

To test hypothesis 2b that BM risk for psychopathology moderates the association between PAE and child externalizing problems, hierarchical multiple regressions were conducted with the aforementioned covariates. The PAE x BM psychopathology interaction term was not significant in predicting parent or teacher reports of child externalizing problems (see Table 7).

Table 7. Regression results; hypothesis 2: Influence of prenatal alcohol exposure (PAE) on child externalizing (parent/teacher report), as moderated by birth mother psychopathology

	CBCL Externalizing Parent Report			TRF Externalizing Teacher Report		
	B(SE)	**β**	**R^2 Change**	**B(SE)**	**β**	**R^2 Change**
Prenatal tobacco exposure	.20(.12)	.11†		-.42(1.88)	-.02	
Other substance exposure	-.07(.13)	-.03		-1.27 (2.13)	-.05	
Adoptive family income	.00(.00)	-.13*		.00(.01)	-.01	
Openness	-.03(.06)	-.03		-.01(.91)	.00	
Obstetric complications	.01(.01)	.03	.03†	.06(.15)	.03	.02
Prenatal alcohol exposure (PAE)	-.08(.13)	-.04	.00	1.61(2.04)	.06	.00
Birth mother (BM) psychopathology	.03(.04)	.05	.01	-.16(.59)	-.02	.00
PAE x BM psychopathology	.10(.08)	.08	.01	.56(1.25)	.04	.00
F	1.86†			F	.17	
R	.22			R	.08	
R^2	.05			R^2	.01	

*Denotes significance at $p < .05$

†Denotes significance at $p < .10$

6.6 DIRECT EFFECTS OF PAE ON IC AND IC ON CHILDREN'S EXTERNALIZING

To test hypothesis 3 that associations between PAE and child externalizing would be mediated by child IC, a series of hierarchical regressions were computed to examine whether

PAE would be related to IC (A → B path; hypothesis 3a), whether IC would be related to children's externalizing (B → C; hypothesis 3b), and finally whether direct associations between PAE and child externalizing problems would be mediated by IC (hypothesis 3c). After accounting for covariates, PAE and IC were not found to be significantly related to one another, but as expected, IC and children's externalizing problems were inversely associated ($\beta = -.13$, $p < .05$, and $\beta = -.23$, $p < .01$, for parent and teacher report, respectively) after accounting for covariates. As reported above, PAE was not directly related to either parent or teacher reports of early school-age externalizing problems or child IC; hence, it was not possible to test whether IC mediated the association between PAE and child externalizing problems.

6.7 INTERACTIVE EFFECTS OF PAE AND AP DEPRESSIVE SYMPTOMS ON CHILDREN'S IC

Finally, to test hypothesis 3d that AP depressive symptoms would moderate the pathway between PAE and children's IC, four hierarchical multiple regressions were computed, alternating between AM and AF depressive symptoms and parent and teacher reports of child externalizing problems. The same covariates were used as described above, with the addition of the BM psychopathology variable, to integrate the genetic risk component into the final model. No significant interaction effect was found between PAE and depressive symptoms in relation to IC for either adoptive parent's depressive symptoms (see Table 8).

Table 8. Regression results; hypothesis 3: Influence of prenatal alcohol exposure (PAE) on child inhibitory control, as moderated by maternal/paternal depressive symptoms

Maternal Depressive Symptoms				Paternal Depressive Symptoms			
Inhibitory Control (Stroop)							
	B(SE)	β	R^2 Change		B(SE)	β	R^2 Change
Birth mother (BM) psychopathology	-.01(.02)	-.03		BM psychopathology	-.01(.02)	-.03	
Prenatal tobacco exposure	.06(.07)	.05		Prenatal tobacco exposure	.07(.07)	.04	
Other substance exposure	.06(.08)	.05		Other substance exposure	.06(.08)	.04	
Adoptive family income	.00(.00)	-.01		Adoptive family income	.00(.00)	-.01	
Openness	.13(.03)	.21*		Openness	.13(.03)	.21*	
Obstetric complications	-.01(.01)	-.10†	.05*	Obstetric	-.01(.01)	-.10	.05
Prenatal alcohol exposure (PAE)	-.11(.11)	-.08	.00	PAE	-.11(.10)	-.08	.00
Adoptive mother (AM) BDI	.00(.01)	-.01	.00	Adoptive father (AF) BDI	-.01(.01)	-.03	.00
PAE x AM BDI	.01(.02)	.05	.00	PAE x AF BDI	.02(.02)	.06	.00
F	2.28*			F	2.30*		
R	.24			R	.24		
R^2	.06			R^2	.06		

*Denotes significance at $p < .05$

†Denotes significance at $p <. 10$

6.8 INTERACTIVE EFFECTS OF IC AND AP DEPRESSIVE SYMPTOMS ON CHILDREN'S EXTERNALIZING PROBLEMS

To test the hypothesis that AP depressive symptoms would moderate the pathway between IC and children's externalizing, four additional hierarchical multiple regressions were conducted with the relevant covariates, again with the inclusion of BM psychopathology. With BM psychopathology included in the model as a covariate, IC continued to predict externalizing in three out of the four regressions (in all but the equation with AF depressive symptoms and parent report of externalizing; see Table 9). In the model with AM depressive symptoms and parent report of externalizing, AM depressive symptoms no longer predicted child externalizing (β = -.32, *ns*). By contrast, in the model with AF depressive symptoms and parent report of child externalizing, there was a negative effect of AF depressive symptoms on externalizing, where lower paternal depressive symptoms predicted higher child externalizing. AM depressive symptoms moderated the pathway between IC and parent-reported children's externalizing at a marginally significant level (β = .41, p = .051; see Table 9). No significant interaction was found between child IC and AF depressive symptoms, or for child IC and AM depressive symptoms when teacher report was used instead of parent report.

To understand the pattern of the marginally significant interaction between IC and AM depression in relation to child externalizing, the effect of maternal depressive symptoms on child externalizing was calculated at low (1 SD below mean), medium (mean) and high (1 SD above mean) levels of child IC. When children's IC was high, maternal depressive symptoms were associated with greater levels of child behavior problems. At low levels of child IC, the opposite pattern was found— lower levels of maternal depressive symptoms were associated with a *higher* likelihood of child behavior problems (see Figure 5).

Table 9. Regression results; hypothesis 3: Influence of inhibitory control (IC) on child externalizing (parent/teacher report), as moderated by maternal/paternal depressive symptoms

	Maternal Depressive Symptoms						**Paternal Depressive Symptoms**					
	CBCL Externalizing Parent Report			**TRF Externalizing Teacher Report**			**CBCL Externalizing Parent Report**			**TRF Externalizing Teacher Report**		
	B(SE)	**β**	**R^2 Change**	**B(SE)**	**β**	**R^2 Change**		**B(SE)**	**β**	**R^2 Change**	**B(SE)**	**β**	**R^2 Change**
Birth mother psychopathology	.04(.04)	.08		-.19(.55)	-.03		BM psychopathology	.04(.04)	.08		-.28(.55)	-.04	
Prenatal tobacco exposure	.17(.12)	.09		-.02(1.90)	.00		Prenatal tobacco exposure	.20(.12)	.11		.17(1.915)	.01	
Other substance exposure	.00(.13)	.00		.47 (2.20)	.02		Other substance exposure	-.03(.13)	-.02		.26 (2.21)	.01	
Adoptive family income	.00(.00)	-.09		.01(.02)	.03		Adoptive family income	.00(.00)	-.08		.01(.02)	.04	
Openness	-.01(.06)	-.01		.86 (.97)	.07		Openness	.00(.06)	.00		.86 (.97)	.07	
Obstetric complications	.01(.01)	.07	.03	.01(.17)	.01	.00	Obstetric complications	.01(.01)	.04	.04	.01(.16)	.01	.00
Inhibitory control (IC)	-.35 (.14)	-.23*	.01†	-5.11(2.10)	-.26*	.06*	IC	-.26(.13)	-.17†	.01†	-6.33(2.06)	-.32*	.06*
Adoptive mother (AM) BDI	-.09(.05)	-.32	.00	.34(.80)	.10	.01	Adoptive father (AF) BDI	-.02(.06)	-.07	.02*	-.53(.73)	-.14	.00
IC x AM BDI	.06(.03)	.41†	.01†	.03(.42)	.02	.00	IC x AF BDI	.04(.03)	.23	.00	.36(.41)	.19	.00
F	1.94*			F	1.57		F	2.19*			F	1.41	
R	.25			R	.27		R	.26			R	.29	
R^2	.06			R^2	.08		R^2	.07			R^2	.08	

*Denotes significance at $p < .05$
†Denotes significance at $p <. 10$

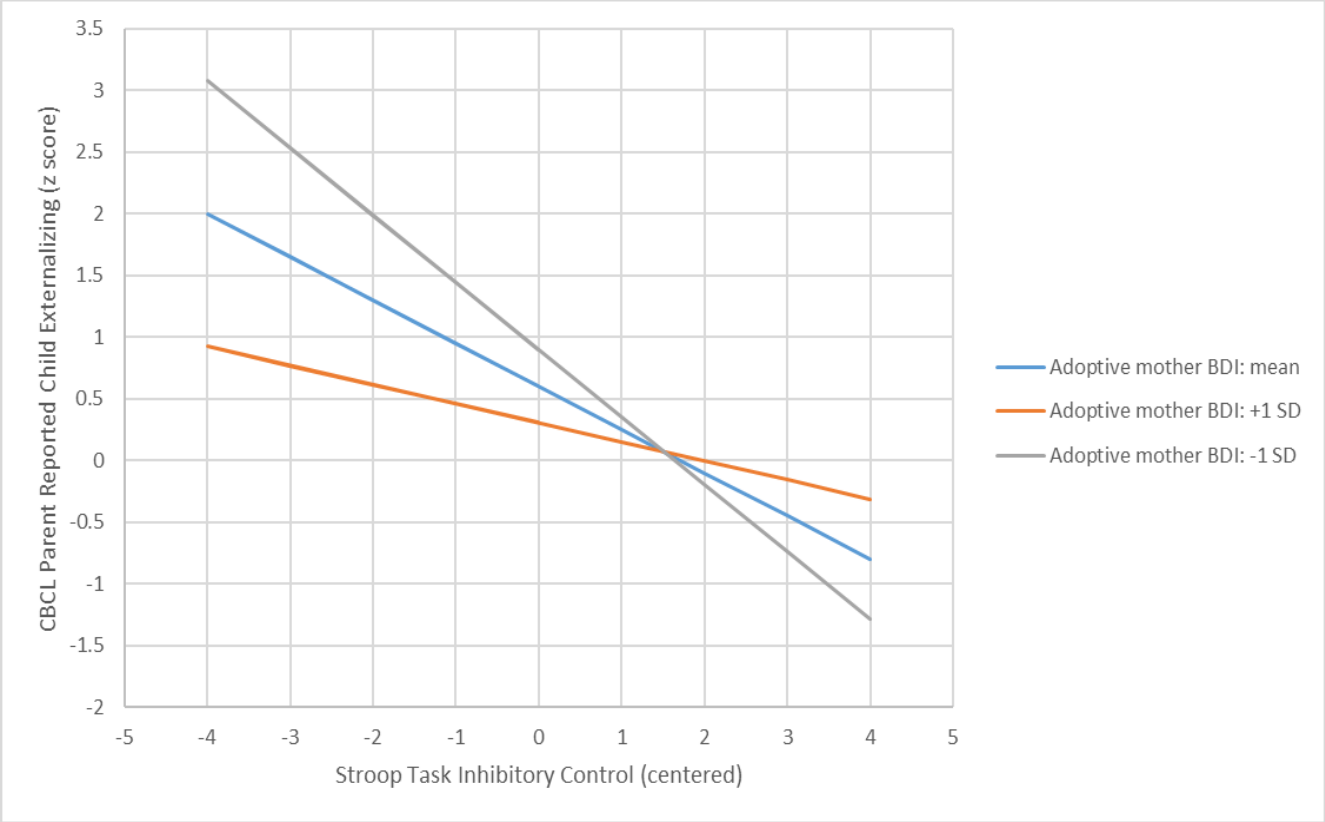

Figure 5. Moderating effects of adoptive mother depressive symptoms on the association between child inhibitory control and parent-reported child externalizing

7.0 DISCUSSION

In November 2015, the American Academy of Pediatrics (AAP) issued a well-publicized report recommending that "no amount of alcohol intake should be considered safe" during pregnancy (Williams & Smith, 2015)z. The article's authors highlight the many known consequences associated with consuming alcohol during pregnancy, while acknowledging that the effects of low doses of alcohol (i.e., an average consumption of less than one standard drink per day) remain unknown. Findings from the current study suggest that in the domain of child behavior problems and IC, there is no evidence that low levels of alcohol exposure are associated with behavior problems during early childhood, or that interactions between PAE and postnatal adoptive parent depression are linked to early child problem behavior.

Findings from the current study also did not support the hypothesis that BM psychopathology has a direct or interactive effect with PAE on children's early externalizing problem behavior. Additionally, although prenatal and genetic risk factors were not found to predict children's early school-age externalizing problems, both the quality of children's early family environment and children's early IC were directly linked to later externalizing symptoms. Consistent with past literature on biologically-related families (Brennan et al., 2000; Kim-Cohen et al., 2005), AM depressive symptoms were associated with parent *and* teacher reports of child externalizing problem behavior. Importantly, AF depressive symptoms also were linked to parent reports of externalizing, with both adoptive parents' depressive symptoms contributing

independent variance to parent reports of child externalizing. Also as expected, an observational measure of child IC at 27 months was negatively related to parent and teacher reports of child externalizing problems at age six.

7.1 NULL FINDINGS BETWEEN PAE AND CHILD EXTERNALIZING BEHAVIOR PROBLEMS

Although much of the published literature has focused on the association between PAE and risk for child behavior problems, not all studies have established a positive relationship between light to moderate PAE and child maladaptive outcomes. In fact, there is a general lack of consensus on whether light to moderate drinking should be considered a risk for pregnant women and their offspring, and the debate about how to address this issue in the arena of public policy has been fairly contentious.

Kelly and colleagues (2013; 2010; 2009) did not find any relationship between light PAE (i.e., 1-2 drinks per week) and high levels of child conduct problems at ages 3, 5, or 7, although there was an association between *heavy* drinking (at least 7 drinks per week) and child conduct problems at age three. Other researchers found a similar pattern in which "low" PAE (defined as less than or equal to 6 drinks per week) was not associated with increased child externalizing problems at ages 2, 5, or 8, but higher levels of exposure were (Colleen M. O'Leary et al., 2010). However, both studies used a clinical cutoff for conduct problems as opposed to a continuous scale, which may obscure smaller but possibly meaningful effects. In addition, a prospective, longitudinal study in which researchers followed children from birth to age 14 found that light drinking (defined as 2-6 drinks per week) in the first trimester was not associated with an

increased risk of externalizing problems (Robinson et al., 2010). Interestingly, Robinson and colleagues also did not find a relationship between heavy PAE and externalizing behavior problems. Another study did not find an effect of low to moderate PAE (1-8 drinks per week) on children's executive functioning at age five (Skogerbø et al., 2012).

In a review article, O'Leary and Bower (2012) remark that although there is a lack of compelling evidence to indicate that low levels of PAE are associated with risk of adverse outcomes for children, the best policy is likely to communicate the message that the safest choice is to abstain from alcohol consumption while pregnant. Other researchers have issued similar statements acknowledging the lack of evidence that low levels of PAE are truly a risk factor for children, but expressing wariness about the possible consequences of recommending a minimum safe dosage of alcohol other than abstinence (Kodituwakku & Ceccanti, 2010). This was likely the approach of the AAP in crafting their recommendation. However, the authors' statement that, based on the studies they reviewed, "the healthiest choice regarding alcohol use during pregnancy is to abstain" may be misleading, as none of the articles cited in the AAP's report indicate that there is any risk of adverse outcomes for children in relation to light drinking. Recommending complete abstinence may further stigmatize those women who choose to drink lightly and infrequently while pregnant.

7.2 EFFECTS OF MATERNAL AND PATERNAL DEPRESSIVE SYMPTOMS ON CHILD EXTERNALIZING

The finding that post-natal maternal depressive symptoms was associated with child externalizing behavior problems is consistent with prior literature (Kim-Cohen et al., 2005; Shaw

et al., 2012; Shaw & Shelleby, 2014). However, unlike most prior research in this area, genetic transmission as a mechanism for the association between maternal depression and child behavior problems (Kim-Cohen et al., 2005) can be ruled out in the current study because of the genetically-informed, adoption design. The finding that paternal depressive symptoms contributed independent variance to child externalizing according to parents' report is relatively novel. The association of both AM and AF depressive symptoms with toddler externalizing problems at age 27 months has previously been established in the current sample, although only the first cohort (i.e., 361 of the 561) was included in the study (Pemberton et al., 2010).

7.3 INHIBITORY CONTROL, MATERNAL DEPRESSIVE SYMPTOMS, AND CHILD EXTERNALIZING

The negative association between child IC at 27 months and externalizing behaviors at 6 years is consistent with many prior studies. For example, research by Eisenberg and colleagues (2009) suggests that effortful control (a construct similar to IC, measured by persistence on an observed task) is longitudinally associated with improvement in children's externalizing symptoms over time. Thus, the current findings corroborate prior research in this area.

It was surprising that paternal depressive symptoms had a negative association with child externalizing symptoms in the regression equation evaluating the moderating effect of paternal depressive symptoms on child IC in predicting externalizing. This contradicts the finding that fathers' depressive symptoms have a *positive* relationship with children's externalizing, both independently and after accounting for maternal depressive symptoms. The pattern of the marginally significant interaction between child IC and AM depressive symptoms was also

surprising. In seems that in biologically unrelated parents, after accounting for some of the genetic components (i.e., BM psychopathology and IC) that may impact children's externalizing problems, low paternal depressive symptoms may be a risk factor, and also low maternal depressive symptoms, but only for those with low IC.

Prior literature and theory would predict that low IC combined with *high* maternal depression would result in the highest level of child externalizing problems, but findings from the current sample suggest that low IC combined with low levels of maternal depressive symptoms is a risk factor. Research by Lengua and colleagues (2008) suggest that school-age children with low effortful control are more vulnerable to contextual risk; specifically, they found that for children low in effortful control, maternal risk was associated with an increase in internalizing problems over time. It is unclear why the findings from the current study suggest that, contrary to prior literature and theory, the combination of low IC with low maternal depressive symptoms would result in less favorable outcomes for children's behavior. One possibility is that adoptive parents with mild depressive symptoms may be under-reporting externalizing problems in their children with low IC. As the interaction was not corroborated by teacher report of child externalizing and was marginally significant, caution is warranted in interpreting this finding as more than an artifact of biased maternal reporting.

7.4 PAE AND CONTEXTUAL RISK

Findings from the current study underscore the importance of accounting for the postnatal environment when drawing conclusions about the effect of PAE on child outcomes. Some of the studies that have found evidence that low levels of PAE are harmful have not fully accounted for

aspects of the postnatal environment that may be responsible for driving such effects (Jacobson & Jacobson, 2001). For example, findings from one meta-analysis suggest that effects of PAE are substantially reduced when researchers account for covariates such as maternal education and smoking during pregnancy (Testa, Quigley, & Eiden, 2003). In the current study, adoptive families were generally very low-risk, and birth mothers received high-quality prenatal care. These characteristics may have allowed for a closer approximation of the *independent* effect of PAE on children's externalizing problems. If so, findings from the current study suggest that in the absence of high levels of contextual risk, PAE does not seem to be associated with children's problem behavior. However, PAE was low relative to other samples, so it could be that the lack of variability in PAE, in addition to low contextual risk, contributed to null findings.

7.5 LIMITATIONS

One major limitation of the current study is its generalizability to a broader population. This study used an adoption sample as a means to distinguish between the genetic effect of BM psychopathology and the prenatal effect of substance exposure versus the postnatal effects of parental depression on children's outcomes. However, additional research is necessary to determine whether the findings of the current study also apply to children being raised by biological parents. Adoptive families in this study are unlikely to be representative of the environment in which most children with PAE are raised. That is, adoptive parents in the sample were predominantly high-income, heterosexual, Caucasian, and married, so it is unclear whether a similar pattern of results would have been found in a higher-risk sample. Perhaps PAE and/or interaction effects could be observed in less affluent environments where children are exposed to

higher levels of contextual stress. For example, in foster families, children are often raised by genetically unrelated parents, but might still be exposed to greater levels of social adversity.

Another limitation is that relatively low rates of clinically meaningful adoptive parent depression and child externalizing problems were reported in the current study. Even so, several relations were found between risk factors and child externalizing behaviors (child IC and both AM and AF depressive symptoms), suggesting that depressive symptoms do not need to be clinically significant to be linked to children's later disruptive behavior at home and school.

Another notable limitation of the current study and alluded to above, is that the rates of reported PAE were low relative to other community and clinical samples, which made it challenging to examine PAE as a continuous predictor of child externalizing behavior. Studies using large, population-based cohorts often find that 30-40% of women report any drinking during pregnancy (Ethen et al., 2009; Muggli et al., 2016), compared with 23% in the current study. It was expected that birth mothers would have higher rates of drinking during pregnancy than a national sample, especially based on the high rates of conduct disorder (CD) and antisocial personality disorder (ASPD) in the current sample: 44% of birth mothers in the current study endorsed symptoms that would qualify them for a diagnosis of CD and/or ASPD, compared to a national prevalence rate of 1% for women (APA, 2013). As ASPD/CD and substance use are known to be comorbid (Compton, Conway, Stinson, Colliver, & Grant, 2005), it was surprising that the reported rates of drinking while pregnant were so low in the current study.

It is possible that birth mothers in the current study underreported their alcohol use during pregnancy. Underreporting of prenatal alcohol use is relatively common, as evidenced by the higher prevalence estimates when using meconium testing versus self-report (Lange, Shield, Koren, Rehm, & Popova, 2014). Although researchers have previously found that retrospective

reporting of alcohol use during pregnancy usually yields higher rates of reported usage than antenatal reporting (Jacobson, Chiodo, Sokol, & Jacobson, 2002), it is unclear whether this phenomenon also applies to mothers who are not planning to parent their children.

It is also possible that the measure of alcohol use in the current study was not sensitive or detailed enough to detect effects on children. O'Leary and colleagues (2010) recommend use of a composite measure of PAE, including information about the timing, dose, and pattern of drinking. Their research suggests that the use of traditional methods of quantifying PAE such as average alcohol use per week over trimester or entire pregnancy (i.e., the method used in the current study), may obscure associations between low/moderate drinking and child outcomes.

Research by Jacobson & Jacobson (1994b) suggests that on some measures (e.g., Bayley Mental Development Index), there is no clear threshold for a dosage at which PAE begins to exert a harmful effect on children. Specifically, they found differences in infant mental development when comparing mothers categorized as "abstainer" versus "very light" (.02-3.49 drinks/week) drinkers. However, only 10% (n = 58) of the birth mothers in the current study reported drinking more than 1 drink a week on average; perhaps this low level of exposure was not sufficient to detect an effect on child outcomes.

7.6 CONCLUSIONS

Findings from the current study do not provide support for the hypothesis that low to moderate PAE has consequences for children's externalizing problems, independently or in interaction with the postnatal environmental stressor of parental depressive symptoms. However, in the context of low rates of prenatal drinking and adoptive families' relatively high

socioeconomic status, low correlations between PAE and children's externalizing may be expected rather than surprising. Future researchers should continue to assess the risk of low to moderate PAE in a methodologically rigorous way, being careful to adequately account for the many possible confounders in the relationship between PAE and child outcomes.

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