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Executive Dysfunction as a Trait Marker for Depression in Children and Adolescents

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EXECUTIVE DYSFUNCTION AS A TRAIT MARKER FOR DEPRESSION IN CHILDREN AND ADOLESCENTS

by

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ABSTRACT

EXECUTIVE DYSFUNCTION AS A TRAIT MARKER OF DEPRESSION IN CHILDREN AND ADOLESCENTS

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Perinatal depression has been recognized as a public health problem in the United States, which is important because of the demonstrated wide-reaching negative effects of maternal depression on child outcomes. Some evidence suggests that maternal depression is a risk factor for executive dysfunction in children. By contrast, there is abundant evidence that maternal depression is a risk factor for later child depression. Therefore, this study focuses on executive dysfunction in children as a potential trait marker for later depression in childhood and adolescence, utilizing data from the NICHD Study of Early Child Care and Youth Development. Participants were from 10 locations around the United States. Measures assessed postnatal depressive symptoms (Center of Epidemiologic Studies Depression Scale, CES-D), inhibition in children (Conners Continuous Performance Test, CPT), inhibition and information updating in children (Tower of Hanoi, TOH, and Tower of London, TOL), inhibition and set shifting in children (Stroop Test), and internalizing behaviors in children (Child Behavior Checklist, CBCL). Maternal depression was grouped based on trajectory: no depression, postpartum depression, early childhood depression, and chronic depression. A series of ANCOVAs and MANCOVAs were conducted to examine: a) whether early chronic maternal depression would be associated with lower scores on measures of executive function among children in 1st grade, 4th grade, 5th grade, and at 15 years of age; and b)

whether children with depressed mothers who experience executive dysfunction would be more likely to experience subsequent depressive symptoms; that is, whether the relationship between maternal depression and later child internalizing behaviors would be mediated by child executive dysfunction. Overall, findings revealed that all courses of maternal depression were associated with later child depression and child inhibition and information updating deficits at grade 1 in males only. Additionally, early childhood and chronic depression were associated with inhibition and information updating deficits at grade 5 in males. Other exploratory analyses are discussed.

This dissertation is dedicated to my parents, for their unwavering support from 3,000 miles away.

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CHAPTER I

INTRODUCTION

Perinatal depression has been recognized as a public health problem in the United States (Wisner, Chambers, & Sit, 2006). Whereas the prevalence of depression is about 10% in the general female adult population (Kessler et al., 2010), postpartum depression affects about 13% of women (O'Hara & Swain, 1996). The recognition that postpartum depression occurs more frequently than depression in the general female adult population is important because of the negative impacts that maternal depression has on a wide range of child outcomes including difficulty with interpersonal relationships, increased risk for psychopathology, and poorer cognitive development (Beardslee, Versage, & Gladstone, 1998). It is likely that negative parenting practices adversely impact child development, including a lack of positive engagement with the child (Rhoades et al., 2011) and a lack of sensitivity to the child's needs (Kok et al., 2013).

Some evidence suggests that maternal postpartum depression is a risk factor for executive dysfunction in children (Hughes & Ensor, 2009; Hughes, Roman, Hart, & Ensor, 2013), but this has not yet been widely studied. The impact of maternal depression on executive functioning in children is potentially important because children with executive dysfunction experience wide-ranging difficulties with intelligent and goal-driven behavior (Banich, 2009), such as inhibition of responses, information updating, and mental set-shifting (Miyake et al., 2000). Executive function is necessary for self-management and planning and deficits in EF have been linked to increased difficulty with activities of daily living (Grigsby, Kaye, Baxter, Shetterly, & Hamman, 1998; Hanks, Rapport, Millis, Deshpande, 1999; Plehn, Marcopulos, & McClain, 2004).

Although the evidence linking maternal depression to child executive dysfunction is tentative, it has been well established that early maternal depression is a risk factor for later child depression (Cummings & Davies, 1994; Downey & Coyne, 1990). A proposed mechanism by which this occurs is a combination of heritability, dysfunctional neuroregulatory processes, exposure to maternal negative verbalizations, behaviors, and emotions, and stress in the child's life (Gotlib, 1999). It has also been established that executive dysfunction co-occurs with depression (Hughes & Ensor, 2009) in the areas of problem-solving, planning, and inhibition (Fossati et al., 2002). Cognitive-rigidity within depression may be linked to problem-solving impairments and might help maintain depression by preventing patients from coping with stressful life events. Planning deficits suggest that depressed individuals are not motivated to improve performance upon gaining corrective feedback. Inhibition deficits are likely related to psychomotor retardation and a lack of cognitive resources (Fossati et al., 2002).

Multiple studies have identified executive dysfunction as a trait marker for depression (Christensen, Kyvik, & Kessing, 2006; Hsu, Young-Wolff, Kendler, Halberstadt, & Prescott, 2013). Specifically, a twin study showed that in monozygotic twins (MZ) with one depressed and one non-depressed twin, the non-depressed twin had lower executive functioning capabilities than in twins not affected by depression (Hsu et al., 2014). This suggests potential heritability of executive dysfunction, which might make an individual more vulnerable to later depression.

This study aims to explore the relationship between early maternal depression and later child executive dysfunction and depression. Because maternal depression is a risk factor for both executive dysfunction and depression, the present study examines the

effect of maternal depression on later child depression, as potentially mediated by child executive dysfunction. In order to gain a better understanding of the developmental processes that link early maternal depression to child executive dysfunction, this study utilizes a national longitudinal data set (i.e., the Study of Early Child Care and Youth Development [SECCYD]).

Executive Functioning

Despite an interest in understanding executive functioning (EF) in the cognitive development literature, the construct currently lacks a widely-accepted operational definition (Barkley, 2012). In fact, Sargeant, Geurts, and Oosterlaan (2002) note that in the current literature there are 33 different definitions of EF. One broad definition of EF that seems to encompass more specific definitions is the following:

> In general, executive function can be thought of as the set of abilities required to effortfully guide behavior toward a goal, especially in nonroutine situations. Various functions are thought to fall under the rubric of executive function. These include prioritizing and sequencing behavior, inhibiting familiar and stereotyped behaviors, creating and maintaining an idea of what task or information is most relevant for current purposes (often referred to as an attentional or mental set), providing resistance to information that is distracting or task irrelevant, switching behavior task goals, utilizing relevant information in support of decision making, categorizing or otherwise abstracting common elements across items, and handling novel information or situations. As can be seen from this list, the functions that fall under the category of executive function are indeed wide ranging. (Banich, 2009, p.89)

Although researchers have not yet agreed upon a universal definition of EF, the general consensus is that EF exists on a continuum and is essential for self-directed behavior (Miyake et al., 2000). Multiple studies have found that the greater the executive dysfunction, the poorer the ability to live independently (Grigsby et al., 1998; Hanks et al., 1999; Plehn et al., 2004).

In the literature, there are two predominant theories that explain mechanisms underlying EF: the theory of unity and the theory of non-unity, both of which have empirical support (Jurado & Rosselli, 2007). Because EF is associated with the frontal lobe (Alvarez & Emory, 2006), studying traumatic brain injury and frontal lobe lesions has been particularly helpful in understanding EF. Duncan, Emslie, Williams, Johnson, and Freer (1996) studied traumatic brain injury, and more specifically goal neglect, to argue for the theory of unity. Goal neglect can be defined as an individual disregarding a task requirement, even though the individual both understood and remembered that requirement. Colloquially it can be described as "slipping one's mind" and is particularly apparent in novel tasks and in multiple concurrent tasks. It has been theorized that there is one central factor underlying EF, that is, general intelligence (De Frais, Dixon, & Strauss, 2006; Duncan et al., 1996). To examine the underlying factor of EF, De Frais and colleagues (2006) utilized a sample of older healthy adults each of whom was administered four indicators of EF. De Frais and colleagues tested both a 2-factor (inhibition and shifting) and a single-factor model. Findings indicated that the single-factor model at the latent construct level fit the data well, whereas the 2-factor model did not. Additionally, the single executive factor was associated with a measure of fluid intelligence. De Frais and colleagues (2006) concluded that fluid intelligence could

underlie the different EF tasks.

Although there is empirical evidence for the theory of unity (De Frais, Dixon, & Strauss, 2006; Duncan et al., 1996; Duncan & Miller, 2002) there is also evidence for distinct components of EF (Burgess et al. 2007; Robbins 1996; Stuss & Alexander, 2007). Stuss and Alexander (2007) argue that data do not support a central executive or undifferentiated supervisory system. Rather, they argue that there are functionally and anatomically independent frontal lobe processes. By studying impairments in functioning based on lesions in different frontal lobe regions, they have identified three regions associated with distinct processes: energization occurs in the superior medial region, task setting in the left lateral region, and monitoring in the right lateral region.

Although articles continue to be published supporting separate theories of unity and non-unity, to make sense of the conflicting outcomes, some authors have tried to integrate the two ideas. For example, one theory suggests that the structure of EF may change across the life span from a multidimensional construct in young college students to a more unidimensional one in typical aging adults (De Frais, 2006), but EF processes are particularly difficult to examine due to a problem of task-impurity in the measures used to tap into these functions (Phillips, 1997). One integrative theory that has gained credence in the literature is that there are both unitary and diverse aspects of executive functioning which contribute to complex frontal lobe tasks (Miyake et al., 2000).

A common approach to defining and measuring distinct aspects of EF can be seen in a study by Miyake and colleagues (described below). They identified the following components: *inhibition of responses, information updating, and mental set shifting*. An example of each component and the way it is typically measured is as follows: *inhibition*

of responses can be defined as an individual's ability to purposefully inhibit automatic responses, such as naming the word itself rather than the color of the word in the Stroop Task (Miyake et al., 2000). *Information updating* utilizes working memory to encode incoming information and determine its relevance to the current task while simultaneously disposing of old, irrelevant information and replacing it with the new information (Morris & Jones, 1990). For example, the N-back Task measures information updating by presenting participants with a series of images on a screen and the participant has to identify whether the image is in the same or a different location than it appeared on the previous screens. The N stands for the number of previous screens the participant will have to consider when determining whether the current image appears in the same or a different location. If a participant were asked to consider the previous screen, it would be called the 1-back task, whereas the 2-back task would require participants to consider the location of the image as compared to that screen which appeared two screens prior. Finally, *mental set shifting* is the ability to shift attention back and forth between different tasks (Allport, Styles, & Hsieh, 1994). *Mental set shifting* can be measured using the Trail Making Test, Part B, in which a participant is asked to draw a line connecting numbers in order, but switching by incorporating letters in order (1, A, 2, B, 3, C, etc.). This requires the participant to switch sets between letters and numbers.

Miyake and colleagues (2000) were among the first to introduce this idea into the literature (see also Duncan, Johnson, Swales, & Freer, 1997; Teuber, 1972); they used a latent variable analysis to study individual differences in executive functioning domains. Miyake and colleagues (2000) used this approach to statistical analysis due to the

potential problems that can arise when using the typical correlational, factor-analytic method that many previous studies have employed. Specifically, correlational or factor-analytic approaches have been used in the past to find low correlations between tasks examining EFs, but this is not necessarily indicative of independent EFs. Instead, it is possible that vast differences in nonexecutive processes that are utilized during EF tasks have disguised some true underlying commonalities between EF domains. As stated previously, other cognitive processes are utilized during these EF measures making it difficult to discern between unity and diversity in EF (Miyake et al., 2000).

Due to limitations of other statistical methods, Miyake and colleagues aimed to decrease the task impurity problem by using latent variable analysis to study the separability of three EF domains. These include *inhibition of responses*, *information updating*, and *mental set shifting*, and were chosen because they are simpler EF that can be more precisely defined, as compared to a higher-level function such as planning.

To examine how different measures related to the three postulated domains of EF, Miyake and colleagues (2000) utilized a sample of college students who performed tasks that are considered to target each EF area: the Wisconsin Card Sorting Test (WCST), Tower of Hanoi (TOH), random number generation (RNG), operation span, and dual tasking (Miyake et al., 2000). The WCST is a measure that asks participants to match individually-presented target cards to reference cards according to three stimulus attributes: color, number, or shape. Participants were told that only one stimulus attribute was correct for each card. Target cards were presented and participants were given feedback as to whether their sorting was correct or incorrect. Participants were told that the sorting criteria changed over time, but they were not told how many correctly-sorted

cards were to be achieved before the criterion would change. The WCST was measured by number of perseverative errors, that is, number of times the participant failed to change sorting strategies when the categories changed. The TOH is an activity in which participants were shown an ending configuration of four different disks of varying size arranged on three pegs and were then presented with a starting configuration. Participants were then asked to make the starting configuration look like the ending one by using the fewest moves and least time possible, while following rules such as moving only one disk at a time, keeping each disk placed on a peg, and never placing a larger disk on top of a smaller one. Scores were based on the number of total moves that the participant took to complete the target problems. During the RNG, participants were presented with a beep and asked to say a random number from 1 to 9 aloud. Randomness was explained by asking them to pretend they were pulling a number out of a hat and then returning it after each pull. Randomness was scored based on an analysis of participants' responses, including redundancy and adjacency. Operation Span is a task that requires individuals to read aloud a simple math equation, answer whether it is true or false aloud, and then read a single presented word on a screen, such as "king." At the end of the trial, the participant was asked to recall all of the words from the set of equation-word pairs, with one stipulation that the word from the last presented pair should not be recalled first. Operation Span was scored based on the number of correct words recalled. In the Dual Task, participants were asked to complete as many mazes as possible in 3 minutes, then they were asked to complete a word generation task for 3 minutes during which they were presented with a letter and asked to generate as many words as possible beginning with that letter. The final stage required participants to

complete these tasks simultaneously. This final stage was scored using a specific equation to determine the proportion of decrement in performance observed from the individual to the dual task.

Using latent variable analysis, Miyake and colleagues (2000) found that different measures related to distinct EF domains in the following ways: Wisconsin Card Sorting Test related to *shifting*, Tower of Hanoi to *inhibition*, Random Number Generator to *inhibition/updating*, and operation span to *updating*. Dual tasking was not related to any of the three EF areas. Although the domains were clearly distinguishable, they were not completely independent; rather, they seemed to share some commonality. This was evident because the full three-factor model better fit the data than the three-factor model that assumed complete separability between the EF domains. Overall, this suggests both unity and diversity of executive functioning (Miyake et al., 2000).

Although studies supporting the theory of unity (Duncan et al., 1996) and the theory of non-unity (Stuss & Alexander, 2007) continue to be published, it is likely that EF does not exist on either end of the continuum (Miyake et al., 2000). Rather, it is likely that EF has both a factor or factors that underlie all processing, and that it also has distinct areas of EF that are separable (Miyake et al., 2000). General intelligence has been proposed to be the one factor underlying processing (De Frais, Dixon, & Strauss, 2006; Duncan et al., 1996), whereas inhibition, updating, and set shifting are major examples of independent factors (Miyake et al., 2000).

Maternal Depression and the Development of Executive Functioning

Although not widely studied, some evidence suggests that maternal postpartum depression is a risk factor for deficits in children's EF development (Hughes et al., 2013).

This potential association is important because perinatal mothers are at an increased risk for depression, compared with the general female adult population (Stowe & Nemeroff, 1995) and early development of EF in children is more vulnerable to environmental influences as compared to other neurocognitive functions (Noble, Norman, & Farah, 2005).

Hughes, Roman, Hart, and Ensor (2013) considered the chronicity of depression in mothers as having a potentially important impact on child EF development. Maternal depression was measured at 4 time points between child ages 2 and 6 years. Findings indicated that maternal postpartum depression had a negative and enduring impact on child EF from age 2 through age 6. Specifically, maternal depression was significantly predictive of children's EF over a 4-year period; children with depressed mothers scored lower on measures of EF. Also, levels and chronicity of depression predicted unique variances in EF scores at age 6. Covariates included maternal education, maternal scaffolding, and the stability of EF over time. These covariates might be important because they can all influence EF development in children and could potentially confound the research question.

Although there is evidence that maternal depression negatively impacts child EF, there is also evidence to the contrary (Klimes-Dougan et al., 2006; Micco et al., 2009; Rhoades, Greenberg, Lanza, & Blair, 2011). Two studies (Klimes-Dougan et al., 2006; Micco et al., 2009) had null results when looking at the impact of maternal depression on child EF development. One explanation that could account for disparate findings between these authors and Hughes and colleagues (2013) could be age. Both Klimes-Dougan and colleagues (2006) and Micco and colleagues (2009) studied the impact of

maternal depression on older children and adolescents. Maternal depression during older childhood and adolescence was measured, but early maternal depression was not considered. In both studies, environmental factors such as SES and overall family stress were considered, although they did not consider some factors known to be associated with child development such as parental education. It is possible that older children and adolescents spend less time with their mothers and may therefore be less impacted by their depression (Hughes et al., 2013). It is also possible that the older children and adolescents who were exposed to maternal depression were less vulnerable than their younger counterparts to disruption in EF development. A study by Rhoades and colleagues (2011) also found null results when looking at the impact of maternal depression on child EF development. Similar to Hughes and colleagues (2013), Rhoades and colleagues (2011) examined EF development in young children; however, Rhoades and colleagues differed from Hughes and colleagues (2013) in their sample. The former sample consisted of 67% African-Americans, whereas the latter was almost exclusively White. It is possible that the effects of maternal depression on early child EF differ across ethnicities.

Although there are no studies that have examined differences in EF development between males and females with depressed mothers, the literature on general cognitive development in children can serve to inform potential gender differences in EF development. Specifically, gender differences have been observed in vulnerability to maternal depression. Multiple studies have found that maternal postpartum depression has a negative impact on male cognitive development, but the same is not true for females (Murray, 1992; Sharp et al., 1995). It is possible that a similar pattern might

exist in EF development. One hypothesis for gender differentiation in both EF and cognitive development is that females have a maturational advantage in the development of language and social skills, and are therefore more protected against the negative impacts of maternal depression (Berk 1997). If true, this association may explain why male children are more vulnerable to the effects of maternal depression. Also, it is possible that mothers interact differently with their male infants (Murray, 1992). Finally, it is feasible that male infants act in such a way that prolongs maternal postpartum depression which might negatively impact male cognitive development due to exposure to more chronic maternal depression (Sharp et al., 1995).

Another important concern when considering the impact of maternal depression on child executive functioning development is whether executive dysfunction in children could prolong maternal depression. In the current literature on assessing executive function, it appears that the measurements have been created to assess executive functioning in preschool children ages 3-5 years old; however, these assessments are sparse and their psychometric properties are not considered adequate enough to be used in clinical settings (Garon, Bryson, & Smith, 2008; Isquith, Crawford, Espy, & Gioia, 2005). Also, the current study considers maternal depressive symptoms during infancy and early childhood, during which time there do not exist measures of executive function, nor is it known whether executive dysfunction is detectable in this age group. Therefore, at the present moment it is not possible to determine whether executive dysfunction in infants might prolong maternal depression. There are, however, known diagnoses with a constellation of symptoms including executive dysfunction such as Autism Spectrum Disorders (Happe, Booth, Charlton, & Hughes, 2006), but these also include other

symptoms such as lack of bonding, which could be responsible for prolonging maternal depression. With that in mind, though, Autism Spectrum Disorders are not usually diagnosed until age 3, with some researchers attempting to diagnose as early as 19 months without clear stability of diagnosis (Guthrie, Swineford, Nottke, & Wetherby, 2013). Therefore, even if an Autism Spectrum Disorder were to develop, there is not current evidence that symptoms would show in early infancy, and would subsequently prolong maternal depression.

There is conflicting evidence in the literature about whether early maternal depression has a negative impact on child EF development. However, this is to be expected given the different samples that were used in the few studies that have examined this topic, one with Caucasian toddlers (Hughes et al., 2013), two with adolescents (Klimes-Dougan et al., 2006; Micco et al., 2009), and one with a large proportion of African-Americans (Rhoades et al., 2011). The current study is most similar to Hughes and colleagues (2013). Also, the possibility of early executive dysfunction in children impacting the course of depression has not been studied and currently does not seem feasible due to lack of measurement of executive dysfunction and associated diagnoses in early infancy (Isquith et al., 2005; Happe et al., 2006; Garon et al., 2008; Guthrie et al., 2013).

How Maternal Depression Affects Child Executive Functioning Development

Very few studies have examined environmental factors that impact EF development in children, but the few that have examined this association have found that the child's mother plays a role (Kok et al., 2013; Rhoades et al., 2011). Rhoades and colleagues looked at how ecological risks related to child EF development and found that

socioeconomic risk was significantly associated with children's EF abilities at 36 months; this was partially mediated by parenting behaviors. Specifically, Caucasian mothers who were in the *Poor/Married* and *Poor/Unmarried* groups were often engaging in more intrusiveness with their infants and less positive engagement, as compared to mothers in the *Low Risk/Married* group. These negative parenting practices were then related to lower EF in children. Additionally, Kok and colleagues (2013) found that maternal sensitivity in particular was related to child EF development. Specifically, mothers who behaved more sensitively to their children had children with fewer EF problems in preschool.

Although neither of these studies directly related problematic parenting behaviors with depression, depression is a well-established risk factor for problematic parenting behaviors, specifically negative/coercive behavior toward infants and a lack of sensitivity (Lovejoy, Graczyk, O'Hare, & Neuman, 2000). For example, Stein and colleagues (1991) examined interactions between mothers and their infants at 19 months. Findings suggested that mothers with postpartum depression were less affectionate, initiated less socialization of the child with a stranger, and were less involved in the overall facilitation of their children's lives.

Additionally, contingent stimulation provided by a mother is thought to help a child engage with and learn from their environments. Hay (1997) found that depressed mothers are less likely to consistently use contingent stimulation with their children, which is problematic for a child's learning. A form of operant conditioning, contingent stimulation occurs when a parent responds to an infant based on the initial behavior of the infant. This aids in learning because it allows an infant to understand the world by

predicting responses to his or her behavior. Given findings by Hay and Kumar (1995), it is possible that mothers with fewer years of education are less likely to provide children with an environment rich in contingent stimulation because mothers with fewer years of education have been associated with poorer cognitive outcomes in children. Ramey and Finkelstein (1978) examined contingent stimulation as a tool to enhance learning abilities in children who were at risk for socioculturally-caused intellectual deficits. The groups received either contingent auditory-visual responses to infant vocalizations, non-contingent auditory-visual responses, or no responses. Results suggested that children in the contingent stimulation group had enhanced learning abilities. Although contingent stimulation has proven important in cognitive outcomes, it is likely that it is also important in EF development.

Other Etiologies of Executive Dysfunction

Although the present study focuses on maternal depression as an environmental stressor that can negatively impact child executive functioning development, it is important to consider other risk factors for executive dysfunction. First, it is important to acknowledge the heritability of executive dysfunction, which has been demonstrated in twin studies on ADHD (Freitah, Rohde, Lempp, & Romanos, 2010). However, the majority of twins are reared together and are potentially exposed to the same types of environmental disturbances making it difficult to parse out true heritability from shared environmental experiences (Freitah et al., 2010). Experiences that are known to negatively impact executive functioning development occur both prenatally and postnatally. These negative experiences include exposure to alcohol and/or marijuana in utero (Connor et al., 2000; Fried & Smith, 2001), neonatal hypoxia and anoxia

(inadequate oxygen; Decker & Rye, 2002; Sullivan & Brake, 2003), and being born pre-term (Edgin et al., 2008). The present study utilizes an existing national longitudinal data set from which participants with birth complications were excluded. This makes executive dysfunction more likely to occur due to hypothesized environmental stressors (i.e., maternal depressive symptoms), or heritability. See *Participants* below for exclusion criteria.

Maternal Depression and Later Child Depression

Maternal depression is a well-established risk factor for later child depression (Cummings & Davies, 1994; Downey & Coyne, 1990). As stated previously, this is of particular importance because new and expecting mothers are at an increased risk for developing depression (Stowe & Nemeroff, 1995). In fact, Hammen and Brennan (2003) found that children with depressed mothers were twice as likely to have diagnosable depression at age 15 than children with never-depressed mothers. Hammen and Brennan (2003) considered severity, chronicity, and timing of maternal depression on child outcomes and found that after controlling for demographics, maternal depression severity was a better predictor of child depression than chronicity, and timing did not predict risk in the child. Overall, they found that one episode of depression in the mother during the child's first 10 years predicted later child depression, independent of the timing of that depressive episode. Of note, the chronicity of maternal depression was unimportant; brief maternal major depression as well as more extended mild depression were both predictive of children's depression at age 15.

Although maternal depression is an established risk factor for later child depression, the mechanisms for this transmission are not well-documented. Goodman

and Gotlib (1999) proposed a model to understand this transmission considering the role of development in clarifying the risk in children. They proposed an integrative model for understanding this risk using four mechanisms: 1) depression heritability, 2) dysfunctional neuroregulatory processes, 3) exposure to negative maternal verbalizations, behaviors, and emotions, and 4) stress in the children's lives.

After reviewing genetics research on depression, Goodman and Gotlib (1999) concluded that substantial heritability for depression exists in adults, which is greater for early-onset rather than late-onset depression. However, results are less clear for childhood- and adolescent-onset depression and seem to vary based on factors such as severity and affected gender. For example, for more severe depression in males, environmental factors seem to play a bigger role as compared to less severe depression in females in which heritability appears to be a stronger factor. Additionally, the genetic risk for children seems to be non-specific because children of depressed mothers have been found to struggle with substance abuse and conduct disordered behavior. Regarding the second proposed mechanism, Goodman and Gotlib (1999) concluded that there is not substantial evidence to determine that abnormal neuroendocrine functioning during pregnancy mediates maternal depression on later child depression. There is more evidence for the third mechanism that depressed mothers are unable to meet the social and emotional needs of their children, which then limits the development of cognitive and social skills in children. Additionally, children of depressed mothers have been found to display cognitions, affect, and behaviors that broadly mirror those of their depressed mothers. Finally, children of depressed mothers are exposed to more stressful environments than those with non-depressed mothers (mechanism four), but this has not

been established as a direct mediator of maternal depression on child depression.

Gibb, Uhrlass, Grassia, Benas, and McGeary (2009) proposed another integrative model for intergenerational transmission of depression that considers genetic, cognitive, and environmental factors. Rather than a literature review like that of Goodman and Gotlib (1999) above, Gibb and colleagues (2009) used hierarchical linear modeling to examine maternal depression, child depression, and expressed emotion criticism. Expressed-emotion in families has been found to be an environmental risk factor leading to relapse of mental illness, with expressed-emotion criticism being most important among pediatric samples (Nelson, Hammen, Brennan, & Ullman, 2003). Findings suggested that mothers' current depressive symptoms were significantly related to levels of expressed-emotion criticism toward children, but the same was not true for mothers with a history of Major Depressive Disorder (MDD) but no current depression. Also, maternal expressed-emotion criticism toward children was not stable across mothers with a history of MDD without a current episode. Gibb and colleagues also examined an integrated gene by cognition by environment interactional model of risk. Results lent partial support to their proposed model. They found that children who assumed negative self-characteristics, that is, those who after presented with a hypothetical negative event assumed negative characteristics about themselves or their role in that event, were more likely to display depressive reactions when criticized by their mothers than children who did not assume negative self-characteristics. This finding was only true, though, for children with one or two copies of the 5-HTTLPR alleles. For children who were homozygous for the 5-HTTLPR allele, there was no indication of an interaction between cognitive vulnerability and stress. They found a dose-response effect in which the

number of lower expressing alleles affected the magnitude of the cognitive vulnerability and stress relationship. Overall, findings suggested that mothers with a history of MDD were more likely to exhibit current depressive symptoms. In turn, these mothers were more likely to exhibit expressed-emotion criticism toward their children; children in a subgroup with negative inferential styles about self-characteristics who carried one or two 5-HTTLPR alleles were more likely to experience elevated depressive symptoms with exposure to maternal criticism.

Although maternal depression is a well-established risk factor for later depression in children (Cummings & Davies, 1994; Downey & Coyne, 1990; Hammen & Brennan, 2003), the mechanisms through which depression risk is conveyed to offspring are less clear. Goodman and Gotlib (1999) as well as Gibb and colleagues (2009) both proposed models through which depression is transmitted intergenerationally. Both sets of authors considered heritability, cognitive factors, and stress in the child's environment, all of which seem to contribute to the development of a child's mental health. Gibb and colleagues (2009) expounded upon Goodman and Gotlib's work (1999) by looking at specific genetic markers. They found that children who carried one or two 5-HTTLPR alleles were more likely to react with depressive symptoms to expressed-emotion criticism, which was more likely to come from depressed mothers.

Executive Dysfunction During Depression

Given that some studies have shown maternal depression to be associated with executive dysfunction in children (Hughes & Ensor, 2009; Hughes, Roman, Hart, & Ensor, 2013), and that maternal depression is also associated with depression in children (Cummings & Davies, 1994; Downey & Coyne, 1990), it is important to look at the

profile of executive functioning for individuals with depression to establish a potential relationship between these two. There are currently many more studies on executive functioning in adults with depression, so a wide age range will be considered, which may be useful in informing an understanding of child executive dysfunction.

A review by Fossati, Ergis, and Allilaire (2002) noted that frontal lobe dysfunction, and therefore executive dysfunction, is likely prominent in depression; a meta-analysis noted that there is a reliable relationship between depression and executive dysfunction with effect sizes typically ranging from .32 to .97 (Snyder, 2013). The studies that Fossati and colleagues (2002) reviewed used measures known to be sensitive to frontal lobe damage such as verbal fluency tests, Stroop Test, Wisconsin and California Card Sorting Tests, Tower of London, and Trail Making Test. Fossati and colleagues (2002) noted that depressed patients typically exhibited problem-solving impairments, planning deficits, and inhibition deficits. Problem-solving impairments may stem from cognitive-rigidity and may help maintain depression by preventing patients from coping with stressful life events. Planning deficits suggest that depressed individuals are not motivated to improve performance upon gaining negative feedback. Inhibition deficits are likely related to psychomotor retardation and a lack of cognitive resources; depressed individuals might then be more likely to process irrelevant environmental information, which could limit their ability to regulate mood changes (Fossati et al., 2002).

To increase the understanding of how executive dysfunction during depression might impact children, it is important to consider studies using pediatric samples. As compared to studies of executive dysfunction in adult depression, studies on children

have mixed results. Cataldo and colleagues (2005) aimed to explore whether depressed children and adolescents have levels of cognitive impulsivity similar to what has been found in adults. Findings suggested that depressed children and adolescents displayed a conservative response style, that is, they took more time to attempt answers, than controls; however, depressed youth did not answer less accurately. This finding suggests that the cognitive style of depressed children and adolescents is not characterized by impulsivity. However, when sustained attention was tested, depressed children and adolescents had longer reaction times, responded less consistently, and made more omission errors than controls. An omission error occurs when a target is present, but the participant fails to respond to it by clicking the mouse. This typically indicates sluggish responding or lack of attention. Children and adolescents also displayed an interference effect on the Stroop Test; the participants had difficulty naming the color of the word rather than what the word said, as evidenced by errors and/or taking more time to respond. Although impulsivity was not affected in depressed children and adolescents, it appeared that other areas of executive control, such as sustained attention and inhibition, were impaired (Cataldo, et al., 2005).

By contrast, in a study by Favre and colleagues (2008), children and adolescents diagnosed with MDD who were administered measures of intelligence and EF were found to have no difference in performance compared to controls. It is important to note, however, that mental processing speed in the depressed group was slower than in the control, and the depressed group performed below average on a test of set-shifting (Trail Making Test). It is possible that researchers did not find more global EF deficits due to their small sample.

It is also possible that there is a gender difference in executive functioning in child and adolescent depression. Emerson, Mollet, and Harrison (2005) examined executive function in boys experiencing anxious-depression, a co-occurring disorder of both anxiety and depression. The researchers chose to examine anxious depression because anxiety and depression have both been associated with impairment in EF. They compared a group of boys with anxious-depression to controls on the following measures: Trail Making Test (Forms A and B) and the Concept Formation portion of the Cognitive Abilities subsection of the Woodcock-Johnson. Although they did not study girls, findings suggested that boys with anxious-depression had deficits in problem-solving tasks, sequencing, and alternation as evidenced by lengthier times to completion and more response errors (Emerson et al., 2005).

Whereas executive dysfunction in adults experiencing depression is well-documented (Fossati, et al., 2002; Snyder, 2013), there are few studies that have considered executive dysfunction in pediatric samples with depression. Of the studies that do exist, results are mixed (Cataldo et al., 2005; Emerson et al., 2005 Favre et al., 2008). It is possible that consistent results with pediatric samples do not exist due to the different age groups, different measures, and differences in the severity of depression, or mixed anxiety and depression.

Executive Functioning Deficits as Trait Markers for Depression

Although there is strong empirical support for the co-occurrence between executive dysfunction and depression, the timing of these co-occurring phenomena is less clear (Fossati, et al., 2002; Snyder, 2013). If dysfunction exists prior to the onset of depression, it could be identified as an endophenotype, a heritable trait present both

during and in the absence of a psychiatric illness, which additionally can be found in non-affected family members at higher rates than in the general population (Gottesman & Gould, 2003).

To answer these questions, Christensen and colleagues (2006) considered both monozygotic (MZ) and dizygotic (DZ) twins both with and without a co-twin who had been diagnosed with an affective disorder. Healthy MZ and DZ twins with co-twins diagnosed with unipolar depressed scored lower on measures of cognitive functioning, as compared with healthy twins with an unaffected co-twin. These measures included sustained attention, selective attention, executive function, language processing, and declarative and working memory. Researchers claimed that cognitive impairment is likely present before the onset of depression, which is evidence for heritability of this trait (Christensen, 2008). It is possible that cognitive dysfunction is genetically transmitted, but it is also possible that environmental factors contributed to these findings.

By contrast, Hsu and colleagues (2013) used only MZ twins, discordant for a history of depression, to examine whether neuropsychological dysfunction could be an endophenotype for depression or whether depression causes prolonged neuropsychological dysfunction, even after symptoms remit. Monozygotic twin pairs were assessed using a structured clinical interview, and measures from the WAIS-III and WMS-III. Results suggested that twins with a history of depression and their unaffected co-twins scored similarly on measures of working memory, verbal memory, attention, and visuo-spatial processing. When compared with twins from pairs with no history of depression, unaffected twins in discordant pairs scored lower. This result was true only

for measures of general knowledge and attention, and after researchers controlled for sex and age. Overall, this finding suggests that part of the familial risk for depression might be conveyed in executive dysfunction (Hsu et al., 2013).

Additional evidence that executive dysfunction precedes the onset of depression is in the high comorbidity between a disorder of executive function, Attention Deficit Hyperactivity Disorder (ADHD), and depression, with ADHD typically existing first (Burleson, 2008). In a literature review, Burleson (2008) reported that depression occurs at a significantly higher rate in children and adolescents who have been diagnosed with ADHD, which likely occurs as a result of environmental challenges faced by children with ADHD. Because executive dysfunction occurs on a continuum, individuals with mild dysfunction would not likely be diagnosed with ADHD. Thus, it is possible that executive dysfunction goes undetected prior to the onset of depression and directly influences an individual's vulnerability to developing depression.

Although there is not an abundance of evidence, some evidence suggests that neuropsychological dysfunction occurs prior to depression in at-risk individuals, based on family history (Christensen et al., 2008; Hsu et al., 2013) or in individuals with ADHD (Burleson, 2008). This highlights the possibility that neuropsychological dysfunction could make an individual more vulnerable to experiencing depression. Although Christensen and colleagues (2008) and Hsu and colleagues (2013) argue for genetic heritability of neuropsychological dysfunction, the present study will examine environmental factors that might predispose an individual to neuropsychological dysfunction and later depression.

The Present Study and Hypotheses

The present study uses The Study of Early Childcare and Youth Development (SECCYD), a national longitudinal data set, to examine executive functioning in children exposed to early maternal depression. Because there is evidence that maternal depression negatively impacts child EF development, that maternal depression is related to later child depression, and that executive dysfunction might be a trait marker for later depression, this study explores maternal depression as an environmental influence on EF and potential later depression in children (for a conceptual diagram, please see Figure 1 below). Maternal education is an important factor that has been considered in past studies and is considered in this study given that fewer years of maternal education has been associated with poorer outcomes in child EF (Hughes et al., 2013) and overall cognitive abilities (Hay & Kumar, 1995). Other covariates related to child outcomes include total family income, child age at testing, and site of data collection (Watamura, Phillips, Morrissey, McCartney, & Bub, 2011). The following hypotheses were tested: 1) chronic maternal depression will have a negative and enduring impact on measures of executive function in children in 1st grade, 4th grade, 5th grade, and at 15 years of age; and 2) children with chronically depressed mothers, as defined by mothers who experience depression both during the postpartum and early childhood periods, who experience executive dysfunction will be more likely to experience subsequent internalizing behaviors; that is, the relationship between chronic maternal depression and later child internalizing behaviors will be partially mediated by child executive dysfunction.

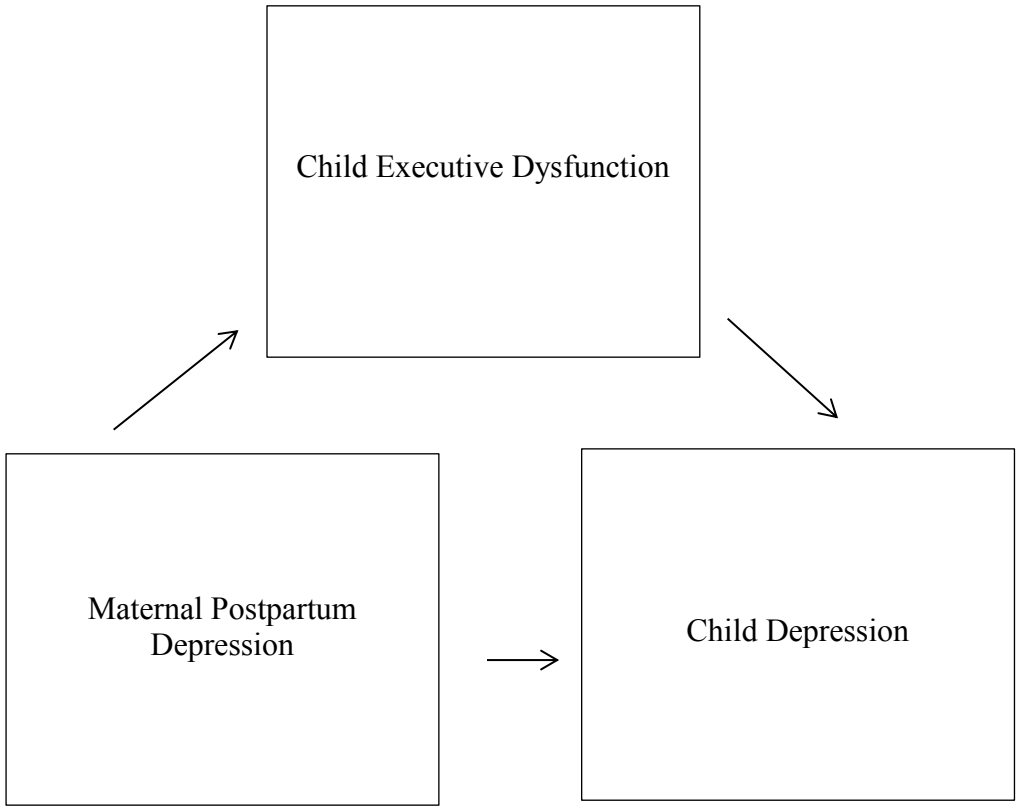

Figure 1. Conceptual relationship of partial mediation model (Hypothesis 2). The maternal education, family income, site of data collection, and child age at testing covariates are not graphically represented.

CHAPTER II

METHOD

Participants

The present study uses data from Phases I-IV of the Study of Early Child Care and Youth Development (SECCYD), sponsored by the National Institute of Child Health and Human Development (NICHD). Data collection began in 1991 and continued through 2007. The SECCYD followed 1,364 children from birth through age 15 at 10 data collection sites around the United States. Data collection sites included: University of Arkansas; University of California, Irvine; University of Kansas; University of New Hampshire; Pennsylvania State University; Temple University; University of Virginia; University of Washington; Western Carolina Center; and University of Wisconsin. The following criteria excluded families from participating in the study: those with mothers under the age of 18, those with maternal medical or substance abuse problems, those who anticipated moving, those with multiple birth infants who had disabilities or health concerns, and those residing in dangerous neighborhoods. During selected 24 hour periods, women giving birth at one of the ten locations were screened for willingness and eligibility to participate. During the sampling period, 8,986 mothers gave birth and 5,416 (60%) met eligibility requirements and agreed to be contacted by the study team. Of the 5,416 who agreed to be contacted, the team used a conditional random sample of 3,015 to be called two weeks later. This method allowed for adequate representation, at least 10%, of ethnicity minority mothers and mothers without a high school diploma. At the two-week call, families were excluded if the infant had complications causing him or her to be hospitalized for more than 7 days, if the family was planning to move in the next 3

years, or the family could not be reached during the first 3 attempts to contact. It is important to note that because of the exclusion criteria, this sample did not include many children experiencing various potential risk conditions and the developmental experiences associated with these risk conditions. As related to the present study, children predisposed to executive dysfunction due to birth complications (see *Other Etiologies of Executive Dysfunction* above) were excluded from the study.

Measures

Please see Table 1 for an outline of each measure and the time point at which it was measured.

Table 1

Variable Measures, the Time at Which They Were Measured, and Sample Sizes

Var	1m	6m	15m	24m	36m	1g	3g	4g	5g	6g	15y
CES-D	968	968	968	968	953						
CPT						820		747			
TOH						814			788		
Stroop											731
TOL											736
CBCL						842	816	809	807	809	768

Note. Var = variable, m = month, g = grade, y = years, CES-D = Center for Epidemiological Studies Depression Scale, CPT = Connors Continuous Performance Test, TOH = Tower of Hanoi, TOL = Tower of London, CBCL = Child Behavior Checklist; numbers denote sample sizes

Maternal Depression. Depressive symptoms in mothers were measured at child ages 1 month, 6 months, 15 months, 24 months, and 36 months with one of the most widely used self-report depression measures for non-clinical populations, the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977). Maternal depressive symptoms were not measured prior to or during pregnancy. Respondents report the frequency of 20 different depressive symptoms over the past week. Response categories include: 0 (*rarely or none of the time [less than 1 day]*), 1 (*some or little of the time [1-2*

days]), 2 (*occasionally or a moderate amount of the time [3-4 days]*), and 3 (*most or all of the time [5-7 days]*). The CES-D assesses symptoms such as mood, appetite, and self-esteem with items such as, "I felt I was just as good as other people," and "I was bothered by things that usually don't bother me." Scores range from 0 to 60 with scores of 16 or above suggesting a need for further assessment. The mean score in the general population is typically 7-9, while the mean score in a clinical population is typically 24-27. Internal consistency reliability was .90 in the clinical sample and .85 in the general population. Across two weeks, test-retest reliability correlations (n = 139) were .51; across 4 weeks (n = 105), .67; across 6 weeks (n = 97), .59; and across 8 weeks (n = 78), .59 (Radloff, 1977). The total test-retest correlation (n = 419) was .57. Correlations between other measures of depressive symptoms and the CES-D are high, which is evidence of the validity of this measure. Means on the CES-D are also higher for psychiatric samples than for nonclinical adults. In a study comparing CES-D scores in clinical and nonclinical samples, 70% of psychiatric patients scored above the cutoff, whereas 21% of the nonclinical sample scored above the cutoff. In a college sample, the CES-D was found to have a sensitivity of 86.7, specificity of 76.6, positive predictive value of 41.9, and negative predictive value of 96.7 (Shean & Baldwin, 2008).

Child Executive Function. Although many measures of EF assess more than one domain, this study strives to recognize measures of EF according to Miyake and colleagues' (2000) theory of EF as comprised by inhibition of responses, information updating, and mental set shifting. These classifications are different than those of the SECCYD; please see Table 2 for Miyake and colleagues' (2000) classification of each measure used in the present study. Additionally, it is important to note that this data set

does not provide information about the child's parents' executive functioning, so it is impossible to parse out heritability versus environmental influences.

Table 2

Measures of EF Based on Miyake and Colleagues' (2000) Model

Measure	Domain(s) of EF Measured	Source
CPT	Inhibition	Wilcutt, Doyle, Nigg, Faraone, & Pennington, 2005
TOH	Inhibition	Miyake et al., 2000
	Information Updating	Hull, Martin, Beier, Lane, & Hamilton, 2008
TOL	Inhibition	Baughman & Cooper, 2007
	Information Updating	Miyake et al., 2000
Stroop	Inhibition	Archibald & Kerns, 1999; Miyake et al., 2000
	Set-shifting	Spreen & Strauss, 1998

Note. CPT = Connors Continuous Performance Test, TOH = Tower of Hanoi, TOL = Tower of London

Inhibition. Inhibition in children was measured at 54 months, 1st grade, and 4th grade using the Conners Continuous Performance Task (CPT) for young children

(Mirsky et al., 1991). The CPT is a computer-generated task during which pictures of familiar objects (e.g. fish, butterfly, flower) are presented to the child on a 2 inch screen. The child was instructed to press a button every time one of the target stimuli appeared. At 54 months, ten stimuli were presented in each block over a course of 22 separate blocks. The stimulus appeared for 500 milliseconds and the interval between stimuli was 1500 milliseconds. The target stimulus was randomly shown twice within each block. The length of the test was approximately 7 minutes and 20 seconds. For the 1st grade assessment, stimuli were presented in 30 blocks, with 10 stimuli in each block. The stimulus appeared for 200 milliseconds and the interval between stimuli was 1500 milliseconds. Similar to 54 months, the target stimuli was randomly presented twice within each block. The length of this test was approximately 8.5 minutes. During the 4th grade assessment, stimuli were presented in 45 blocks of 12 stimuli. The target stimulus was the letter X appearing after the letter A. The stimulus appeared for 200 milliseconds and the interval between stimuli was 1500 milliseconds. The target stimulus was randomly presented twice during each block, and the overall task took about 15 minutes.

For the above tests, the following scores were provided: mean response time for target responses (hit reaction time), the number of targets to which the child did not respond (errors of omission), and the number of times the child responded to a non-target (errors of commission). The SECCYD chose this measure because it is the most widely used measure of sustained attention, there is evidence of reliability and validity across a wide age range of children, and it is a more pure measure of sustained attention than an observation of a child during solitary play or on an achievement task, both of which can be influence by knowledge or creativity (NICHD, 1993).

Halperin, Sharma, Greenblatt, and Schwartz (1991) examined the psychometric properties of this measure on a sample of non-referred boys ages 7-11 years from diverse sociocultural and ethnic backgrounds. These authors found that measures of impulsivity and inattention derived from the CPT had test reliability of .65-.74, which is in the adequate range. As a measure of attention, the CPT has good content and predictive validity (Halperin et al., 1991). Additionally, the CPT has been shown to predict cognitive function in school-age children and is sensitive to individual differences such as in those with ADHD and learning disorders (Barkley, DuPaul, & McMurray, 1990; Campbell et al., 1994).

Inhibition and Information Updating. Inhibition and information updating were measured using the Tower of Hanoi (TOH) at 1st grade and 5th grade and the Tower of London (TOL) at age 15. The TOH asked the child to transform an initial configuration of rings into a goal state. Specifically, this task required that the child move three rings of different colors and diameters along three vertical pegs. The rings were provided to the child in an initial configuration and the goal was for the child to move the rings along the three pegs to construct a tower on a specified peg with the rings ordered from largest to smallest, with the largest on the bottom. Children completing this task were bound by three rules: 1) they may only move one ring at a time, 2) they may not place larger rings on smaller rings, and 3) a ring must either be in the child's hand or on a peg. The goal was to construct the tower in the fewest number of moves.

The child's age was considered when administering this task. At first grade, the child began a task with 3 rings and 4 moves. However, at 3rd and 5th grade, the child began at the second task (3 rings, 5 moves). At 3rd and 5th grade, unlike at 1st grade, a

seventh task was added (4 rings, 15 moves) if the child successfully completed the sixth task (4 rings, 11 moves). A research assistant recorded the child's performance by considering each move the child made during each trial. The number of trials was determined by the number of times the research assistant placed the rings in the starting configuration, while each move occurred when the child lifted a ring off of a peg and placed in either on the same or a different peg. A total planning efficiency score was computed.

The TOH was chosen because it is an unfamiliar task for children, not tied to a specific knowledge base (NICDH, 1993); therefore, children from diverse backgrounds could approach this task with an equal opportunity for success. Additionally, the TOH has been found to be sensitive to age differences in normally-developing children (Welsh, 1991), and has been found to discriminate between normally-developing children and those with cognitive disabilities (Welsh, Pennington, Ozonoff, Rouse, & McCabe, 1990).

Ahonniska, Ahonen, Aro, Tolvanen, and Lyytinen (2000) examined the reliability and age effects of the Tower of Hanoi. They used a sample of two groups of children (7.7 years and 11.6 years) who completed the task three times each, with test-retest time intervals of 2 months. In both samples, they found improved performance and decreased performance time in repeating the assessments. Older children improved their performance more quickly than did younger children. Scores maintained stability through all the assessments and the reliability of all scores was satisfactory. Regarding validity, the Tower of Hanoi has been found to be sensitive to differences in neurological, developmental, and intellectual differences (Welsh et al., 1990), which provides construct validity.

The Tower of London (TOL) was also used to measure inhibition and information updating in study participants at age 15 (Berg & Byrd, 2002). The TOL task required participants to work on a computer to complete a puzzle-like activity. Specifically, they were asked to move three balls appearing on the screen from their starting positions to target positions, based on the goal that also appeared on the screen. The boards presented had three pegs. The tall peg held up to 3 balls, the middle peg up to 2, and the short peg only 1. Each puzzle could be solved in various numbers of minimum moves ranging from one move to seven moves. Participants were instructed to solve the puzzles in the fewest number of moves possible and as quickly as possible. Lifting the ball off of one peg and placing it on another counted as one move. Each participant was given a maximum of 4 minutes to complete each of 20 test trials. The TOL was chosen for this study because of support for its use in measuring the response inhibition aspect of cognitive planning (Asato, Sweeney, & Luna, 2006).

Multiple studies have found validity for the TOL task and its sensitivity to frontal lobe dysfunction. The TOL has been found to discriminate between adults with and without frontal lobe lesions, to detect Parkinson's patients with dopamine depletion in the frontal cortex, and to identify frontal lobe lesion volume in children (Schnirman, Welsh, & Retzlaff, 1998). Additionally, in a sample of children with ADHD, the TOL task produced the highest loading on an Executive Planning and Inhibition factor as compared to Psychometric Intelligence, Memory, and Executive Concept Formation and Flexibility (Culbertson & Zillmer, 1998). In a sample of college students, the TOL has found to have adequate internal consistency reliability ($\alpha = .79$) and test-retest reliability ($r = .70$) across two administrations (Schnirman et al., 1998). One notable weakness is that this

task was administered to college students, so it is difficult to generalize these psychometric properties to a wider age range.

Inhibition and Set-Shifting. The Stroop Task (Stroop, 1935) was used to measure inhibition and set-shifting at age 15. This was a computerized tasked during which participants were instructed to press a button matching the color of the presented word while ignoring what the word said. Color responses included blue, green, yellow, and red. Until the participant reached 75% accuracy, practice trials were displayed; if the child did not exceed 75% accuracy by the second practice trial block, the task was discontinued. Ninety-six trials composed the complete tasks, and within each block there were neutral trials and incongruent trials. Neutral trials occurred when a neutral word (add, divide, equal, or math) was provided in one of the four possible colors. Incongruent trials occurred when the words blue, green, red, or yellow were written in a different color of ink than the word indicated. Responses were scored for speed and accuracy. A Total Interference Score on All Trials was calculated by subtracting the average response time for neutral trials from the average response time for incongruent trials. This number was then adjusted for baseline differences in response time by dividing the difference score by the average response time for neutral trials. Lower scores indicated less interference and therefore better performance.

The Stroop task is one of the most commonly used measures of inhibitory control (MacLeod, 1991) and has been associated with resistance to interference from outside stimuli, cognitive flexibility, psychopathology, and creativity. When a conflict between the meaning of the word and the color of the word appears, the Stroop effect demonstrates a delayed processing of the word's color, which leads to slower reaction

times and more mistakes. In test-retest reliability intervals of three minutes, one day, and one week, an individual Stroop Test administration was found to have the following reliabilities: .86, .82, and .73, respectively (Jensen, 1965).

Child Internalizing Symptoms. The Child Behavior Checklist (CBCL; Achenbach, 1991) was used to measure internalizing behaviors in children at 3rd grade, 4th grade, 5th grade, 6th grade, and age 15. It is the most widely-used screening tool for identifying and tracking the emergence of problematic behavior in children ages 4-18. The CBCL is highly reliable and internally consistent and there is extensive evidence of validity. Children in a clinical sample have been found to receive elevated scores on this measure; further, elevated scores are predictive of both the onset and continuation of problematic behaviors. The person completing the CBCL (either a parent or teacher) rates the child's behavior on a 3 point scale. A computer program generates both broad band syndrome scores (such as internalizing and externalizing behaviors) and narrow band scores (such as delinquency, attention problems, and aggression). This study will use the Internalizing Behaviors subscale to capture depressive symptoms in children. Internalizing Behaviors is a broad-band subscale comprised of the following narrow-band subscales: Anxiety/Depression, Withdrawn, and Somatic Complaints. A broad-band subscale will capture a broader sample of children struggling with depressive symptoms.

During SECCYD data collection in 2001, a newer version of the CBCL was introduced (CBCL/6-18; Achenbach & Rescorla, 2001). Major differences include an updated normative sample, a change in the lower limit of the age range, and an addition of six items. However, Achenbach and Rescorla (2001) noted that, "most children's scores would rank at nearly the same level on the new and 1991 versions," and, "if a

child's functioning has not changed much between assessments on the 1991 and new versions of a form, the child's syndrome scores should be equivalent to about the same percentiles and T scores on each version" (p. 166).

Contextual Variables. Maternal education and family income have been associated with child EF and general cognitive development (Hay & Kumar, 1995; Hughes et al., 2013), and are used as covariates in this study. Maternal education was reported in number of years, and family income was reported in dollars earned per year for the total household. Although Hughes and colleagues (2013) also identified maternal scaffolding, and the stability of EF at age 2 as important covariates, this data set did not capture these variables. Additional covariates important to this data set include site of data collection and child age of testing (Watamura et al., 2011).

Depression Grouping. For purposes of grouping mothers based on their depression trajectories, the CES-D was turned into a binary variable based on a cutoff score of 16 which indicates significant depressive symptomatology (Radloff, 1977). Depressive symptomatology was used as a categorical rather than continuous variable for the purpose of examining factors such as the timing and chronicity of the depressive symptoms. The current study strives to categorize depression into groups based on previous studies. One previous study of perinatal depressive trajectories categorized depression into 5 groups: 1) *non-depressed*, 2) *antepartum only*, 3) *postpartum only*, resolving after the first year, 4) *late*, present at 25 months postpartum, and 5) *chronic*, or always depressed (Mora et al., 2009). However, given the available data in the current study, depression is categorized into 4 groups: 1) *non-depressed,* 2), *postpartum only*, resolving after the first year, 3) *early childhood only*, present after the first year, and 4)

chronic, or depressed both during the postpartum and early childhood periods. Also, the current study utilized data with more measurement points of maternal depressive symptoms than the above study and will classify *early childhood only* as existing after the postpartum period, rather than 25 months postpartum.

Analytic Approach

All statistical analyses were conducted using SPSS Statistics Software (IBM Corp, 2010).

Hypothesis 1. *Early chronic maternal postpartum depression will have a negative and enduring impact on measures of executive function in children in 1st grade, 4th grade, 5th grade, and at 15 years of age.* Operationally, this hypothesis predicted that maternal postpartum depression would be associated with lower scores in 1st grade on the CPT (Proportion of Correct Responses) and Tower of Hanoi (Total Planning Efficiency Score), 4th grade on the CPT (Proportion of Correct Responses), 5th grade on the Tower of Hanoi (Total Planning Efficient Score) and age 15 on the Tower of London (Total Percent of Trials Solved) and Stroop Test (Interference Score on All Trials). CES-D scores were converted into binary variables based on a cutoff score of 16. Mothers were categorized as depressed or not depressed based on the score for each measurement (at five different time points: 1 month, 6 months, 15 months, 24 months, and 36 months). Then, the number and timing of the depressive episodes were considered to create four groups: *not depressed*, *postpartum depression* (the first year after birth), *early childhood depression* (beginning after the first year after birth), and *chronic depression* (both postpartum and early childhood depression). The initial analytic plan was to consider a neuropsychological profile using a MANCOVA with the child's CPT and Tower of

Hanoi scores at 1st grade, CPT score at 4th grade, Tower of Hanoi at 5th grade, Tower of London at age 15, and Stroop Test at age 15. I then planned to use five separate ANCOVAs to examine the impact of maternal depression on each EF task separately because each EF measure had a different sample size, so separating the analysis would allow for the most power within each model.

However, this plan was modified when it became clear that the EF measures had low correlations. The MANCOVA approach was abandoned in favor of separate ANCOVAs. Covariates included maternal education, total family income, site of data collection, and age at assessment.

Hypothesis 2. *Children in the chronic depression group who experience executive dysfunction will be more likely to experience subsequent depressive symptoms; that is, the relationship between early chronic maternal depression and later child depression will be partially mediated by child executive dysfunction* (See Figure 2 below for a graphical depiction). According to the methods suggested by Baron and Kenny (1986), multiple models should be separately tested and then, together, combined to perform a test of mediation. Initial analytic plan was as follows:

Model 1. I planned to run one MANCOVA looking first at the effect of chronic maternal depression (none, postpartum depression, early childhood depression, or chronic depression) on EF measures at 1st grade (TOH and CPT). I then planned to run a separate MANCOVA looking at neuropsychological profiles at 1st grade (TOH and CPT) on child depression at 3rd, 4th, 5th, and 6th grades, and at age 15 which would have provided me with regression coefficients for the association between maternal depression and child EF (and its standard error) and coefficients for the association between child EF and child

depression (and its standard error). Children experiencing depression at 1st grade would be dropped to ensure that lower EF scores during that time were not due to depression.

However, as previously stated, EF measures were not correlated enough to run a MANCOVA, so ANCOVAs were used instead when EF measures were used as DVs (see Results section). Because child internalizing symptoms were highly correlated, MANCOVAs were used when CBCL scores were used as DVs. Covariates included maternal education, total family income, site of data collection, and age at assessment.

Model 2. Next, I planned to run a MANCOVA looking at the effect of chronic maternal depression (none, postpartum depression, early childhood depression, or chronic depression) on neuropsychological profiles at 4th grade (CPT) and 5th grade (TOH) and then a separate model looking at these profiles on child depression at 6th grade and 15. I planned to run *Model 1* and *Model 2* separately to examine only potential depressive symptoms occurring after executive dysfunction. *Model 2* would have provided me with regression coefficients for the association between maternal depression and child EF (and its standard error) and coefficients for the association between child EF and child depression (and its standard error). Children experiencing depression at 4th and 5th grades would be dropped to ensure that lower EF scores during that time were not due to depression.

However, as previously stated, EF measures were not correlated enough to run a MANCOVA, so ANCOVAs were used instead when EF measures were used as DVs (see Results section). Because child internalizing symptoms were highly correlated, MANCOVAs were used when CBCL scores were used as DVs. Covariates included maternal education, total family income, site of data collection, and age at assessment.

Sobel Tests. With the regression coefficients and standard errors for both models above, I planned to utilize a program to calculate the critical ratio to determine whether the indirect effect of maternal depression on child depression via child executive dysfunction was significantly different than zero. However, after initial results were null, it became unnecessary to examine a mediation model.

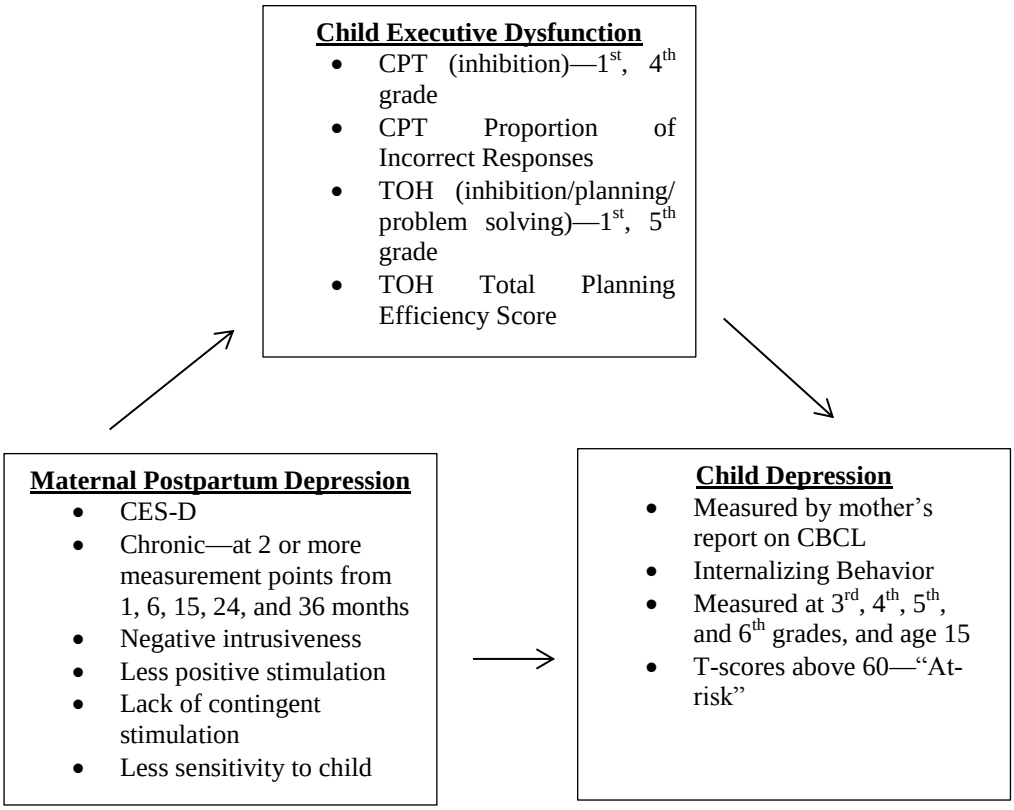

Figure 2. Relationship of partial mediation model (Hypothesis 2). The maternal education and family income covariates are not graphically represented.

CHAPTER III

RESULTS

Prior to the analyses for main hypotheses, frequencies and descriptive statistics for primary variables were calculated (see Appendix A). Assumptions for the proposed models were then checked and models were evaluated with some post-hoc respecification (described below) where justified by the conceptual framework of the study.

Descriptive Statistics

Descriptive statistics were screened for extreme outliers, floor and ceiling effects, and distribution shape. Although other variables showed roughly normal distributions with no significant outliers, total family income had a strong positive skew. Descriptive statistics and frequencies for demographic and primary study variables can be found in Appendix A. Covariates used in all models include total family income, maternal education, site of data collection, and child's age at assessment.

The initial planned analyses included two MANCOVAs, but upon examination of dependent variable correlations, ANCOVAs seemed to be more appropriate. Correlations between dependent variables were lower than expected, given that all measures assessed executive functioning. This presents some evidence for disparate executive functioning skills, as opposed to one underlying unified skill. See table below for dependent variable correlations. Besides low correlations between dependent variables, greater N sizes for each model provided further support for individual ANCOVAs.

Table 3

Correlations Between Dependent Variables

Variable	CPT G1	TOH G1	CPT G4	TOH G5	TOL A15	Stroop A15
CPT G1	1	.118	.276	.142	.150	-.071
TOH G1	.118	1	.132	.345	.233	-.026
CPT G4	.276	.132	1	.199	.178	-.087
TOH G5	.142	.345	.199	1	.260	-.013
TOL A15	.138	.233	.178	.260	1	-.031
Stroop A15	-.071	-.026	-.087	-.013	.013	1

Note. CPT = Connors Continuous Performance Test, TOH = Tower of Hanoi, TOL = Tower of London, G = Grade, A = Age

All models were tested for assumptions. In many cases there were no violations. When assumptions were violated, follow-up examination indicated that the violations were an artifact of the large sample size (see Appendix C).

Prior to examining the hypotheses, maternal depression's impact on child depression was examined to replicate past studies (Downey & Coyne, 1990; Cummings & Davies, 1994). The sample size was 677 and the following covariates were included: maternal education, site of data collection, and total family income. The independent variable (depression) and aforementioned covariates were examined to determine if any interactions were present between independent variables and covariates. There were no

significant interactions, so this assumption was not violated. The test of equality of covariance matrices was not significant, Box's M = 1030.285, F(735, 22730.820) = 1.082, p = .065. Results indicated that maternal depression was significantly associated with child depression at all time points, with the largest effect at grade 1 and the smallest at age 15 (see below).

Table 4

MANCOVA Results for Maternal Depression on Child Depression

Variable	*Variable*	*df*	*F*	*p*	*partial* η^2
Dep Pattern	CBCL G1	3	12.050	.000	.054
	CBCL G3	3	10.364	.000	.047
	CBCL G4	3	7.951	.000	.036
	CBCL G5	3	8.117	.000	.037
	CBCL G6	3	9.882	.000	.045
	CBCL 15	3	6.685	.000	.031
	Error	635			

For descriptive statistics, please see Table 5; for graphic depiction, please see Figure 3.

Table 5

Maternal Depression Group Differences on Later Child Depression

CBCL Internalizing	*Maternal Depression*	*Mean T Score*	*95 % CI* *Lower Bound*	*Upper Bound*
G1	None	47.019	46.159	47.880
	Postpartum	46.361	44.460	48.262
	Ear. Childhood	49.399	47.692	51.105
	Chronic	52.225	50.621	53.830
G3	None	46.966	45.973	47.960
	Postpartum	47.307	45.112	49.501
	Ear. Childhood	47.920	45.950	49.890
	Chronic	52.908	51.055	54.761
G4	None	46.562	45.595	47.528
	Postpartum	46.449	44.314	48.585
	Ear. Childhood	47.551	45.634	49.468
	Chronic	51.568	49.765	53.370
G5	None	46.952	45.991	47.914
	Postpartum	48.376	46.252	50.501
	Ear. Childhood	48.683	46.775	50.590
	Chronic	52.099	50.306	53.892
G6	None	43.879	42.846	44.911
	Postpartum	45.007	42.726	47.288
	Ear. Childhood	45.688	43.640	47.736

Table 5 Cont'd

	Chronic	48.547	46.621	50.472
Age 15	None	45.474	44.492	46.457
	Postpartum	45.439	43.269	47.609
	Ear. Childhood	47.236	45.288	49.183
	Chronic	50.095	48.263	51.927

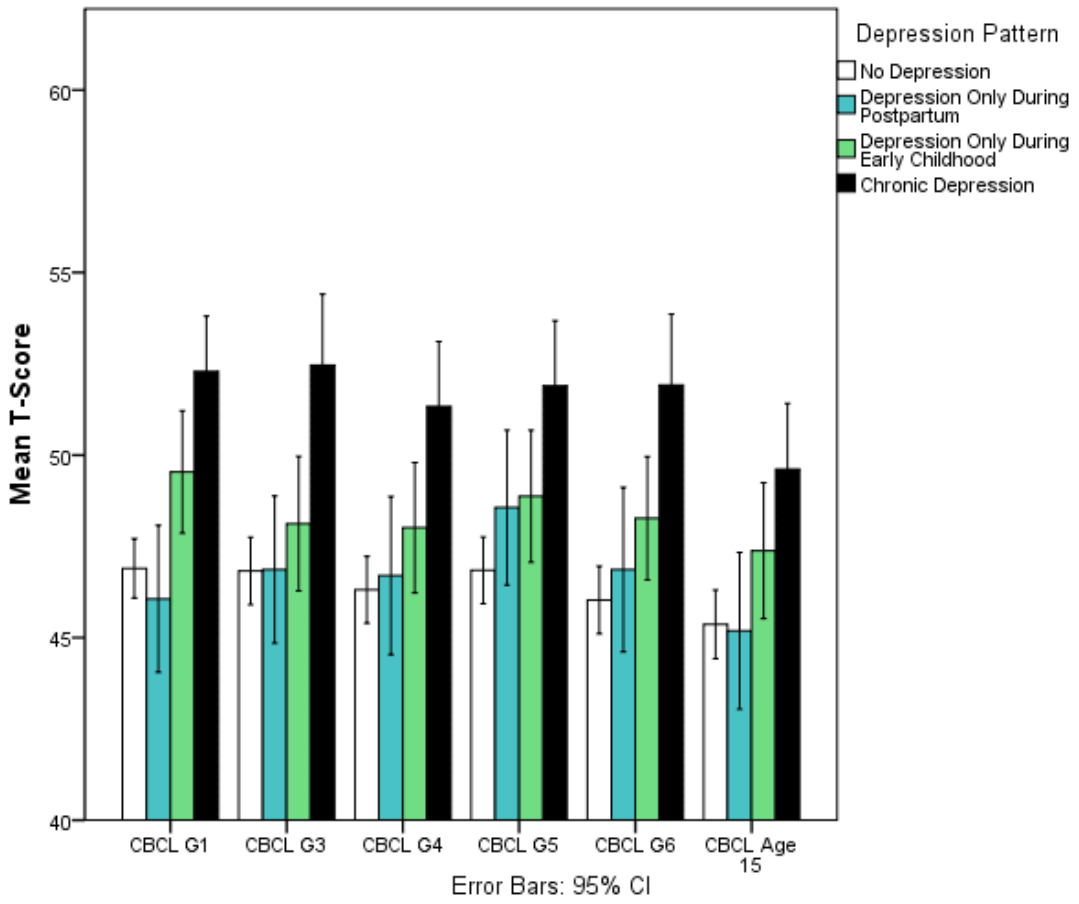

Figure 3. Maternal Depression Group Differences on Later Child Internalizing.

Hypothesis One: Chronic maternal depression will have a negative and enduring impact on measures of executive function in children in 1st grade, 4th grade, 5th grade, and at 15 years of age.

Summary of Results. Overall, maternal depression pattern was not associated with child executive functioning scores, as hypothesized. However, an exploratory model revealed that males with mothers who experienced depression at any time scored significantly poorer on the TOH at grade 1 than males with non-depressed mothers, and males with mothers who had postpartum or chronic depression also scored significantly poorer on the TOH at grade 5 (see Figures 7 and 8). Additionally, Caucasian, Non-Hispanic children with mothers who had postpartum depression scored significantly better on the CPT at grade 4 than children with chronically depressed mothers (see Figure 4).

Results. The first hypothesis included six separate ANCOVAs with maternal depression pattern as the independent variable and the following dependent variables (one per model): CPT 1st grade (Model 1A), TOH 1st grade (Model 1B), CPT 4th grade (Model 1C), TOH 5th grade (Model 1D), TOL Age 15 (Model 1E) and Stroop Age 15 (Model 1F). The original number of participants in the data set was 1364. After accounting for missing data, the N for each model appears below in Table 6.

Table 6

N Values for Each Model

Model (DV)	N
1A (CPT G1)	552
1B (TOH G1)	805
1C (CPT G4)	585
1D (TOH G5)	778
1E (TOL Age 15)	726
1F (Stroop Age 15)	721

In addition to the 411 cases with missing data on depression, the following issues accounted for missingness (from months 1 – 36): 400 from the CPT at grade 1, 146 from the TOH at grade 1, 368 from the CPT at grade 4, 173 from the TOH at grade 5, 225 from the TOL at age 15, 230 from the Stroop at age 15, 2 Family Income, 141 from CPT age at grade 1, 146 from TOH age at grade 1, 213 from CPT age at grade 4, 193 from TOH at grade 5, 224 from TOL at age 15, and 224 from Stroop at age 15. Extreme outliers were present in CPT Grade 1 and CPT Grade 4. Because these two variables had a negative skew, data was transformed by reverse scoring items (CPT was reverse scored using 1 – Proportion of Correct Responses) and then taking the natural log of those items. These were then reverse scored again (0 – Score) to make values easier to interpret, with a higher number indicating a better score. No extreme outliers were present in TOH

Grade 1, TOH Grade 5, or TOL at age 15, so these data were not transformed.

Histograms of all DVs indicated that the data were unimodal and normally distributed, with skewness and kurtosis values within the acceptable range (less than 2). See Table 7 below for skewness and kurtosis values.

Table 7

Skewness and Kurtosis Values for Hypothesis 1 Dependent Variables

Variable	Skewness	Kurtosis
CPT, Grade 1 (Transformed)	-.64	-.40
TOH, Grade 1	.36	-.40
CPT, Grade 4 (Transformed)	-.33	-.38
TOH, Grade 5	-.77	.34
TOL, Age 15	.13	-.15
Stroop, Age 15	.30	.12

All models were tested for assumptions. In most cases, there were no violations. However, when significant IV and CV interactions were noted, they were interpreted and included in the final models.

Model 1 (Proposed) – Effects of Maternal Depression Pattern on Child Executive Functioning Scores.

Model 1A Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1A (DV of CPT G1) to determine if any interactions were present between independent variables and covariates (see Appendix C). A significant interaction was noted between depression and site of data collection, $F(27, 500) = 1.612$, $p = .028$, *partial* $\eta^2 = .080$. Upon visual examination of an interaction plot, it was determined that there was no meaningful interpretation, so it was excluded. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 512) = 1.232$, $p = .163$.

Model 1A Results. An ANCOVA with depression pattern as the IV and CPT G1 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in children at grade 1, $F(3, 536) = 1.044$, $p = .373$, *partial* $\eta^2 = .006$.

Model 1B Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1B (DV of TOH G1) to determine if any interactions were present between independent variables and covariates (see Appendix C). There were no significant interactions, so this assumption was not violated. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 765) = 0.951$, $p = .558$.

Model 1B Results. An ANCOVA with depression pattern as the IV and TOH G1 as the DV revealed that maternal depression did not have a significant association with TOH scores in children at grade 1, $F(3, 789) = 2.498$, $p = .059$, *partial* $\eta^2 = .009$.

Model 1C Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1C (DV of CPT G4) to determine if any interactions were present between independent variables and covariates (see Appendix C). Significant interactions were noted between depression pattern and maternal education and depression pattern and total income Model 1C (see Appendix C). Because these interactions were small in magnitude (*partial* η^2 = .017 and .024, respectively), they were retained as covariates and the interaction terms were left in the model to capture this variance. Visual examination of interaction plots indicated that children with mothers with early childhood depression and chronic depression scored higher on the CPT G4 when their mothers were more educated than the mean level of maternal education. Regarding the income and depression interaction, children from higher income families scored higher on the CPT G4 when their mothers had postpartum or chronic depression, but lower when their mothers had early childhood depression. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 545) = .831$, $p = .759$.

Model 1C Results. An ANCOVA with depression pattern as the IV and CPT G4 as the DV revealed that maternal depression did not have a significant association with CPT scores in children at grade 4, $F(3, 563) = 2.365$, $p = .070$, *partial* η^2 = .012.

Model 1D Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1D (DV of TOH G5) to determine if any interactions were present between independent variables and covariates (see Appendix C

for tables). There were no significant interactions, so this assumption was not violated. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 738) = 1.360$, $p = .073$.

Model 1D Results. An ANCOVA with depression pattern as the IV and TOH G5 as the DV revealed that maternal depression did not have a significant association with TOH scores in children at grade 5, $F(3, 762) = 1.037$, $p = .376$, *partial* $\eta^2 = .004$.

Model 1E Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1E (DV of TOL Age 15) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions, so this assumption was not violated. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 686) = 1.029$, $p = .423$.

Model 1E Results. An ANCOVA with depression pattern as the IV and TOL Age 15 as the DV revealed that maternal depression did not have a significant association with TOH scores in children at age 15, $F(3, 710) = 1.005$, $p = .390$, *partial* $\eta^2 = .004$.

Model 1F Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 1F (DV of Stroop Age 15) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions, so this assumption was not violated. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 678) = 1.014$, $p = .448$.

Model 1F Results. An ANCOVA with depression pattern as the IV and Stroop Age 15 as the DV revealed that maternal depression did not have a significant association with Stroop scores in children at age 15, $F(3, 702) = 1.126$, $p = .338$, *partial* $\eta^2 = .005$.

Exploratory Results Summary (Models 2 and 3). As no significant findings support the primary hypotheses, additional analyses were done in order to understand potential differences based on participant characteristics. The following models are exploratory in nature and are interpreted with caution, especially in cases where *p* values are close to .05. This section serves as a synthesis of results to follow. Please see specific model results for *F* strings.

Although specific model findings are presented below, overall results suggested that males with depressed mothers in any group scored significantly poorer on the TOH at grade 1 than males with non-depressed mothers, and males with mothers who had early childhood or chronic depression also scored significantly poorer on the TOH at grade 5 (see Figures 8 and 9 in Discussion). Additionally, Caucasian, Non-Hispanic children with mothers who had postpartum depression scored significantly better on the CPT at grade 4 than children with chronically depressed mothers (see Figure 4 below).

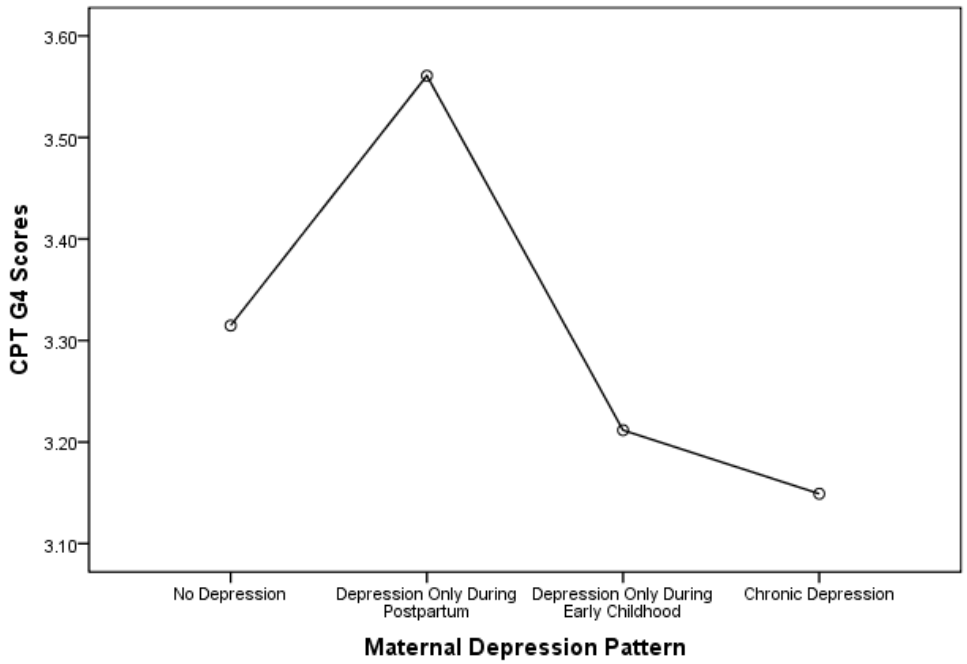

Figure 4. Depression Pattern on Caucasian, Non-Hispanic CPT G4 Scores.

A significant interaction was observed between depression and total family income on female TOH scores at grade 5, indicating that lower income was associated with poorer TOH G5 scores when the mother had postpartum or chronic depression (see Figure 5 below). Moreover, a significant interaction was observed between depression and maternal education for male Stroop scores at age 15, indicating that children with mothers who had more education scored more poorly than children with less educated mothers when the mother had postpartum depression but better when the mother was depressed during early childhood (see Figure 6 below). Also, there was a significant interaction between depression and child age at testing for a model examining Caucasian

children's CPT scores at grade 1. Visual examination of a plot indicated that younger children performed better on the CPT G1 during all maternal depression groups, but poorer with no maternal depression (see Figure 7 below). There were significant interactions between depression and maternal education, and depression and total income, in a model examining Caucasian children's CPT G4 scores. Visual examination of plots indicated that children with more educated mothers scored better on the CPT G4 when their mothers had early childhood and chronic depression (see Figure 8), and children from higher income families scored better than children from low income families when the mother had postpartum or chronic depression, but not early childhood depression (see Figure 9).

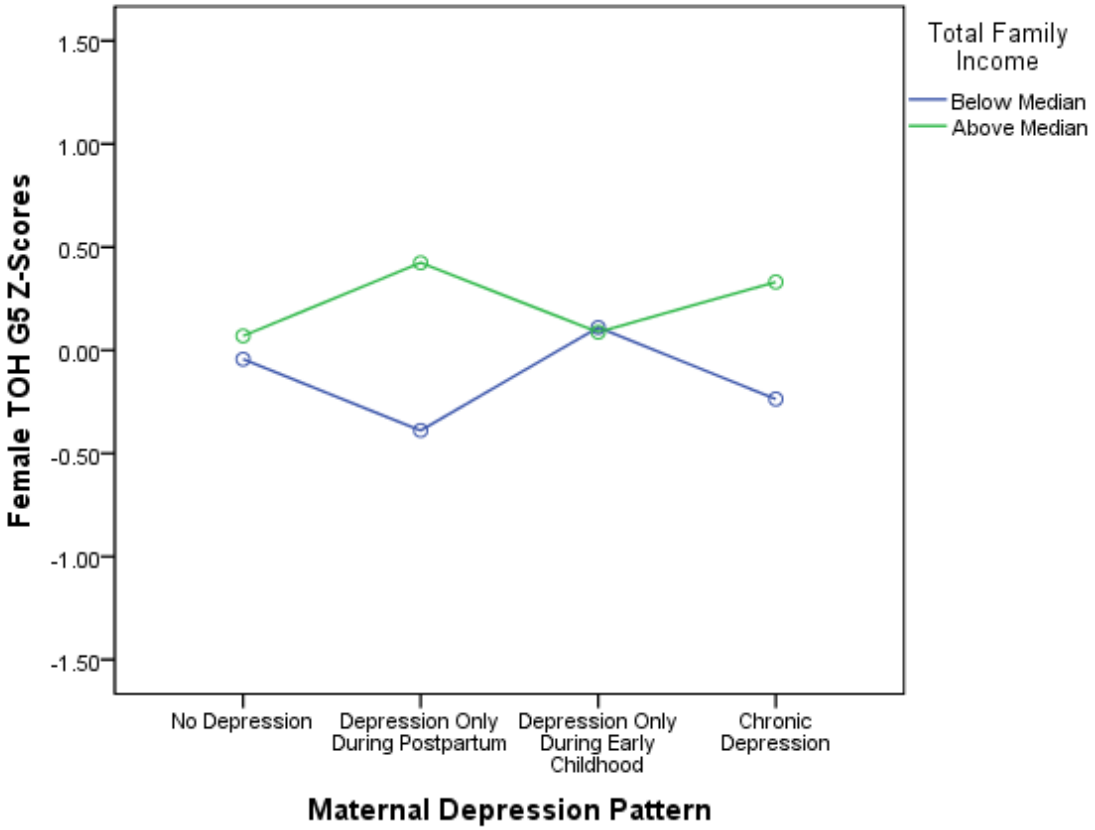

*Figure 5. Depression*Total Family Income on Female TOH G5 Z-Scores.*

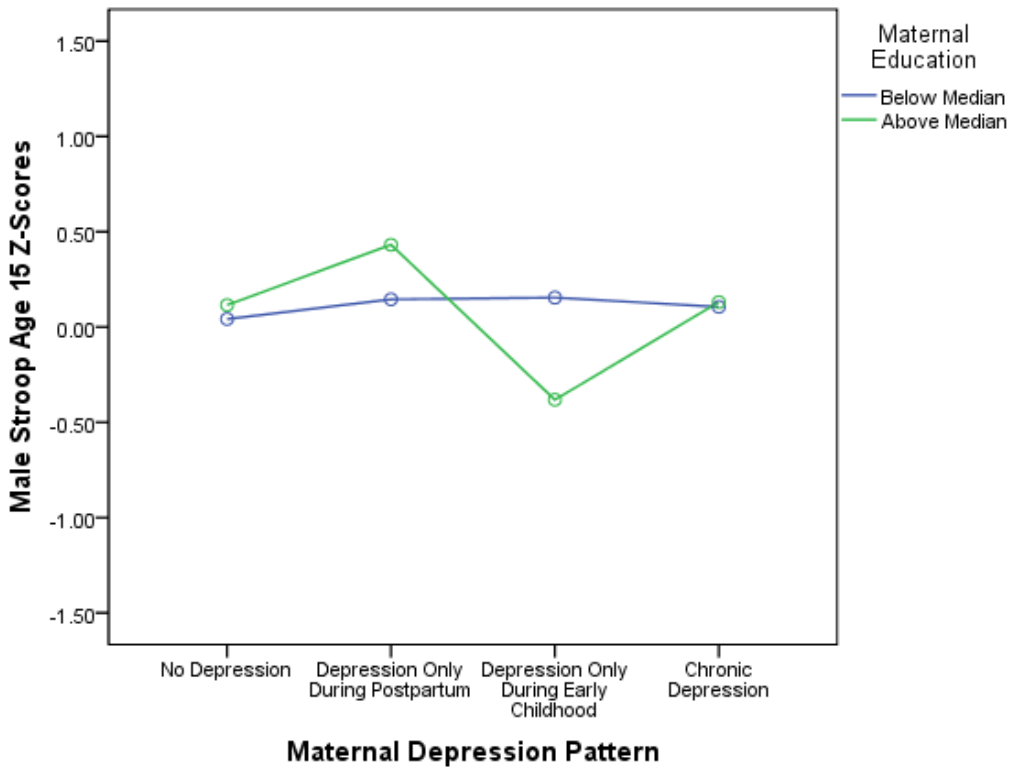

*Figure 6. Depression*Maternal Education on Male Stroop Z-Scores at Age 15.*

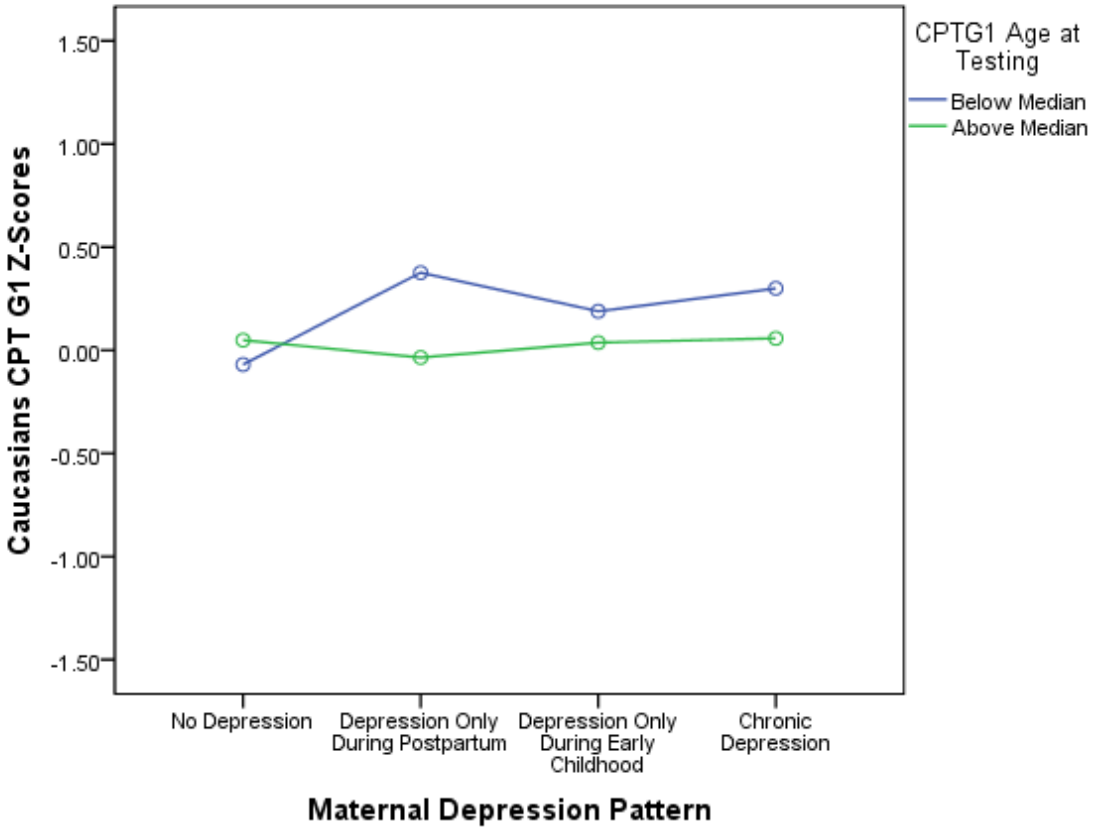

*Figure 7. Depression*Child Age at Testing on Caucasian CPT G1 Z- Scores.*

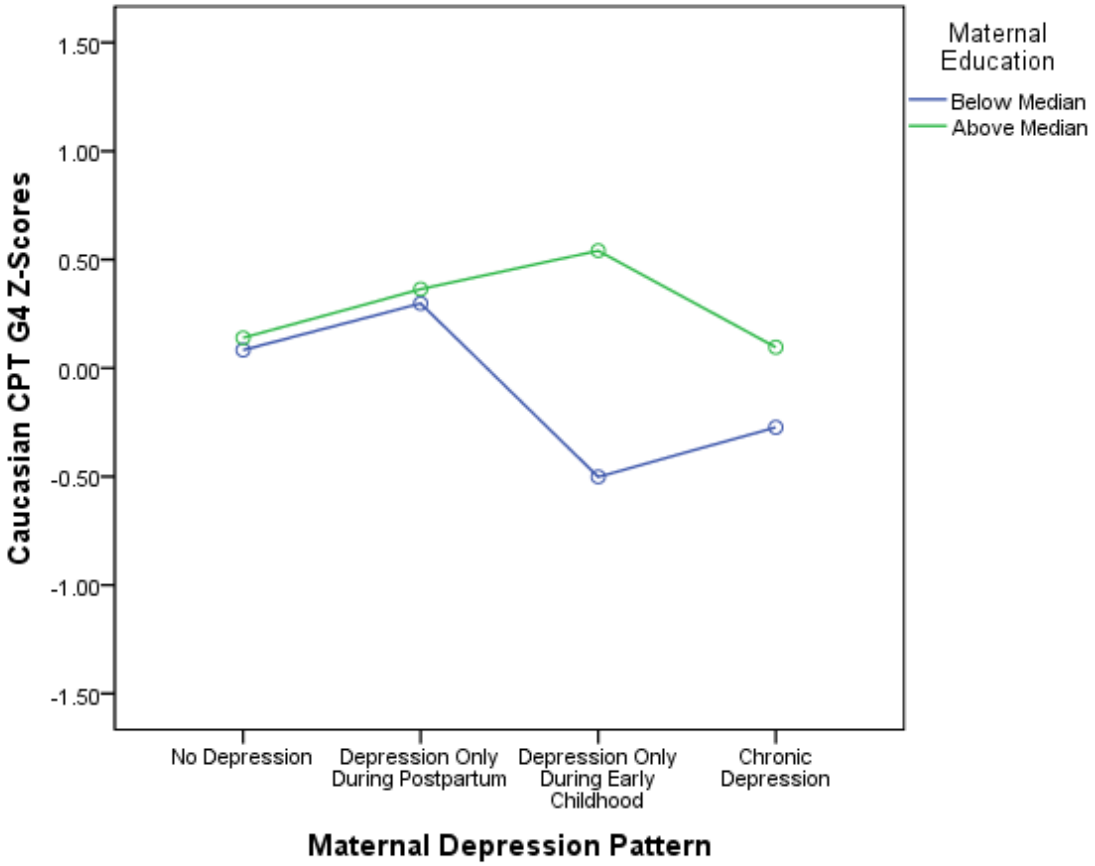

*Figure 8. Depression*Maternal Education on Caucasian CPT G4 Z-Scores.*

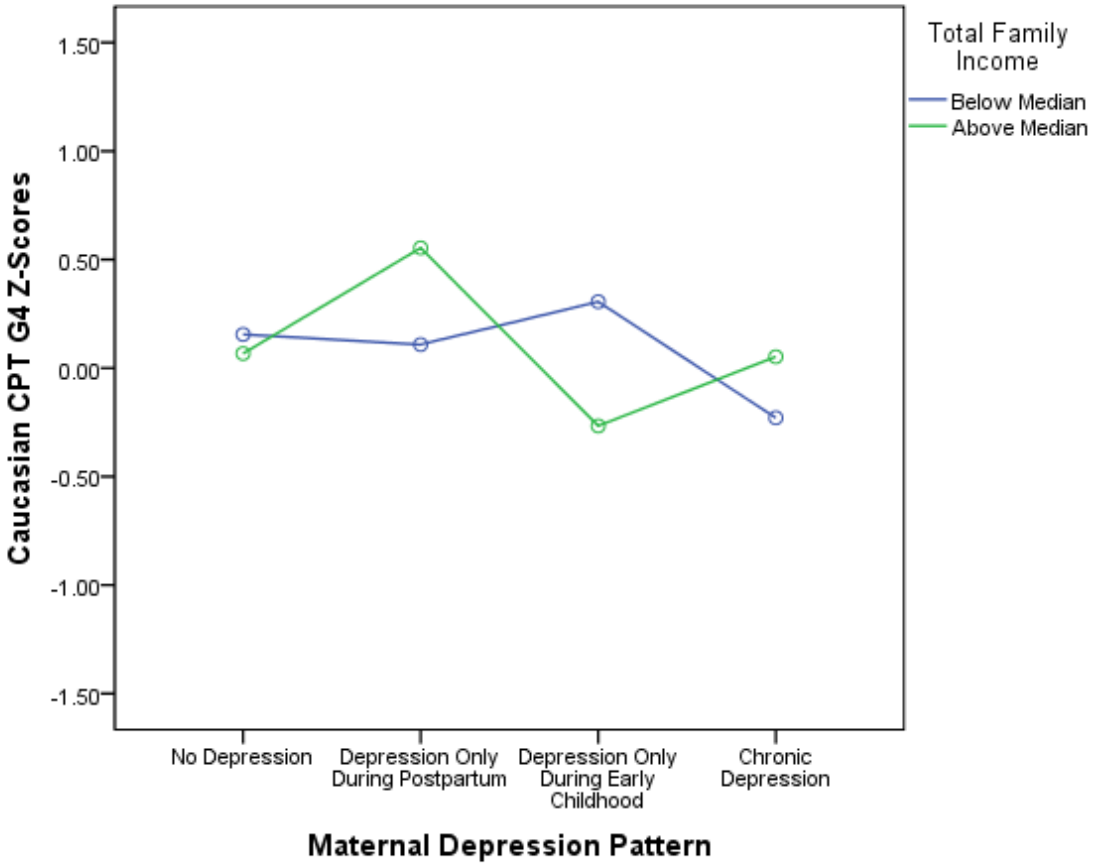

*Figure 9. Depression*Total Family Income on Caucasian CPT G4 Z-Scores.*

Model 2 (Exploratory) – Effects of Maternal Depression Pattern on Male Versus Female Child Executive Functioning Scores.

Although there is some evidence that maternal depression negatively impacts child executive functioning development (Hughes et al., 2013), potential gender differences have not been delineated. By contrast, in the cognitive development literature, maternal depression has been shown to have a detrimental effect on male cognitive development, but not that of females (Murray, 1992; Sharp et al., 1995). This

cognitive development literature support was applied in an exploratory model to examine potential gender differences in executive functioning in children with depressed mothers.

This exploratory analysis included twelve separate ANCOVAs with maternal depression pattern as the independent variable and the following dependent variables (one per model): CPT 1st grade, Males (Model 2A, Males), CPT 1st Grade, Females (Model 2A, Females), TOH 1st grade, Males (Model 2B, Males), TOH 1st grade, Females (Model 2B, Females), CPT 4th grade, Males (Model 2C, Males), CPT 4th grade, Females (Model 2C, Females), TOH 5th grade, Males (Model 2D, Males), TOH 5th grade, Females (Model 2D, Females), TOL Age 15, Males (Model 2E, Males), TOL Age 15, Females (Model 2E, Females), Stroop Age 15, Males (Model 2F, Males), and Stroop Age 15, Females (Model 2F, Females). The original number of participants in the data set was 1364. Please see Appendix C for assumptions. After accounting for missing data, the N for each model was as follows.

Table 8

N Values for Each Model

Model (DV)	N
2A, Males (CPT G1)	278
2A, Females (CPT G1)	274
2B, Males (TOH G1)	397
2B, Females (TOH G1)	408
2C, Males (CPT G4)	287
2C, Females (CPT G4)	298
2D, Males (TOH G5)	383
2D, Females (TOH G5)	395
2E, Males (TOL Age 15)	348
2E, Females (TOL Age 15)	378
2F, Males (Stroop Age 15)	343
2F, Females (Stroop, Age 15)	375

Model 2A Males Results. A males-only ANCOVA model with depression pattern as the IV and CPT G1 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in male children at grade 1, $F(3, 262) = .011$, $p = .999$, *partial* $\eta^2 < .001$.

Model 2A Females Results. A females-only ANCOVA model with depression pattern as the IV and CPT G1 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in female children at grade 1, $F(3, 255) = .042$, $p = .988$, *partial* $\eta^2 = .000$.

Model 2B Males Results. A males-only ANCOVA model with depression pattern as the IV and TOH G1 as the DV revealed that there was a significant association between maternal depression pattern and TOH scores in male children at grade 1, $F(3, 381) = 5.625$, $p = .001$, *partial* $\eta^2 = .042$. Pairwise comparisons suggested that males with non-depressed mothers had significantly better scores (M=15.13) than males with mothers experiencing depression only during postpartum (M= 12.07, $p = .003$), males with mothers experiencing depression only during early childhood (M = 12.31, p =.006) and males with chronically depressed mothers (M = 12.67, p =.007). There were no significant differences between male TOH scores in the different depression groups.

Model 2B Females Results. A females-only ANCOVA model with depression pattern as the IV and TOH G1 as the DV revealed that maternal depression pattern did not have a significant association with TOH scores in female children at grade 1, $F(3, 389) = 1.589$, $p = .191$, *partial* $\eta^2 = .012$.

Model 2C Males Results. A males-only ANCOVA model with depression pattern as the IV and CPT G4 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in male children at grade 4, $F(3, 271) = 1.138$, $p = .334$, *partial* $\eta^2 = .012$.

Model 2C Females Results. A females-only ANCOVA model with depression pattern as the IV and CPT G4 as the DV revealed that maternal depression pattern was

significantly associated with CPT scores in female children at grade 4, $F(3, 279) = 2.662$, $p = .048$, *partial* $\eta^2 = .028$. However, there were no significant pairwise comparisons, likely due to smaller N in the pairwise comparisons than in the omnibus test (no depression vs. depression postpartum, $p = .950$; no depression vs. depression only during early childhood, $p = .622$; no depression vs. chronic depression, $p = .939$; depression postpartum vs. early childhood depression, $p = .671$; postpartum depression vs. chronic depression, $p = .994$; early childhood depression vs. chronic depression, $p = .646$; see Appendix A for means tables).

Model 2D Males Results. A males-only ANCOVA model with depression pattern as the IV and TOH G5 as the DV revealed that there was a significant association between maternal depression pattern and TOH scores in male children at grade 5, $F(3, 367) = 3.363$, $p = .019$, *partial* $\eta^2 = .027$. Pairwise comparisons suggested that males with non-depressed mothers had significantly better scores (M=24.17) than males with mothers experiencing depression only during early childhood ($M = 20.94$, $p = .010$) and males with chronically depressed mothers ($M = 21.85$, $p = .026$), but not different than males with mothers experiencing depression only during postpartum ($M = 22.24$, $p = .103$). There were no significant differences between male TOH scores in the different depression groups.

Model 2D Females Results. A females-only ANCOVA model with depression pattern as the IV and TOH G5 as the DV revealed that maternal depression pattern had a significant association with TOH scores in female children at grade 5, $F(3, 376) = 3.952$, $p = .009$, *partial* $\eta^2 = .031$. However, when post hoc pairwise comparisons were examined, there were no significant differences (no depression vs. depression

postpartum, p = .793; no depression vs. depression only during early childhood, p = .515; no depression vs. chronic depression, p = .581; depression postpartum vs. early childhood depression, p = .799; postpartum depression vs. chronic depression, p = .838; early childhood depression vs. chronic depression, p = .964; see Appendix A for means tables). A significant main effect without significant pairwise comparisons could have occurred due to loss of N with pairwise comparisons. A significant interaction was observed between depression and total family income, $F(3,376) = 5.349$, p = .001, *partial* η^2 = .041. Visual examination of the interaction indicated that lower income was associated with poorer TOH G5 scores when the mother had postpartum or chronic depression.

Model 2E Males Results. A males-only ANCOVA model with depression pattern as the IV and TOL Age 15 as the DV revealed that maternal depression pattern did not have a significant association with TOL scores in male children at age 15, $F(3, 332) = .996$, p = .395, *partial* η^2 = .009.

Model 2E Females Results. A females-only ANCOVA model with depression pattern as the IV and TOL Age 15 as the DV revealed that maternal depression pattern did not have a significant association with TOL scores in female children at age 15, $F(3, 362) = .852$, p = .466, *partial* η^2 = .007.

Model 2F Males Results. A males-only ANCOVA model with depression pattern as the IV and Stroop Age 15 as the DV revealed that maternal depression pattern had a significant association with Stroop scores in male children at age 15, $F(3, 321) = 3.386$, p = 018., *partial* η^2 = .031. However, when post hoc pairwise comparisons were examined, there were no significant differences (no depression vs. depression postpartum, p = .312; no depression vs. depression only during early childhood, p = .150;

no depression vs. chronic depression, p = .817; depression postpartum vs. early childhood depression, p = .058; postpartum depression vs. chronic depression, p = .507; early childhood depression vs. chronic depression, p = .172; see Appendix A for means tables). A significant main effect without significant pairwise comparisons could have occurred due to loss of N with pairwise comparisons. A significant interaction was observed between depression and maternal education, $F(3, 321) = 3.458$, p = .017, *partial* η^2 = .031. Visual examination of a plot indicated that children with mothers who had more education scored better than children with less educated mothers when the mother had postpartum depression but worse when the mother was depressed during early childhood.

Model 2F Females Results. A females-only ANCOVA model with depression pattern as the IV and Stroop Age 15 as the DV revealed that maternal depression pattern did not have a significant association with Stroop scores in female children at age 15, $F(3, 356) = 1.660$, p = .175, *partial* η^2 = .014.

Model 3 (Exploratory) – Effects of Maternal Depression Pattern on Caucasian Versus Minority Child Executive Functioning Scores.

The effect of maternal depression on child executive functioning development in different racial groups has not yet been examined. However, different processes and mechanisms through which racial differences (African Americans versus Latinos), maternal depression, parenting practices, and poverty on child outcomes have been demonstrated for behavioral problems and cognitive development (Pachter, Auinger, Palmer, & Weitzman, 2006). It is important to interpret the cognitive development findings keeping in mind that cognitive assessments can be culturally biased in favor of Caucasian children (Brooks-Gunn, Klebanov, & Duncan, 2008). Measures of

neuropsychological functioning, even those assessing nonverbal skills, have demonstrated cultural bias, but that bias is typically based on education level (Rosselli & Ardila, 2003), which is not applicable to the current study because the children were assessed at matched grade levels. The literature support for different cognitive development will be applied in an exploratory model to examine potential racial differences in executive functioning in children with depressed mothers. Because the SECCYD data was largely representative of Caucasian children, all minority ethnicities were grouped into one to preserve power.

This exploratory analysis included twelve separate ANCOVAs with maternal depression pattern as the independent variable and the following dependent variables (one per model): CPT 1st grade, Caucasian Non-Hispanic (Model 3A, Caucasian), CPT 1st Grade, Minority (Model 3A, Minority), TOH 1st grade, Caucasian Non-Hispanic (Model 3B, Caucasian), TOH 1st grade, Minority (Model 3B, Minority), CPT 4th grade, Caucasian Non-Hispanic (Model 3C, Caucasian), CPT 4th grade, Minority (Model 3C, Minority), TOH 5th grade, Caucasian Non-Hispanic (Model 3D, Caucasian), TOH 5th grade, Minority (Model 3D, Minority), TOL Age 15, Caucasian Non-Hispanic (Model 3E, Caucasian), TOL Age 15, Minority (Model 3E, Minority), Stroop Age 15, Caucasian Non-Hispanic (Model 3F, Caucasian), and Stroop Age 15, Minority (Model 3F, Minority). The original number of participants in the data set was 1364. After accounting for missing data, the N for each model was as follows.

Table 9

N Values for Each Model

Model (DV)	N
3A, Caucasians (CPT G1)	435
3A, Minorities (CPT G1)	117
3B, Caucasians (TOH G1)	665
3B, Minorities (TOH G1)	140
3C, Caucasians (CPT G4)	462
3C, Minorities (CPT G4)	123
3D, Caucasians (TOH G5)	635
3D, Minorities (TOH G5)	143
3E, Caucasians (TOL Age 15)	592
3E, Minorities (TOL Age 15)	134
3F, Caucasians (Stroop Age 15)	584
3F, Minorities (Stroop Age 15)	134

Model 3A Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and CPT G1 as the DV revealed that maternal depression pattern had a significant association with CPT scores in Caucasian Non-Hispanic children at grade 1, $F(3, 416) = 4.221$, $p = .006$, *partial* $\eta^2 = .030$. However, no pairwise comparisons were significant (no depression vs. postpartum depression, $p = .169$; no depression vs. early childhood depression, $p = .420$, no

depression vs. chronic depression, p = .167; postpartum depression vs. early childhood depression, p = .602; postpartum depression vs. chronic depression, p = .892; early childhood depression vs. chronic depression, p = .665; see Appendix A for means tables). It is possible that there was a main effect with no significant pairwise comparisons because N is lost with pairwise comparisons. There was a significant interaction between depression and child age at testing $F(3, 416) = 4.056$, p = .007, *partial* η^2 = .028. Visual examination of a plot indicated that younger children performed better on the CPT G1 during all maternal depression groups, but worse with no maternal depression.

Model 3A Minorities Results. An ANCOVA model with individuals of racial minority status with depression pattern as the IV and CPT G1 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in Minority children at grade 1, $F(3, 101) = .518$, p = .671, *partial* η^2 = .015.

Model 3B Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and TOH G1 as the DV revealed that maternal depression pattern did not have a significant association with TOH scores in Caucasian Non-Hispanic children at grade 1, $F(3, 649) = 2.200$, p = .087, *partial* η^2 = .010.

Model 3B Minorities Results. An ANCOVA model comprised of individuals of racial minority status with depression pattern as the IV and TOH G1 as the DV revealed that maternal depression pattern did not have a significant association with TOH scores in Other Race children at grade 1, $F(3, 124) = .557$, p = .645, *partial* η^2 = .013.

Model 3C Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and CPT G4 as the DV revealed that

maternal depression pattern had a significant association with CPT scores in Caucasian Non-Hispanic children at grade 4, $F(3, 440) = 3.513$, $p = .015$, *partial* $\eta^2 = .023$. Pairwise comparisons suggested that children with mothers depressed during the postpartum period ($M = 3.583$) scored significantly better than those with chronically depressed mothers ($M = 3.111$), $p = .019$. There were significant interactions between depression and maternal education, $F(3, 440) = 3.722$, $p = .012$, *partial* $\eta^2 = .025$, and depression and total income, $F(3, 440) = 2.703$, $p = .045$, *partial* $\eta^2 = .018$. Visual examination of plots indicated that children with more educated mothers scored better on the CPT G4 when their mothers had early childhood and chronic depression. Additionally, children from higher income families scored better than children from low income families when the mother had postpartum or chronic depression, but not early childhood depression.

Model 3C Minorities Results. An ANCOVA model comprised of individuals of racial minority status with depression pattern as the IV and CPT G4 as the DV revealed that maternal depression pattern did not have a significant association with CPT scores in Other Race children at grade 4, $F(3, 107) = .418$, $p = .741$, *partial* $\eta^2 = .012$.

Model 3D Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and TOH G4 as the DV revealed that maternal depression pattern did not have a significant association with TOH scores in Caucasian Non-Hispanic children at grade 5, $F(3, 619) = .102$, $p = .959$, *partial* $\eta^2 = .000$.

Model 3D Minorities Results. An ANCOVA model comprised of individuals of racial minority status with depression pattern as the IV and TOH G5 as the DV revealed that maternal depression pattern did not have a significant association with TOH scores in Other Race children at grade 5, $F(3, 127) = 2.005$, $p = .117$, *partial* $\eta^2 = .045$.

Model 3E Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and TOL Age 15 as the DV revealed that maternal depression pattern did not have a significant association with TOL scores in Caucasian Non-Hispanic children at age 15, $F(3, 576) = .918$, $p = .432$, *partial* $\eta^2 = .005$.

Model 3E Minorities Results. An ANCOVA model comprised of individuals of racial minority status with depression pattern as the IV and TOL Age 15 as the DV revealed that maternal depression pattern did not have a significant association with TOL scores in Other Race children at age 15, $F(3, 118) = 2.205$, $p = .091$, *partial* $\eta^2 = .053$.

Model 3F Caucasians Results. An ANCOVA model comprised of Caucasian individuals with depression pattern as the IV and Stroop Age 15 as the DV revealed that maternal depression pattern did not have a significant association with Stroop scores in Caucasian Non-Hispanic children at age 15, $F(3, 568) = .620$, $p = .602$, *partial* $\eta^2 = .003$.

Model 3F Minorities Results. An ANCOVA model comprised of individuals of racial minority status with depression pattern as the IV and Stroop Age 15 as the DV revealed that maternal depression pattern did not have a significant association with Stroop scores in Other Race children at age 15, $F(3, 118) = 1.194$, $p = .315$, *partial* $\eta^2 = .029$.

Hypothesis Two: Children with chronically depressed mothers, as defined by mothers who experience depression both during the postpartum and early childhood periods, who experience executive dysfunction will be more likely to experience subsequent internalizing behaviors; that is, the relationship between chronic maternal depression and later child internalizing behaviors will be partially mediated by child executive dysfunction.

Summary of Results. Overall, child executive functioning scores were not associated with later child depression, as proposed. However, exploratory analyses suggested that CPT G1 scores of children of minority status had a significant positive relationship with CBCL Internalizing scores at age 15 such that children who scored better on the CPT at G1 displayed more internalizing symptoms (see Figure 10). Also in children of minority status, TOH G1 scores were significantly associated with CBCL Internalizing scores at grade 3 such that better TOH scores were predictive of lower internalizing behavior scores (see Figure 11).

Results. The second hypothesis included four separate MANCOVAs with the following dependent variables: child internalizing behaviors, as reported by the mother at grade 3, grade 4, grade 5, grade 6, and age 15. Child internalizing scores were only used after the time at which the IV executive functioning measured was assessed (e.g., EF score at grade 1 would predict depression at grades 3, 4, 5, 6, and age 15, but EF score at grade 5 would only predict depression at grades 6 and age 15). Each model had a different independent variable: CPT 1st grade (Model 4A), TOH 1st grade (Model 4B), CPT 4th grade (Model 4C), TOH 5th grade (Model 4D). TOL and Stroop models were not included because depression was measured concurrently, making interpretation of causal direction more difficult. Covariates included maternal education, total family income, site of data collection, and age of testing. The original number of participants in the data set was 1364. After accounting for missing data, the N for each model was as follows.

Table 10

N Values for Each Model

Model (IV)	N
4A (CPT G1)	450
4B (TOH G1)	658
4C (CPT G4)	526
4D (TOH G5)	705

In addition to missing data outlined in hypothesis 1, data were missing for the following reasons: 145 from CBCL grade 3, 153 from CBCL grade 4, 154 from CBCL grade 5, 152 from CBCL grade 6, and 192 CBCL age 15. There were no extreme outliers for CBCL scores at any of the time points measured.

Histograms of all DVs indicated that the data were unimodal and normally distributed, with skewness and kurtosis values within the acceptable range (less than 2). See Table 11 below for skewness and kurtosis values.

Table 11

Skewness and Kurtosis Values for CBCL Internalizing Scales

Variable	Skewness	Kurtosis
CBCL, Grade 3	.372	-.443
CBCL, Grade 4	.403	-.150
CBCL, Grade 5	.322	-.212
CBCL, Grade 6	.426	-.030
CBCL, Age 15	.343	-.088

Prior to assessing assumptions, bivariate correlations of CBCL scores were examined because MANCOVA requires moderate correlation between DVs. Correlations ranged from .477 to .738, which were within an acceptable range.

Model 4 (Proposed) – Effect of Child Executive Functioning Scores on Later Child Depression.

Model 4A Results. A MANCOVA model with CPT G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that CPT G1 was not significantly associated with child internalizing scores across time points, *Wilks' λ* = .988, F (6, 432) = 1.035, p = .396, *partial* η^2= .012.

Model 4B Results. A MANCOVA model with TOH G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that TOH G1 was not significantly

associated with child internalizing scores across time points, *Wilks' λ* = .989, *F* (5, 640) = .1.416, *p* = .216, *partial* η^2= .011.

Model 4C Results. A MANCOVA model with CPT G4 as the IV and CBCL G5, G6, and age 15 as the DVs revealed that CPT G4 was not significantly associated with child internalizing scores across time points, *Wilks' λ* = .997, *F* (3, 514) = .480, *p* = .696, *partial* η^2= .003.

Model 4D Results. A MANCOVA model with TOH G5 as the IV and CBCL G6 and age 15 as the DVs revealed that TOH G5 was not significantly associated with child internalizing scores across time points, *Wilks' λ* = .999, *F* (2, 703) = .415, *p* = .660, *partial* η^2= .001.

Exploratory Results Summary (Models 5 and 6). The following models are exploratory in nature and are interpreted with caution, especially in cases where *p* values are close to .05. This section serves as a synthesis of results to follow. Please see specific model results for *F* strings.

Results suggest that the CPT G1 scores of children of minority status had a significant positive relationship with CBCL Internalizing scores at age 15 (see Figure 10 below). Also in children of minority status, TOH G1 scores were significantly associated with CBCL Internalizing scores at grade 3 such that better TOH scores were predictive of lower CBCL Internalizing scores (see Figure 11 below).

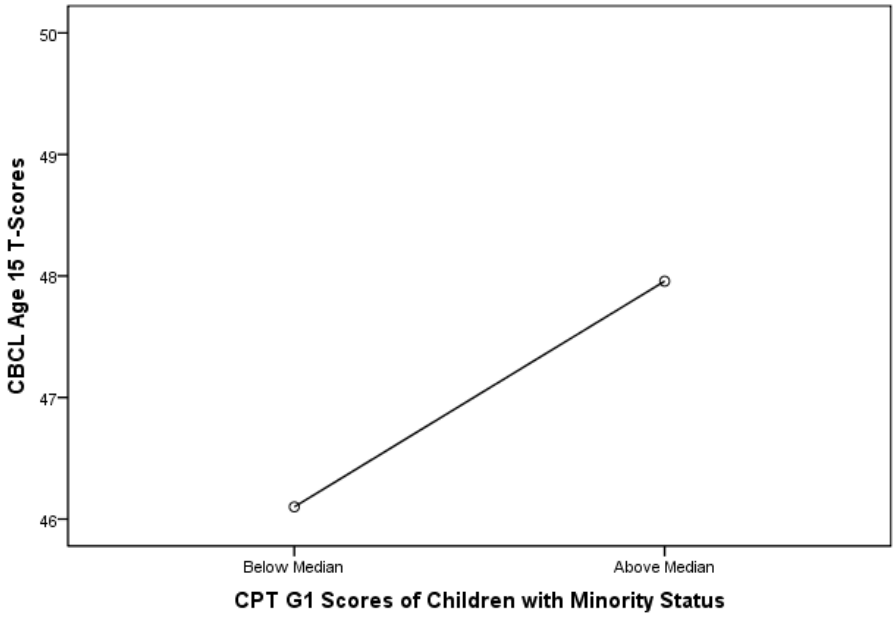

Figure 10. CPT G1 Scores on CBCL Age 15 in Children of Minority Status.

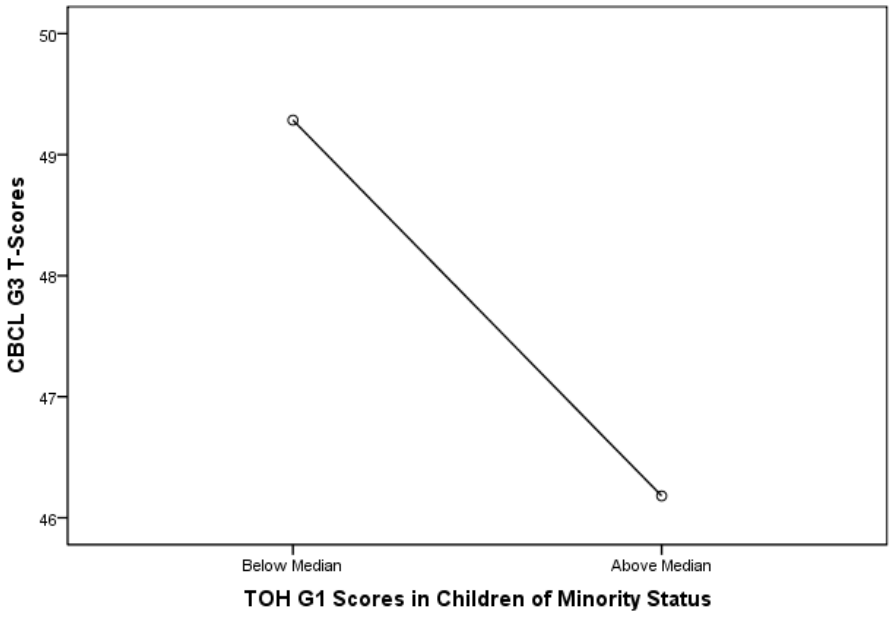

Figure 11. TOH G1 Scores on CBCL G3 in Children of Minority Status.

Model 5 (Exploratory) – Effects of Male Versus Female Child Executive Functioning Scores on Later Child Depression.

Because gender and ethnicity differences were examined above (Models 2 and 3), they will again be examined here (Model 5 – Gender, Model 6 – Ethnicity). This exploratory analysis includes eight separate MANCOVAs with a DV of child internalizing behaviors, as reported by the mother at grade 3, grade 4, grade 5, grade 6, and age 15. Child internalizing scores were only used after the time at which the IV executive functioning measured was assessed. Each model has a different independent variable: CPT 1st grade, Males (Model 5A Males), CPT 1st Grade, Females (Model 5A Females), TOH 1st Grade, Males (Model 5B Males), TOH 1st grade, Females (Model 5B Females), CPT 4th Grade, Males (Model 5C Males), CPT 4th Grade, Females (Model 5C Females), TOH 5th Grade, Males (Model 5D Males), TOH 5th Grade, Females (Model 5D Females). TOL and Stroop models were not included because depressed was measured concurrently, so they would not be predictive of depression. Covariates included maternal education, total family income, site of data collection, and age of testing. The original number of participants in the data set was 1364. After accounting for missing data, the N for each model was as follows.

Table 12

N Values for Each Model

Model (IV)	N
5A Males (CPT G1)	215
5A Females (CPT G1)	233
5B Males (TOH G1)	310
5B Females (TOH G1)	345
5C Males (CPT G4)	256
5C Females (CPT G4)	274
5D Males (TOH G5)	352
5D Females (TOH G5)	366

Model 5A Males Results. A males only MANCOVA model with CPT G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that CPT G1 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .981, *F* (6, 196) = .646, *p* = .693, *partial* η^2= .019.

Model 5A Females Results. A females only MANCOVA model with CPT G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that CPT G1 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .967, *F* (65 215) = 1.448, *p* = .208, *partial* η^2= .033.

Model 5B Males Results. A males only MANCOVA model with TOH G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that TOH G1 was not

significantly associated with child internalizing scores at any time point, *Wilks' λ* = .985, *F* (6, 291) = .646, *p* = .611, *partial* η^2= .015.

Model 5B Females Results. A females only MANCOVA model with TOH G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that TOH G1 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .981, *F*(5, 327) = 1.294, *p* = .266, *partial* η^2= .019.

Model 5C Males Results. A males only MANCOVA model with CPT G4 as the IV and CBCL G5, G6, and age 15 as the DVs revealed that CPT G4 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .999, *F* (3, 240) = .987, *p* = .611, *partial* η^2= .001.

Model 5C Females Results. A females only MANCOVA model with CPT G4 as the IV and CBCL G5, G6, and age 15 as the DVs revealed that CPT G4 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .984, *F* (3, 258) = 1.434, *p* = .233, *partial* η^2= .016.

Model 5D Males Results. A males only MANCOVA model with TOH G5 as the IV and CBCL G6, and age 15 as the DVs revealed that TOH G5 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .999, *F* (2, 337) = .090, *p* = .914, *partial* η^2= .001.

Model 5D Females Results. A females only MANCOVA model with TOH G5 as the IV and CBCL G6, and age 15 as the DVs revealed that TOH G5 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .999, *F* (2, 337) = .090, *p* = .914, *partial* η^2= .001.

Model 6 (Exploratory) – Effects of Caucasian Versus Minority Child Executive Functioning Scores on Later Child Internalizing.

This exploratory analysis of race differences in executive functioning scores on depression includes eight separate MANCOVAs with a DV of child internalizing behaviors, as reported by the mother at grade 1, grade 3, grade 5, grade 5, grade 6, and age 15. Child internalizing scores were only used after the time at which the IV executive functioning measured was assessed. Each model has a different independent variable: CPT 1st grade, Caucasian Non-Hispanic (Model 6A, Caucasians), CPT 1st Grade, Minority (Model 6A, Minorities), TOH 1st Grade, Caucasian Non-Hispanic (Model 6B, Caucasians) TOH 1st grade, Minority (Model 6B, Minorities), CPT 4th Grade, Caucasian Non-Hispanic (Model 6C, Caucasians), CPT 4th Grade, Minority (Model 6C Minorities), TOH 5th Grade, Caucasian Non-Hispanic (Model 6D Caucasians), TOH 5th Grade, Minority (Model 6D Minorities). TOL and Stroop models were not included because depressed was measured concurrently, so they would not be predictive of depression. Covariates included maternal education, total family income, site of data collection, and age of testing. The original number of participants in the data set was 1364. After accounting for missing data, the N for each model was as follows.

Table 13

N Values for Each Model

Model (IV)	N
6A, Caucasians (CPT G1)	355
6A, Minorities (CPT G1)	95
6B, Caucasians (TOH G1)	538
6B, Minorities (TOH G1)	120
6C, Caucasians (CPT G4)	423
6C, Minorities (CPT G4)	107
6D, Caucasians (TOH G5)	588
6D, Minorities (TOH G5)	130

Model 6A Caucasians Results. A MANCOVA model comprised of Caucasian individuals with CPT G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that CPT G1 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .988, F (5, 337) = .837, p = .524, *partial* η^2= .012.

Model 6A Minorities Results. A MANCOVA model comprised of individuals of racial minority status with CPT G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that CPT G1 was significantly associated with child internalizing scores, *Wilks' λ* = .819, F (5, 76) = 3.368, p = .008, *partial* η^2= .181. Pairwise comparisons suggested that CPT G1 scores were only significantly related to CBCL Internalizing scores at Age 15, F (1, 80) = 4.53, p = .036, *partial* η^2= .054, with better

CPT G1 scores being associated with higher CBCL scores (more internalizing symptoms). There was a significant interaction between income and CPT G1 scores, *Wilks' λ* = .797, *F* (5, 76) = 3.862, *p* = .004, *partial* η^2= .203. However, there were no significant pairwise comparisons, likely due to smaller N in the pairwise comparisons than in the omnibus test (total income*CBCL G3, *p* = .570, total income*CBCL G4, *p* = .351, total income*CBCL G5, *p* = .559, total income*CBCL G6, *p* = .750, and total income*CBCL Age 15, *p* = .051).

Model 6B Caucasians Results. A MANCOVA model comprised of Caucasian individuals with TOH G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that TOH G1 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .990, *F(*5, 520) = 1.086, *p* = .367, *partial* η^2= .010.

Model 6B Minorities Results. A MANCOVA model comprised of individuals of racial minority status with TOH G1 as the IV and CBCL G3, G4, G5, G6, and age 15 as the DVs revealed that TOH G1 was significantly associated with child internalizing scores, *Wilks' λ* = .860, *F*(5, 102) = 3.329, *p* = .008, *partial* η^2= .140. TOH G1 was only significantly associated with CBCL Internalizing Scores at grade 3, *F*(1, 106) = 7.669, *p* = .007, *partial* η^2= .067. Higher TOH scores were predictive of lower CBCL scores.

Model 6C Caucasians Results. A MANCOVA model comprised of Caucasian individuals with CPT G4 as the IV and CBCL G5, G6, and age 15 as the DVs revealed that CPT G4 was not significantly associated with child internalizing scores at any time point, *Wilks' λ* = .991, *F* (3, 407) = 1.281, *p* = .280, *partial* η^2= .009.

Model 6C Minorities Results. A MANCOVA model comprised of individuals of racial minority status with CPT G4 as the IV and CBCL G5, G6, and age 15 as the DVs

revealed that CPT G4 was significantly associated with child internalizing scores, *Wilks'* λ = .912, F (3, 91) = 2.932, p = .038, *partial* η^2= .088. However, CPT G4 was not significantly associated with any individual CBCL scores.

Model 6D Minorities Results. A MANCOVA model comprised of Caucasian individuals with TOH G5 as the IV and CBCL G6, and age 15 as the DVs revealed that TOH G5 was not significantly associated with child internalizing scores at any time point, *Wilks'* λ = .997, F (2, 573) = .916, p = .401, *partial* η^2= .003.

Model 6D Minorities Results. A MANCOVA model comprised of individuals of racial minority status with TOH G5 as the IV and CBCL G6, and age 15 as the DVs revealed that TOH G5 was not significantly associated with child internalizing scores at any time point, *Wilks'* λ = .991, F (2, 115) = .525, p = .593, *partial* η^2= .009.

CHAPTER IV

DISCUSSION

The present study examined the effects of maternal depression on child executive functioning development and the potential mediating role that child executive dysfunction could play in the relationship between maternal depression and later child depression. It was hypothesized that chronic maternal depression would have a negative and enduring impact on children's depression from 1st grade through age 15, and children who experienced executive dysfunction would be more likely to experience later depression. The proposed hypotheses were not supported, but results included a number of exploratory analyses that revealed some of these effects in certain populations. Additionally, results replicated findings in the literature that maternal depression is significantly associated with later child internalizing behaviors, as well as provided evidence of non-unity of EF.

Executive Functioning

The present study lends support to the theory of non-unity of executive functioning which states that executive functioning is comprised of distinct skills (Burgess et al. 2007; Robbins 1996) that likely do not have an underlying "central executive" (Stuss & Alexander, 2007). Non-unity was evidenced by low correlations between the various measures of executive function (CPT, TOH, TOL, and Stroop). Literature on this topic has been mixed, with evidence for both one central underling factor of EF (hypothesized to be general intelligence; De Frais, Dixon, & Strauss, 2006; Duncan et al., 1996) as well as distinct components that are functionally and anatomically independent (Burgess et al. 2007; Robbins 1996; Stuss & Alexander, 2007). Another

theory in the literature is that EF may change across the lifespan from a multidimensional construct in younger individuals to a unidimensional one in aging adults (De Frais, 2006). However, studying EF has been particularly difficult due to task-impurity of existing measures used to examine these functions (Phillips, 1997).

Maternal Depression and Later Child Internalizing Behaviors

The present study replicated a number of previous findings that link maternal depression to child internalizing behaviors **(**Downey & Coyne, 1990; Brennan et al., 2000; Cummings & Davies, 1994). Results indicated that all groups of maternal depression (postpartum, early childhood, and chronic) were predictive of later child internalizing behaviors at all time points (grades 1, 3, 4, 5, 6, and age 15), with the largest effect at grade 1 and the smallest at age 15. As compared to maternal postpartum and early childhood depression, chronic depression was consistently associated with higher mean scores for children on the CBCL at all measurement points (indicating more internalizing behaviors), consistent with past literature that suggests that chronic maternal depression is more detrimental to child development than shorter courses of depression (Goodman et al., 2011). Although the current study did not explore mechanisms through which maternal depression affects child internalizing behaviors, other researchers have proposed methods of transmission. These hypothesized transmission mechanisms include: 1) heritability, 2) innate dysfunctional neuroregulation, 3) exposure to negative maternal behavior, and 4) stressful environments (Goodman & Gotlib, 1999). Potential protective factors include: 1) father involvement, b) course of maternal depression, and 3) child characteristics (Goodman & Gotlib, 1999). Although transmission mechanisms were outside the scope of the present study, current results support the course of maternal

depression as a protective factor; that is, shorter courses of maternal depression were associated with fewer child internalizing behaviors.

Maternal Depression and Child Executive Functioning Development

There was no statistically significant difference between executive functioning in children with depressed mothers versus children with non-depressed mothers. Past literature on this topic has been mixed, with some evidence that maternal depression negatively impacts child executive functioning development (Hughes et al., 2013) and other null results (Klimes-Dougan et al., 2006; Micco et al., 2009; Rhoades et al., 2011).

This study explored gender differences in executive functioning development, which had not been done in prior research. These exploratory models were based on findings in the cognitive development literature demonstrating that maternal depression negatively impacts male cognitive development, but not female cognitive development (Murray, 1992; Sharp et al., 1995). After accounting for potentially influential factors (maternal education, total family income, age at assessment, and data collection site), results suggested that any maternal depression negatively impacted male TOH scores at 1st grade (see Figure 12) and early childhood and chronic depression negatively influenced male TOH scores at 5th grade (see Figure 13). Although postpartum depression approached significance, it is possible that as the child ages, the effect of postpartum depression on child's planning skills begins to remit before other courses of maternal depression.

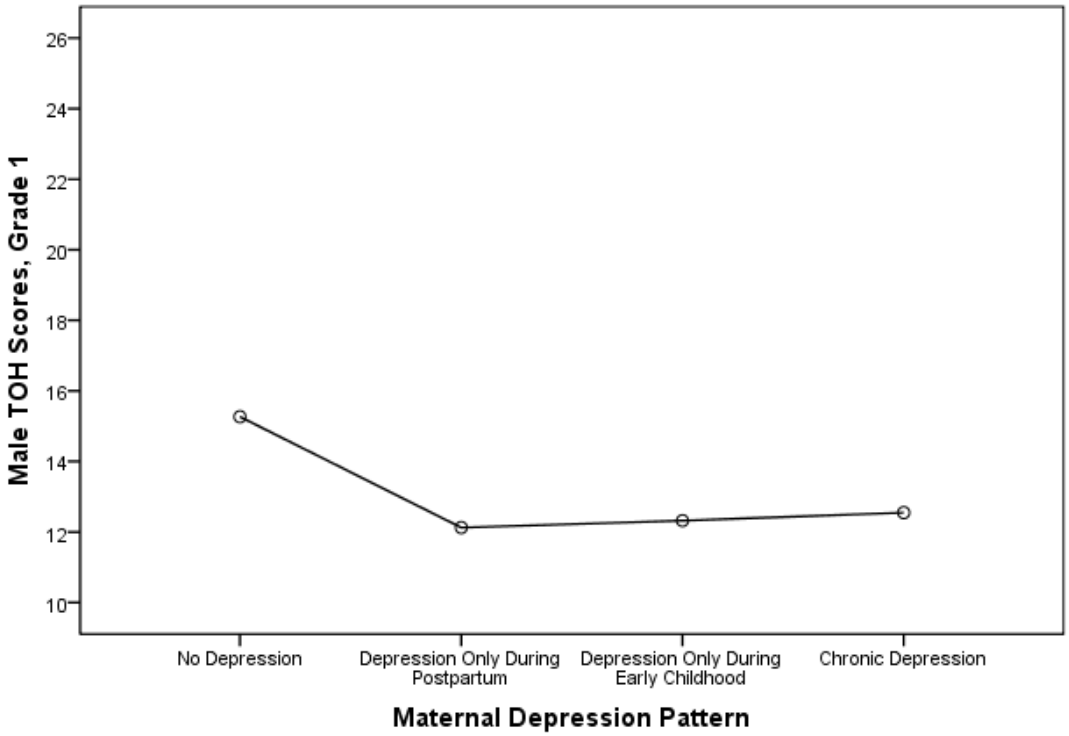

Figure 12. Maternal Depression Pattern and Male TOH Scores at Grade 1.

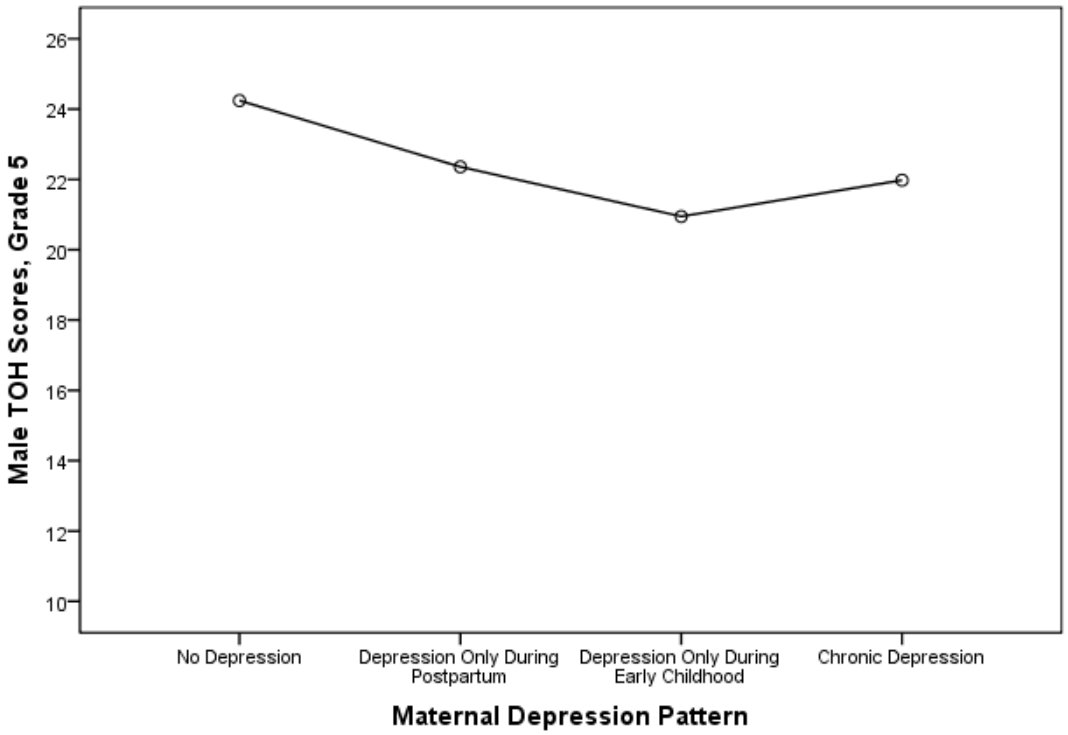

Figure 13. Maternal Depression Pattern and Male TOH Scores at Grade 5.

Maternal depression pattern was significantly associated with poorer TOH G1 scores for males with the *partial* η^2 being over four times as large for male children as it was in the overall sample (.042 and .009, respectively) and notably larger than the *partial* η^2 for females (.042 and .012, respectively). Additionally, maternal depression pattern was associated with significantly poorer TOH G5 scores for males with the *partial* η^2 being over six times as large for male children as it was in the overall sample (.027 and .004, respectively). The *partial* η^2 for males (.027) was comparable to that of females (.031), but the association between maternal depression and child scores in females was in the

opposite direction (females with depressed mothers scored better). The larger effect size of depression pattern at 1st grade (.042) as compared to at 5th grade (.027), presents evidence that the impact of depression can be seen more clearly at grade 1 than grade 5; in other words, by grade 5, children have other influences on their development that allow them to catch up to peers. Over time, the effect of postpartum depression dropped off, whereas early childhood depression and chronic depression had an enduring impact on child planning abilities. The TOH measures higher-order EF functioning abilities that require planning, organization, working memory, and pattern detection skills to work collaboratively (these rely on inhibition and information updating; Miyake et al., 2000), whereas the CPT is more straightforward and only measures inhibition (Miyake et al., 2000).

Because gender differences in executive functioning development have not yet been examined, there are no hypotheses in the literature that propose the mechanisms behind this phenomenon. However, we can look to the cognitive development literature for some hypotheses. First, depressed mothers potentially treat their sons differently than their daughters (Murray, 1992). For example, studies have found that depressed mothers engage in more intrusive behaviors with boys (Hart, Field, de Valle, & Pelaez-Nogueras, 1998) as well as use less infant-focused speech (Murray, Kempton, Woolgar, & Hooper, 1996). Second, as compared to males, females have a maturational advantage with language and social skills development, which could act as a protective factor from their mothers' illness (Berk, 1997). This suggests that males might have a greater need for a healthy caregiver's attention to help with emotion regulation. Third, it is possible that there are behavioral differences between male and females that have not yet been

identified. These behaviors could potentially prolong maternal depression, although there is some evidence that the course of the mother's depression is independent of child gender (Sharp et al., 1995). Finally, any combination of the above explanations could explain gender differences in cognitive development.

Additionally, exploratory analyses indicated that Caucasian, Non-Hispanic children with mothers who had postpartum depression scored significantly better on the CPT at grade 4 than children with chronically depressed mothers. This aligns with past studies that have shown chronic depression to be more detrimental to child executive functioning development than shorter courses of depression (Hughes et al., 2013; Shaw, Connell, Dishion, Wilson, & Gardner, 2009). However, a question emerges as to why children would score better on the CPT when their mothers are depressed during the postpartum year. It is possible that mothers receive more support during the postpartum period than during early childhood, in the form of help from family and friends and visits to the pediatrician. Past studies have shown that social support during the postpartum period is related to maternal mental health (Gjerdingen, Froberg, & Fontaine, 1991). Moreover, social support and visits to the pediatrician have been associated with improved home environment and maternal-infant parenting skills (Shaw, Levitt, & Wong, 2006). It is also possible that during this time the infant is more likely to interact with other, non-depressed adults, and that exposure acts as a buffer to the effects of the mother's depression on the child's executive functioning development, but this is an area for future study.

Regarding race differences, there is no explanation in the literature for this specific association. However, it has been noted that African Americans and Latinas are

at risk for reporting more early postpartum depressive symptoms compared to Caucasian mothers (Howell, Mora, Horowitz, & Leventhal, 2005). This unmeasured effect of depression frequency and/or severity might help explain lower Caucasian mother postpartum depression and better child CPT G4 scores.

A significant interaction was observed between depression and total family income on female TOH scores at grade 5, indicating that lower income was associated with poorer TOH G5 scores when the mother had postpartum or chronic depression. The negative effects of maternal depression on child development have been shown to be exacerbated by low-income, with affluence acting as a buffer to negative child outcomes (Petterson & Albers, 2001). This could potentially be a threshold effect; that is, at a certain income level, maternal depression has less of an impact on child development. However, this potential threshold effect and child gender differences are areas for future study.

Moreover, a significant interaction was observed between depression and maternal education for male Stroop scores at age 15, indicating that children with mothers who had more education scored more poorly than children with less educated mothers when the mother had postpartum depression but higher when the mother was depressed during early childhood. This stands in contrast to previous studies that have found that maternal education is associated with better child outcomes (Christian, Morrison, & Bryant, 1998; Downer & Pianta, 2006) and acts as a buffer to the negative effects of maternal depression on child cognitive development (Hay 1997; Hay & Kumar, 1995).

Also, there was a significant interaction between depression and child age at testing for a model examining Caucasian children's CPT scores at grade 1. Younger children performed better on the CPT G1 during all maternal depression groups, but poorer with no maternal depression. This could be partially explained by younger children having less exposure to maternal depression than older children, keeping in mind that chronicity of exposure to depression is associated with poorer outcomes (Hughes et al., 2013; Shaw et al., 2009). However, race difference is an area for future study.

There were also significant interactions between depression and maternal education, and depression and total income, in a model examining Caucasian children's CPT G4 scores. Maternal education was a protective factor for children's inhibition scores when the mother had early childhood and chronic depression. Also, higher income acted as a protective factor for children when the mother had postpartum or chronic depression. This is consistent with literature highlighting the importance of maternal education (Hay 1997; Hay & Kumar, 1995) and income (Petterson & Albers, 2001) for favorable child outcomes, however the interaction between specific depression groups for different child genders is an unexplored area.

Overall, children's scores on a measure of inhibition at multiple different time points were better when the mother was chronically depressed. It is possible that children with chronically depressed mothers adapt to an environment of consistent depression such that it does not have a negative impact on inhibition skills. As children got older, known protective factors (maternal education and family income) were associated with higher scores on a measure of inhibition, so it is possible that these protective factors act as more of a buffer as the child ages

Child Executive Functioning and Later Internalizing

There was no statistically significant association between child executive functioning scores and later child internalizing, as hypothesized. Although this is a new area of study, there is some literature to suggest that executive dysfunction occurs prior to depression in individuals at-risk based on family history (Christensen et al., 2008; Hsu et al., 2013). Exploratory results suggest that the CPT G1 scores of children of minority status had a significant positive relationship with CBCL Internalizing scores at age 15 such that better CPT G1 scores were associated with more internalizing symptoms. This is inconsistent with prior literature that has found poorer executive functioning to be associated with higher scores in internalizing scales (Burleson, 2008; Christensen et al., 2008; Hsu et al., 2013). However, consistent with prior literature, in children of racial minority status better TOH G1 scores were significantly associated with fewer internalizing symptoms scores at grade 3.

Child Executive Functioning as a Mediator Between Maternal Depression and Later Child Internalizing

Because the proposed models were non-significant, executive dysfunction as a mediator between maternal depression and later child depression was not examined. This mediation model was hypothesized based on two prior twin studies that found evidence of executive dysfunction existing prior to depression (Christensen et al., 2008; Hsu et al., 2013) and the high comorbidity between ADHD and depression, with ADHD typically existing first (Burleson, 2008). This is a new area of study and one with a small research literature. Additionally, the current study was the first of its kind to examine environmental factors contributing to child executive functioning and depression. It is

possible that executive dysfunction as a trait marker of depression is only genetically transmitted. However, it is difficult to parse out genetic versus environmental factors because of the interaction between the two. Although these preliminary findings were null, this is still an important area for future study. This study did not capture maternal depression severity, which has been found to predict child depression above and beyond chronicity and timing (Hammen and Brennan, 2003). It would be important to measure this in a future study.

Strengths

The most notable strength of the present study is the longitudinal nature of the SECCYD data. Data on child development over the course of 15 years is unique and presented a special opportunity to examine specific contributors to child development including child executive functioning, maternal depression, and child depression.

Additionally, because maternal depression was measured at multiple time points, it could be grouped into different trajectory patterns, no depression, postpartum depression, early childhood depression, and chronic depression. The CES-D, a widely-used and validated measure of depression, was used to measure maternal depression, which likely makes these findings generalizable to maternal depression in the population at large. Additionally, the CBCL is the most utilized screening tool for tracking the emergence of problematic behavior in children ages 4-18, and is highly reliable and valid, making it a strong tool to measure and generalize child internalizing behaviors. Finally, the EF measures have been widely studied and have good reliability and validity data to support them.

Limitations

One major limitation of the current study is that countless variables contribute to child development and it is difficult to parse out the most meaningful contributors. This is especially true for outcomes such as executive dysfunction and depression, which have environmental, genetic, and epigenetic components that can be difficult to measure. More specifically, prenatal depression is a known risk factor for postpartum and chronic depression, but the SECCYD did not measure depression in mothers during pregnancy, so this lack of data is a limitation of the current study.

Additionally, this study did not examine how much time the child spent in the care of his or her mother. Differences in time spent with a depressed mother could impact both executive dysfunction and later depression in the child. Additionally, the current study did not account for children who were cared for in other settings (e.g., center-based daycare, family daycare, other caregiver), or cared for by the father. Moreover, the SECCYD did not assess depression in fathers, which we know exists in about 10% of new fathers and has a moderate correlation with maternal depression (Paulson & Bazemore, 2010).

Although the full SECCYD data set was large, the present study looked at subsets to examine differences in genders and ethnicities. By reducing the sample size in these analyses, power to detect small effect sizes was reduced. Additionally, sample sizes for each measure of executive functioning were variable, so it is possible that models with significant findings resulted from a larger sample size. Also, maternal depression was broken into different postnatal trajectories. By virtue of depression prevalence, the non-depressed group had larger N than the other three depression groups. This also likely

contributed to reducing power to detect effects. Further, the SECCYD sample is largely Caucasian, so examining minorities resulted in a less robust group. Due to a small sample of minorities, all minority groups were considered together, which limits the interpretation of results.

Another limitation of the results is that many notable findings came from exploratory analyses. By using multiple exploratory analyses, the chance of spurious findings increases and results are interpreted with caution. It is important to note that the large sample size in the study had the power to detect small affects. There is a chance that some of the exploratory analysis findings are spurious due to multiple analyses and will not replicate in future studies.

Implications

The present study replicated previous findings that maternal depression after childbirth is associated with later child depressive symptoms (Cummings & Davies, 1994; Downey & Coyne, 1990; Hammen & Brennan, 2003). Additionally, these findings mimicked those in the cognitive development literature (Murray, 1992; Sharp et al., 1995) in that maternal depression was associated with lower TOH scores in male children at both grade 1 and grade 5. All of the maternal depression groups were associated with poorer TOH scores at grade 1, and early childhood and chronic depression was associated with poorer TOH at grade 5. This presents some evidence that the effects of postpartum depression drops off over time, however early childhood and chronic depression continue to negatively affect boys at grade 5. Considering that these results were exploratory, and with other limitations of this study in mind, it is still safe to recommend routine screening for maternal depression through early childhood by primary care physicians,

gynecologists, and/or pediatricians (Wisner, Parry, & Piontek, 2002). This is important for the mother's well-being, for the potentially negative implications for the child's executive functioning development, and for environmental parenting factors that might contribute to later child depression.

This study found that maternal depression negatively impacted male executive functioning in the realm of planning. However, with small to medium effect sizes, differences in functioning might not be noticeable. Also, the TOH was not measured past 5th grade, so it is difficult to determine whether maternal depression would continue to impact boys' planning abilities, or whether boys with early deficits would catch up to peers over time.

Given the limitations of the present study and the conflicting results for the effects of executive functioning on later child depression, it is difficult to make conclusions about executive dysfunction as a potential trait marker for later depression.

Future Directions

The hypothesized questions are inherently difficult to address due to the number and intricacy of variables that affect child executive functioning development and child depression. The present study failed to capture a number of these variables that can likely be measured in future studies: time spent with the caretaker, depression in other caretakers besides the mother, and qualitative measures of interaction between caretaker and child to further explore mechanisms through which depression affects child executive functioning and child depression. It would important to measure specific parenting behaviors known to be associated with both child executive functioning development and child depression. There is evidence to suggest that depression in mothers and fathers can

negatively impact child cognitive development through decreased positive enrichment activities such as reading, singing, and telling stories (Paulson, Dauber, & Leiferman, 2006), as well as contingent stimulation, or responsiveness to the child (Hay, 1997). Withdrawn, harsh, and inconsistent parenting behaviors have been associated with later child depression (Lovejoy et al., 2000). Additionally, it would be helpful to use the same measure of executive functioning at each time point to be able to use a repeated measures design and look at longitudinal neuropsychological profiles. More specifically, the Tower of Hanoi task would be most appropriate considering that planning was negatively impacted by maternal depression in the present study.

Future studies will benefit from alterations in data collection such as over-representing racial minorities and mothers with risk factors for depression. The group of minorities was much smaller than Caucasians, as was depressed mothers versus non-depressed mothers. It would be ideal to sample large groups of different racial minorities so that they would not need to be grouped into one to create enough power to detect small affects. Additionally, it would be helpful for mothers to complete depression questionnaires more often to gain a better idea of whether a mother truly suffered from chronic depression or multiple separate episodes. Furthermore, a larger depression group would allow the CES-D to be broken down into different groups examining the severity of depressive symptoms and to look at potential interactions between severity and chronicity.

Additionally, it would be helpful to gather corroborating data on child internalizing behaviors. Maternal depression has been observed to significantly affect their ratings of their children on the CBCL (Friedlander, Weiss, & Traylor, 1986), so it

would be beneficial to administer questionnaires to other caretakers (i.e., the father), school teachers, and when the child is old enough, the child him or herself.

CHAPTER V

CONCLUSIONS

The present study used the SECCYD, a national longitudinal data set, to examine the relationship between maternal depression and child executive functioning and later depression, and whether executive dysfunction could mediate the relationship between maternal depression and later child depression. Maternal education, total family income, site of data collection, and child age at assessment were used as covariates because of past evidence of their impact on child development. Overall, findings revealed that male executive functioning development at grade 1 was negatively impacted by postpartum depression, early childhood depression, and chronic depression. Additionally, maternal early childhood depression and chronic depression negatively impacted male executive functioning at grade 5; however, the effect of postpartum depression no longer impacted males at this measurement point. Executive dysfunction did not mediate the relationship between maternal depression and child depression. Future research will benefit from measuring depression in other caregivers, the amount of time the child spends with the caregiver, consistent executive functioning tasks over time, and corroborating reports of child depression. Additionally, it will be helpful to sample a larger number of depressed mothers and individuals of racial minority status.

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APPENDIX A

DESCRIPTIVE STATISTICS

Table A1

Descriptive Statistics, Model 1A, IV: Depression Pattern, DV: CPT G1

Depression Pattern	*Mean*	*SD*	*N*
None	3.225	.789	308
Postpartum	3.322	.861	67
Early Childhood	3.182	.881	77
Chronic	3.224	.902	100
Total	3.23	.831	552

Table A2

Descriptive Statistics, Model 1B, IV: Depression Pattern, DV: TOH G1

Depression Pattern	*Mean*	*SD*	*N*
None	15.32	6.600	461
Postpartum	14.41	6.793	98
Early Childhood	13.32	6.276	100
Chronic	13.63	7.148	136
Total	14.65	6.715	805

Table A3

Descriptive Statistics, Model 1C, IV: Depression Pattern, DV: CPT G4

Depression Pattern	*Mean*	*SD*	*N*
None	3.320	.854	320
Postpartum	3.312	.914	76
Early Childhood	3.162	.872	78
Chronic	3.066	.932	111
Total	3.251	.883	585

Table A4

Descriptive Statistics, Model 1D, IV: Depression Pattern, DV: TOH G5

Depression Pattern	*Mean*	*SD*	*N*
None	24.12	6.977	437
Postpartum	22.66	7.989	94
Early Childhood	22.80	8.144	108
Chronic	22.24	7.285	139
Total	23.42	7.359	778

Table A5

Descriptive Statistics, Model 1E: IV: Depression Pattern, DV: TOL Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	54.765	12.950	408
Postpartum	51.440	12.788	83
Early Childhood	52.848	13.034	105
Chronic	52.402	15.502	130
Total	53.685	13.468	726

Table A6

Descriptive Statistics, Model 1F, IV: Depression Pattern, DV: Stroop Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	.094	.072	405
Postpartum	.079	.075	83
Early Childhood	.088	.066	103
Chronic	.089	.073	127
Total	.091	.072	718

Table A7

Descriptive Statistics, Model 2A Males, IV: Depression Pattern, DV: CPT G1

Depression Pattern	*Mean*	*SD*	*N*
None	3.252	.795	152
Postpartum	3.268	.846	36
Early Childhood	3.140	.872	35
Chronic	3.147	.948	55
Total	3.220	.840	278

Table A8

Descriptive Statistics, Model 2A Females, IV: Depression Pattern, DV: CPT G1

Depression Pattern	*Mean*	*SD*	*N*
None	3.798	.785	156
Postpartum	3.389	.888	31
Early Childhood	3.216	.898	42
Chronic	3.319	.844	45
Total	3.242	.823	274

Table A9

Descriptive Statistics, Model 2B Males, IV: Depression Pattern, DV: TOH G1

Depression Pattern	*Mean*	*SD*	*N*
None	15.32	6.356	227
Postpartum	12.26	5.763	50
Early Childhood	12.25	6.622	48
Chronic	12.33	6.711	72
Total	14.02	6.535	397

Table A10

Descriptive Statistics, Model 2B Females, IV: Depression Pattern, DV: TOH G1

Depression Pattern	*Mean*	*SD*	*N*
None	15.31	6.842	234
Postpartum	16.65	7.112	48
Early Childhood	14.15	5.917	62
Chronic	15.08	7.394	64
Total	15.25	6.839	408

Table A11

Descriptive Statistics, Model 2C Males, IV: Depression Pattern, DV: CPT G4

Depression Pattern	*Mean*	*SD*	*N*
None	3.289	.839	149
Postpartum	3.348	.729	41
Early Childhood	3.200	.889	34
Chronic	2.957	.961	63
Total	3.214	.866	287

Table A12

Descriptive Statistics, Model 2C Females, IV: Depression Pattern, DV: CPT G4

Depression Pattern	*Mean*	*SD*	*N*
None	3.349	.869	171
Postpartum	3.286	1.103	35
Early Childhood	3.132	.867	44
Chronic	3.209	.881	48
Total	3.267	.900	298

Table A13

Descriptive Statistics, Model 2D Males, IV: Depression Pattern, DV: TOH G5

Depression Pattern	*Mean*	*SD*	*N*
None	24.45	6.766	214
Postpartum	22.27	7.460	49
Early Childhood	20.64	9.599	45
Chronic	21.61	7.304	75
Total	23.17	7.465	383

Table A14

Descriptive Statistics, Model 2D Females, IV: Depression Pattern, DV: TOH G5

Depression Pattern	*Mean*	*SD*	*N*
None	23.80	7.175	223
Postpartum	23.09	8.591	45
Early Childhood	24.33	6.582	63
Chronic	22.98	7.250	64
Total	23.67	7.257	395

Table A15

Descriptive Statistics, Model 2E Males, IV: Depression Pattern, DV: TOL Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	55.747	13.447	196
Postpartum	52.046	12.328	39
Early Childhood	55.135	14.423	43
Chronic	51.627	15.570	70
Total	54.428	13.961	348

Table A16

Descriptive Statistics, Model 2E Females, IV: Depression Pattern, DV: TOL Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	53.858	12.435	212
Postpartum	50.902	13.302	44
Early Childhood	51.261	11.840	62
Chronic	53.307	15.504	60
Total	53.001	12.980	378

Table A17

Descriptive Statistics, Model 2F Males, IV: Depression Pattern, DV: Stroop Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	.0846	.069	194
Postpartum	.071	.061	39
Early Childhood	.087	.066	42
Chronic	.082	.080	68
Total	.083	.070	343

Table A18

Descriptive Statistics, Model 2F Females, IV: Depression Pattern, DV: Stroop Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	.103	.073	211
Postpartum	.087	.086	44
Early Childhood	.089	.066	61
Chronic	.098	.065	59
Total	.098	.072	375

Table A19

Descriptive Statistics, Model 3A Caucasians, IV: Depression Pattern, DV: CPT G1

Depression Pattern	*Mean*	*SD*	*N*
None	3.256	.789	257
Postpartum	3.402	.856	47
Early Childhood	3.258	.810	62
Chronic	3.324	.812	69
Total	3.283	.802	435

Table A20

Descriptive Statistics, Model 3A Minorities, IV: Depression Pattern, DV: CPT G1

Depression Pattern	*Mean*	*SD*	*N*
None	3.070	.776	51
Postpartum	3.135	.865	20
Early Childhood	2.867	1.107	15
Chronic	3.003	1.058	31
Total	3.037	.909	117

Table A21

Descriptive Statistics, Model 3B Caucasians, IV: Depression Pattern, DV: TOH G1

Depression Pattern	*Mean*	*SD*	*N*
None	15.56	6.587	398
Postpartum	14.85	6.992	75
Early Childhood	13.43	6.176	94
Chronic	14.66	7.308	98
Total	15.04	6.714	665

Table A22

Descriptive Statistics, Model 3B Minorities, IV: Depression Pattern, DV: TOH G1

Depression Pattern	*Mean*	*SD*	*N*
None	13.81	6.537	63
Postpartum	12.96	6.011	23
Early Childhood	12.96	7.021	16
Chronic	10.95	6.018	38
Total	12.76	6.414	140

Table A23

Descriptive Statistics, Model 3C Caucasians, IV: Depression Pattern, DV: CPT G4

Depression Pattern	*Mean*	*SD*	*N*
None	3.344	.845	271
Postpartum	3.583	.810	53
Early Childhood	3.171	.899	64
Chronic	3.111	.922	74
Total	3.310	.869	462

Table A24

Descriptive Statistics, Model 3C Females, IV: Depression Pattern, DV: CPT G4

Depression Pattern	*Mean*	*SD*	*N*
None	3.193	.902	49
Postpartum	2.713	.865	23
Early Childhood	3.121	.761	14
Chronic	2.975	.958	37
Total	3.030	.905	123

Table A25

Descriptive Statistics, Model 3D Caucasians, IV: Depression Pattern, DV: TOH G4

Depression Pattern	*Mean*	*SD*	*N*
None	24.28	6.903	377
Postpartum	24.17	7.485	69
Early Childhood	23.47	7.597	91
Chronic	23.47	6.757	98
Total	24.03	7.041	635

Table A26

Descriptive Statistics, Model 3D Minorities, IV: Depression Pattern, DV: TOH G4

Depression Pattern	*Mean*	*SD*	*N*
None	23.08	7.404	60
Postpartum	18.48	7.985	25
Early Childhood	19.18	10.120	17
Chronic	19.32	7.741	41
Total	20.73	8.128	143

Table A27

Descriptive Statistics, Model 3E Caucasians, IV: Depression Pattern, DV: TOL Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	54.619	12.933	351
Postpartum	52.040	13.274	60
Early Childhood	52.987	13.251	89
Chronic	54.730	15.532	92
Total	54.130	13.445	592

Table A28

Descriptive Statistics, Model 3E Minorities, IV: Depression Pattern, DV: TOL Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	55.665	13.133	57
Postpartum	49.874	11.554	23
Early Childhood	52.075	12.125	16
Chronic	46.766	14.085	38
Total	51.719	13.445	134

Table A29

Descriptive Statistics, Model 3F Caucasians, IV: Depression Pattern, DV: Stroop Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	.091	.072	348
Postpartum	.079	.070	60
Early Childhood	.089	.069	87
Chronic	.091	.075	89
Total	.090	.071	584

Table A30

Descriptive Statistics, Model 3F Minorities, IV: Depression Pattern, DV: Stroop Age 15

Depression Pattern	*Mean*	*SD*	*N*
None	.113	.066	57
Postpartum	.081	.090	23
Early Childhood	.080	.046	16
Chronic	.086	.071	38
Total	.096	.071	134

Table A31

Descriptive Statistics, Model 4A, IV: CPT G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	47.89	9.562	446
CBCL G4	47.47	9.396	446
CBCL G5	48.33	9.459	446
CBCL G6	47.83	9.867	446
CBCL Age 15	46.77	9.506	446

Table A32

Descriptive Statistics, Model 4B, IV: TOH G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	48.08	9.711	649
CBCL G4	47.43	9.498	649
CBCL G5	48.19	9.498	649
CBCL G6	47.79	9.640	649
CBCL Age 15	46.35	9.610	649
Total			

Table A33

Descriptive Statistics, Model 4C , IV: CPT G4, DVs: CBCL G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G5	48.38	9.663	530
CBCL G6	47.54	9.936	530
CBCL Age 15	46.31	9.668	530

Table A34

Descriptive Statistics, Model 4D, IV: TOH G5, DVs: CBCL G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G6	47.48	9.778	718
CBCL Age 15	46.25	9.576	718

Table A35

Descriptive Statistics, Model 5A Males, IV: CPT G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G1	48.01	8.503	215
CBCL G3	47.87	9.835	215
CBCL G4	47.07	9.356	215
CBCL G5	48.27	9.697	215
CBCL G6	47.22	10.155	215
CBCL Age 15	46.20	9.431	215

Table A36

Descriptive Statistics, Model 5A Females, IV: CPT G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	47.94	9.288	233
CBCL G4	47.86	9.407	233
CBCL G5	48.42	9.251	233
CBCL G6	48.39	9.551	233
CBCL Age 15	47.27	9.605	233

Table A37

Descriptive Statistics, Model 5B Males, IV: TOH G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G1	48.00	8.925	310
CBCL G3	47.96	10.041	310
CBCL G4	47.16	9.735	310
CBCL G5	47.86	9.734	310
CBCL G6	46.95	9.967	310
CBCL Age 15	45.62	9.492	310

Table A38

Descriptive Statistics, Model 5B Females, IV: TOH G1, DVs: CBCL G3, G4, G5, G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	48.27	9.365	345
CBCL G4	47.73	9.240	345
CBCL G5	48.55	9.277	345
CBCL G6	48.03	9.345	345
CBCL Age 15	47.01	9.680	345

Table A39

Descriptive Statistics, Model 5C Males, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G5	48.25	10.020	256
CBCL G6	47.38	10.326	256
CBCL Age 15	45.61	9.528	256

Table A40

Descriptive Statistics, Model 5C Females, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G5	48.50	9.333	274
CBCL G6	47.70	9.573	274
CBCL Age 15	49.96	9.769	274

Table A41

Descriptive Statistics, Model 5D Males, IV: TOH G5, DVs: CBCL G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G6	46.99	10.067	352
CBCL Age 15	45.58	9.438	352

Table A42

Descriptive Statistics, Model 5D Females, IV: TOH G5, DVs: CBCL G5, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G6	47.96	9.482	366
CBCL Age 15	46.89	9.677	366

Table A43

Descriptive Statistics, Model 6A Caucasians, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	47.67	9.525	355
CBCL G4	47.61	9.363	355
CBCL G5	48.22	9.595	355
CBCL G6	47.88	10.073	355
CBCL Age 15	46.80	9.571	355

Table A44

Descriptive Statistics, Model 6A Minorities, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	48.67	9.633	95
CBCL G4	46.86	9.415	95
CBCL G5	48.59	9.022	95
CBCL G6	47.43	9.031	95
CBCL Age 15	46.48	9.446	95

Table A45

Descriptive Statistics, Model 6B Caucasians, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	48.01	9.570	538
CBCL G4	47.62	9.327	538
CBCL G5	48.24	9.432	538
CBCL G6	47.54	9.614	538
CBCL Age 15	46.33	9.583	538

Table A46

Descriptive Statistics, Model 6B Minorities, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G3	48.57	10.200	120
CBCL G4	46.83	10.186	120
CBCL G5	48.03	9.8442	120
CBCL G6	47.29	9.838	120
CBCL Age 15	46.39	9.757	120

Table A47

Descriptive Statistics, Model 6C Caucasians, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G5	48.33	9.554	423
CBCL G6	47.54	9.967	423
CBCL Age 15	46.34	9.652	423

Table A48

Descriptive Statistics, Model 6C Minorities, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G5	48.57	10.125	107
CBCL G6	47.57	9.859	107
CBCL Age 15	46.19	9.778	107

Table A49

Descriptive Statistics, Model 6D Caucasians, IV: TOH G5, DVs: CBCL G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G6	47.52	9.803	588
CBCL Age 15	46.27	9.575	588

Table A50

Descriptive Statistics, Model 6D Minorities, IV: TOH G5, DVs: CBCL G6, Age 15

Depression Pattern	*Mean*	*SD*	*N*
CBCL G6	47.35	9.702	130
CBCL Age 15	46.14	9.619	130

APPENDIX B

DEMOGRAPHICS

N= 953 unless otherwise noted

Table B1

Gender and Ethnicity of the Sample

Variable	*n*	*%*
Child Gender		
Male	480	50.4
Female	473	49.6
Child Ethnicity		
American Indian, Eskimo, Aleut	4	0.4
Asian or Pacific Islander	9	0.9
Black or Afro-American	88	9.2
Caucasian/White	812	85.0
Other	40	4.2
Child Ethnicity		
Non-Hispanic	906	95.1
Hispanic	47	4.9
Mother Ethnicity		
American Indian, Eskimo, Aleut	6	0.6
Asian or Pacific Islander	17	1.8
Black or Afro-American	86	9.0

Table B1 Continued

Variable	*n*	*%*
Caucasian/White	830	87.1
Other	14	1.5
Mother Ethnicity		
Non-Hispanic	918	96.3
Hispanic	35	3.7
Father Ethnicity		
American Indian, Eskimo, Aleut	3	0.3
Asian or Pacific Islander	13	1.4
Black or Afro-American	98	10.3
Caucasian/White	822	86.3
Other	16	1.7
Father Ethnicity		
Non-Hispanic	927	97.3
Hispanic	25	2.6

Table B2

Mother's Marital Status at 1 Month

Variable	*n*	*%*
Married, living together	809	84.9
Partnered, living together	70	7.3
Separated, not living together	5	0.5
Divorced, not living together	2	0.2
Never married, have a continuous romantic relationship, not living together	35	3.7
Never married, not involved romantically, not living together	26	2.7
Other	5	0.5

Note: $N = 952$

Table B3

CES-D Scores for Each Depression Group at Measurement Points

Variable	*M*	*SD*
1 month		
Not Depressed	6.35	4.02
Postpartum Depression	19.47	8.28
Early Childhood Depression	8.28	3.84
Chronic Depression	20.68	8.87
6 months		
Not Depressed	4.83	3.64
Postpartum Depression	12.33	8.48
Early Childhood Depression	7.73	3.73
Chronic Depression	17.99	9.62
15 months		
Not Depressed	4.81	3.75
Postpartum Depression	7.31	4.05
Early Childhood Depression	12.56	8.27
Chronic Depression	18.03	8.90
24 months		
Not Depressed	4.96	3.74
Postpartum Depression	7.48	4.02
Early Childhood Depression	16.13	9.35

Table B3 Continued

Variable	*M*	*SD*
Chronic Depression	17.78	9.92
36 months		
Not Depressed	4.84	3.88
Postpartum Depression	7.42	3.98
Early Childhood Depression	14.89	8.82
Chronic Depression	17.46	9.29

Note: Not Depressed N = 550, Postpartum Depression N = 114, Early Childhood Depression N = 125, and Chronic Depression N = 164.

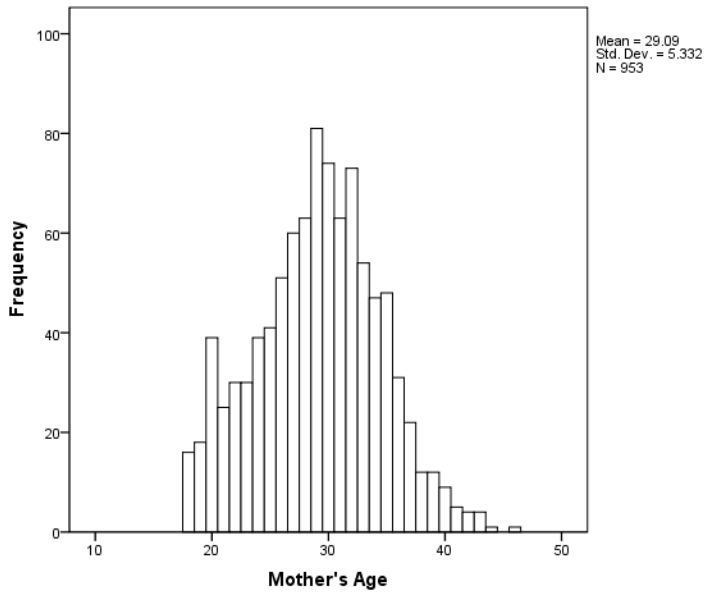

Figure B1. Distribution of Mother Age in Years.

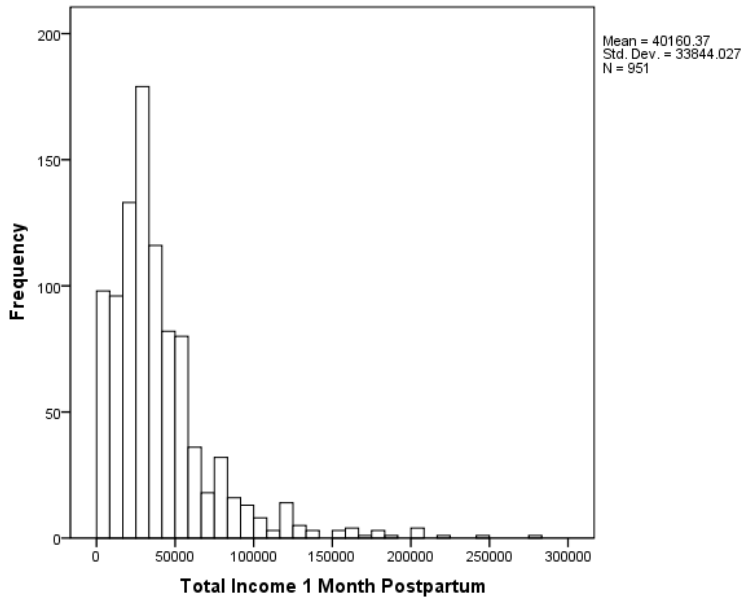

Figure B2. Distribution of Total Family Income at 1 Month in Dollars.

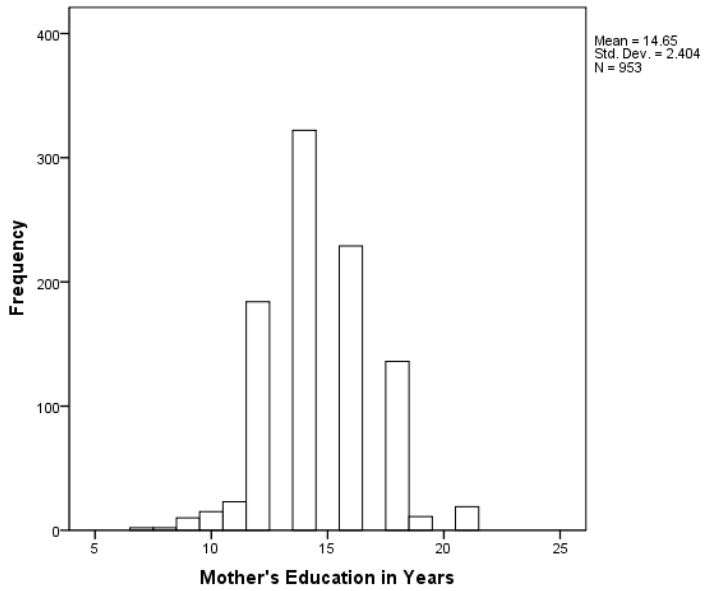

Figure B3. Distribution of Maternal Education in Years.

APPENDIX C

ASSUMPTIONS

Table C1

Independence of IV and CV Test, Model 1A, IV: Depression Pattern, DV: CPT G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.108	.955
Depression*Total Family Income	3	1.773	.151
Depression*Site	27	1.612	.028
Depression*Child Age at Test	3	2.324	.074
Error	500		

Table C2

Independence of IV and CV Test, Model 1B, IV: Depression Pattern, DV: TOH G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.027	.994
Depression*Total Family Income	3	.529	.620
Depression*Site	27	1.298	.143
Depression*Child Age at Test	3	.593	.620
Error	753		

Table C3

Independence of IV and CV Test, Model 1C, IV: Depression Pattern, DV: CPT G4

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	3.010	.030
Depression*Total Family Income	3	4.354	.005
Depression*Site	27	1.215	.212
Depression*Child Age at Test	3	1.911	.127
Error	533		

Table C4

Independence of IV and CV Test, Model 1D, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.166	.322
Depression*Total Family Income	3	1.306	.271
Depression*Site	27	.965	.517
Depression*Child Age at Test	3	.282	.839
Error	726		

Table C5

Independence of IV and CV Test, Model 1E IV: Depression Pattern, DV: TOL Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.524	.666
Depression*Total Family Income	3	1.297	.275
Depression*Site	27	1.097	.336
Depression*Child Age at Test	3	.977	.403
Error	674		

Table C6

Independence of IV and CV Test, Model 1F, IV: Depression Pattern, DV: Stroop Age 15

Source	*df*	*F*	*P*
Depression Pattern*Maternal Education	3	1.059	.366
Depression*Total Family Income	3	.650	.583
Depression*Site	27	1.009	.453
Depression*Child Age at Test	3	.654	.581
Error	666		

Table C7

Independence of IV and CV Test, Model 2A Males, IV: Depression Pattern, DV: CPT G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.799	.495
Depression*Total Family Income	3	1.523	.209
Depression*Site	27	1.432	.084
Depression*Child Age at Test	3	1.184	.371
Error	226		

Model 2A Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2A, Males (DV of CPT G1) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no statistically significant interactions, suggesting no evidence of problems with this assumption. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's Test, $F(39, 238) = .907$, $p = .632$.

Table C8

Independence of IV and CV Test, Model 2A Females, IV: Depression Pattern, DV: CPT G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.803	.147
Depression*Total Family Income	3	.371	.774
Depression*Site	27	1.644	.028
Depression*Child Age at Test	3	1.212	.306
Error	222		

Model 2A Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2A, Females (DV of CPT G1) to determine if any interactions were present between independent variables and covariates. A significant interaction was noted between depression and total family income (*partial* η^2 = .167), indicating that children from lower income families scored consistently lower than children from higher income families, with the exception of children with depressed mothers during early childhood. Total family income was retained as a covariate and the interaction term was left in the model to capture this variance (see Appendix C for tables). There were no violated assumptions related to homogeneity of variance, as

assessed by a Levene's test, $F(39, 234) = .839$, $p = .740$.

Table C9

Independence of IV and CV Test, Model 2B Males, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.314	.270
Depression*Total Family Income	3	2.110	.099
Depression*Site	27	.695	.872
Depression*Child Age at Test	3	1.304	.273
Error	345		

Model 2B Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2B, Males (DV of TOH G1) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no statistically significant interactions, suggesting no evidence of problems with this assumption. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 357) = .934$, $p = .586$.

Table C10

Independence of IV and CV Test, Model 2B Females IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.326	.807
Depression*Total Family Income	3	2.077	.103
Depression*Site	27	1.768	.012
Depression*Child Age at Test	3	2.855	.037
Error	356		

Model 2B Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2B, Females (DV of TOH G1) to determine if any interactions were present between independent variables and covariates (see Appendix C). A significant interaction was noted between depression and site and depression and child age at testing (*partial* η^2= .011 and .023, respectively). Visual examination of the depression by site interaction indicated that there was no meaningful interpretation, so it was excluded from the model. The depression and child age at testing interaction indicated that children who were older at the time of testing scored higher than their younger counterparts when their mothers had no depression or chronic depression. This

interaction was retained in the model to capture this variance. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 368) = .812$, $p = .783$.

Table C11

Independence of IV and CV Test, Model 2C Males, IV: Depression Pattern, DV: CPT G4

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	2.290	.079
Depression*Total Family Income	3	2.184	.091
Depression*Site	27	1.243	.3197
Depression*Child Age at Test	3	.617	.605
Error	235		

Model 2C Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2C (DV of CPT G4) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions indicating that this assumption was not violated.

There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 247) = 1.154$, $p = .256$.

Table C12

Independence of IV and CV Test, Model 2C Females, IV: Depression Pattern, DV: CPT G4

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	3.860	.010
Depression*Total Family Income	3	1.971	.119
Depression*Site	26	1.197	.239
Depression*Child Age at Test	3	1.452	.228
Error	247		

Model 2C Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2C (DV of CPT G4) to determine if any interactions were present between independent variables and covariates (see Appendix C). A significant interaction was noted between depression and maternal education (*partial* $\eta^2 =$ .045). Visual examination of an interaction plot indicated that children with more

educated mothers scored higher on the CPT G4 than did children with less educated mothers. Maternal education was retained as a covariate and the interaction term was left in the model to capture this variance. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(38, 259) = 1.183$, $p = .224$.

Table C13

Independence of IV and CV Test, Model 2D Males, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.392	.759
Depression*Total Family Income	3	1.179	.318
Depression*Site	27	1.166	.263
Depression*Child Age at Test	3	.353	.787
Error	331		

Model 2D Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2D (DV of TOH G5) to determine if any interactions were present between independent variables and covariates (see Appendix C

for tables). There were no statistically significant interactions, suggesting no evidence of problems with this assumption. The Levene's test was not statistically significant, $F(39,343) = 1.004$, $p = .468$, indicating no problem with heteroscedasticity.

Table C14

Independence of IV and CV Test, Model 2D Females, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.325	.808
Depression*Total Family Income	3	3.333	.020
Depression*Site	27	.991	.481
Depression*Child Age at Test	3	.236	.871
Error	343		

Model 2D Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2D, Females (DV of TOH G5) to determine if any interactions were present between independent variables and covariates (see Appendix C). A significant interaction was noted between depression and total family income

(*partial* η^2 = .028). Visual examination of the interaction indicated that higher income was most protective of child TOH G5 scores when the mother had postpartum or chronic depression. The interaction term was left in the model to capture this variance. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 355) = 1.096$, $p = .326$.

Table C15

Independence of IV and CV Test, Model 2E Males, IV: Depression Pattern, DV: TOL Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.279	.840
Depression*Total Family Income	3	.820	.484
Depression*Site	27	.822	.722
Depression*Child Age at Test	3	.641	.589
Error	296		

Model 2E Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2E, Males (DV of TOL Age 15) to determine if any

interactions were present between independent variables and covariates (see Appendix C for tables). There were no statistically significant interactions, suggesting no evidence of problems with this assumption. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39,308) = 1.330$, $p = .098$.

Table C16

Independence of IV and CV Test, Model 2E Females, IV: Depression Pattern, DV: TOL Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.323	.809
Depression*Total Family Income	3	1.641	.180
Depression*Site	27	1.266	.174
Depression*Child Age at Test	3	1.516	.210
Error	326		

Model 2E Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2E, Females (DV of TOL Age 15) to determine if any interactions were present between independent variables and covariates (see

Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 338) = .997$, $p = .480$.

Table C17

Independence of IV and CV Test, Model 2F Males, IV: Depression Pattern, DV: Stroop Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	4.275	.006
Depression*Total Family Income	3	2.670	.048
Depression*Site	27	.887	.631
Depression*Child Age at Test	3	.651	.583
Error	291		

Model 2F Males Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2F, Males (DV of Stroop Age 15) to determine if any interactions were present between independent variables and covariates. Significant

interactions were noted between depression and total family income and depression and maternal education. Because these interaction were small in magnitude (*partial* η^2 = .027 and .042, respectively), they were retained as covariates and the interaction terms were left in the model to capture this variance. An interaction plot indicated that children from higher income families scored better on the Stroop when their mothers had postpartum or early childhood depression, but not chronic depression. More maternal education only acted as a protective factor for child Stroop scores when she had depression during early childhood. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39,303) = 1.239$, $p = .165$.

Table C18

Independence of IV and CV Test, Model 2F Females, IV: Depression Pattern, DV: Stroop Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.099	.305
Depression*Total Family Income	3	.495	.686
Depression*Site	27	1.610	.031
Depression*Child Age at Test	3	3.380	.019
Error	323		

Model 2F Females Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 2F (DV of Stroop Age 15) to determine if any interactions were present between independent variables and covariates. Significant interactions were noted between depression and site and depression and child age at testing (*partial* η^2 = .119 and .030, respectively). Upon examination of the interaction between site and depression, it was determined that there was no meaningful interpretation and that it would create noise in the final model, so it was excluded. Visual examination of the interaction between age of testing and depression indicated that older children scored lower on the Stroop when their mothers had postpartum or early childhood depression, but higher than younger children when their mothers were depressed. The Levene's test was significant, $F(39, 335) = 1.702$, $p = .007$, but unstandardized residuals were visually examined, indicating no problem with heteroscedasticity.

Table C19

Independence of IV and CV Test, Model 3A Caucasians, IV: Depression Pattern, DV: CPT G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.125	.200
Depression*Total Family Income	3	1.227	.300
Depression*Site	27	1.014	.448
Depression*Child Age at Test	3	3.627	.013
Error	383		

Model 3A Caucasians Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3A, Caucasians (DV of CPT G1) to determine that they were independent (see Appendix C for tables). There was a significant association between depression and child age at testing (*partial* η^2 = .028), but it was retained in the final model to account for this variance. Visual examination of an interaction plot indicated that younger Caucasian children scored higher on CPT G1 for all depression groups except no depression. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 395) = 1.191$, $p = .207$.

Table C20

Independence of IV and CV Test, Model 3A Minorities, IV: Depression Pattern, DV: CPT G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.023	.995
Depression*Total Family Income	3	.409	.747
Depression*Site	22	.944	.542
Depression*Child Age at Test	3	.404	.751
Error	70		

Model 3A Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3A, Minorities (DV of CPT G1) to determine that they were independent (see Appendix C for tables). There were no significant interactions. The Levene's test was not significant, $F(34, 82) = 1.145$, $p = .304$, indicating no problems with heteroscedasticity.

Table C21

Independence of IV and CV Test, Model 3B Caucasians, IV: Depression Pattern, DV: TOH G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.106	.957
Depression*Total Family Income	3	.755	.520
Depression*Site	27	1.055	.390
Depression*Child Age at Test	3	.647	.585
Error	643		

Model 3B Caucasians Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3B, Caucasians (DV of TOH G1) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 625) = .965$, $p = .532$.

Table C22

Independence of IV and CV Test, Model 3B Minorities, IV: Depression Pattern, DV: TOH G1

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.288	.834
Depression*Total Family Income	3	.747	.527
Depression*Site	23	1.209	.258
Depression*Child Age at Test	3	.551	.649
Error	92		

Model 3B Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3B, Minorities (DV of TOH G1) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(35, 104) = .926$, $p = .591$.

Table C23

Independence of IV and CV Test, Model 3C Caucasians, IV: Depression Pattern, DV: CPT G4

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	3.549	.015
Depression*Total Family Income	3	2.755	.042
Depression*Site	27	1.005	.460
Depression*Child Age at Test	3	.971	.406
Error	432		

Model 3C Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3C, Caucasians (DV of CPT G4) to determine that they were independent (see Appendix C for tables). There was a significant association between depression and maternal education and depression and total income (*partial* η^2 = .023 and .020, respectively), but they were retained in the model to capture that variance. The depression by education interaction indicated that more maternal education was protective of child CPT G4 scores when the mother was depressed during early childhood and chronically. The depression by income interaction indicated that children from high income families scored better when their mother was depressed postpartum or

chronically, but lower when she was depressed during the early childhood. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 422)$, = .836, p = .746.

Table C24

Independence of IV and CV Test, Model 3C Minorities, IV: Depression Pattern, DV: CPT G4

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.703	.553
Depression*Total Family Income	3	.352	.788
Depression*Site	23	.886	.615
Depression*Child Age at Test	3	2.083	.110
Error	75		

Model 3C Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3D, Minorities (DV of CPT G4) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as

assessed by a Levene's test, $F(35, 87) = 1.130$, $p = .318$.

Table C25

Independence of IV and CV Test, Model 3D Caucasians, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.949	.120
Depression*Total Family Income	3	1.432	.232
Depression*Site	27	1.369	.102
Depression*Child Age at Test	3	.580	.629
Error	612		

Model 3D Caucasians Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3D, Caucasians (DV of TOH G5) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 595) = 1.096$, $p = .321$.

Table C26

Independence of IV and CV Test, Model 3D Minorities, IV: Depression Pattern, DV: TOH G5

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.454	.715
Depression*Total Family Income	3	.677	.568
Depression*Site	24	1.315	.177
Depression*Child Age at Test	3	.270	.847
Error	94		

Model 3D Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3D, Minorities (DV of TOH G5) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(36, 106) = 1.283$, $p = .165$.

Table C27

Independence of IV and CV Test, Model 3E Caucasians, IV: Depression Pattern, DV: TOL Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.599	.616
Depression*Total Family Income	3	.939	.422
Depression*Site	27	.951	.538
Depression*Child Age at Test	3	.435	.728
Error	566		

Model 3E Caucasians Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3E, Caucasians (DV of TOL Age 15) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 552) = 1.256$, $p = .142$.

Table C28

Independence of IV and CV Test, Model 3E Minorities, IV: Depression Pattern, DV: TOL Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	1.207	.312
Depression*Total Family Income	3	.572	.635
Depression*Site	24	1.050	.416
Depression*Child Age at Test	3	.180	.910
Error	85		

Model 3E Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3E, Minorities (DV of TOL Age 15) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(36, 97) = 1.288$, $p = .165$.

Table C29

Independence of IV and CV Test, Model 3F Caucasians, IV: Depression Pattern, DV: Stroop Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.942	.420
Depression*Total Family Income	3	.398	.754
Depression*Site	27	.723	.846
Depression*Child Age at Test	3	1.164	.323
Error	558		

Model 3F Caucasians Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3F, Caucasians (DV of Stroop Age 15) to determine that they were independent (see Appendix C for tables). There were no significant interactions. There were no violated assumptions related to homogeneity of variance, as assessed by a Levene's test, $F(39, 544) = .975$ $p = .516$.

Table C30

Independence of IV and CV Test, Model 3F Minorities, IV: Depression Pattern, DV: Stroop Age 15

Source	*df*	*F*	*p*
Depression Pattern*Maternal Education	3	.469	.704
Depression*Total Family Income	3	.095	.963
Depression*Site	24	2.454	.001
Depression*Child Age at Test	3	1.044	.377
Error	85		

Model 3F Minorities Assumptions. The independent variable (depression pattern) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 3F, Minorities (DV of Stroop Age 15) to determine that they were independent (see Appendix C for tables). There was a significant interaction between depression and site (*partial* η^2 = .409). Visual examination of the interaction plot indicated that there was no meaningful interpretation, so this interaction term was excluded from the final model. The Levene's test was significant, $F(36, 97) = 2.212$, $p = .001$, but visual examination of an unstandardized residuals plot showed no sign of systematic effects. The residuals were evenly and randomly scattered, indicating no heteroscedasticity.

Table C31

Independence of IV and CV Test, Model 4A, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.990	.859	5	420	.509
CPTG1*Total Family Income	.988	1.050	5	420	.388
CPTG1*Site	.864	1.387	45	1881.865	.046
CPTG1*Child Age at Test	.988	1.022	5	420	.404

Model 4A Assumptions. The independent variable (CPT Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 4A (DV of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There was a significant interaction between depression and site (*partial* η^2= .029; see Appendix C for tables). Visual examination of the interaction indicated no interpretable result, so it was excluded from the final model. The test of equality of covariance matrices was significant, Box's M = 177.764, F(135, 158839.303) = 1.255, p = .024, but none of the Levene's tests of equality of error variances were significant [CBCL G3, F(9, 440) = 1.363, p = .203; CBCL G4, F(9, 440) = .747, p = .666; CBCL G5, F(9, 440) = .443, p = .917; CBCL G6, F(9, 440) = .678, p = .729; CBCL Age 15, F(9, 440) = .397, p = .936].

Table C32

Independence of IV and CV Test, Model 4B, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.990	1.253	5	628	.283
CPTG1*Total Family Income	.991	1.163	5	628	.326
CPTG1*Site	.936	.933	45	2812.299	.599
CPTG1*Child Age at Test	.993	.912	5	628	.473

Model 4B Assumptions. The independent variable (TOH Grade 1) and covariates (total family income, maternal education, site of data collection, child age at testing, and child ethnicity) were examined for Model 4B (DV of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions. The test of equality of covariance matrices was significant, Box's M = 194.664, $F(135, 363788.461) = 1.397$, $p = .002$, but none of the Levene's tests of equality of error variances were significant [CBCL G3, $F(9, 648) = .826$, $p = .592$; CBCL G4, $F(9, 648) = .798$, $p = .619$; CBCL G5, $F(9, 648) = .610$, $p = .789$; CBCL G6, $F(9, 648) = .541$, $p = .845$; CBCL Age 15, $F(9, 648) = .527$, $p = .855$].

Table C33

Independence of IV and CV Test, Model 4C, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.993	1.178	3	502	.317
CPTG1*Total Family Income	.998	.344	3	502	.794
CPTG1*Site	.970	.571	27	1466.741	.962
CPTG1*Child Age at Test	.998	.390	3	502	.760

Model 4C Assumptions. The independent variable (CPT Grade 4) and covariates (total family income, maternal education, site of data collection, child age at testing, and child ethnicity) were examined for Model 4C (DV of CBCL Grades 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions. The test of equality of covariance matrices was non-significant, Box's M = 73.582, F(54, 273766.939) = 1.330, p = .053, and none of the Levene's tests of equality of error variances were significant [CBCL G5, F(9, 520) = .674, p = .733; CBCL G6, F(9, 520) = .291, p = .977; CBCL Age 15, F(9, 520) = .535, p = .850].

Table C34

Independence of IV and CV Test, Model 4D, IV: TOH G5, DVs: CBCL G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.998	.428	3	677	.733
CPTG1*Total Family Income	.996	.894	3	677	.444
CPTG1*Site	.973	.682	27	1977.831	.889
CPTG1*Child Age at Test	.998	.5501	3	677	.682

Model 4D Assumptions. The independent variable (TOH Grade 5) and covariates (total family income, maternal education, site of data collection, child age at testing, and child ethnicity) were examined for Model 4D (DV of CBCL Grade 6 and age 15) to determine if any interactions were present between independent variables and covariates (see Appendix C for tables). There were no significant interactions. The test of equality of covariance matrices was non-significant, Box's M = 36.650, $F(27, 1117382.497)$ = 1.342, p = .110, and none of the Levene's tests of equality of error variances were significant [CBCL G6, $F(9, 708)$ = .754, p = .659; CBCL Age 15, $F(9, 708)$ = .638, p = .765].

Table C35

Independence of IV and CV Test, Model 5A Males, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.975	.957	5	187	.46
CPTG1*Total Family Income	.975	.941	5	187	.456
CPTG1*Site	.733	1.342	45	839	.069
CPTG1*Child Age at Test	.985	.564	5	187	.727

Model 5A Males Assumptions. The independent variable (CPT Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5A, Males (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 191.112, $F(135, 28450.893) = 1.264$, $p = .021$, but none of the Levene's tests of equality of error variances were significant [CBCL G3, $F(9, 207) = 1.324$, $p = .226$; CBCL G4, $F(9, 207) = .366$, $p = .950$; CBCL G5, $F(9, 207) = 1.042$, $p = .407$; CBCL G6, $F(9, 207) = 1.240$, $p = .272$; CBCL Age 15, $F(9, 207) = 1.043$, $p = .407$].

Table C36

Independence of IV and CV Test, Model 5A Females, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.970	1.253	5	203	.286
CPTG1*Total Family Income	.980	.808	5	203	.545
CPTG1*Site	.801	1.027	45	911.171	.425
CPTG1*Child Age at Test	.981	.7992	5	203	.117

Model 5A Females Assumptions. The independent variable (CPT Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5A, Females (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 183.254, F(135, 41873.052) = 1.230, p = .036, but none of the Levene's tests of equality of error variances were significant [CBCL G3, F(9, 223) = 1.187, p = .304; CBCL G4, F(9, 223) = 1.088, p = .372; CBCL G5, F(9, 223) = .706, p = .703; CBCL G6, F(9, 223) = 1.304, p = .236; CBCL Age 15, F(9, 223) = 1.271, p = .254].

Table C37

Independence of IV and CV Test, Model 5B Males, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG1*Maternal Education	.980	1.142	5	283	.338
TOHG1*Total Family Income	.980	1.175	5	283	.321
TOHG1*Site	.826	1.231	45	1269.031	.143
TOHG1*Child Age at Test	.993	.383	5	283	.860

Model 5B Males Assumptions. The independent variable (TOH Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5B Males (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 215.504, $F(135, 68311.174) = 1.484$, $p = .000$, and CBCL G5 and G6 of the Levene's tests of equality of error variances were significant [CBCL G3, $F(9, 303) = 1.214$, $p = .286$; CBCL G4, $F(9, 303) = 1.125$, $p = .344$; CBCL G5, $F(9, 303) = 2.004$, $p = .039$; CBCL G6, $F(9, 303) = 1.962$, $p = .043$; CBCL Age 15, $F(9, 303) = 1.431$, $p = .174$]. Box's M is likely hypersensitive with so

many degrees of freedom; similarly, the Levene's Test is amplified by large N and a big covariance matrix.

Table C38

Independence of IV and CV Test, Model 5B Females, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG1*Maternal Education	.997	.186	5	315	.968
TOHG1*Total Family Income	.977	1.492	5	315	.192
TOHG1*Site	.867	1.021	45	1412.174	.434
TOHG1*Child Age at Test	.993	.457	5	315	.808

Model 5B Females Assumptions. The independent variable (TOH Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5B, Females (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 179.857, F(135, 100824.168) = 1.251, p = .026, but none of the Levene's tests of equality of error variances were significant [CBCL G3, F(9, 335) = .717, p = .693; CBCL G4, F(9, 335) =

1.172, p = .312; CBCL G5, F(9, 335) = .406, p = .931; CBCL G6, F(9, 335) = 1.518, p = .140; CBCL Age 15, F(9, 335) = .659, p = .746].

Table C39

Independence of IV and CV Test, Model 5C Males, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG4*Maternal Education	.966	2.642	3	228	.050
CPTG4*Total Family Income	.978	1.743	3	228	.159
CPTG4*Site	.938	.547	27	666.520	.971
CPTG4*Child Age at Test	.989	.841	3	228	.473

Model 5C Males Assumptions. The independent variable (CPT Grade 4) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5C, Males (DVs of CBCL Grades 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's M = 69.524, F(54, 48353.208) = 1.218, p = .131, and the CBCL G6 Levene's tests of equality of error variances was

significant [CBCL G5, $F(9, 246) = 1.327$, $p = .223$; CBCL G6, $F(9, 246) = 1.939$, $p = .047$; CBCL Age 15, $F(9, 246) = .852$, $p = .569$].

Table C40

Independence of IV and CV Test, Model 5C Females, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG4*Maternal Education	.980	1.695	3	246	.169
CPTG4*Total Family Income	.996	.353	3	246	.787
CPTG4*Site	.945	.520	27	719.089	.980
CPTG4*Child Age at Test	.990	.805	3	246	.492

Model 5C Females Assumptions. The independent variable (CPT Grade 4) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5C, Females (DVs of CBCL Grades 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's $M = 86.070$, $F(54, 77539.690) = 1.519$, $p = .008$, as was the CBCL G6 Levene's tests of equality of error variances [CBCL G5, $F(9, 264) = .848$, $p = .572$; CBCL G6, $F(9, 264) = 2.034$, $p = .036$; CBCL

Age 15, $F(9, 264) = 1.267$, $p = .255$]. Box's M is likely hypersensitive with so many degrees of freedom; similarly, the Levene's Test is amplified by large N and a big covariance matrix.

Table C41

Independence of IV and CV Test, Model 5D Males, IV: TOH G5, DVs: CBCL G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG4*Maternal Education	.999	.157	3	318	.925
CPTG4*Total Family Income	.989	1.142	3	318	.332
CPTG4*Site	.958	.514	27	929.366	.982
CPTG4*Child Age at Test	.995	.518	3	318	.670

Model 5D Males Assumptions. The independent variable (TOH Grade 5) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5D, Males (DVs of CBCL Grades 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's $M = 46.361$, $F(27, 258705.071) = 1.676$, $p = .015$, and the CBCL G6 Levene's tests of equality of error variances was significant [CBCL G6, $F(9, 342) = 1.913$, $p = .049$; CBCL Age 15, $F(9, 342) = 1.164$, $p = .318$].

Box's M is likely hypersensitive with so many degrees of freedom; similarly, the Levene's Test is amplified by large N and a big covariance matrix.

Table C42

Independence of IV and CV Test, Model 5D Females, IV: TOH G5, DVs: CBCL G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG5*Maternal Education	.992	.919	3	331	.432
TOHG5*Total Family Income	.991	1.052	3	331	.370
TOHG5*Site	.938	.793	27	967.333	.765
TOHG5*Child Age at Test	.996	.459	3	331	.711

Model 5D Females Assumptions. The independent variable (TOH Grade 5) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 5D, Females (DVs of CBCL Grades 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 46.014, $F(27, 257641.568) = 1.665$, $p = .016$, but none of the Levene's tests of equality of error variances were

significant [CBCL G6, $F(9, 356) = 1.304$, $p = .233$; CBCL Age 15, $F(9, 356) = .445$, $p = .910$].

Table C43

Independence of IV and CV Test, Model 6A Caucasians, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.983	1.1515	325	.333	.017
CPTG1*Total Family Income	.974	1.763	5	325	.120
CPTG1*Site	.844	1.251	45	1456.907	.125
CPTG1*Child Age at Test	.996	.273	5	325	.928

Model 6A Caucasians Assumptions. The independent variable (CPT Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6A, Caucasians (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's $M = 162.529$, $F(135, 98249.727) = 1.132$, $p = .141$, and none of the Levene's tests of equality of error

variances were significant [CBCL G3, $F(9, 345) = .677$, $p = .730$; CBCL G4, $F(9, 345) = .430$, $p = .918$; CBCL G5, $F(9, 345) = .667$, $p = .739$; CBCL G6, $F(9, 345) = 1.262$, $p = .257$; CBCL Age 15, $F(9, 345) = .329$, $p = .965$].

Table C44

Independence of IV and CV Test, Model 6A Minorities, IV: CPT G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG1*Maternal Education	.967	.449	5	65	.813
CPTG1*Total Family Income	.832	2.618	5	65	.032
CPTG1*Site	.708	.524	45	293.864	.995
CPTG1*Child Age at Test	.925	1.049	5	65	.397

Model 6A Minorities Assumptions. The independent variable (CPT Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6A, Minorities (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There was a significant interaction between income and CPT scores (*partial* $\eta^2 = .075$). Visual examination indicated that children from higher income families

scored lower on the CPT at G1. The interaction term was retained in the final model. The test of equality of covariance matrices was non-significant, Box's M = 158.588, $F(105, 3350.469) = 1.101$, $p = .230$, but CBCL G4, G5, and Age 15 of the Levene's tests of equality of error variances were significant [CBCL G3, $F(9, 85) = 1.834$, $p = .074$; CBCL G4, $F(9, 85) = 2.696$, $p = .008$; CBCL G5, $F(9, 85) = .706$, $p = 2.860$; CBCL G6, $F(9, 85) = .361$, $p = .950$; CBCL Age 15, $F(9, 85) = 3.491$, $p = .001$]. The Levene's Test is amplified by large N and a big covariance matrix.

Table C45

Independence of IV and CV Test, Model 6B Caucasians, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG1*Maternal Education	.987	1.381	5	508	.230
TOHG1*Total Family Income	.982	1.867	5	508	.098
TOHG1*Site	.920	.953	45	2275.510	.562
TOHG1*Child Age at Test	.993	.665	5	508	.650

Model 6B Caucasians Assumptions. The independent variable (TOH Grade 1) and covariates (total family income, maternal education, site of data collection, and child age

at testing) were examined for Model 6C, Caucasians (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 193.068, F(135, 233394.411) = 1.375, p = .003, but none of the Levene's tests of equality of error variances were significant [CBCL G3, F(9, 528) = .355, p = .956; CBCL G4, F(9, 528) = .467, p = .897; CBCL G5, F(9, 528) = .256, p = .985; CBCL G6, F(9, 528) = 1.439, p = .168; CBCL Age 15, F(9, 528) = .252, p = .986].

Table C46

Independence of IV and CV Test, Model 6B Minorities, IV: TOH G1, DVs: CBCL G3, G4, G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG1*Maternal Education	.907	1.853	5	90	.111
TOHG1*Total Family Income	.911	1.764	5	90	.128
TOHG1*Site	.570	1.207	45	405.695	.177
TOHG1*Child Age at Test	.931	1.329	5	90	.259

Model 6B Minorities Assumptions. The independent variable (TOH Grade 1) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6D, Minorities (DVs of CBCL Grades 3, 4, 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's M = 178.914, F(120, 4176.759) = 1.138, p = .147, but CBCLT G4 and Age 15 of the Levene's tests of equality of error variances were significant [CBCL G3, F(9, 110) = .1.553, p = .139; CBCL G4, F(9, 110) = 2.035, p = .042; CBCL G5, F(9, 110) = 2.083, p = .037; CBCL G6, F(9, 110) = .480, p = .885; CBCL Age 15, F(9, 110) = 3.743, p = .000]. The Levene's Test is amplified by large N and a big covariance matrix.

Table C47

Independence of IV and CV Test, Model 6C Caucasians, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG4*Maternal Education	.997	.402	3	395	.752
CPTG4*Total Family Income	.998	.244	3	395	.866
CPTG4*Site	.954	.698	27	1154.246	.874
CPTG4*Child Age at Test	.997	.412	3	395	.745

Model 6C Caucasians Assumptions. The independent variable (CPT Grade 4) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6C, Caucasians (DVs of CBCL Grades 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was significant, Box's M = 81.955, F(54, 172255.807) = 1.471, p = .014, but none of the Levene's tests of equality of error variances were significant [CBCL G5, F(9, 413) = .405, p = .932; CBCL G6, F(9, 413) = 1.107, p = .357; CBCL Age 15, F(9, 413) = .296, p = .976].

Table C48

Independence of IV and CV Test, Model 6C Minorities, IV: CPT G4, DVs: CBCL G5, G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
CPTG4*Maternal Education	.952	1.341	3	79	.267
CPTG4*Total Family Income	.970	.824	3	79	.485
CPTG4*Site	.812	.632	27	231.363	.922
CPTG4*Child Age at Test	.990	.260	3	49	.854

Model 6C Minorities Assumptions. The independent variable (CPT Grade 4) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6C Minorities (DVs of CBCL Grades 5, 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's M = 64.976, F(48, 6081.852) = 1.168, p = .200, but the CBCL Age 15 Levene's tests of equality of error variances was significant [CBCL G5, F(9, 97) = 1.880, p = .064; CBCL G6, F(9, 97) = .789, p = .627; CBCL Age 15, F(9, 97) = 2.334, p = .020]. The Levene's Test is amplified by large N and a big covariance matrix.

Table C49

Independence of IV and CV Test, Model 6D Caucasians, IV: TOH G5, DVs: CBCL G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG5*Maternal Education	.996	.773	3	550	.509
TOHG5*Total Family Income	.994	1.192	3	550	.312
TOHG5*Site	.961	.812	27	1606.962	.741
TOHG5*Child Age at Test	.995	.888	3	550	.447

Model 6D Caucasians Assumptions. The independent variable (TOH Grade 5) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6D, Caucasians (DVs of CBCL Grades 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's M = 37.047, F(27, 756807.552) = 1.353, p = .104, and none of the Levene's tests of equality of error variances were significant [CBCL G6, F(9, 578) = 1.475, p = .153; CBCL Age 15, F(9, 578) = .325, p = .967].

Table C50

Independence of IV and CV Test, Model 6D Minorities, IV: TOH G5, DVs: CBCL G6, Age 15

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*
TOHG5*Maternal Education	.982	.599	3	99	.617
TOHG5*Total Family Income	.964	1.222	3	99	.306
TOHG5*Site	.726	1.241	27	289.773	.195
TOHG5*Child Age at Test	.951	1.717	3	99	.168

Model 6D Minorities Assumptions. The independent variable (TOH Grade 5) and covariates (total family income, maternal education, site of data collection, and child age at testing) were examined for Model 6D, Minorities (DVs of CBCL Grades 6, and age 15) to determine if any interactions were present between independent variables and covariates. There were no significant interactions (see Appendix C for tables). The test of equality of covariance matrices was non-significant, Box's M = 34.976, F(27, 7324.604) = 1.180, p = .238, but the CBCL Age 15 Levene's tests of equality of error variances was significant [CBCL G6, F(9, 120) = 1.004, p = .441; CBCL Age 15, F(9, 120) = 3.916, p = .000].

APPENDIX D

ANCOVA TABLES

Table D1

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on CPT Scores at Grade 1 (Model 1A)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	2.078	3	.693	1.044	.373	.006
Total Family Income	4.060	1	4.060	6.118	.014	.011
Maternal Education	6.841	1	6.841	10.307	.001	.019
Site	2.952	9	.328	.494	.879	.008
Child Age at Test	3.402	1	3.402	5.126	.024	.009
Error	355.748	536	.664			

Table D2

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on TOH Scores at Grade 1 (Model 1B)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	321.048	3	107.016	2.498	.059	.009
Total Family Income	55.544	1	55.544	1.296	.255	.002
Maternal Education	177.216	1	177.216	4.136	.042	.005
Site	1218.719	9	135.413	3.161	.001	.035
Child Age at Test	153.740	1	153.740	3.589	.059	.009
Error	33802.683	789	42.842			

Table D3

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on CPT Scores at Grade 4 (Model 1C)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	5.209	3	1.736	2.365	.070	.012
Total Family Income	2.813	1	2.813	3.832	.051	.007
Maternal Education	10.372	1	10.372	14.130	.000	.024
Site	6..633	9	.737	1.004	.435	.016
Child Age at Test	.041	1	.041	.054	.816	.000
Dep Pattern * Mat Ed	5.881	3	1.960	2.671	.047	.014
Dep Pattern * Income	8.217	3	2.739	3.731	.011	.019
Error	413.271	563	.734			

Table D4

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on TOH Scores at Grade 5 (Model 1D)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	159.971	3	53.324	1.037	.376	.004
Total Family Income	350.127	1	350.127	6.807	.009	.009
Maternal Education	407.461	1	407.461	7.921	.005	.010
Site	1137.785	9	126.421	2.458	.009	.028
Child Age at Test	24.780	1	24.780	.482	.488	.001
Error	39195.312	762	51.437			

Table D5

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on TOL at Age 15 (Model 1E)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	526.525	3	175.508	1.005	.390	.004
Total Family Income	332.974	1	332.974	1.906	.168	.003
Maternal Education	2518.321	1	2518.321	14.418	.000	.020
Site	2470.367	9	2470.367	1.571	.120	.020
Child Age at Test	12.770	1	12.770	.073	.787	.000
Error	124014.752	710	174.669			

Table D6

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Stroop Scores at Age 15 (Model 1F)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.017	3	.006	1.126	.338	.005
Total Family Income	.000	1	.000	.003	.954	.000
Maternal Education	.000	1	.000	.062	.804	.000
Site	.096	9	.096	2.109	.027	.026
Child Age at Test	.005	1	.005	.891	.346	.001
Error	3.561	702	.005			

Table D7

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male CPT Scores at Grade 1 (Model 2A Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.022	3	.007	.011	.999	.000
Total Family Income	.204	1	.204	.296	.587	.001
Maternal Education	2.779	1	2.779	4.035	.046	.015
Site	5.176	9	.575	.835	.584	.028
Child Age at Test	1.617	1	1.617	2.347	.127	.009
Error	180.470	262	.689			

Table D8

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female CPT Scores at Grade 1 (Model 2A Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.080	3	.027	.042	.988	.000
Total Family Income	6.753	1	6.753	10.689	.001	.040
Maternal Education	2.637	1	2.637	4.174	.042	.016
Site	6.833	9	.759	1.202	.294	.041
Child Age at Test	2.978	1	2.978	4.713	.031	.018
Dep * Income	2.827	3	.942	1.492	.217	.017
Error	161.104	255	.632			

Table D9

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male TOH Scores at Grade 1 (Model 2B Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	672.128	3	224.043	5.625	.001	.042
Total Family Income	5.774	1	5.774	.145	.704	.000
Maternal Education	8.996	1	8.996	.225	.635	.001
Site	600.089	9	66.677	1.674	.093	.038
Child Age at Test	152.775	1	152.775	3.836	.051	.010
Error	15175.127	381	39.830			

Table D10

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female TOH Scores at Grade 1 (Model 2B Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	208.921	3	69.640	1.589	.191	.012
Total Family Income	57.171	1	57.171	1.305	.254	.003
Maternal Education	224.688	1	224.688	5.128	.024	.013
Site	1035.431	9	115.048	2.626	.006	.057
Child Age at Test	2.128	1	2.128	.049	.826	.000
Depression * Child Age	213.150	3	71.050	1.622	.184	.012
Error	17044.104	389	43.815			

Table D11

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male CPT Scores at Grade 4 (Model 2C Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	2.504	3	.835	1.138	.334	.012
Total Family Income	.578	1	.578	.788	.376	.003
Maternal Education	7.241	1	7.241	9.867	.002	.035
Site	2.324	9	.258	.352	.956	.012
Child Age at Test	.461	1	.461	.629	.428	.002
Error	198.860	271	.734			

Table D12

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female CPT Scores at Grade 4 (Model 2C Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	6.000	3	2.000	2.662	.048	.028
Total Family Income	4.743	1	4.743	6.315	.013	.022
Maternal Education	11.264	1	11.264	14.995	.000	.051
Site	9.742	9	1.082	1.441	.170	.044
Child Age at Test	.040	1	.040	.053	.818	.000
Dep * Mat Ed	5.761	3	1.920	2.557	.056	.027
Error	209.583	279	.751			

Table D13

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male TOH Scores at Grade 5 (Model 2D Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	565.376	3	188.459	3.952	.009	.031
Total Family Income	612.036	1	312.036	12.834	.000	.033
Maternal Education	454.333	1	454.333	9.527	.002	.025
Site	889.488	9	98.832	2.072	.031	.047
Child Age at Test	23.046	1	23.046	.483	.487	.001
Dep * Income	764.843	3	254.948	5.346	.001	.041
Error	17930.669	376	47.688			

Table D14

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female TOH Scores at Grade 5 (Model 2D Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	41.065	3	13.688	.277	.842	.002
Total Family Income	197.171	1	197.171	3.997	.046	.010
Maternal Education	559.048	1	559.048	11.333	.001	.029
Site	880.329	9	97.814	1.983	.040	.045
Child Age at Test	46.579	1	46.579	.944	.332	.002
Error	18695.512	379	49.329			

Table D15

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male TOL Scores at Age 15 (Model 2E Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	559.670	3	186.557	.996	.395	.009
Total Family Income	677.362	1	677.362	3.616	.058	.011
Maternal Education	485.954	1	485.954	2.594	.108	.008
Site	2476.809	9	275.201	1.469	.158	.038
Child Age at Test	.013	1	.013	.000	.993	.000
Error	62194.580	332	187.333			

Table D16

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female TOL Scores at Age 15 (Model 2E Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	416.726	3	138.909	.955	.478	.023
Total Family Income	.255	1	.255	.002	.968	.000
Maternal Education	2326.856	1	2326.856	14.266	.000	.038
Site	1401.216	9	155.691	.955	.478	.023
Child Age at Test	34.904	1	34.904	.214	.644	.001
Error	59044.052	362	163.105			

Table D17

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Male Stroop Scores at Age 15 (Model 2F Males)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.048	3	.004	.939	.018	.031
Total Family Income	.000	1	.000	.007	.933	.000
Maternal Education	.003	1	.003	.733	.393	.000
Site	.083	9	.009	1.949	.045	.052
Child Age at Test	.004	1	.004	.939	.333	.003
Dep * Income	.028	3	.009	1.971	.118	.018
Dep * Mat Ed	.049	3	.016	3.458	.017	.031
Error	1.515	321	.005			

Table D18

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Female Stroop Scores at Age 15 (Model 2F Females)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.016	3	.005	1.029	.380	.009
Total Family Income	.000	1	.000	.053	.817	.000
Maternal Education	.003	1	.003	.629	.428	.002
Site	.083	9	.009	1.769	.073	.042
Child Age at Test	.003	1	.003	.655	.419	.002
Error	1.862	359	.005			

Table D19

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic CPT G1 (Model 3A Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	7.852	3	2.617	4.221	.006	.030
Total Family Income	3.222	1	3.222	5.198	.023	.012
Maternal Education	1.643	1	1.643	2.651	.104	.006
Site	2.877	9	.320	.516	.863	.011
Child Age at Test	.583	1	.583	.940	.333	.002
Dep * Age	7.545	3	2.515	4.056	.007	.028
Error	279.511	443	.631			

Table D20

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race CPT G1 (Model 3A Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	1.198	3	.399	.518	.671	.015
Total Family Income	.105	1	.105	.136	.713	.001
Maternal Education	7.486	1	7.486	9.719	.002	.088
Site	4.363	9	.485	.629	.770	.053
Child Age at Test	6.907	1	6.907	8.967	.003	.082
Error	77.800	101	.770			

Table D21

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic TOH G1 (Model 3B Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	286.277	3	95.737	2.200	.087	.010
Total Family Income	6.038	1	6.038	.139	.709	.000
Maternal Education	104.224	1	104.224	2.402	.122	.004
Site	1049.914	9	116.657	2.689	.004	.036
Child Age at Test	92.737	1	92.737	2.138	.144	.003
Error	28155.755	649	43.383			

Table D22

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race TOH G1 (Model 3B Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	64.330	3	21.443	.557	.645	.013
Total Family Income	21.203	1	21.203	.550	.460	.004
Maternal Education	30.904	1	30.904	.802	.372	.006
Site	519.208	9	57.690	1.498	.156	.098
Child Age at Test	76.611	1	76.611	1.989	.161	.016
Error	4776.338	124	38.519			

Table D23

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic CPT G4 (Model 3C Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	7.515	3	2.505	3.513	.015	.023
Total Family Income	.535	1	.535	.751	.387	.002
Maternal Education	7.999	1	7.999	11.218	.001	.025
Site	4.112	9	.457	.641	.762	.013
Child Age at Test	.144	1	.144	.202	.653	.000
Dep * Mat Ed	7.963	3	2.654	3.722	.012	.025
Dep * Income	5.783	3	1.928	2.703	.045	.018
Error	313.750	440	.713			

Table D24

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race CPT G4 (Model 3C Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.926	3	.309	.418	.741	.012
Total Family Income	1.880	1	1.880	2.543	.114	.023
Maternal Education	3.011	1	3.011	4.074	.046	.037
Site	12.718	9	1.413	1.912	.058	.139
Child Age at Test	.001	1	.001	.001	.978	.000
Error	79.074	107	.739			

Table D25

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic TOH G5 (Model 3D Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	14.838	3	4.946	.102	.959	.000
Total Family Income	167.157	1	167.157	3.448	.064	.006
Maternal Education	203.419	1	203.419	4.196	.041	.007
Site	615.762	9	68.419	1.411	.179	.020
Child Age at Test	21.350	1	21.350	.440	.507	.001
Error	30012.071	619	48.485			

Table D26

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race TOH G5 (Model 3D Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	346.702	3	115.567	2.005	.117	.045
Total Family Income	.000	1	.000	.000	.999	.000
Maternal Education	61.733	1	61.733	1.071	.303	.008
Site	1413.984	9	157.109	2.726	.006	.162
Child Age at Test	3.836	1	3.836	.067	.797	.001
Error	7318.772	127	57.628			

Table D27

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic TOL 15 (Model 3E Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	486.663	3	162.221	.918	.432	.005
Total Family Income	272.470	1	272.470	1.543	.215	.003
Maternal Education	1796.162	1	1796.164	10.169	.002	.017
Site	1874.164	9	208.240	1.179	.306	.018
Child Age at Test	33.973	1	33.973	.192	.661	.000
Error	101736.239	576	176.625			

Table D28

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race TOL 15 (Model 3E Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	1016.612	3	338.871	2.205	.091	.053
Total Family Income	47.957	1	47.957	.312	.577	.003
Maternal Education	780.645	1	780.645	5.080	.026	.041
Site	3389.923	9	376.658	2.451	.014	.157
Child Age at Test	.298	1	.298	.002	.965	.000
Error	18133.728	118	153.676			

Table D29

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Caucasian Non-Hispanic Stroop 15 (Model 3F Caucasians)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.010	3	.003	.620	.602	.003
Total Family Income	.001	1	.001	.143	.706	.000
Maternal Education	.001	1	.001	.237	.627	.000
Site	.078	9	.009	1.703	.085	.026
Child Age at Test	.000	1	.000	.000	.998	.000
Error	2.906	568	.005			

Table D30

Analysis of Covariance for Depression Pattern, Total Family Income, Maternal Education, Site, and Child Age at Test on Other Race Stroop 15 (Model 3F Minorities)

Source	*SS*	*df*	*MS*	*F*	*p*	*partial* η^2
Depression Pattern	.019	3	.006	1.364	.257	.031
Total Family Income	.001	1	.001	.310	.579	.002
Maternal Education	.020	1	.020	4.270	.041	.033
Site	.021	1	.021	4.547	.035	.035
Child Age at Test	.014	1	.014	3.089	.081	.024
Error	.590	126	.005			

Table D31

Multivariate Analysis of Covariance for Model 4A

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G1	.988	1.035	5	432	.396	.012
Maternal Education	.995	.437	5	432	.822	.005
Total Income	.989	.990	5	432	.423	.011
Site	.885	1.195	45	1935.544	.177	.024
Child Age at Test	.992	.709	5	432	.617	.008

Table D32

Multivariate Analysis of Covariance for Model 4B

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G1	.989	1.416	5	640	.216	.011
Maternal Education	.993	.919	5	640	.468	.007
Total Income	.998	.298	5	640	.914	.002
Site	.918	1.234	45	2865.978	.138	.017
Child Age at Test	.995	.624	5	640	.681	.005

Table D33

Multivariate Analysis of Covariance for Model 4C

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G4	.997	.480	3	514	.696	.003
Maternal Education	.998	.266	3	514	.850	.002
Total Income	.996	.724	3	514	.538	.004
Site	.923	1.544	27	1501.787	.037	.026
Child Age at Test	.994	1.105	3	515	.347	.006

Table D34

Multivariate Analysis of Covariance for Model 4D

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G5	.999	.415	2	703	.660	.001
Maternal Education	.998	.584	2	703	.558	.002
Total Income	.993	2.482	2	703	.759	.001
Site	.964	1.462	18	1406	.095	.018
Child Age at Test	.993	2.482	2	703	.084	.001

Table D35

Multivariate Analysis of Covariance for Model 5A Males

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G1	.981	.646	6	196	.693	.019
Maternal Education	.996	.127	6	196	.993	.004
Total Income	.981	.629	6	196	.707	.019
Site	.733	1.166	54	1004.002	.196	.050
Child Age at Test	.972	.949	6	196	.461	.028

Table D36

Multivariate Analysis of Covariance for Model 5A Females

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G1	.967	1.448	5	215	.208	.033
Maternal Education	.976	1.055	5	215	.386	.024
Total Income	.962	1.703	5	215	.135	.038
Site	.864	.713	45	964.850	.922	.029
Child Age at Test	.980	.865	5	215	.506	.020

Table D37

Multivariate Analysis of Covariance for Model 5B Males

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G1	.988	.720	5	295	.609	.012
Maternal Education	.991	.517	5	295	.764	.009
Total Income	.992	.463	5	295	.804	.008
Site	.846	1.116	45	1322.709	.278	.033
Child Age at Test	.986	.855	5	295	.512	.014

Table D38

Multivariate Analysis of Covariance for Model 5B Females

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G1	.981	1.294	5	327	.266	.019
Maternal Education	.992	.540	5	327	.746	.008
Total Income	.989	.714	5	327	.614	.011
Site	.889	.867	45	1465.853	.721	.023
Child Age at Test	.981	.829	5	327	.530	.013

Table D39

Multivariate Analysis of Covariance for Model 5C Males

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G4	.999	.045	3	240	.987	.001
Maternal Education	.995	.440	3	240	.725	.005
Total Income	.997	.268	3	240	.848	.003
Site	.839	1.606	27	701.566	.027	.057
Child Age at Test	.993	.573	3	240	.633	.007

Table D40

Multivariate Analysis of Covariance for Model 5C Females

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G4	.984	1.434	3	258	.233	.016
Maternal Education	.999	.062	3	258	.980	.001
Total Income	.985	1.321	3	258	.268	.015
Site	.898	1.046	27	754.135	.402	.035
Child Age at Test	.992	.663	3	258	.576	.008

Table D41

Multivariate Analysis of Covariance for Model 5D Males

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G5	.999	.090	2	337	.914	.001
Maternal Education	.999	.103	2	337	.902	.001
Total Income	.998	.380	2	337	.684	.002
Site	.913	1.738	18	674	.029	.044
Child Age at Test	.986	2.460	2	337	.087	.014

Table D42

Multivariate Analysis of Covariance for Model 5D Females

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G5	.994	.985	2	351	.375	.006
Maternal Education	.998	.370	2	351	.691	.002
Total Income	.997	.537	2	351	.585	.003
Site	.947	1.076	18	702	.372	.027
Child Age at Test	.996	.722	2	351	.486	.004

Table D43

Multivariate Analysis of Covariance for Model 6A Caucasians

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G1	.988	.837	5	337	.524	.012
Maternal Education	.988	.807	5	337	.545	.012
Total Income	.982	.1224	5	337	.298	.018
Site	.868	1.075	45	1510.586	.341	.028
Child Age at Test	.998	.361	5	337	.875	.005

Table D44

Multivariate Analysis of Covariance for Model 6A Minorities

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G1	.819	3.368	5	76	.008	.181
Maternal Education	.821	3.319	5	76	.009	.179
Total Income	.783	4.216	5	76	.002	.217
Site	.451	1.486	45	343.069	.028	.147
Child Age at Test	.930	1.143	5	76	.345	.070
CPT * Income	.797	3.862	5	76	.004	.203

Table D45

Multivariate Analysis of Covariance for Model 6B Caucasians

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G1	.990	1.086	5	520	.367	.010
Maternal Education	.991	.990	5	520	.423	.009
Total Income	.995	.498	5	520	.778	.005
Site	.912	1.083	45	2324.189	.327	.018
Child Age at Test	.994	.585	5	520	.711	.006

Table D46

Multivariate Analysis of Covariance for Model 6B Minorities

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G1	.860	3.329	5	102	.008	.140
Maternal Education	.962	.816	5	102	.541	.038
Total Income	.990	.203	5	102	.961	.010
Site	.551	1.455	45	549.374	.033	.112
Child Age at Test	.924	1.666	5	102	.149	.076

Table D47

Multivariate Analysis of Covariance for Model 6C Caucasians

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G4	.991	1.281	3	407	.280	.009
Maternal Education	.997	.456	3	407	.713	.003
Total Income	.995	.664	3	407	.575	.005
Site	.921	1.266	27	1189.292	.165	.027
Child Age at Test	.996	.516	3	407	.672	.004

Table D48

Multivariate Analysis of Covariance for Model 6C Minorities

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
CPT G4	.912	2.932	3	91	.038	.088
Maternal Education	.984	.489	3	91	.691	.016
Total Income	.982	.570	3	91	.636	.018
Site	.695	1.307	27	266.409	.148	.114
Child Age at Test	.966	1.081	3	91	.361	.034

Table D49

Multivariate Analysis of Covariance for Model 6D Caucasians

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G5	.997	.916	2	573	.401	.003
Maternal Education	.999	.215	2	573	.807	.001
Total Income	.999	.402	2	573	.669	.001
Site	.966	1.119	18	1146	.327	.017
Child Age at Test	.993	1.969	2	573	.141	.007

Table D50

Multivariate Analysis of Covariance for Model 6D Minorities

Source	*Wilks' Lambda*	*F*	*Hyp df*	*Error df*	*p*	*partial* η^2
TOH G5	.991	.525	2	115	.593	.009
Maternal Education	.983	1.004	2	115	.370	.017
Total Income	.996	.211	2	115	.810	.004
Site	.825	1.289	18	230	.196	.092
Child Age at Test	.958	2.491	2	115	.087	.042

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EDUCATION

2011 – 2016	**Virginia Consortium Program in Clinical Psychology** Ph.D., expected August, 2016
2011 – 2013	**Old Dominion University** M.S., Experimental Psychology
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CLINICAL TRAINING

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PRESENTATIONS

Maduro, R. S., **Oettinger, E.**, & Paulson, J.F. (2014, November). *The Effects of Cumulative Risk for Maternal Harsh Parenting Practices on Child Cognitive Outcomes Over Time*. Poster presented at the Association for Behavioral and Cognitive Therapies Annual Convention, Philadelphia, Pennsylvania.

Oettinger, E., Paulson, J.F. (2014, August). *Moderating Effects of Care Quality on Cognitive Development of Male Children with Depressed Mothers*. Poster presented at the American Psychological Association Annual Convention, Washington DC.

Oettinger, E., Maduro, R.S., & Paulson, J.F. (2014, May). *Effects of Early Maternal Depression on Male Cognitive Development*. Poster presented at the Association for Psychological Science Annual Convention, San Francisco, California.

Oettinger, E., Paulson, J.F., & Maduro, R.S. (2013, August). *Moderating Effects of Day Care on Cognitive Development of Male Children with Depressed Mothers*. Poster presented at the American Psychological Association Annual Convention, Honolulu, Hawaii.