Genetic and Environmental Influences on Executive Functioning in Middle Childhood:

The Role of Early Adversity

by

Gianna Rea-Sandin

A Thesis Presented in Partial Fulfillment
of the Requirements for the Degree
Master of Psychology

Approved August 2018 by the
Graduate Supervisory Committee:

Kathryn Lemery-Chalfant, Chair
Robert Bradley
Kit Elam

ARIZONA STATE UNIVERSITY

December 2018

ABSTRACT

This study examined whether early adversity at 30-months moderated the heritability of common and individual components of EF at 8 years. It was hypothesized that early adversity would not moderate the common EF factor, but instead moderate individual EF components. The sample included 208 twin pairs from the Arizona Twin Project. Early Adversity, assessed at 30 months of age, included Parenting Daily Hassles, low perceived MOS social support, punitive punishment (Parental Responses to Child Misbehavior), home chaos (Confusion, Hubbub, and Order Scale), CES-D maternal depression, and low maternal emotional availability. EF at 8 years included the Eriksen Flanker Task, Continuous Performance Task, Digit Span Forward and Backward, and parent-reported Attentional Focusing and Inhibitory Control (Temperament in Middle Childhood Questionnaire). For both early adversity and EF, the first principal components were extracted as composites. A confirmatory factor analysis was also conducted to index common EF. Genetic analyses were tested on the common EF composites as well as each individual task using umx. Univariate models revealed genetic influences on all individual measures and common EF, with broad sense heritability from .22 (Digit Span Backwards) to .61 (parent-reported inhibitory control). Shared environmental influences were found for the Flanker Task (.13) and parent-reported inhibitory control (.24), and E was moderate to high (.40-.73) for all measures except parent-report inhibitory control (.15) and attentional focusing (.31). Moderation of heritability was not observed in for Digit Span Forward, Digit Span Backward, and Attentional Focusing. However, the nonshared environment was moderated for Common

EF, and the Flanker Task, and additive genes and the nonshared environment were moderated for the Continuous Performance Task and Inhibitory Control. Generally, total variance decreased as early adversity increased, suggesting that homes with low levels of adversity may allow children to interact with more proximal processes that can promote EF development.

TABLE OF CONTENTS

Page

Page

LIST OF TABLES

Table Page

LIST OF FIGURES

Figure Page

Early environmental experiences are important for children's development. When children are exposed to stressful early family experiences (e.g. physically punitive parental discipline, high levels of interparental conflict and violence, and cold, unsupportive parenting), they are at an elevated risk for the development of later cognitive and behavioral adjustment issues, including a wide range of mental and physical health problems (Cummings, Davies, & Campbell, 2002; Repetti, Taylor, & Seeman, 2002). Seeman, Singer, Horwitz, and McEwen (1997) found that stressful and threatening family environments increased the likelihood of biological dysregulations that lead to a buildup of allostatic load, which refers to premature physiological aging of the organism. Over time, physiological systems may lose their ability to function efficiently as the body adapts to the demands of the environment (Taylor, Way, & Seeman, 2011). One system that is implicated in the stress response is the hypothalamic-pituitary-adrenal (HPA) axis, which involves the release of corticosteroids, like cortisol. In the short term, this system is thought to have evolved to handle acute, life-threatening stressors by shifting energy for immediate use while suppressing nonessential systems (Sapolsky, 2002). Importantly, chronic activation of the HPA axis can lead to adverse long-term effects on health, like compromised immune function, increases in blood pressure, and neurochemical imbalances (McEwen & Seeman, 2003). Elevated levels of stress hormones can disrupt the brain's developing architecture (Shonkoff, Duncan, Fisher, Magnuson, & Raver, 2011). More specifically, it has been shown that stressful experiences alter the size and structure of the amygdala, hippocampus, and prefrontal

cortex, which can lead to functional differences in learning, memory, and executive functioning (Garner, Shonkoff, Siegel, Dobbins, Earls, et al., 2012).

The current study adds to this literature by using a genetically informed twin design to examine whether early adversity moderates the genetic and environmental influences on executive functioning in middle childhood. The Bioecological Theory of Nature-Nurture Effects (Bronfenbrenner & Ceci, 1994) posits that the heritability of a trait is dependent on an individual's interaction with the environment, therefore it is possible that exposure to early adversity could impact the genetic influence on later executive functioning.

Past research has established the effect of early home stress on adverse outcomes in middle childhood. For example, children who were unable to regulate physical aggression during early childhood had mothers that previously exhibited antisocial behaviors, parents with low income, and parents that experienced high levels of conflict with one another (Tremblay, Nagin, Seguin, Zoccolillo, Zelazo, et al., 2004). Along with externalizing behaviors, children were found to exhibit dysregulated affect, over-reactivity, and impulsivity if their parents used corporal punishment and had high aggression (Schwartz, Dodge, Pettit, & Bates, 1997; Shields & Cicchetti, 2001). Early adversity has also been correlated with poor academic performance. Home environments characterized by strict parenting, corporal punishment, and exposure to violence were linked with declines in grade point averages longitudinally (Dubow, Boxer & Huesman, 2009). However, these studies are correlational, therefore the direction of effect is unknown. These studies also do not elucidate whether these associations are genetic or environmental in nature.

However, many animal studies have shown the causal links between early life stress and altered hippocampal and HPA axis functioning. In one example, male rats that experienced daily separations from the dam showed increased HPA axis responsiveness to stressors (Aisa, Tordera, Lasheras, Del Rio, & Ramirez, 2007). Similarly, early social deprivation was causally linked with cognitive deficits in rhesus monkeys (Sanchez, Hearn, Do, Rilling, & Herndon, 1998), which has strong implications, as impairments of memory could potentially affect how an individual handles new stressors. Taken together, human and animal studies support the important role of exposure to early life stress and altered brain functioning and dysregulated emotional and behavioral outcomes.

One aspect of cognitive development of particular importance likely influenced by early adversity is executive functioning (EF). EF refers to higher order cognitive processes that underlie adaptive responses to novel, complex, or ambiguous situations (Hughes, Graham, & Grayson, 2005) and goal-directed behaviors, like inhibitory control, working memory, planning, and set shifting (Garon, Bryson, & Smith, 2008; Miyake, Friedman, Emerson, Witzki, & Howerter, 2000). Research has shown that EF is related to a multitude of outcomes: children's math and arithmetic proficiency (Blair & Razza, 2007), reading ability (Clark, Prior, & Kinsella, 2002), academic achievement (Biederman, Monuteaux, Doyle, Seidman, Wilens, Ferrero, et al., 2004), and emotion regulation (Carlson & Wang, 2007).

From the temperament literature, there is a related construct referred to as effortful control, which is formally defined as the ability to willfully or voluntarily inhibit, activate, or modulate attention and behavior, as well as EF constructs of planning, detecting errors, and integrating information relevant to selecting behavior (Eisenberg et

al., 2004). Effortful control involves a focus on automatic or nonconscious aspects of emotional reactivity and regulation, and is commonly assessed using parent report of Inhibitory Control and Attentional Focusing (Blair & Razza, 2007; Eisenberg et al., 2004). We include parent report assessments of Effortful Control in our study in an attempt to integrate these two related literatures, with a focus on working memory, inhibitory control, and attentional focusing. Overall, although phenotypic studies have established the relationship between early adversity and later negative outcomes, it is unclear how adverse environments affect the genetic and environmental etiology of EF.

The Development of Executive Functioning

EF, which is tied to the prefrontal cortex, can be identified as early as infancy (Carpenter, Nagell, & Tomasello, 1998), with accelerated periods of development between 2 and 5 years of age, and again at puberty (Anderson, Anderson, Northam, Jacobs, & Catroppa, 2001; Zelazo & Müller, 2002). Rutter and Rutter (1993) suggest that the transition from childhood to adulthood is characterized by dramatic shifts in cognitive flexibility. Similarly, Magnetic Resonance Imaging studies have consistently shown that compared to children, adolescents showed a higher volume of white matter (compared to gray matter) in the frontal cortex and parietal cortex, which reflects increased axonal myelination in these areas (e.g. Barnea-Goraly, Menon, Eckert, Tamm, Bammer, et al., 2005; Sowell, Peterson, Thompson, Welcome, Henkenius et al., 2003; Sowell, Thompson, Tessner, & Toga, 2001). Research on environmental influences on EF has primarily focused on preschool-aged children and adolescents. However, middle childhood is one developmental period that is understudied, therefore little is known about whether environments influence EF during this stage.

In the following section, we define the specific EF components: working memory, inhibitory control, and attentional focusing, as well as review research that examines these components in relation to various aspects of early home stress.

Working Memory

Working memory is comprised of many systems that actively maintain information amidst distractions and/or ongoing processing (Conway, Kane, Bunting, Hambrick, Wilhelm, & Engle, 2005; Miyake et al., 2000). Working memory is correlated with the superior frontal and intraparietal cortex, with the magnitude of the association increasing from childhood into early adulthood (Gathercole, Pickering, Ambridge, & Wearing, 2004; Klingberg, Forssberg, & Westerberg, 2002). Working memory has been associated with many facets of cognition, including comprehension, reasoning, problem solving, and language comprehension (e.g. Daneman & Carpenter, 1996; Engle, 2002). Similarly, Fitzpatrick, Archambault, Janosz, and Pagani (2015) found that a one-point increase in working memory skills assessed at preschool predicted a 26% reduction in the odds of being at risk for dropping out of high school (measured when the participants were in 7th grade). Studies have shown that working memory is moderately correlated with general cognitive ability (r = .26-.44) (Ando, Ono, & Wright, 2001), and similarly, Engle, Tuholski, Laughlin, and Conway (1999) found that measures of working memory capacity strongly predicted IQ, particularly fluid ability.

Researchers found that children who were observed to be more securely attached with their mothers between 15 months and 2 years showed higher performance on the backward digit span task, a common assessment of working memory (Matte-Gagné, Bernier, Sirois, Lalonde, & Hertz, 2017). In another study, children that experienced

childhood abuse or neglect had lower scores on working memory tasks compared to children that had no history of abuse or neglect (Perna & Kiefner, 2013).

Inhibitory Control

Inhibitory control refers to the ability to deliberately inhibit dominant, automatic, or prepotent responses when necessary (Miyake et al., 2000). These processes can be identified in late toddlerhood and continue to improve through childhood into adulthood (Bell & Livesey, 1985; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Studies utilizing functional magnetic resonance imaging have found that the anterior cingulate cortex (ACC), inferior frontal gyrus (IFG), the dorsal lateral prefrontal cortex, the frontal eye field (FEF), the posterior parietal cortex, the striatum, and the cerebellum are brain regions involved in inhibitory control (Curtis, Cole, Rao, D'Esposito, 2005; Rubia, Smith, Brammer, & Taylor, 2003). Lower inhibitory control has been associated with ADHD (Wilcutt, Doyle, Nigg, Faraone, & Pennington, 2005), poorer academic achievement (McClelland, Cameron, Duncan, Bowles, Acock, et al., 2014), and impaired theory of mind performance (Carlson & Moses, 2001).

Overall, studies have shown that parenting is related to performance on tasks that tap into inhibitory control abilities. One task that is commonly used to measure inhibitory control and attentional focusing is the Eriksen Flanker Task (Eriksen & Eriksen, 1974), which investigates the limits of visual selective attention as well as the efficiency of the frontal network (Eriksen & Eriksen, 1974; Stins et al., 2004). In one example, Matte-Gagné et al. (2018) found that children who were observed to be more securely attached to their mothers between 15 months and 2 years showed higher performance on the Flanker Task in kindergarten. Similarly, older children and early adolescents that had

high error rates on the Flanker Task also had parents that reported more inconsistent discipline, whereas participants with fewer errors on the EF task had parents with high parental involvement or parental responsibility (Sosic-Vasic, Kroner, Schneider, Vasic, Spitzer, et al., 2017).

Attentional Focusing

Attentional focusing refers to the ability to voluntarily focus or shift attention as needed (Eisenberg et al., 2004). Eisenberg, Zhou, Spinrad, Valiente, Fabes, et al. (2005) have found that attentional focusing can be identified in infancy and toddlerhood, and has been shown to be stable by early to middle childhood. The anterior cingulate gyrus is involved in appropriate attention allocation (Eisenberg, Guthrie, Fabes, Shepard, Losoya, et al., 2000; Posner & Rothbart, 1998). Difficulties with attention regulation have been linked with poorer academic performance (Raver, Smith-Donald, Hayes, & Jones, 2005), more problem behaviors (Rothbart & Bates, 2006), and emotional dysregulation (McClelland, Acock, & Morrison, 2006).

Young children that are exposed to chronic early life stress are more likely to have activation in the amygdala that promotes the influence of anxiety and fear on attentional and executive processes (Davis, Walker, & Lee, 1997). Other studies that examine the association between environmental stressors and attentional focusing yield similar results. In Kindergarten-aged children, chaos in the home was associated with limited attentional focusing abilities (Dumas, Nissley, Nordstrom, Smith, Prinz, et al., 2005). Similarly, maternal stress was shown to impact children's attentional focusing longitudinally from infancy to middle childhood (Pesonen, Räikkönen, Heinonen, Komsi,

Järvenpää, et al., 2008). Overall, these studies suggest that early adversity can have a lasting impact on the development of children's executive functioning.

Changing Executive Functioning

Although it has not yet been shown that early home stress has a lasting impact on children's EF, there are studies that suggest the effectiveness of interventions on EF. For example, using a computerized training program, working memory abilities improved in 7-9-year-old children with ADHD (Klingberg, Fernell, Olesen, Johnson, Gustaffson, et al., 2005). In another study, Chang, Shaw, Dishion, Gardner, and Wilson (2014) examined the effects of a parenting intervention on the development of children's inhibitory control from toddlerhood to middle childhood. The intervention condition underwent the Family Check-Up program, which focuses on improving parenting practices. Overall, based on parent report of their children's inhibitory control, children in the intervention condition showed higher levels of growth compared to the control condition. Lastly, in another study, researchers administered a computerized progressive attentional training program to 6- to 13-year-old children with ADHD (Shalev, Tsal, & Mevorach, 2007). Compared to the control group, the training group showed significant improvements in reading comprehension and parent-reported attentiveness.

Although these intervention studies demonstrate that EF skills may be improved by intervention throughout childhood and early adolescence, reviews of these studies suggest some caveats. In a systematic review, Diamond and Ling (2016) found that EF can be improved, however, wide transfer does not seem to occur. For example, working memory training programs improves working memory only, but not other skills like flexibility or self-control (e.g., Bergman Nutley et al., 2011; Harrison et al., 2013).

Therefore, in creating new EF interventions, it is necessary to develop trainings that tap into broader EF skills, as opposed to solely improving on a particular EF task (e.g., Stroop).

The Twin Design

The twin design allows us to address the extent to which EF is influenced by genetics or if it can be influenced by the developmental context, including early adversity. More specifically, when utilizing a genetically informed design, additive genetic (A), shared environmental (C), and nonshared environmental (E) influences on a particular trait are estimated. Additive genetic influences refer to the effects of multiple genes on a trait. The shared environment contributes to twin similarities, which may include the family environment, sharing the same classroom, etc. However, experiences that would cause the twins to become dissimilar are considered nonshared environmental influences. Examples may include the twins playing different sports, attending different schools, or one twin suffering an injury. Note that the estimate of the nonshared environment also includes measurement error.

Monozygotic (MZ; identical) twins share 100% of their segregating genes, whereas dizygotic (DZ; fraternal) twins share 50% of their segregating genes, on average. If within pair correlations are higher for MZs than DZ twins, then that suggests that there is a genetic influence on the phenotype. However, a DZ correlation that is more than half the MZ correlation shows that the shared environment is playing a role. The nonshared environment can be seen when MZ correlations are less than 1.0. The twin design also allows researchers to distinguish between common and unique genetic and environmental covariance across multiple phenotypes.

Genetic and Environmental Influences on Executive Functioning

Cognitive ability can be studied in terms of general cognitive ability (G) or specific cognitive abilities. More specifically, *G* can be the composite score of many tasks or the assessment of IQ, whereas specific cognitive abilities consist of verbal and spatial abilities, memory, and speed (Friedman, Miyake, Young, DeFries, Corley, & Hewitt, 2008). Twin studies have found that *g* is moderately heritable in children (between 41-50%), with the remaining variance being evenly split between the shared and nonshared environment (e.g. Chipuer, Rovine, & Plomin, 1990; Haworth, Wright, Luciano, Martin, De Geus, et al., 2010). However, the shared environment has been shown to play a larger role in early childhood, with heritability increasing over time (Knopik, Neiderhiser, DeFries, & Plomin, 2016). In late adolescence, studies have shown that genes account for roughly 70% of the variance in IQ (Haworth, Wright, Luciano, Martin, De Geus, et al., 2010; McGue, Bouchard, Iacono, & Lykken, 1993).

In contrast, studies have consistently found high heritability for a common EF factor. In third- through eighth-grade twins and triplets, Engelhardt, Briley, Mann, Harden, and Tucker-Drob (2015) found that the common EF factor was 100% heritable. Similarly, Friedman, Miyake, Young, DeFries, Corley, and Hewitt (2008) found that executive functions are influenced by a highly heritable (99%) common factor in young adults. It is surprising that this phenotype has been shown to be consistently highly heritable, considering the fact that other phenotypes (like intelligence) are modestly heritable in childhood and increase with age (Haworth et al., 2010).

There are a few genetically informed studies on working memory in early childhood and adolescence, but studies focusing on working memory in middle childhood

could not be identified. The studies that do exist suggest that working memory is moderately heritable, even at different age ranges. For example, Stins, de Sonneville, Groot, Polderman, van Baal, and Boomsma (2004) studied the heritability of working memory tasks in preschoolers (age = 5.8 years) and found that working memory reaction time could be explained by an additive genetic (A) and nonshared environmental (E) model (A = .54, E = .46). In another study, 12-year-old twins completed the digit span task and an AE model fit best (A = .56, E = .44; Polderman, Stins, Posthuma, Gosso, Verhulst, & Boomsma, 2006). Similarly, Ando, Ono, and Wright (2001) found that spatial and verbal working memory tasks were moderately heritable in Japanese twins ranging from 16 to 29 years of age (.46). Overall, studies show modest heritability and no influence of the shared environment for individual EF tasks, and high heritability for a common factor across tasks.

Twin studies on inhibitory control consistently show moderate heritability. A study using infant twins revealed that parent-report inhibitory control was primarily influenced by genes (.58) and the shared environment (.26), and observer report revealed no influence of the shared environment (A = .38, E = .62; Gagne & Saudino, 2010). Similar results were found in older children. Using the Stroop (1935) task, Polderman, de Geus, Hoekstra, Bartels, van Leeuwen, et al., (2009) found that inhibitory control was heritable for 9-, 12-, and 18-year cohorts (heritability was .36, .51, .51, respectively).

The Eriksen Flanker Task (Eriksen & Eriksen, 1974) is frequently used to measure inhibitory control and attentional focusing, however, this research has been conducted primarily in adolescents and adults. Using a sample of 12-year-old twins, Stins, van Baal, Polderman, Verhulst, and Boomsma (2004) found little evidence of

heritability on flanker performance. Interestingly, they reported higher twin intraclass correlations for opposite sex pairs (.55) than identical pairs (.38 MZ males; .35 MZ females) for the overall flanker interference effect, which refers to the reaction time (RT) of incongruent conditions minus the RT for congruent conditions. This pattern of twin correlations suggested no genetic influence on flanker performance. In another study using both adolescent and adult twins, the overall flanker interference effect was found to be highly heritable (.89; Fan, Wu, Fossella, & Posner, 2001). However, the sample size was very small (26 MZ and 26 same sex DZ pairs), therefore these results should be taken with caution. Also, the participants in this study ranged from 14 and 42 years of age, therefore it is difficult to draw conclusions from this study.

Parent-report of attentional focusing reveals moderate to high genetic influence. In one study, Lemery-Chalfant, Doelger, and Goldsmith (2008) found that observer report of attentional control was highly heritable (83%), with no influence of the shared environment. Other twin studies utilizing parent-report examine attentional focusing within effortful control. In one example, parent-reported effortful control for 3 to 7-year old children was moderately influenced by genetics (.43), the shared environment (.12), and the nonshared environment (.45; Goldsmith, Buss, & Lemery, 1997). Another study examining twins during middle childhood found significant genetic and modest shared and non-shared environmental effects on both mother-reported (A = .69, C = .10, E = .21) and father-reported (A = .50, C = .34, E = .17) effortful control (Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009). Together, these studies consistently demonstrate that genes have a moderate to high impact on attentional focusing.

Moderation of Genetic and Environmental Influences on Child Development

Overall, research on general and specific EF shows at least modest genetic influence. However, it is possible that in addition to direct main effects, the magnitude of genetic influence on EF could differ as a function of environmental context, which is referred to as moderated heritability. Similarly, Jensen (1981) and Scarr (1981) suggest that there could be a nonlinear effect of the family environment on cognitive ability. The concept of gene-environment interaction is also in line with the Bioecological Theory of Nature-Nurture Effects (Bronfenbrenner & Ceci, 1994). More specifically, this theory argues against the concept of a "single" heritability. Instead, the expression of any genetic trait is dependent on proximal processes, which are interactions between children and their environments. Understanding the moderation of genetic and environmental influences on a trait can help provide a more fine-grained approach to understanding how a measured environmental factor can impact individual differences across a particular phenotype. In one study, researchers observed the moderating role of parental education level on the genetic and environmental contributions to variation in verbal IQ in a diverse adolescent twin sample (Rowe et al., 1999). As parental education level increased, the heritability of verbal IQ increased and the influence of the shared environment decreased. In a similar study, Turkheimer, Haley, Waldron, D'Onofrio, and Gottesman (2003) found that the heritability of IQ was moderated by socioeconomic status in a highly impoverished sample of 7-year-old twins. Similar to Rowe et al., (1999), as socioeconomic status increased, the heritability of IQ increased (as the influence of the shared environment decreased). Results from these studies are in-line with one prediction from the bioecological theory (Bronfenbrenner & Ceci, 1994) that enhanced

environments will increase heritability because they allow genetic potentials to be more fully realized. If this is the case, then it is imperative to develop policies and interventions that focus on providing supportive environments for children, which could then promote their cognitive development.

The Current Study

The first goal of this study is to disentangle the genetic and environmental contributions to multiple components of EF in middle childhood. Based on previous literature (Polderman et al., 2006; Stins et al., 2004), we hypothesize that working memory will be influenced by genes and the non-shared environment. We hypothesize that both genes and the shared environment will have an influence on parent-report inhibitory control (Gagne & Saudino, 2010). We anticipate that parent-reported attentional focusing will be influenced by genes and the non-shared environment (Goldsmith et al., 1997; Lemery-Chalfant et al., 2008). Lastly, we predict that tasks that tap into both inhibitory control and attentional focusing will be influenced by genes and the shared environment (Fan et al., 2001; Stins et al., 2004).

Fitting a multivariate common pathway model, we will examine whether a common factor of EF includes effortful control, or whether they are two separate factors. This goal also includes testing the extent to which the same or different genetic and environmental influences impact various components of EF. Effortful control is referred to as the efficiency of executive attention (Rothbart & Bates, 2006), therefore we hypothesize that it will be included in the common factor. Further, we predict that the common factor will be highly heritable (Engelhardt et al., 2015; Friedman et al., 2008), whereas the residual individual components will be more influenced by the environment.

The final goal of the study involves examining whether the heritability of EF abilities is moderated by early adversity. This involves testing each component of EF separately, then utilizing the common factor. Based on previous research, we hypothesize that at lower levels of early adversity, heritability estimates will be higher for the individual tasks and scales (e.g. Rowe et al, 1999; Turkheimer et al., 2003). However, since we anticipate the heritability of the common factor to be high, we do not expect the genetic influence of the common factor to be moderated by early adversity.

Method

Participants

The study consisted of 416 twins (208 pairs) from the ongoing longitudinal Arizona Twin Project (Lemery-Chalfant, Clifford, McDonald, O'Brien, & Valiente, 2013). The Office of Vital Records, within the Division of Public Health Services, mailed letters (in English and Spanish) to a random sample of mothers over the age of 18 who had given birth to live twins in an Arizona hospital between July 2007 and July 2008. The twins and their mothers first participated when the twins were 12 months of age. The current sample consisted of 26.5% monozygotic (MZ), 37.1% same-sex dizygotic (DZ), and 36.5% opposite-sex DZ twins. The participants were 22.3% Hispanic, 58.7% Caucasian, 5.0% Asian, 2.8% African American, and 11.2% mixed race or other. Total household income ranged from less than $20,000 to over $150,000, with a median of $95,000. Parental education ranged from less than a high school diploma to a professional degree (median educational level was a college degree).

Procedure

When the twins were 30 months of age, primary caregivers completed telephone interviews about early adversity, parenting and their twins' health and development. Home visits were conducted when the twins were about 8 years of age (M = 8.41, SD = .40), which involved research assistants going into the home to collect data about the home environment and the twins' health, sleep behaviors, and temperament. During the home visit, each twin independently completed EF tasks on a laptop computer.

Measures

Zygosity Questionnaire for Young Twins

The Zygosity Questionnaire for Young Twins (Goldsmith, 1991) was administered to mothers when the twins were 12 months of age. This 32-item measure assesses zygosity by asking mothers about their pregnancies (e.g. the use of In Vitro Fertilization) and physical characteristics of their twins (e.g. hair and eye color). Compared to genotyping, studies have shown that parent-reported zygosity is over 95% accurate in assessing twin zygosity (e.g. Forget-Dubois, Perusse, Turecki, Girard, Billette, et al., 2003; Price, Freeman, Craig, Petrill, Ebersole, & Plomin, 2000). Zygosity information was also gathered from birth medical records, and after the 8-year home visit.

Measures of Early Adversity

Parenting Daily Hassles

The Parenting Daily Hassles (PDH; Crnic & Greenberg, 1990) questionnaire is a 20-item scale that measures everyday life events in parenting and parent-child interactions. Primary caregivers rate the frequency of the given occurrence on a 4-point

scale (1 = rarely, 2 = sometimes, 3 = a lot, 4 = constantly) and how hassled they felt by the event on a 5-point scale (1 = no hassle to 5 = big hassle). The PDH contains two subscales: Parenting Tasks Factor and Challenging Behavior Factor which are correlated r = .78. Reliability for both the frequency of each occurrence and the intensity of each occurrence was high (alpha = .81 and alpha = .90). Examples of items include "The twins are constantly under foot or in the way" and "The twins are difficult to manage in public places." Note that each question was changed to indicate "twins" instead of a singleton child. Also, for this study, we will only be using the Parenting Tasks Factor subscale in order to isolate the frequency of hassles that the parent experiences on a daily basis.

MOS Social Support Survey

The 6-item MOS Social Support Survey (Sherbourne & Stewart, 1991) assessed parental satisfaction with the availability of emotional and tangible support. Primary caregivers rate the frequency of support that was available on a 5-point Likert scale (1 = none of the time to 5 = all of the time). Items include "Someone to help you with daily chores if you need it" and "Someone who shows you love and affection." The items are totaled to create a sum score. Reliability for this measure is high (alpha = .81).

Parental Responses to Child Misbehavior

The Parental Responses to Child Misbehavior (PCRM; Holden & Zambarano, 1992) questionnaire was designed to determine techniques and frequency used by parents in response to their children's misbehaviors in an average week over the past month. The measure includes 12 discipline strategies, including reasoning, ignoring, and spanking with hand. The harsh discipline scale was used, and each item is scored on a 5-point Likert scale ranging from 0 (never) to 6 (nine or more times), and the items are totaled to

create a sum score. The PCRM has well-established criterion validity. More specifically, high parental discipline was associated with low positive maternal involvement and high levels of negativity (Holden & Zambarano, 1992).

Confusion, Hubbub, and Order Scale

The short version of the Confusion, Hubbub, and Order Scale (CHAOS; Matheny et al., 1995) assesses parents' perceptions of disorganization in the home. Parents indicate either 'true' or 'false' to items such as "Our home is a good place to relax" and "First thing in the day, we have a regular routine at home." One total score was derived per family. Reliability for this scale was acceptable (alpha = .79), and the CHAOS was correlated with parent-child interactions in the predicted direction, which demonstrates that it is a valid measure (Matheny et al., 1995).

Center for Epidemiological Studies Depression Scale

The Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) is a 20-item self-report measure of adult depressive symptoms in non-clinical populations. Respondents are asked to rate the frequency of each occurrence of each symptom in the past week on a 4-point scale, which ranges from rarely or none of the time (less than 1 day) to most or all of the time (5-7 days). Responses are totaled, and a score of 16 or higher is considered to have clinical significance. Reliability for this measure was high (alpha = .88; Orme, Reis, & Herz, 1986).

Emotional Availability Scale

The Emotional Availability Scale (EAS; Biringen, Robinson, & Emde, 2000) is a 28-item parent report questionnaire that assesses six dimensions of the emotional availability of the parent towards the young child and vice versa. The parental dimensions

are Sensitivity, Structuring, Nonintrusiveness, and Nonhostility, and the child dimensions include Child Responsiveness to the Parent and Child's Involvement of the Parent. Responses are totaled, and higher scores show more emotional availability. For our study, Cronbach's alpha was acceptable (alpha = .74). In this study, the questions ask about each twin separately.

Principal Component Representing Early Adversity

For the 30-month assessment, we included six standardized variables for a principal components analysis (PCA) without rotation (Table 1). The 30-month assessment produced two components. The first component, which accounted for 37.96% of the variance (eigenvalue = 2.28, loadings from .46 - .71), was retained. Standardized component scores were saved as a composite measure of Early Adversity, with higher scores representing more adversity.

Temperament in Middle Childhood Questionnaire

The Temperament in Middle Childhood Questionnaire (TMCQ; Simonds & Rothbart, 2006) is a parent-report measure used to assess temperament in young children. The TMCQ has 15 scales that tap various dimensions of temperament. For our study, the Attentional Focusing (alpha = .75) and Inhibitory Control (alpha = .72) scales were used. Primary caregivers rated their children on a 7-point scale ranging from 1 = *extremely untrue of your child* to 7 = *extremely true of your child*. An example of an item from the Attentional Focusing scale is, "When building or putting something together, becomes very involved in what s/he is doing, and works for long periods," and "Is good at following instructions" is an example item from the Inhibitory Control scale.

Digit Span

The Wechsler Intelligence Scale for Children (WISC-IV; Wechsler, 2003) digit span task is a test of working memory. We administered both the Digit Span Forward and Digit Span Backwards components of the task. Digit Span Forward requires that children repeat series of numbers read aloud by the experimenter exactly as they hear them. In contrast, in the Digit Span Backwards portion, the experimenter reads series of numbers aloud, and then children must repeat them to the experimenter backwards. The first set of digits in the forward task consists of three digits (and two for the backwards task), and the series increases by one digit every two trials. The task is stopped once the child misses both trials within a series. One point is given for each correct trial, and the raw scores on Digit Span Forward and Digit Span Backward were used for analysis. The Digit Span Forward component taps into short-term auditory memory, sequencing, and simple verbal expression (Hale, Hoeppner, & Fiorello, 2002), whereas the Digit Span Backward component is a sensitive measure of deficits in working memory (Rosenthal, Riccio, Gsanger, & Jarratt, 2006).

Psychology Experiment Building Language (PEBL)

The Psychology Experiment Building Language (PEBL; Mueller, 2013) is an open-source software system of behavioral test paradigms that researchers can use in their own studies (Mueller & Piper, 2014). For our study, we administered the Continuous Performance Task and the Eriksen Flanker Task using the PEBL system.

Continuous Performance Task

The Continuous Performance Task (CPT; Conners, 2000) is administered on a laptop computer in which a continuous series of stimuli (letters of the alphabet) are presented on a screen, and participants must either respond to target stimuli by pressing a

key or inhibit a response to non-target stimuli. There are multiple variations of the CPT, and we used the CPT-Not X (also known as Connor's CPT), where the participant must press a key in response to all letters of the alphabet except X. The 14-minute long task consisted of 360 letters (in 18 consecutive blocks of 20 trials) that appeared one at a time on the screen for approximately 250 milliseconds. Various measures can be generated from this task: RT to correct responses, standard error of hit RT, errors of omission, and errors of commission. Errors of omission refer to instances where the child presses the spacebar when the letter X is presented, whereas errors of commission refer to instances where the child fails to press the spacebar when the target (not X) is presented. In our study, these two variables were highly correlated (r = .87), therefore they were reversed and combined to represent errors on the task, where higher scores represent fewer errors.

This version of the CPT has been shown to result in a high number of responses, more errors of commission, more accurate and reliable measures of reaction time, and greater demand on the participants' ability to inhibit a response (Conners, Epstein, Angold, & Klaric, 2003). Split half reliability for all of the CPT performance measures range between .73 and .95, and test-retest reliabilities range from .55 and .84 after a 3-month interval (Conners, 2000).

Flanker Task

The flanker task (Eriksen & Eriksen, 1974) is a computer-administered paradigm where participants respond with a left or right key press to a central target arrow while ignoring congruent (e.g. >>>) or incongruent (e.g. <><) flanker arrows (Mayr, Awh, & Lawrey, 2003). It has been suggested that previous experience with the paradigm has little impact on performance, although researchers have seen reduced reaction time with

repeated exposure (Fan et al., 2001). The stimuli were presented on a laptop computer and participants responded using either the left or right shift key. To ensure the child understood the task, there were 12 practice trials before the actual task began, and the actual task lasted about 10 minutes. For each trial, RT and whether the response was correct was recorded. Flanking arrows pointing in the same direction were coded as congruent trials, whereas arrows pointing in the opposite direction were coded as incongruent trials. The overall flanker interference effect is the most commonly used measure of the Flanker task, which is the RT of incongruent trials minus the RT for congruent trials. However, it is less likely than the Linear Integration Speed Accuracy Score (LISAS; Vandierendonck, 2017) score to capture a greater proportion of variance than RT and proportion of error. The LISAS measure, which is used in this study, takes RT and proportion of error into account, along with the standard deviations of both measures which gives RT and proportion of error an equal weight. Scores were reversed, where higher scores indicate higher efficiency.

Principal Component Representing Common Executive Functioning

For the 8-year assessment, we included six standardized variables for a principal components analysis (PCA) without rotation (Table 2). The PCA was done without rotation to maximize the amount of variance accounted for in the first component. The 8-year assessment produced two components, with the first component explaining 33.69% of the variance (eigenvalue = 2.70, loadings from -.57 to .76). Standardized component scores from the first component were saved as a composite measure of EF, with higher scores representing better EF performance.

Common Pathway Models

Along with forming a PCA composite indexing common EF, a common pathway model was conducted to extract common EF from the variables as well as to examine ACE influences on the common factor and residual variables using OpenMx (Table 8), and fit statistics are described in Table 9. The first model, with all EF variables included, produced a common factor that was moderately heritable (.66). However, the paths from the common factor to the individual tasks were extremely small, with the exception of Attentional Focusing (.90). Given that Attentional Focusing was the only variable that was phenotypically related to Inhibitory Control, it is possible that the inclusion of Inhibitory Control was inflating the influence of Attentional Focusing in the common pathway model. Therefore, another model was conducted without Inhibitory Control. With this model, all of the paths contributed more equally, and the common factor revealed higher heritability estimates (.79).

A confirmatory factor analysis (CFA) was conducted in MPlus 7.11 without Inhibitory Control to extract a common factor score in order to conduct twin analyses. Loadings and fit statistics can be found in Table 10. Twin ICCs for the CFA common factor were similar to the PCA common factor (MZ = .56, DZ = .30). Univariate ACE estimates were also similar to the PCA common factor, with an AE model fitting the data best (A = .64, E = .36). There was significant moderation ($\Delta\chi2(2) = 4.02$, $p = .13$), such that E was lowest at high levels of early adversity. At the mean level of adversity, E was .04 (.07 at -1 SD, .01 at +1 SD).

Ultimately, given that both a PCA and CFA produced similar heritability estimates, we opted to use the PCA because each task contributed more equally to the score compared to the CFA.

Covariates

Covariates included child sex and child age. The effects of child sex and child age were regressed out of each variable and the residual scores were utilized for twin biometric analysis, as the models become too complex with multiple covariates (McGue & Bouchard, 1984).

Statistical Approach

Table 1 will include the mean, SD, minimum, maximum, skewness, and kurtosis for each variable. Variables with skewness above 2 or kurtosis above 7 will be transformed to approximate normality (West, Finch, & Curran, 1995). Univariate and multivariate outliers will be identified using Mahalanobis D^2 (Cohen, Cohen, West & Aiken, 2003), and zero order correlations will be conducted using MPlus 7.0 while controlling for twin interdependence. If zero order correlations show that certain tasks are highly related, then we will combine them to form a composite which will be used in our genetic analyses.

Estimating the roles of genes and the environment on a given factor is possible through the twin design (Neale & Maes, 2004). Within-pair (i.e. intraclass) correlations for MZ and DZ twins are used in order to estimate sources of variability (Falconer, 1989). Genes are said to play a role if MZ intraclass correlations are greater than DZ correlations. However, if the MZ correlation is less than twice the DZ correlation, then that suggests the shared environment is also significant.

Structural equation model fitting was conducted using OpenMX (Neale, et al., 2016) to obtain full univariate twin models which decompose the variance in a variable into latent additive genetic (A, linear effect of multiple genes), shared environmental (C, environmental experiences that increase cotwin similarity), and non-shared environmental (E, environmental experiences that cause twins to become dissimilar as well as measurement error) factors. MZ twins share 100% of their segregating DNA, therefore the latent A (also known as heritability) influence is correlated 1.00 between cotwins, and .50 for DZ twins, because DZ cotwins share 50% of their segregating genes. 'A' can be estimated by doubling the difference between MZ and DZ twin intraclass correlations. The shared environmental influence is calculated by subtracting the MZ correlation from double the DZ correlation. Lastly, because the total variance explained cannot exceed 1.00 (100%), the influence of the nonshared environment is calculated by subtracting the MZ intraclass correlation from 1.00. Note that this method can be used to assess quick estimates of genetic and environmental influence on a phenotype; however, we will be using Structural Equation Modeling to calculate these values, which also takes sample size and standard errors into account. Figure 1 depicts the univariate ACE model, which is used to ascertain the genetic and environmental influences on general EF as well as the specific EF tasks. After full models are fit, parameters are systematically dropped, and the fit of the reduced models are compared to the full model using the -2 log likelihood chi-square test of fit. A significant loss of fit indicates that the dropped path is required to represent the data, whereas a nonsignificant loss of fit implies that the reduced model represents the observed data as well as the full model.

A common pathway model (Figure 2; Neale & Maes, 2004) is used to test whether there is a common EF factor among the variables, or if EF and effortful control are distinct constructs. The covariance among each variable is represented as a latent factor (e.g. Common EF). This common factor has single estimates of A, C, and E, with each variable loading on this factor. There are direct phenotypic paths from the common factor to each variable, where genetic, shared environmental, and nonshared environmental variance that is independent of the common factor is also estimated.

Figure 3 depicts a moderated model such that the variance components attributable to latent genetic, shared, and nonshared environmental effects are a function of an environmental moderator (Purcell, 2002). More specifically, the first phenotype (the moderator, M) moderates the ACE components of the second phenotype (the trait, T). This model is used to test whether early adversity (the moderator) moderates the ACE estimates for general EF and specific EF tasks. Path coefficients represent the magnitude of the effect; therefore, they are expressed as linear functions of the moderator. If β_x is significantly non-zero, this represents an interaction between the path coefficient and the moderator (Purcell, 2002).

It is possible that the early adversity variables could be correlated with the genetic influences on executive functioning (gene-environment correlation; r_{GE}) rather than moderating the genetic influences. The moderated heritability model controls for gene-environment correlation by allowing for a main effect, which removes genetic effects shared between the trait and the moderator from the covariance model. In other words, r_{GE} will appear as a main effect, and any detected interactions will reflect interactions between the moderator and variance components specific to the trait (Purcell, 2002).

Results

Preliminary Analyses

Descriptive statistics and correlations for the early adversity variables are presented in Table 1. All of the early adversity measures were positively correlated with one another, and all variables were within acceptable ranges for skewness (+-2.00) and kurtosis (+-7.00; Muthen & Kaplan, 1985). Descriptive statistics and correlations for the executive functioning variables are presented in Table 2. For the CPT and Flanker variables, if a participant's value was greater than 3 SD from the mean, it was brought down to 3 SD from the sample mean for that condition (Pe, Vandekerckhove & Kuppens, 2013). For the CPT variables, 22 cases were adjusted, and 21 cases were adjusted for the Flanker variables. All EF variables were at least modestly positively correlated with one another, with the exception of Inhibitory Control, which was only significantly correlated with Flanker Congruent, Flanker Incongruent, and parent-reported Attentional Focusing. Results showed that CPT errors of omission and errors of commission, as well as Flanker Congruent and Flanker Incongruent, were highly correlated (.87 and .79, respectively). Therefore, two composites were created: the first was a CPT mean composite of CPT errors of omission and CPT errors of commission, and the second was a Flanker mean composite consisting of Flanker Congruent trials and Flanker Incongruent trials.

Quantitative Genetic Analyses

Twin Intraclass Correlations

Twin intraclass correlations (ICCs) are provided in Table 5. MZ twins were more similar than DZ twins for all variables, suggesting the role of additive genetic influences.

The ICCs also revealed the influence of the non-shared environment (and measurement error) on the task-based EF variables, as the MZ correlations were less than 1.00. Also, the DZ ICC for Attentional Focusing was less than half the MZ ICC, demonstrating that dominant genetic influences (D) may also contribute to trait variation.

Saturated Models

Saturated models were conducted for the common EF and individual EF variables to test for sex differences as well as rater contrast and assimilation effects. For common EF and the task-based variables, fully saturated models that freely estimate means, variances, and covariances for MZ and DZ twins were compared to models that constrained means and variances to be equal across twin pairs and zygosity groups and means, variances, and covariances to be equal across sex. Assimilation effects can be observed when DZ phenotypic variance is higher relative to the MZ group, whereas lower DZ variance suggests imitation effects (Neale & Maes, 2004). Means, variances, and covariances could be equated across sex, and means and variances across zygosity, with the exception of the Flanker Task. More specifically, means and variances could not be equated across twin order and zygosity, with MZ twins having more variance than DZ twins in this case.

Univariate ACE and ADE Models

Standardized estimates of A, C (or D), and E factors for common EF and for each individual EF measure, as well as fit statistics of the full and best fitting reduced model, are presented in Table 6. Univariate models revealed genetic influences on all individual measures and common EF, with broad sense heritability ranging from .22 (Digit Span

Backwards) to .61 (parent-reported inhibitory control). Shared environmental influences were found for the Flanker Task (.13) and parent-reported inhibitory control (.24), and E was moderate to high (.40-.73) for all measures except parent-report inhibitory control (.15) and attentional focusing (.31). For Digit Span Backward, we did not have the power to differentiate between AE and CE models, therefore the full model was retained as the best model.

Moderated Heritability Models

Model fit statistics for the moderation models are provided in Table 7. First, direct prediction of the mean was tested for each full model, which would be indicative of gene-environment correlation. If prediction of the mean is non-significant, then that indicates that there is no significant relationship between the moderator and the variable. However, if dropping the path results in a significant loss of fit for the model, then there is a relationship between the moderator and the variable due to gene-environment correlation and the means model is retained in the final model to control for gene-environment correlation (Price & Jaffee, 2008).

Moderation of the means for CPT, Flanker, Digit Span Forward, and Digit Span Backward were dropped, since these paths could be dropped without a significant loss of fit for these variables. Once moderation of the mean was dropped for these variables, the reduced model was used as the comparison model to examine moderation on the other paths. For CPT, there was significant moderation of A and E ($\Delta\chi2(1) = .03$, $p = .87$) such that A was highest at higher levels of adversity. Similarly, there was significant

moderation of E ($\Delta\chi2(2) = .51$, $p = .78$), such that E was lowest at higher levels of early adversity.

In Table 7, the results are based on using the full model as the comparison (with or without including the means model based on its significance). After moderation of the mean was tested, full moderation was tested for each variable (i.e., moderation of A, C and E estimates), and then various moderation paths were dropped. If dropping a particular path results in a non-significant p-value (>.05), then it can be dropped because it does not result in a significant loss of fit. Significant moderation of heritability was observed for common EF, CPT, Flanker, and Inhibitory Control (Figure 4). More specifically, for common EF, E was moderated ($\Delta\chi2(2) = 5.17$, $p = .08$) such that E was lowest at higher levels of early adversity. At the mean of early adversity, E was .41 (.84 at -1 SD, .13 at +1 SD). There was full moderation of all paths for CPT ($\chi2(264) = 1681.62$), such that A was highest and E was lowest at higher levels of early adversity, however estimating moderation on C produced a very large negative change in -2LL, therefore these results should be taken with caution. At the mean of early adversity, A was 7.02 (1.56 at -1 SD, 42.90 at +1 SD) and E was 18.66 (44.09 at -1 SD, 4.00 at +1 SD). For Flanker, there was moderation on E ($\Delta\chi2(2) = .85$, $p = .65$), such that E was lowest at higher levels of early adversity. At the mean level of early adversity, E was 5163.76 (8497.15 at -1 SD, 2656.37 at +1 SD). Lastly, for Inhibitory Control, there was moderation on A and E ($\Delta\chi2(1) = .53$, $p = .47$), such that A was highest and E was lowest at higher levels of early adversity. At the mean of early adversity, A was .20 (.11 at -1 SD, .32 at +1 SD) and E was .06 (.11 at -1 SD, .03 at +1 SD).

Influence of ADHD Symptomology

Executive Functioning may be highly related to symptoms of ADHD. A boxplot was used to assess the distribution of the ADHD composite of the Health and Behavior Questionnaire (HBQ; Armstrong & Goldstein, 2003). Children in the upper quartile were removed (n=6) and analyses were conducted to examine whether these extreme cases were driving any of the effects. Excluding these individuals did not impact results, with phenotypic and intraclass correlations remaining the same magnitude and level of significance. Therefore, these individuals were retained in the study.

Discussion

There were three main goals of this study. The first was to examine the genetic and environmental influences on executive functioning (Continuous Performance Task, Eriksen Flanker Task, Digit Span Forward, and Digit Span Backward), and effortful control (parent-reported Attentional Focusing and Inhibitory Control) in middle childhood. The second goal of the study was to extract and assess the etiology of a common factor of EF, and the main goal of the study was to examine whether the heritability of EF abilities in middle childhood was moderated by early adversity measured approximately six years earlier in toddlerhood. The study also aimed to examine the overlap between objective EF and parent-reported effortful control measures. Generally, individual differences in EF in middle childhood could be explained by additive genetics and the nonshared environment. Similarly, a common factor of EF, extracted using both principal components analysis and confirmatory factor analysis, was influenced by genes and the nonshared environment. Finally, early adversity moderated the etiology of the common EF factor, the Continuous Performance Task, the Eriksen Flanker Task, and parent-reported inhibitory control. Together, the common factor indicated that the EF tasks were indicators of the same latent construct. Similarly, although these variables were influenced by genetics, our study demonstrates the role of the nonshared environment in explaining differences in EF, as well as the ability of early adversity to moderate the etiology of this heritable trait.

Heritability of EF in Middle Childhood

Generally, we found that genes and the nonshared environment explained individual differences for all EF tasks, with the exception of Digit Span Backward and parent-reported Attentional Focusing and Inhibitory Control. Digit Span Backward and parent-reported Inhibitory Control were influenced by additive genetics and the shared environment, whereas dominant genetic effects influenced variation in parent-reported Attentional Focusing. Both common factors of EF were influenced by genes and the nonshared environment to the same degree of magnitude, demonstrating that the two methods are tapping into aspects of EF that are shared across the tasks and parent-reported measures.

The majority of our findings were in-line with previous EF research. More specifically, the Continuous Performance Task and the Eriksen Flanker Task, tasks that assess attentional focusing and inhibitory control, were influenced by genes and the nonshared environment, fitting previous literature (Fan et al., 2001; Gagne & Saudino, 2010; Stins et al., 2004). Also concordant with previous research (Gagne & Saudino, 2010), parent-reported inhibitory control was influenced by additive genes and the shared environment. The shared environmental influence on parent-reported inhibitory control could be a result of the measure tapping into inhibitory control behaviors that are emphasized in the home, like waiting for one's turn or being able to keep secrets, given that primary caregivers are the ones teaching and communicating these types of behaviors. Conversely, it is possible that task-based inhibitory control centers on child-specific inhibitory abilities that twins might differ on. For example, twins could receive differential inhibitory control training through being in different classrooms or interacting

with various games and activities on a tablet, contributing to reduced shared environmental influences.

We found that Digit Span Forward and Backward were modestly correlated (r = .26), suggesting that they measure different aspects of working memory. Other studies support this assertion, finding that these tasks indicate two distinct types of memory processes and should be studied separately (e.g., Reynolds, 1997). In our study, genetically-informed analyses indicated that individual differences on Digit Span Forward were explained by additive genes and the nonshared environment, fitting the extant literature (Polderman et al., 2006; Stins et al., 2004). We did not have enough power to differentiate between an AE and CE model for Digit Span Backward, therefore the full ACE model was retained, however, the twin intraclass correlation for DZ twins was more than half the correlation for MZ twins, indicating the role of the shared environment. Other research also found that working memory tasks are more difficult than inhibitory control tasks for children in middle childhood (Davidson, Amso, Anderson, & Diamond, 2006), therefore it is possible that heritability will increase with age and mastery.

Next, for parent-reported Attentional Focusing, about 69% of the variance was due to broad sense heritability, which is very consistent with previous literature examining the heritability of effortful control in middle childhood (Lemery-Chalfant et al., 2008). Therefore, our study adds to the literature demonstrating the moderate to high heritability of Attentional Focusing in middle childhood.

Our results indicated that the common factor of EF was moderately heritable (67%). Similar studies of older children resulted in much higher estimates: Friedman et al. (2008) and Engelhardt et al. (2015) found that a genetic factor accounted for 99% and 100% of the variance in a common EF factor, respectively. There are several potential explanations for these differences. First, our sample was younger than the two published studies, therefore the challenging nature of these tasks for this age group could have restrained the heritability of the common factor. With mastery, we expect that our sample will perform better on these tasks, thereby exercising their genetic potential as they enter adolescence. Regardless, our findings, along with the two previously mentioned studies, suggest that there is a core genetic factor that widely impacts higher order executive processes. Similarly, researchers have posited that childhood EF acts as a developmental endophenotype that influences psychological, social, and health outcomes (Engelhardt et al., 2015).

We hypothesized that measures of effortful control would be included in the common factor of EF. In the original CFA with all variables included, the path from the common factor to parent-reported attentional focusing was the strongest, and the remaining paths were extremely small. However, the path coefficients became more comparable once parent-reported inhibitory control was removed. Parent-reported inhibitory control was only strongly correlated with parent-reported attentional focusing, perhaps inflating the role of attentional focusing in the common factor model. This suggests that the effortful control measure of inhibitory control could be tapping into a different type of inhibition that is separate from inhibitory behavior that is required with EF tasks. These differences could be indicative of *hot* and *cool* systems of self-regulation,

with the hot system consisting of emotional processing (parent-reported inhibitory control) and the cool system specializing in complex cognitive processing in response to neutral stimuli (EF tasks tapping into inhibitory control; Zhou, Chen, & Main, 2012). Although it appears that there are differences in EF tasks and parent-reported inhibitory control, it has been argued that hot and cool systems are not separate, but instead ends of a continuum (Zelazo & Cunningham, 2007). This provided rationale for utilizing the common EF factor created using PCA, as this method appears to be identifying core EF evenly across task-based and parent-reported EF that could be involved with both hot and cool systems of self-regulation.

We consistently found that the nonshared environment played a large role in explaining differences across all EF variables, including the common factor. Given that we collected EF data in the home, variability in testing environments could have contributed to the reduced genetic influence and the increased role of the nonshared environment. Other studies examining the heritability of EF found that the common factor was almost 100% heritable (e.g., Engelhardt et al., 2015; Friedman et al., 2008), however, these studies used older samples of twins, so it is possible that measuring EF after an accelerated period of development in adolescence could have contributed to lower estimates of the nonshared environment where EF abilities are more stable.

Moderation of Heritability of EF by Early Adversity

In our study, the etiology of the common factor, Flanker, CPT, and parent-reported inhibitory control was moderated by early adversity measured at 30 months of age. Importantly, as early adversity increased, the amount of total variance decreased.

Our phenotypic analyses also demonstrated that higher experiences of stress in the home during toddlerhood were related to lower scores on EF tasks in middle childhood. Children in homes with high levels of stress are likely to experience more instability and live in disadvantaged neighborhoods with poorer quality schools and fewer resources, potentially leading to decreased trait-relevant variance in these environments (Evans, 2004). This could then limit the ability for children to interact with proximal processes and maximize their genetic potential that can then bolster EF abilities (Bronfenbrenner & Ceci, 1994; Evans, 2004).

More specifically, the non-shared environment was moderated by early adversity for these variables, such that E decreased as early adversity increased. The heritability of CPT and parent-reported inhibitory control was moderated as well, with the role of additive genes increasing with early adversity. These findings demonstrate that the etiology of EF can differ as a function of early home stress, as well as highlight that a heritable trait is not immutable by the environment. Instead, our results suggest that a child's early home environment can have lasting effects on the degree to which genes and the nonshared environment play a role in explaining individual differences in EF in middle childhood. The environment contributed to individual differences more so under contexts with low adversity. More specifically, in homes with low stress, parents might have more opportunities to be increasingly emotionally available to teach and demonstrate effective self-regulatory abilities. For example, research has found that parental responsiveness and warmth was positively associated with children's effortful control abilities (von Suchodoletz, Trommsdorff, & Heikamp, 2011). In examining homes with high stress, another study found an indirect association between chaos in the

home in toddlerhood and EF two years later, mediated by parent responsivity and acceptance (Vernon-Feagans, Willoughby, & Garrett-Peters, 2016). This research, along with our study, highlight the long-term importance of the rearing environment on the development of EF throughout childhood.

There is great interest in using research to better inform interventions that bolster EF abilities in children. Previous research has consistently demonstrated that individuals with the poorest EF display the greatest improvement from programs that improve EF, showing that EF training might give previously disadvantaged children the opportunity to catch up (Diamond & Ling, 2016). Our results are consistent with this assertion, suggesting that improving and maximizing the trait relevant environment could give children that have experienced high stress in the home greater opportunities to interact with proximal processes, thereby fostering their EF skills. Diamond and Ling (2016) suggested that along with including training components to improve EF abilities, interventions should also address emotional, social, and physical needs in an attempt to provide long-lasting improvements, which could contribute to the broader transfer of EF skills. As our study demonstrated, EF does not develop in isolation, but instead is influenced by other external factors that could be modified through intervention.

This is the first genetically informed study to longitudinally examine moderation of genetic and environmental influences on EF by early adversity. Strengths included the use of a multimethod approach and the ability to observe the long-lasting role of early adversity on the later etiology of EF. This study also had several limitations. First, administering EF tasks in the home could have introduced error into our results, inflating

the effect of the environment on performance on our EF tasks. Although the home environments varied widely, the magnitude of environmental influences from the univariate models was comparable to other studies that administered EF tasks in a laboratory setting (e.g., Fan et al., 2001; Friedman et al., 2008).

Another concern is that it could be difficult to generalize results to singleton children, given that our sample consists of twins. Shared environmental influences could be inflated due to the fact that twins share a prenatal environment and are the same age (Friedman et al., 2008). However, we only found shared environmental influences for Digit Span Backward and parent-reported Inhibitory Control in our study, therefore it is unlikely that this bias was consistently occurring. Also, perhaps families with twins experience more stress. Contrary to that prediction, families in our study reported similar levels of chaos in the home as other phenotypic studies with singleton children (e.g., Bridgett, Burt, Laake, & Oddi, 2013), demonstrating that families with twins are not experiencing drastically more stress in the home than families with singleton children.

Future directions include examining this association at other ages. One study found that prenatal maternal stress was significantly associated with lower inhibitory control abilities in girls and lower working memory abilities in boys and girls in middle childhood (Buss, Davis, Hobel, & Sandman, 2011), therefore it is possible that prenatal stress could also influence the later etiology of EF. Similarly, another future goal is to examine if early adversity in toddlerhood moderates the heritability of EF when our sample enters into adolescence. In a sample of low-income adolescents, researchers found that household chaos was related to behavioral regulation, both measured

concurrently (Evans, Gonnella, Marcynyszyn, Gentile, & Salpekar, 2005), however, it is unknown whether early life stress influences the heritability of EF during an accelerated period of EF development.

Overall, our study, along with other behavior genetic research, supports the hypothesis by Bronfenbrenner and Ceci (1994) that heritability is not static, but instead can fluctuate as a function of interactions with the environment. More importantly, our study contributes to the literature by demonstrating that even moderate levels of stress in the home can have a long-term impact on the development of EF in middle childhood.

References

Aisa, B., Tordera, R., Lasheras, B., Del Río, J., & Ramírez, M. J. (2007). Cognitive impairment associated to HPA axis hyperactivity after maternal separation in rats. *Psychoneuroendocrinology*, *32*(3), 256-266. doi: 10.1016/j.psyneuen.2006.12.013

Anderson, V. A., Anderson, P., Northam, E., Jacobs, R., & Catroppa, C. (2001). Development of executive functions through late childhood and adolescence in an Australian sample. *Developmental Neuropsychology*, *20*(1), 385-406. doi: 10.1207/S15326942DN2001_5

Ando, J., Ono, Y., & Wright, M. J. (2001). Genetic structure of spatial and verbal working memory. *Behavior Genetics*, *31*(6), 615-624. doi: 10.1023/A:1013353613591

Armstrong, J. M., & Goldstein, L. H. (2003). Manual for the MacArthur health and behavior questionnaire (HBQ 1.0). *MacArthur Foundation Research Network on Psychopathology and Development, University of Pittsburgh*.

Barnea-Goraly, N., Menon, V., Eckert, M., Tamm, L., Bammer, R., Karchemskiy, A., Dant, C.C., & Reiss, A.L. (2005). White matter development during childhood and adolescence: A cross-sectional diffusion tensor imaging study. *Cerebral Cortex*, 15, 1848–1854. doi: 10.1093/cercor/bhi062

Bell, J. A., & Livesey, P. J. (1985). Cue significance and response regulation in 3-to 6-year-old children's learning of multiple choice discrimination tasks. *Developmental Psychobiology*, *18*(3), 229-245. doi: 10.1002/dev.420180304

Bergman Nutley, S., Söderqvist, S., Bryde, S., Thorell, L. B., Humphreys, K., & Klingberg, T. (2011). Gains in fluid intelligence after training non-verbal reasoning in 4-year-old children: A controlled, randomized study. *Developmental Science*, *14*(3), 591-601. doi: 10.1111/j.1467-7687.2010.01022.x

Biederman, J., Monuteaux, M. C, Doyle, A. E., Seidman, L. J., Wilens, T. E., Ferrerò, F., et al. (2004). Impact of executive function deficits and ADHD on academic outcomes in children. *Journal of Consulting and Clinical Psychology*, 72, 757-766. doi: 10.1037/0022-006X.72.5.757

Biringen, Z., Robinson, J. L., & Emde, R. N. (2000). Appendix B: The emotional availability scales (an abridged infancy/early childhood version). *Attachment & Human Development*, 2(2), 256-270. doi: 10.1080/14616730050085626

Blair, C, & Razza, R. P. (2007). Relating effortful control, executive function, and false belief understanding to emerging math and literacy ability in kindergarten. *Child Development*, 78, 647-663. doi: 10.1111/j.1467-8624.2007.01019.x

Bridgett, D. J., Burt, N. M., Laake, L. M., & Oddi, K. B. (2013). Maternal self-regulation, relationship adjustment, and home chaos: Contributions to infant negative emotionality. *Infant Behavior and Development*, *36*(4), 534-547. doi: 10.1016/j.infbeh.2013.04.004

Bronfenbrenner, U., & Ceci, S. J. (1994). Nature-nurture reconceptualized in developmental perspective: A bioecological model. *Psychological Review*, *101*(4), 568-586. doi: 10.1037/0033-295X.101.4.568

Buss, C., Davis, E. P., Hobel, C. J., & Sandman, C. A. (2011). Maternal pregnancy-specific anxiety is associated with child executive function at 6–9 years age. *Stress*, *14*(6), 665-676.

Carlson, S. M., & Moses, L. J. (2001). Individual differences in inhibitory control and children's theory of mind. *Child Development*, *72*(4), 1032-1053. doi: 10.1111/1467-8624.00333

Carlson, S. M., & Wang, T. S. (2007). Inhibitory control and emotion regulation in preschool children. *Cognitive Development*, 22, 489-510. doi: 10.1016/j.cogdev.2007.08.002

Carpenter, M., Nagell, K., Tomasello, M., Butterworth, G., & Moore, C. (1998). Social cognition, joint attention, and communicative competence from 9 to 15 months of age. *Monographs of the Society for Research in Child Development*, i-174. doi: 10.2307/1166214

Chang, H., Shaw, D. S., Dishion, T. J., Gardner, F., & Wilson, M. N. (2014). Direct and indirect effects of the family check-up on self-regulation from toddlerhood to early school-age. *Journal of Abnormal Child Psychology*, *42*(7), 1117-1128. doi: 10.1007/s10802-014-9859-8

Chipuer, H. M., Rovine, M. J., & Plomin, R. (1990). LISREL modeling: Genetic and environmental influences on IQ revisited. *Intelligence*, *14*(1), 11-29. doi: 10.1016/0160-2896(90)90011-H

Clark, C, Prior, M., & Kinsella, G. (2002). The relationship between executive function abilities, adaptive behaviour, and academic achievement in children with externalising behaviour problems. *Journal of Child Psychology and Psychiatry*, 43, 785-796. doi: 10.1111/1469-7610.00084

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). Multiple regression/correlation analysis for the behavioral sciences (3rd Ed.) Mahwah, NJ: Lawrence Erlbaum.

Conners, C. K., Epstein, J. N., Angold, A., & Klaric, J. (2003). Continuous Performance Test performance in a normative epidemiological sample. *Journal of Abnormal Child Psychology, 31*(5), 555-562. doi: 10.1023/A:1025457300409

Conners, C. K. (2000). *Conners' continuous performance test II: Technical guide*. Toronto, Canada: Multi-Health Systems. Retrieved from http://www.pearsonclinical.co.uk/Psychology/ChildMentalHealth/ChildADDADHDBehaviour/ConnersContinuousPerformanceTestIIVersion5forWindows(CPTIIV5)/PDFReports/Profile.pdf

Conway, A. R., Cowan, N., Bunting, M. F., Therriault, D. J., & Minkoff, S. R. (2002). A latent variable analysis of working memory capacity, short-term memory capacity, processing speed, and general fluid intelligence. *Intelligence*, *30*(2), 163-183. doi: 10.1016/S0160-2896(01)00096-4

Conway, A. R., Kane, M. J., Bunting, M. F., Hambrick, D. Z., Wilhelm, O., & Engle, R. W. (2005). Working memory span tasks: A methodological review and user's guide. *Psychonomic Bulletin & Review, 12*(5), 769-786. doi: 10.3758/BF03196772

Crnic, K. and Greenberg, M. (1990). Minor parenting stresses with young children. *Child Development, 61*(5), 1628-1637. doi: 10.1111/j.1467-8624.1990.tb02889.x

Cummings, E. M., Davies, P. T., & Campbell, S. B. (2002). *Developmental psychopathology and family process: Theory, research, and clinical implications*. Guilford Press.

Curtis, C. E., Cole, M. W., Rao, V. Y., & D'esposito, M. (2004). Canceling planned action: an FMRI study of countermanding saccades. *Cerebral Cortex*, *15*(9), 1281-1289. doi: 10.1093/cercor/bhi011

Daneman, M., & Merikle, P. M. (1996). Working memory and language comprehension: A meta-analysis. *Psychonomic Bulletin & Review*, *3*(4), 422-433. doi: 10.3758/s13423-013-0565-2

Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia*, *44*(11), 2037-2078. doi: 10.1016/j.neuropsychologia.2006.02.006

Davis, M., Walker, D. L., & Lee, Y. (1997). Roles of the amygdala and bed nucleus of the stria terminalis in fear and anxiety measured with the acoustic startle reflex.

Annals of the New York Academy of Sciences, 821, 305–331. doi: 10.1111/j.1749-6632.1997.tb48289.x

Diamond, A., & Ling, D. S. (2016). Conclusions about interventions, programs, and approaches for improving executive functions that appear justified and those that, despite much hype, do not. *Developmental Cognitive Neuroscience*, *18*, 34-48. doi: 10.1016/j.dcn.2015.11.005

Dubow, E. F., Boxer, P., & Huesmann, L. R. (2009). Long-term effects of parents' education on children's educational and occupational success: Mediation by family interactions, child aggression, and teenage aspirations. *Merrill-Palmer Quarterly (Wayne State University. Press)*, *55*(3), 224. doi: 10.1353/mpq.0.0030

Dumas, J. E., Nissley, J., Nordstrom, A., Smith, E. P., Prinz, R. J., & Levine, D. W. (2005). Home chaos: Sociodemographic, parenting, interactional, and child correlates. *Journal of Clinical Child and Adolescent Psychology*, *34*(1), 93-104. doi: 10.1207/s15374424jccp3401_9

Eisenberg, N., Guthrie, I. K., Fabes, R. A., Shepard, S., Losoya, S., Murphy, B., ... & Reiser, M. (2000). Prediction of elementary school children's externalizing problem behaviors from attentional and behavioral regulation and negative emotionality. *Child Development*, *71*(5), 1367-1382. doi: 10.1111/1467-8624.00233

Eisenberg, N., Spinrad, T. L., Fabes, R. A., Reiser, M., Cumberland, A., Shepard, S. A., ... & Thompson, M. (2004). The relations of effortful control and impulsivity to children's resiliency and adjustment. *Child Development*, *75*(1), 25-46. doi: 10.1111/j.1467-8624.2004.00652.x

Eisenberg, N., Zhou, Q., Spinrad, T. L., Valiente, C., Fabes, R. A., & Liew, J. (2005). Relations among positive parenting, children's effortful control, and externalizing problems: A three-wave longitudinal study. *Child Development*, *76*(5), 1055-1071. doi: 10.1111/j.1467-8624.2005.00897.x

Engle, R. W. (2002). Working memory capacity as executive attention. *Current Directions in Psychological Science*, *11*(1), 19-23. doi: 10.1111/1467-8721.00160

Engle, R. W., Tuholski, S. W., Laughlin, J. E., & Conway, A. R. (1999). Working memory, short-term memory, and general fluid intelligence: a latent-variable approach. *Journal of Experimental Psychology: General*, *128*(3), 309. doi: 10.1037/0096-3445.128.3.309

Engelhardt, L. E., Briley, D. A., Mann, F. D., Harden, K. P., & Tucker-Drob, E. M. (2015). Genes unite executive functions in childhood. *Psychological Science*, *26*(8), 1151-1163.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Attention, Perception, & Psychophysics*, *16*(1), 143-149. doi: 10.3758/BF03203267

Evans, G. W. (2004). The environment of childhood poverty. *American Psychologist*, *59*(2), 77.

Evans, G. W., Gonnella, C., Marcynyszyn, L. A., Gentile, L., & Salpekar, N. (2005). The role of chaos in poverty and children's socioemotional adjustment. *Psychological Science*, *16*(7), 560-565. doi: 10.1111/j.0956-7976.2005.01575.x

Falconer, D. S. (1989). Introduction to quantitative genetics. Essex, England: Longman Scientific and Technical.

Fan, J., Wu, Y., Fossella, J. A., & Posner, M. I. (2001). Assessing the heritability of attentional networks. *BMC Neuroscience*, *2*(1), 14. doi: 10.1186/1471-2202-2-14

Fitzpatrick, C., Archambault, I., Janosz, M., & Pagani, L. S. (2015). Early childhood working memory forecasts high school dropout risk. *Intelligence*, *53*, 160-165. doi: 10.1016/j.intell.2015.10.002

Forget-Dubois, N., Pérusse, D., Turecki, G., Girard, A., Billette, J. M., Rouleau, G., ... & Tremblay, R. E. (2003). Diagnosing zygosity in infant twins: Physical similarity, genotyping, and chorionicity. *Twin Research*, *6*(06), 479-485. doi: 10.1375/twin.6.6.479

Friedman, N. P., Miyake, A., Young, S. E., DeFries, J. C., Corley, R. P., & Hewitt, J. K. (2008). Individual differences in executive functions are almost entirely genetic in origin. *Journal of Experimental Psychology: General*, *137*(2), 201. doi: 10.1037/0096-3445.137.2.201

Gagne, J. R., & Saudino, K. J. (2010). Wait for it! A twin study of inhibitory control in early childhood. *Behavior Genetics*, *40*(3), 327-337. doi: 10.1007/s10519-009-9316-6

Garner, A. S., Shonkoff, J. P., Siegel, B. S., Dobbins, M. I., Earls, M. F., McGuinn, L., ... & Committee on Early Childhood, Adoption, and Dependent Care. (2012). Early childhood adversity, toxic stress, and the role of the pediatrician: translating developmental science into lifelong health. *Pediatrics*, *129*(1), e224-e231. doi: 10.1542/peds.2011-2662

Garon, N., Bryson, S. E., & Smith, I. M. (2008). Executive function in preschoolers: A review using an integrative framework. *Psychological Bulletin*, 134, 31-60. doi: 10.1037/0033-2909.134.1.31

Gathercole, S. E., Pickering, S. J., Ambridge, B., & Wearing, H. (2004). The structure of working memory from 4 to 15 years of age. *Developmental Psychology*, *40*(2), 177. doi: 10.1037/0012-1649.40.2.177

Goldsmith, H. H. (1991). A zygosity questionnaire for young twins: A research note. *Behavior Genetics, 21*(3), 257-269. doi: 10.1007/BF01065819

Goldsmith, H. H., Buss, K. A., & Lemery, K. S. (1997). Toddler and childhood temperament: Expanded content, stronger genetic evidence, new evidence for the importance of environment. *Developmental Psychology*, *33*(6), 891. doi: 10.1037/0012-1649.33.6.891

Hale, J. B., Hoeppner, J. A. B., & Fiorello, C. A. (2002). Analyzing digit span components for assessment of attention processes. *Journal of Psychoeducational Assessment*, *20*(2), 128-143. doi: 10.1177/073428290202000202

Harrison, T. L., Shipstead, Z., Hicks, K. L., Hambrick, D. Z., Redick, T. S., & Engle, R. W. (2013). Working memory training may increase working memory capacity but not fluid intelligence. *Psychological Science*, *24*(12), 2409-2419. doi: 10.1177/0956797613492984

Haworth, C. M., Wright, M. J., Luciano, M., Martin, N. G., De Geus, E. J. C., Van Beijsterveldt, C. E. M., ... & Kovas, Y. (2010). The heritability of general cognitive ability increases linearly from childhood to young adulthood. *Molecular Psychiatry*, *15*(11), 1112. doi: 10.1038/mp.2009.55

Holden, G. W., & Zambarano, R. J. (1992). Passing the rod: similarities between parents and their young children in orientations toward physical punishment. Retrieved from: http://psycnet.apa.org/record/1992-97915-006

Hughes, C., Graham, A., & Grayson, A. (2005). Executive function in childhood: Development and disorder. In J. Oates (Ed.), Cognitive Development (pp. 205–230). Milton Keynes: Open University Press. Retrieved from: http://irep.ntu.ac.uk/id/eprint/8754/

Jensen, A. R. (1981). Raising the IQ: The Ramey and Haskins study. *Intelligence*, *5*(1), 29-40. doi: 10.1016/0160-2896(81)90015-5

Klingberg, T., Forssberg, H., & Westerberg, H. (2002). Increased brain activity in frontal and parietal cortex underlies the development of visuospatial working memory capacity during childhood. *Journal of Cognitive Neuroscience*, *14*(1), 1-10. doi: 10.1162/089892902317205276

Knopik, V. S., Neiderhiser, J. M., DeFries, J. C., & Plomin, R. (2016). *Behavioral genetics*. Macmillan Higher Education.

Lemery-Chalfant, K., Clifford, S., McDonald, K., O'Brien, T. C., & Valiente, C. (2013). Arizona twin project: A focus on early resilience. *Twin Research and Human Genetics*, *16*(1), 404-411. doi: 10.1017/thg.2012.107

Lemery-Chalfant, K., Doelger, L., & Goldsmith, H. H. (2008). Genetic relations between effortful and attentional control and symptoms of psychopathology in middle childhood. *Infant and Child Development*, *17*(4), 365-385. doi: 10.1002/icd.581

Matheny, A. P., Wachs, T. D., Ludwig, J. L., & Phillips, K. (1995). Bringing order out of chaos: Psychometric characteristics of the confusion, hubbub, and order scale. *Journal of Applied Developmental Psychology*, *16*, 429– 444. doi: 10.1016/0193-3973(95)90028-4

Matte-Gagné, C., Bernier, A., Sirois, M. S., Lalonde, G., & Hertz, S. (2018). Attachment Security and Developmental Patterns of Growth in Executive Functioning During Early Elementary School. *Child Development*. doi: 10.1111/cdev.12807

Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature Neuroscience*, *6*(5), 450-452. doi: 10.1038/nn1051

McClelland, M. M., Acock, A. C., & Morrison, F. J. (2006). The impact of kindergarten learning-related skills on academic trajectories at the end of elementary school. *Early Childhood Research Quarterly*, 21, 471-490. doi: 10.1016/j.ecresq.2006.09.003

McClelland, M. M., Cameron, C. E., Duncan, R., Bowles, R. P., Acock, A. C., Miao, A., & Pratt, M. E. (2014). Predictors of early growth in academic achievement: The head-toes-knees-shoulders task. *Frontiers in Psychology*, *5*. doi: 10.3389/fpsyg.2014.00599

McEwen, B. S., & Seeman, T. E. (2003). Stress and affect: Applicability of the concepts of allostasis and allostatic load. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), Handbook of affective sciences (pp. 1117–1137). New York: Oxford University Press.

McGue, M., & Bouchard, T.J. (1984). Adjustment of twin data for the effects of age and sex. *Behavioral Genetics,14*; 325-43. doi: 10.1007/BF01080045

McGue, M., Bouchard, T. J., Jr., Iacono, W. G., & Lykken, D. T. (1993). Behavioral genetics of cognitive abilities: A life-span perspective. In R. Plomin & G. E. McClearn (Eds.), Nature, nurture, and psychology (pp. 59–76). Washington, DC: American Psychological Association. doi: 10.1037/10131-003

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., & Howerter, A. (2000). The unity and diversity of executive functions and their contribution to complex '

'frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49-100. doi: 10.1006/cogp.1999.0734

Mueller, S. T. (2013). The Psychology Experiment Building Language (Version 0.13) [Software]. Available from http://pebl.sourceforge.net

Mueller, S. T., & Piper, B. J. (2014). The Psychology Experiment Building Language (BEBL) and PEBL test battery. *Journal of Neuroscience Methods, 222*, 250-259. doi: 10.1016/j.jneumeth.2013.10.024

Mullineaux, P. Y., Deater-Deckard, K., Petrill, S. A., Thompson, L. A., & DeThorne, L. S. (2009). Temperament in middle childhood: A behavioral genetic analysis of fathers' and mothers' reports. *Journal of Research in Personality*, *43*(5), 737-746. doi: 10.1016/j.jrp.2009.04.008

Muthén, B., & Kaplan, D. (1985). A comparison of some methodologies for the factor analysis of non-normal Likert variables. *British Journal of Mathematical and Statistical Psychology*, *38*(2), 171-189. doi: 10.1111/j.2044-8317.1985.tb00832.x

Neale, M. C., Hunter, M. D., Pritikin, J. N., Zahery, M., Brick, T. R., Kirkpatrick, R. M., ... & Boker, S. M. (2016). OpenMx 2.0: Extended structural equation and statistical modeling. *Psychometrika*, *81*(2), 535-549. doi: 10.1007/s11336-014-9435-8

Neale M. C., Maes H. M. Methodology for Genetic Studies of Twins and Families. 2004 Retrieved March 2, 2004, http://www.vipbg.vcu.edu/~vipbg/ mx/book2004a.pdf.

Orme, J. G., Reis, J., & Herz, E. J. (1986). Factorial and discriminant validity of the Center for Epidemiological Studies Depression (CES-D) scale. *Journal of Clinical Psychology*, *42*(1), 28-33. doi: 10.1002/1097-4679(198601)42:1<28::AID-JCLP2270420104>3.0.CO;2-T

Pe, M. L., Vandekerckhove, J., & Kuppens, P. (2013). A diffusion model account of the relationship between the emotional flanker task and rumination and depression. *Emotion*, *13*(4), 739.

Perna, R. B., & Kiefner, M. (2013). Long-term cognitive sequelae: Abused children without PTSD. *Applied Neuropsychology: Child*, *2*(1), 1-5. doi: 10.1080/09084282.2011.595460

Pesonen, A. K., Räikkönen, K., Heinonen, K., Komsi, N., Järvenpää, A. L., & Strandberg, T. (2008). A transactional model of temperamental development: Evidence of a relationship between child temperament and maternal stress over five years. *Social Development*, *17*(2), 326-340. doi: 10.1111/j.1467-9507.2007.00427.x

Polderman, T. J., de Geus, E. J., Hoekstra, R. A., Bartels, M., van Leeuwen, M., Verhulst, F. C., ... & Boomsma, D. I. (2009). Attention problems, inhibitory control, and intelligence index overlapping genetic factors: a study in 9-, 12-, and 18-year-old twins. *Neuropsychology*, *23*(3), 381-391. doi: 10.1037/a0014915

Polderman, T. J., Stins, J. F., Posthuma, D., Gosso, M. F., Verhulst, F. C., & Boomsma, D. I. (2006). The phenotypic and genotypic relation between working memory speed and capacity. *Intelligence*, *34*(6), 549-560. doi: 10.1016/j.intell.2006.03.010

Posner, M. I., & Rothbart, M. K. (2007). Research on attention networks as a model for the integration of psychological science. *Annual Review of Psychology, 58*, 1–23 doi: 10.1146/annurev.psych.58.110405.085516

Price, T. S., Freeman, B., Craig, I., Petrill, S. A., Ebersole, L., & Plomin, R. (2000). Infant zygosity can be assigned by parental report questionnaire data. *Twin Research*, *3*(03), 129-133. doi: 10.1375/twin.3.3.129

Price, T. S., & Jaffee, S. R. (2008). Effects of the family environment: gene-environment interaction and passive gene-environment correlation. *Developmental Psychology*, *44*(2), 305. doi: 10.1037/0012-1649.44.2.305

Purcell, S. (2002). Variance components models for gene–environment interaction in twin analysis. *Twin Research and Human Genetics*, *5*(6), 554-571. doi: 10.1375/twin.5.6.554

Radloff, L. S. (1977). The CES-D Scale: a self-report depression scale for research in the general population. *Applied Psychological Measurement, 1*, 385-401. doi: 10.1177/014662167700100306

Raver, C. C., Blackburn, E. K., Bancroft, M., & Torp, N. (1999). Relations between effective emotional self-regulation, attentional control, and low-income preschoolers' social competence with peers. *Early Education and Development*, *10*(3), 333-350. doi: 10.1207/s15566935eed1003_6

Repetti, R. L., Taylor, S. E., & Seeman, T. E. (2002). Risky families: family social environments and the mental and physical health of offspring. *Psychological Bulletin*, 128(2), 330. doi: 10.1037/0033-2909.128.2.330

Reynolds, C. R. (1997). Forward and backward memory span should not be combined for clinical analysis. *Archives of Clinical Neuropsychology*, *12*(1), 29-40. doi: 10.1016/S0887-6177(96)00015-7

Rosenthal, E. N., Riccio, C. A., Gsanger, K. M., & Jarratt, K. P. (2006). Digit Span components as predictors of attention problems and executive functioning in children. *Archives of Clinical Neuropsychology*, *21*(2), 131-139. doi: 10.1016/j.acn.2005.08.004

Rothbart, M. K., & Bates, J. E. (2006). Temperament. *Handbook of Child Psychology*. doi: 10.1002/9780470147658.chpsy0303

Rowe D.C., Jacobson K.C., van den Oord E. (1999). Genetic and environmental influences on vocabulary IQ: Parental education level as a moderator. *Child Development*, 70, 1151–1162. doi: 10.1111/1467-8624.00084

Rubia, K., Smith, A. B., Brammer, M. J., & Taylor, E. (2003). Right inferior prefrontal cortex mediates response inhibition while mesial prefrontal cortex is responsible for error detection. *Neuroimage*, *20*(1), 351-358. doi: 10.1016/S1053-8119(03)00275-1

Rutter, M., & Rutter, M. (1993). Developing minds. London: Penguin.

Sánchez, M. M., Hearn, E. F., Do, D., Rilling, J. K., & Herndon, J. G. (1998). Differential rearing affects corpus callosum size and cognitive function of rhesus monkeys. *Brain research*, *812*(1), 38-49. doi: 10.1016/S0006-8993(98)00857-9

Sapolsky, R. M. (2002). Endocrinology of the stress-response. In J. B. Becker, S. M. Breedlove, D. Crews, & M. M. McCarthy (Eds.), Behavioral endocrinology (pp. 409-450). Cambridge, MA: MIT Press.

Scarr, S. (1981). *Race, social class, and individual differences in IQ: New studies of old issues.* Hillsdale, NJ: Erlbaum. Retrieved from: http://hdl.handle.net/10822/548658

Schwartz, D., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1997). The early socialization of aggressive victims of bullying. *Child Development*, 68(4), 665-675. doi: 10.1111/j.1467-8624.1997.tb04228.x

Seeman, T. E., Singer, B., Horwitz, R., & McEwen, B. S. (1997). The price of adaptation–allostatic load and its health consequences: MacArthur studies of successful aging. *Archives of Internal Medicine*, 157, 2259–2268. doi: doi:10.1001/archinte.1997.00440400111013

Shalev, L., Tsal, Y., & Mevorach, C. (2007). Computerized progressive attentional training (CPAT) program: effective direct intervention for children with ADHD. *Child Neuropsychology*, *13*(4), 382-388. doi: 10.1080/09297040600770787

Sherbourne, C. D., & Stewart, A. L. (1991). The MOS Social Support Survey. *Social Science and Medicine, 32,* 705-714. doi: 10.1016/0277-9536(91)90150-B

Shields, A., & Cicchetti, D. (2001). Parental maltreatment and emotion dysregulation as risk factors for bullying and victimization in middle childhood. *Journal of Clinical Child Psychology*, 30(3), 349-363. doi: 10.1207/S15374424JCCP3003_7

Shonkoff, J. P., Duncan, G. J., Fisher, P. A., Magnuson, K., & Raver, C. (2011). Building the brain's "air traffic control" system: How early experiences shape the development of executive function. *Contract*, *11*.

Simonds, J., & Rothbart, M. K. (2006). Temperament in middle childhood questionnaire. *Downloaded in http://www. bowdoin. edu/~ sputnam/rothbart-temperament-questionnaires*.

Sosic-Vasic, Z., Kröner, J., Schneider, S., Vasic, N., Spitzer, M., & Streb, J. (2017). The Association between Parenting Behavior and Executive Functioning in Children and Young Adolescents. *Frontiers in Psychology*, *8*. doi: 10.3389/fpsyg.2017.00472

Sowell, E.R., Peterson, B.S., Thompson, P.M., Welcome, S.E., Henkenius, A.L., & Toga, A.W. (2003). Mapping cortical change across the life span. *Nature Neuroscience*, 6, 309–315. doi: 10.1038/nn1008

Sowell, E.R., Thompson, P.M., Tessner, K.D., & Toga, A.W. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex: Inverse relationships during postadolescent brain maturation. *Journal of Neuroscience,* 21, 8819–8829. Retrieved from: http://www.jneurosci.org/content/jneuro/21/22/8819.full.pdf

Stins, J. F., De Sonneville, L. M., Groot, A. S., Polderman, T. C., Van Baal, C. G., & Boomsma, D. I. (2005). Heritability of selective attention and working memory in preschoolers. *Behavior Genetics*, *35*(4), 407-416. doi: 10.1007/s10519-004-3875-3

Stins, J. F., Van Baal, G. C. M., Polderman, T. J., Verhulst, F. C., & Boomsma, D. I. (2004). Heritability of Stroop and flanker performance in 12-year old children. *BMC Neuroscience*, *5*(1), 49. doi: 10.1186/1471-2202-5-49

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology*, 18, 643–662. doi: 10.1037/h0054651

Taylor, S. E., Way, B. M., & Seeman, T. E. (2011). Early adversity and adult health outcomes. *Development and Psychopathology*, *23*(03), 939-954. doi: https://doi.org/10.1017/S0954579411000411

Tremblay, R. E., Nagin, D. S., Séguin, J. R., Zoccolillo, M., Zelazo, P. D., Boivin, M., ... & Japel, C. (2004). Physical aggression during early childhood: Trajectories and predictors. *Pediatrics*, *114*(1), e43-e50. Retrieved from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2538721/

Turkheimer, E., Haley, A., Waldron, M., d'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies heritability of IQ in young children. *Psychological science*, *14*(6), 623-628. doi: 10.1046/j.0956-7976.2003.psci_1475.x

Vandierendonck, A. (2017). A comparison of methods to combine speed and accuracy measures of performance: A rejoinder on the binning procedure. *Behavior Research Methods*, *49*(2), 653-673. doi: 10.3758/s13428-016-0721-5.

Vernon-Feagans, L., Willoughby, M., & Garrett-Peters, P. (2016). Predictors of behavioral regulation in kindergarten: Household chaos, parenting, and early executive functions. *Developmental Psychology*, *52*(3), 430. doi: 10.1037/dev0000087

Von Suchodoletz, A., Trommsdorff, G., & Heikamp, T. (2011). Linking Maternal Warmth and Responsiveness to Children's Self-regulation. *Social Development, 20*(3), 486-503. doi:10.1111/j.1467-9507.2010.00588.x

Wechsler, D. (2003). Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV). San Antonio, TX: The Psychological Corporation.

West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with nonnormal variables: Problems and remedies.

Willcutt, E.G., Doyle, A.E., Nigg, J.T., Faraone, S.V., & Pennington, B.F. (2005). Validity of the executive function theory of attention deficit/hyperactivity disorder: a metaanalytic review. *Biological Psychiatry*, 57, 1336–1346. doi: 10.1016/j.biopsych.2005.02.006

Williams, B. R., Ponesse, J. S., Schachar, R. J., Logan, G. D., & Tannock, R. (1999). Development of inhibitory control across the life span. *Developmental Psychology*, *35*(1), 205. doi: 10.1037/0012-1649.35.1.205

Zelazo, P. D., & Cunningham, W. (2007). Executive function: Mechanisms underlying emotion regulation. In J. Gross (Ed.), Handbook of emotion regulation (pp. 135–158). New York: Guilford.

Zelazo, P. D., & Müller, U. (2002). Executive function in typical and atypical development. In U. Goswami (Ed.), *Blackwell Handbook of Childhood Cognitive Development*, (pp. 445-469). Malden, MA: Blackwell. doi: 10.1002/9780470996652.ch20

Zhou, Q., Chen, S. H., & Main, A. (2012). Commonalities and differences in the research on children's effortful control and executive function: A call for an integrated model of self-regulation. *Child Development Perspectives*, *6*(2), 112-121. doi: 10.1111/j.1750-8606.2011.00176.x

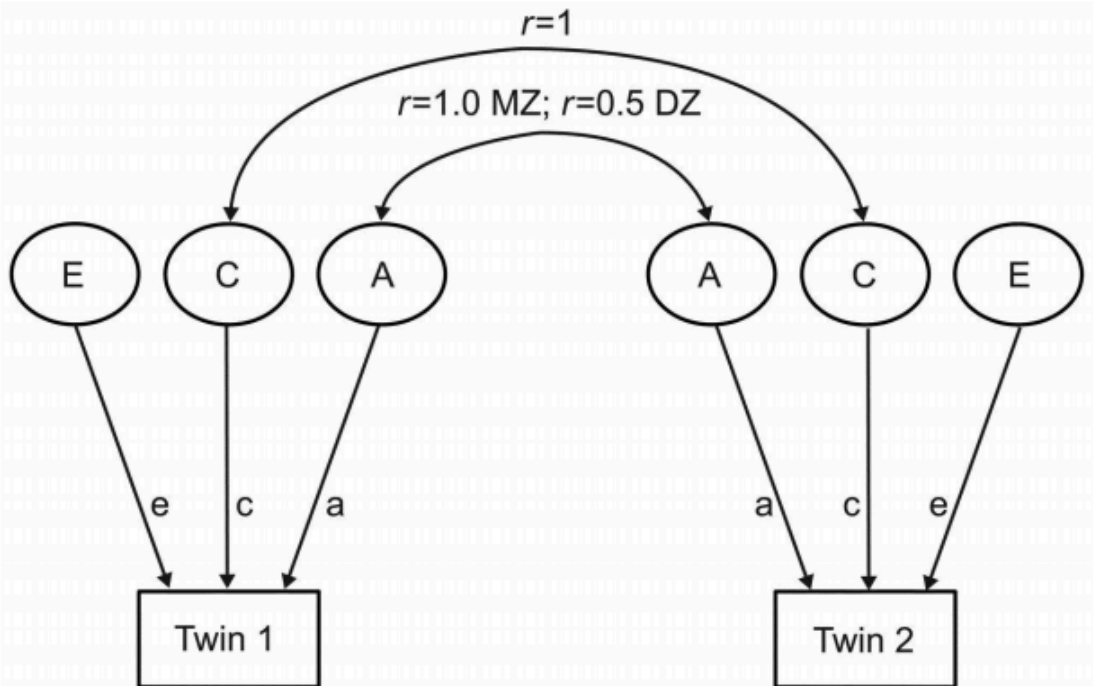


Figure 1. Example univariate biometric model that decomposes the variance into latent additive genetic (A), shared environmental (C), and non-shared environmental (E) factors. The correlations between the latent A factors are set to 1.0 for MZ twins and 0.5 for DZ twins.

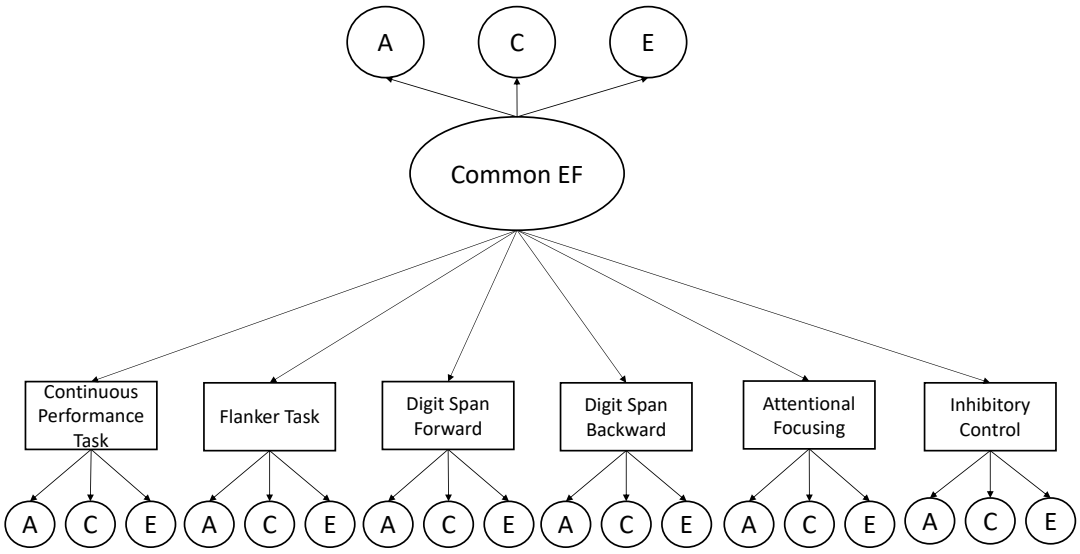


Figure 2. Common pathway model (showing only one twin for simplicity) that tests the assumption that covariance between a set of phenotypes (in this case, Continuous Performance Task, Flanker Task, Digit Span Forward, Digit Span Backward, and parent-reported Inhibitory Control and Attentional Focusing) can be fully accounted for by a single common phenotypic factor (e.g. common Executive Functioning, or EF). It decomposes the variance in the common factor into additive genetic (Ac), shared environmental (Cc), and nonshared environmental (Ec) factors. Also, additive genetic, shared environmental, and nonshared environmental factors that are unique to each phenotype are also estimated.

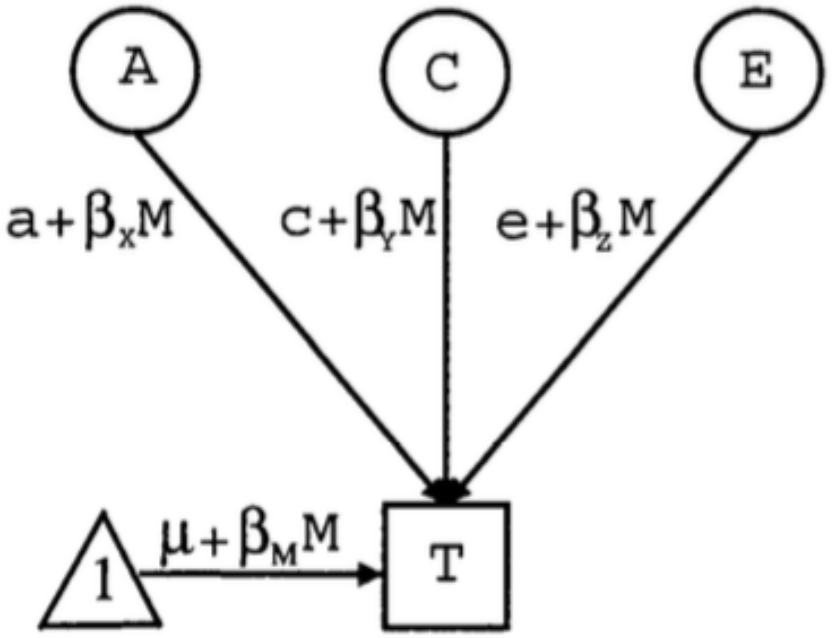


Figure 3. Moderated heritability model (showing only one twin for simplicity) that allows for the moderation of one family-level phenotype (i.e., early adversity) on an individual-level phenotype (i.e., executive functioning). A = additive genetic variance, C = shared environmental variance, E = nonshared environmental variance, M = moderator. Equations next to each path represent the linear relationship between the path coefficient and the moderator. If β_x is significantly non-zero, this represents an interaction between the path coefficient and the moderator (Purcell, 2002).

Table 1. *Principal Component Analysis Results for Early Adversity Composite*

Component Matrix		
	Component	
	1	2
Parenting Daily Hassles	.71	-.36
Social Support (reversed)	.56	.54
Parent Responses to Child Misbehavior	.46	-.43
Chaos in the Home	.71	-.40
Maternal Depression	.64	.35
Emotional Availability (reversed)	.58	.37
Total Variance Explained		
Component	Total	Cumulative %
1	2.28	37.96
2	1.02	55.02

Note. First principal component was used to represent early adversity.

Table 2. *Principal Components Analysis Results for Executive Functioning Composite*

Component Matrix				
	Component			
	1	2	3	4
CPT Errors of omission	.75	-.48	-.38	.00
CPT Errors of commission	.70	-.55	-.38	-.03
Flanker Congruent	.72	.51	-.02	-.34
Flanker Incongruent	.68	.54	-.03	-.37
Digit Span Forward	.32	.36	-.06	.62
Digit Span Backward	.37	.27	-.16	.62
TMCQ Attentional Focusing	.59	-.22	.60	.12
TMCQ Inhibitory Control	.39	-.22	.79	.04

Total Variance Explained		
	Extraction Sums of Squared Loadings	
Component	Total	Cumulative %
1	2.78	34.68
2	1.39	52.02
3	1.29	68.19
4	1.04	81.21

Note. First principal component was used to represent common executive functioning, with higher scores representing higher EF abilities.

Table 3. *Zero-Order Correlations and Descriptive Statistics for Early Adversity Composite*

	1	2	3	4	5	6	7
1. Parenting Daily Hassles	-						
2. Social Support (reversed)	.20**	-					
3. Parent Responses to Child Misbehavior	.22**	.12*	-				
4. Chaos in the Home	.53**	.22**	.25**	-			
5. Maternal Depression	.30**	.34**	.15**	.25**	-		
6. Emotional Availability (reversed)	.22**	.27**	.22**	.25**	.28**	-	
7. Total Early Adversity Composite	.71**	.56**	.50**	.71**	.64**	.58**	-
Mean	2.13	1.85	1.84	0.33	0.74	1.87	0.00
Standard Deviation	0.41	0.83	0.57	0.22	0.31	0.28	1.00
Minimum	1.13	1.00	1.00	0.00	0.27	1.14	-2.21
Maximum	3.71	4.60	3.80	0.90	1.95	2.75	3.24
Skewness	0.50	1.09	0.82	0.47	1.34	0.10	0.49
Kurtosis	0.44	0.53	0.64	-0.36	2.03	-0.19	0.22

Note: ** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Table 4. *Zero-Order Correlations and Descriptive Statistics for Executive Functioning and Early Adversity Composites*

	1	2	3	4	5	6	7	8	9	10
1. CPT errors of omission	-									
2. CPT errors of commission	.87**	-								
3. Flanker congruent	.31**	.22**	-							
4. Flanker incongruent	.28**	.21**	.79**	-						
5. Digit Span Forward	.13*	.08	.19**	.21**	-					
6. Digit Span Backward	.13**	.12*	.21**	.17**	.26**	-				
7. TMCQ Attentional Focusing	.26**	.27**	.26**	.21**	.14**	.13**	-			
8. TMCQ Inhibitory Control	.10	.09	.14**	.12*	.04	.01	.55**	-		
9. Common EF PCA	.75**	.70**	.72**	.68**	.33**	.37**	.59**	.39**	-	
10. Early Adversity Composite	-.06	-.10	-.02	-.06	.02	-.06	-.20**	-.34**	-.19**	-
Mean	-5.86	-16.96	-733.91	-879.48	6.22	4.04	3.31	3.19	0.00	0.00
Standard Deviation	5.11	6.31	150.92	182.23	1.81	1.39	.92	.60	1.00	1.00
Minimum	0.00	-2.00	-442.91	-500.10	0.00	0.00	1.00	1.25	-4.66	-2.21
Maximum	-19.40	-31.40	-1608.98	-1552.65	13.00	9.00	4.86	4.63	2.21	3.24
Skewness	-1.24	-.57	-1.57	-.70	.519	.38	-.37	-.20	-.71	.49
Kurtosis	.87	-.04	4.78	.41	1.15	.28	-.59	-.24	1.13	.45

Note: ** Correlation is significant at the 0.01 level (2-tailed) * Correlation is significant at the 0.05 level (2-tailed).

Table 5. *Twin Intraclass Correlations for Executive Functioning Variables*

	MZ	**DZ**
Common EF PCA	.546**	.289**
Common EF CFA	.562**	.302**
CPT Errors	.366**	.014
Flanker Task	.414**	.257**
Digit Span Forward	.641**	.342**
Digit Span Backward	.313*	.216**
Attentional Focusing	.638**	.185*
Inhibitory Control	.831**	.551**

Note: ** Correlation is significant at the 0.01 level (2-tailed) * Correlation is significant at the 0.05 level (2-tailed).

Table 6. *Univariate ACE models and fit statistics*

Scale	Model	-2LL	*df*	AIC	Δ-2LL	*p*	A	C	E
Common EF PCA	ACE	977.92	353	271.92			.71 (.21-1.13)	-.09 (-.44-.25)	.38 (.25-.59)
	AE	**978.17**	**354**	**270.17**	**0.25**	**.61**	**.60 (.42-.74)**	--	**.40 (.26-.59)**
	CE	984.88	354	276.88	6.96	.01	--	.34 (.20-.47)	.66 (.53-.80)
	E	1006.11	355	296.11	28.19	<.001	--	--	1.00
Common EF CFA	ACE	202.02	470	-737.98			.76 (.38-1.10)	-.01 (-.39-.17)	.34 (.23-.50)
	AE	**202.54**	**471**	**-739.46**	**0.52**	**.47**	**.64 (.49-.75)**	--	**.36 (.25-.51)**
	CE	213.58	471	-728.42	11.55	<.001	--	.35 (.24-.46)	.65 (.54-.76)
	E	245.01	472	-698.99	42.98	<.001	--	--	1.00
CPT Errors	ACE	2461.26	394	1673.26			.72 (.15-1.20)	-.34 (-.74-.06)	.62 (.44-.86)
	AE	**2464.07**	**395**	**1674.07**	**2.81**	**.09**	**.27 (.06-.27)**	--	**.73 (.55-.94)**
	CE[a]	4744.80	394	3956.80	2283.54	<.001			
	E	2470.35	396	1678.35	9.09	.01	--	--	--
Flanker Task	ACE	4743.40	393	3957.40			.29 (-.07-.67)	.14 (-.11- .27)	.56 (.40-.67)
	AE	**4743.59**	**394**	**3955.59**	**0.19**	**.06**	**.50 (.31-.64)**	--	**.50 (.36-.69)**
	CE	4744.80	394	3956.80	1.40	.02	--	.31 (.19 - .43)	.69 (.57-.81)
	E	4764.40	395	3974.40	21.00	<.001	--	--	1.00
Digit Span Forward	ACE	1556.99	394	768.99			.45 (.04-.85)	.13 (-.21-.44)	.42 (.30-.58)
	AE	**1557.58**	**395**	**767.58**	**0.59**	**.43**	**.60 (.46-.71)**	--	**.40 (.29-.54)**
	CE	1561.52	395	771.52	4.54	.03	--	.44 (.32-.55)	.56 (.46-.68)
	E	1604.22	396	812.22	47.23	<.001	--	--	1.00
Digit Span Backward[b]	**ACE**	**1372.09**	**394**	**584.08**			**.22 (-.39-.74)**	**.10 (-.28-.49)**	**.68 (.48-.95)**
	AE	1372.36	395	582.36	0.28	.60	.35 (.16-.52)	--	.65 (.48-.84)
	CE	1372.63	395	582.63	0.54	.46	--	.24 (.10-.36)	.76 (.64-.90)
	E	1384.21	396	592.21	12.13	.002	--	--	1.00
Attentional Focusing[c]	**ADE**	**1152.52**	**448**	**256.52**			**.01 (-.58-.56)**	**.68 (.09-1.3)**	**.31 (.21-.46)**
	AE	1157.69	449	259.69	5.17	.02	.62 (.44-.62)	--	.38 (.25-.56)
	E	1190.80	450	290.80	0.38	<.001	--	--	1.00
Inhibitory Control	**ACE**	**664.72**	**448**	**-231.27**			**.61 (.53-.69)**	**.24 (.10-.38)**	**.15 (.11-.19)**
	AE	668.87	449	-229.13	4.15	.04	.85 (.79-.89)	--	.15 (.11-.21)
	CE	687.54	449	-210.46	22.82	<.001	--	.62 (.53-.70)	.38 (.31-.47)
	E	797.64	450	-102.36	132.92	<.001	--	--	1.00

Note. -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike' s Information Criterion; Δ = change; *p* = probability; A, C, and E are standardized variance components. The most parsimonious final model for each variable is indicated in bold.

[a] Standardized solution could not be estimated.

[b] We were unable to differentiate between AE and CE models, therefore the full model was retained.

[c] DE model was not tested because it would be unlikely that dominance genetic effects would occur without the influence of additive genetic effects.

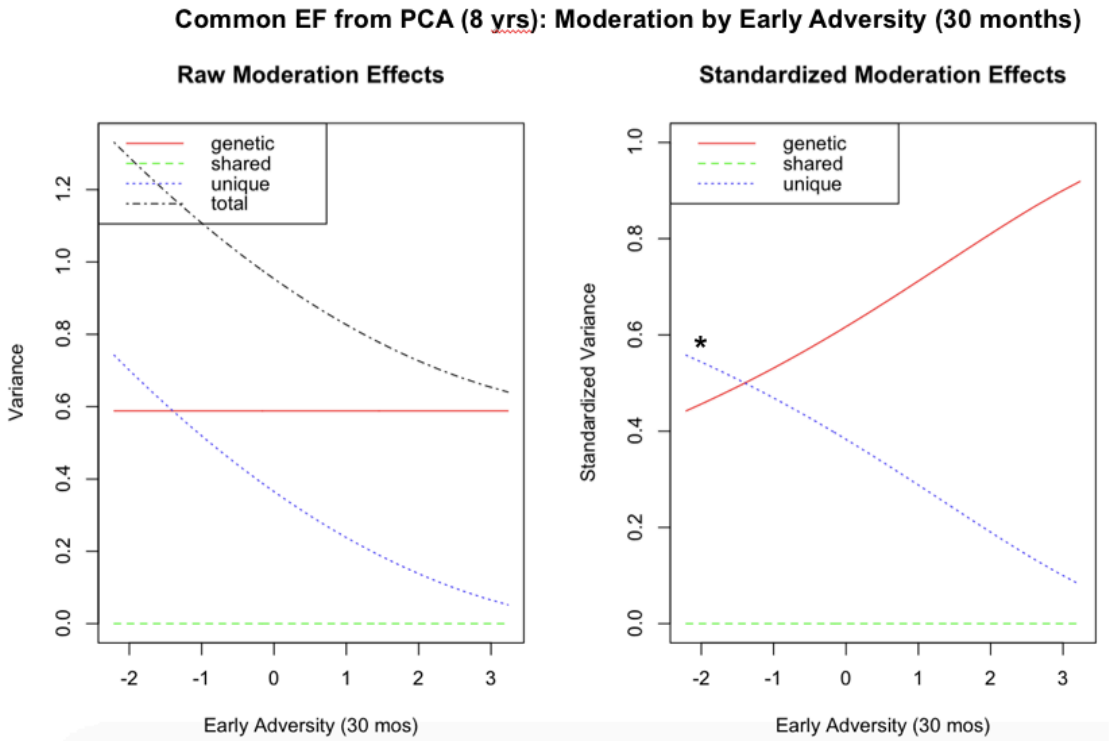
Table 7. *Model fit statistics for moderation models*

Model	**-2LL**	**df**	**AIC**	**Δ-2LL**	**Δdf**	**p**
Common EF PCA	688.05	249	190.05			
No means moderation	693.91	251	191.91	5.86	2	0.05
No moderation on A	691.93	250	191.93	3.88	1	0.04
No moderation on C	691.29	250	191.29	3.24	1	0.07
No moderation on E	695.63	250	195.62	7.58	1	0.01
No C or E moderation	694.70	251	192.70	6.65	2	0.04
No A or C moderation	**693.22**	**251**	**191.22**	**5.17**	**2**	**0.08**
No A or E moderation	696.14	251	194.14	8.09	2	0.02
No moderation	699.82	252	199.79	11.78	3	0.001
Common EF CFA	132.46	321	-509.54			
No means moderation	136.21	323	-507.79	3.75	2	0.15
No moderation on A	135.05	322	-508.95	2.59	1	0.11
No moderation on C	135.56	322	-508.44	3.10	1	0.08
No moderation on E	140.13	322	-504.17	7.67	1	0.007
No C or E moderation	139.50	323	-506.50	7.04	2	0.03
No A or C moderation	**136.48**	**323**	**-509.52**	**4.02**	**2**	**0.13**
No A or E moderation	141.46	323	-504.53	9.00	2	0.01
No moderation	144.11	324	-503.89	11.65	3	0.009
CPT	**1681.62**	**264**	**1153.62**			
No means moderation	1682.54	266	1151.14	0.92	2	0.47
No moderation on A	1687.30	265	1157.30	5.68	1	0.02
No moderation on C	1650.14	265	1120.14	-31.48	1	1.00
No moderation on E	1686.31	265	1156.31	4.69	1	0.03
No C or E moderation	1687.81	266	1155.81	6.19	2	0.05
No A or C moderation	1691.68	266	1159.68	10.06	2	0.01
No A or E moderation	1687.50	266	1155.50	5.88	2	0.05
No moderation	1692.03	267	1158.03	10.41	3	0.02
Flanker	3245.46	264	2717.46			
No means moderation	3246.46	266	2714.46	1.00	2	0.61
No moderation on A	3245.76	265	2715.75	0.30	1	0.64
No moderation on C	3246.30	265	2716.30	0.84	1	0.36
No moderation on E	3249.47	265	2719.47	4.01	1	0.05
No C or E moderation	3250.23	266	2718.23	4.77	2	0.09
No A or C moderation	**3246.31**	**266**	**2714.31**	**0.85**	**2**	**0.65**
No A or E moderation	3250.08	266	2718.08	4.62	2	0.10
No moderation	3252.61	267	2718.61	7.15	3	0.07

Model	-2LL	df	AIC	Δ-2LL	Δdf	p
Digit Span Forward	1056.88	267	522.88			
No means moderation	1060.65	269	522.65	3.77	2	0.15
No moderation on A	1057.45	268	521.45	0.57	1	0.45
No moderation on C	1058.06	268	522.06	1.18	1	0.28
No moderation on E	1056.90	268	520.90	0.02	1	0.88
No C or E moderation	1060.94	269	522.94	4.06	2	0.13
No A or C moderation	1060.55	269	522.55	3.67	2	0.16
No A or E moderation	1058.01	269	520.01	1.13	2	0.57
No moderation	**1060.94**	**270**	**520.94**	**4.06**	**3**	**0.26**
Digit Span Backward	974.76	267	440.76			
No means moderation	976.06	269	438.06	1.30	2	0.52
No moderation on A	974.79	268	438.79	0.03	1	0.87
No moderation on C	974.76	268	438.76	0.001	1	0.97
No moderation on E	974.81	268	438.81	0.05	1	0.82
No C or E moderation	974.81	269	436.81	0.05	2	0.98
No A or C moderation	974.84	269	436.84	0.08	2	0.96
No A or E moderation	974.96	269	436.96	0.20	2	0.91
No moderation	**975.27**	**270**	**435.27**	**0.51**	**3**	**0.92**
Attentional Focusing	819.64	315	189.64			
No means moderation	825.52	317	191.53	5.88	2	0.05
No moderation on A	819.64	316	187.65	0.004	1	0.95
No moderation on C	819.92	316	187.92	0.28	1	0.60
No moderation on E	820.21	316	188.21	0.57	1	0.45
No C or E moderation	820.62	317	186.62	0.98	2	0.61
No A or C moderation	820.02	317	186.02	0.38	2	0.83
No A or E moderation	820.61	317	186.61	0.97	2	0.62
No moderation	**820.70**	**318**	**184.70**	**1.06**	**3**	**0.79**
Inhibitory Control	457.77	315	-172.23			
No means moderation	474.67	317	-159.33	16.90	2	<0.001
No moderation on A	460.17	316	-171.82	2.40	1	0.12
No moderation on C	**458.30**	**316**	**-173.70**	**0.53**	**1**	**0.47**
No moderation on E	462.80	316	-169.20	5.03	1	0.03
No C or E moderation	463.10	317	-170.89	5.33	2	0.07
No A or C moderation	461.14	317	-172.86	3.37	2	0.19
No A or E moderation	463.91	317	-170.09	6.13	2	0.05
No moderation	464.41	318	-171.58	6.64	3	0.08

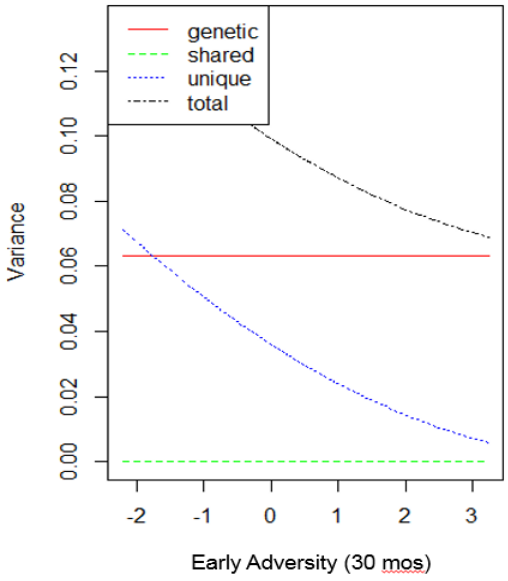
Note. -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike's Information Criterion; Δ = change; *p* = probability. The most parsimonious final model for each variable is indicated in bold.

Figure 4. *Moderation of unstandardized (left) and standardized (right) variance components*

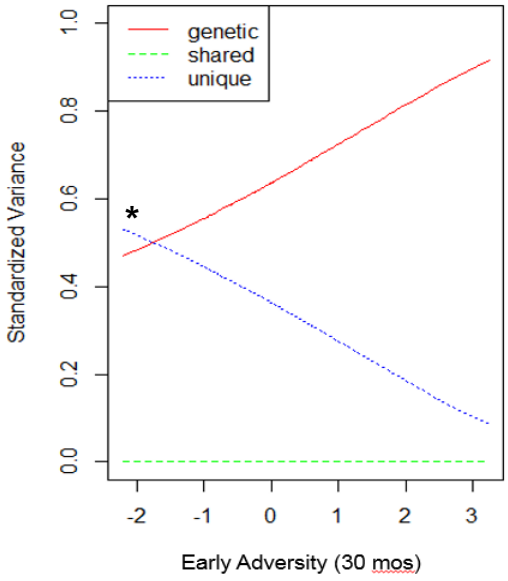


Common EF from CFA (8 yrs): Moderation by Early Adversity (30 mos)

Raw Moderation Effects

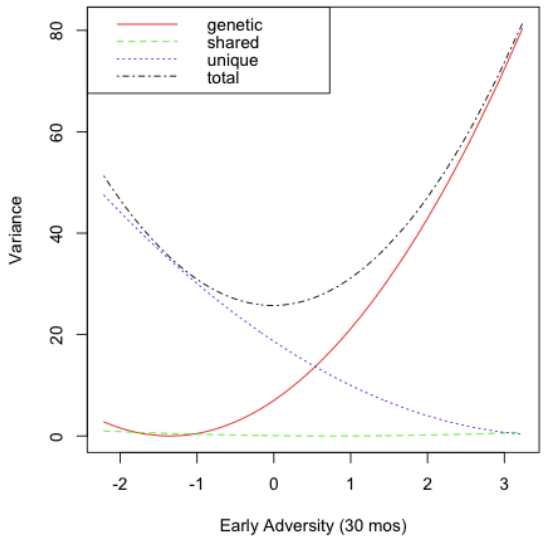


Standardized Moderation Effects

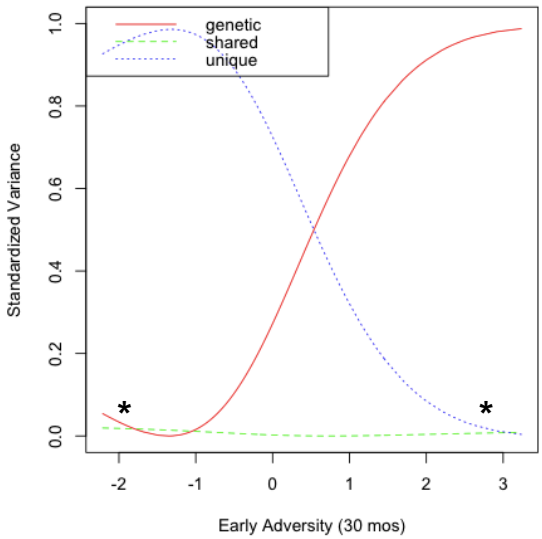


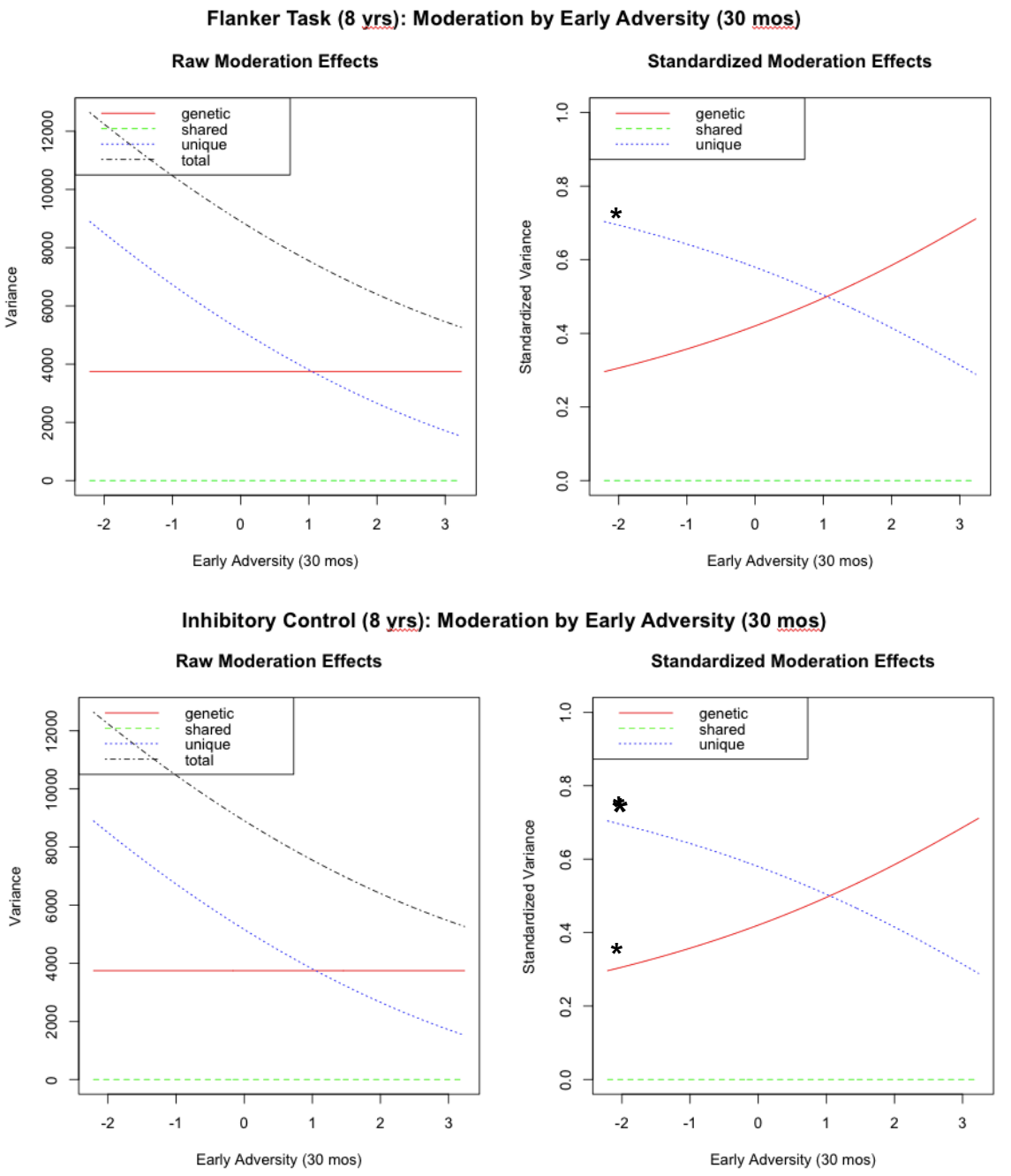
Continuous Performance Task (8 yrs): Moderation by Early Adversity (30 mos)

Raw Moderation Effects



Standardized Moderation Effects





Note: Moderation of unstandardized (left) and standardized (right) variance components. In the unstandardized (left) graph, genetic, shared, unique, and total represent unstandardized additive genetic, shared environmental, nonshared environmental, and total variance, respectively, and in the standardized (right) graph, genetic, shared, and unique represent standardized additive genetic, shared environmental, and nonshared environmental variance, respectively. Paths that are significant are indicated with an asterisk.

Table 8. *Standardized factor loadings from ACE common pathway models*

	Specific ACE Loadings			Loading on Common Factor
One Factor	**A**	**C**	**E**	
Common Factor	.67	--	.33	--
CPT	.23	--	.69	.09
Flanker	.42	--	.42	.09
Digit Span Forward	.45	.12	.40	.03
Digit Span Backward	.18	.11	.69	.02
Attentional Focusing	--	--	.11	.90
Inhibitory Control	.44	.18	.06	.32
One Factor without Inhibitory Control	**A**	**C**	**E**	
Common Factor	.79	--	.21	--
CPT	.10	--	.68	.22
Flanker	.16	--	.41	.43
Digit Span Forward	.49	--	.36	.15
Digit Span Backward	.24	--	.62	.14
Attentional Focusing	.48	--	.33	.19

Table 9. *Fit indices for common pathway models of EF*

Model	**-2LL**	**df**	**AIC**	**Δ-2LL**	**Δdf**	**p vs. Model 1**
1. One common factor	11707.43	2468	6771.43			
2. One common factor without Inhibitory Control	11162.33	2022	7118.33	545.10	446	8.98e-4

Note. -2LL = -2 log likelihood; df = degrees of freedom; AIC = Akaike's Information Criterion; Δ = change; *p* = probability.

Table 10. *Standardized factor loadings and fit statistics from phenotypic confirmatory factor analysis (CFA) without Inhibitory Control*

	Loading
Attentional Focusing	.45
CPT	.45
Flanker	.56
Digit Span Forward	.39
Digit Span Backwards	.38

-2LL	**df**	**RMSEA**	**CFI**	**AIC**
136.11	15	0.04	.926	11649.43

Note. -2LL = -2 log likelihood; df = degrees of freedom; RMSEA = Root Mean Square Error of Approximation; CFI = Confirmatory Fit Index; AIC = Akaike's Information Criterion