Executive functioning in early childhood: etiology and developmental significance

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EXECUTIVE FUNCTIONING IN EARLY CHILDHOOD:
ETIOLOGY AND DEVELOPMENTAL SIGNIFICANCE

by

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EXECUTIVE FUNCTIONING IN EARLY CHILDHOOD: ETIOLOGY AND DEVELOPMENTAL SIGNIFICANCE

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ABSTRACT

Executive functioning (EF) facilitates a wide range of purposeful actions and plays a significant role in adaptive functioning. Despite considerable variability in EF, little is known about the factors underlying individual differences in EF in early childhood. The aims of the present research were to explore the genetic and environmental influences on individual differences in EF and the factors underlying the relations between EF and developmentally-significant outcomes.

The sample comprised 209 4-year-old twin pairs (79 monozygotic, 130 dizygotic). EF was assessed with the NIH Toolbox: Early Childhood Cognitive Battery, a computerized battery of multidimensional measures. Both observers and parents provided ratings of temperament and parents evaluated behavior problems. School readiness was assessed with a standardized test of basic skills.

Model-fitting procedures revealed that variability in set-shifting and inhibitory control could be attributed to both genetic (i.e., 36% and 46%, respectively) and nonshared environmental (i.e., 64% and 54%, respectively) influences. A moderate phenotypic association ($r=.30$) was found between set-shifting and inhibitory control.
Multivariate behavioral genetic models revealed that approximately 85% of the genetic effects on inhibitory control covaried with set-shifting.

Set-shifting and inhibitory control were associated with observer-rated task orientation ($r_s = .29$ and .26, respectively) and school readiness ($r_s = .33$ and .34, respectively). Both task orientation and school readiness were heritable ($h^2 = 28\%$ and $82\%$, respectively) and the correlations between both set-shifting and inhibitory control and these outcomes were due to common genetic influences. Parent-rated temperament was not associated with EF, but a related construct, effortful control, was inversely related to hyperactivity and externalizing behavior problems ($r_s = -.46$ and -.41, respectively). Genetic and environmental factors underlie these associations.

These findings indicate that both facets of EF share common genetic underpinnings and that these effects also underlie their associations with developmental outcomes. The present study contributes novel information about the etiology of early EF, with implications for cognitive, socio-emotional, and behavioral development, and ultimately, prevention and intervention efforts.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acknowledgements</td>
<td>iv</td>
</tr>
<tr>
<td>Abstract</td>
<td>v</td>
</tr>
<tr>
<td>Table of Contents</td>
<td>vi</td>
</tr>
<tr>
<td>List of Tables</td>
<td>xii</td>
</tr>
<tr>
<td>List of Figures</td>
<td>xiii</td>
</tr>
<tr>
<td>List of Abbreviations</td>
<td>xiv</td>
</tr>
<tr>
<td><strong>Chapter 1: Introduction</strong></td>
<td>1</td>
</tr>
<tr>
<td>Defining Executive Functioning</td>
<td>2</td>
</tr>
<tr>
<td><em>Set-Shifting</em></td>
<td>3</td>
</tr>
<tr>
<td><em>Inhibitory Control</em></td>
<td>3</td>
</tr>
<tr>
<td>Theoretical Perspective of Executive Functioning</td>
<td>4</td>
</tr>
<tr>
<td><em>Development of Set-Shifting</em></td>
<td>4</td>
</tr>
<tr>
<td>-Contribution of Set-Shifting to Executive Functioning</td>
<td>7</td>
</tr>
<tr>
<td><em>Development of Inhibitory Control</em></td>
<td>7</td>
</tr>
<tr>
<td>-Contribution of Inhibitory Control to Executive Functioning</td>
<td>9</td>
</tr>
<tr>
<td>Measurement of Executive Functioning in Early Childhood</td>
<td>9</td>
</tr>
<tr>
<td>The Structure of Executive Functioning</td>
<td>10</td>
</tr>
<tr>
<td>Developmental Significance of Executive Functioning</td>
<td>12</td>
</tr>
<tr>
<td><em>Executive Functioning and Temperament</em></td>
<td>12</td>
</tr>
<tr>
<td><em>Executive Functioning and School Readiness</em></td>
<td>14</td>
</tr>
</tbody>
</table>

vii
Executive Functioning and Behavior Problems 16
Etiology 18
Inhibitory Control and Attention 19
Set-Shifting 20
Etiology of Executive Functioning in Early Childhood 21
Genetic and Environmental Overlap 22
Executive Functioning and Developmental Outcomes 22
Current Study 23
Specific Aim 1 23
Specific Aim 2 24
Specific Aim 3 24
Chapter 2: Method 26
Sample 26
Procedure 27
Measures 29
Executive Functioning 29
- Inhibitory Control 29
- Set-Shifting 30
Temperament 32
- Parent-Rated Temperament 32
- Observer-Rated Temperament 33
Behavior Problems 34
School Readiness

Vocabulary

Data Analysis

The Twin Design: Overview

Phenotypic Correlations

Twin Intraclass and Cross Correlations

Model-Fitting Analyses

- Univariate Models

- Multivariate Models

Data Transformations

Chapter Three: Results

Descriptive Statistics

Specific Aim 1

Aim 1a: Univariate Model-Fitting

- Intraclass Correlations

- Model-Fitting Results

Set-Shifting

Inhibitory Control

Aim 1b: Bivariate Model-Fitting

- Phenotypic Correlational Analysis

- Twin Cross-Twin Cross-Trait Correlations

- Multivariate Model-Fitting Results
Specific Aim 2 52

*Phenotypic Correlational Analyses* 52

*Twin Intraclass and Cross-Twin Cross-Trait Correlations* 53

Specific Aim 3 57

*Phenotypic Correlational Analyses* 57

*Twin Intraclass and Cross-Twin Cross-Trait Correlations* 58

*Set-Shifting, Inhibitory Control, and School Readiness* 58

-Multivariate Model-Fitting Results 58

*Hyperactivity, Externalizing Behavior problems and Effortful Control*

-Multivariate Model-Fitting Results 61

**Chapter Four: Discussion** 66

Etiology of Executive Functioning in Early Childhood 66

Executive Functioning and Temperament 74

Executive Functioning and School Readiness 77

Executive Functioning and Behavior Problems 80

Implications 86

Limitations 87

Future Directions 88

Conclusion 88

**Appendices** 91

Appendix A: NIH Toolbox Flanker Administration Instructions 92
# LIST OF TABLES

Table 1. BUTP protocol 28

Table 2. Means (standard deviations) by zygosity and sex 43

Table 3. Twin intraclass and cross-twin cross-trait correlations 44

Table 4. Univariate model-fitting results for set-shifting 46

Table 5. Univariate model-fitting results for inhibitory control 48

Table 6. Bivariate Cholesky decomposition results for set-shifting and inhibitory control 50

Table 7. Trivariate Cholesky decomposition model-fitting results for set-shifting, inhibitory control, and task orientation 54

Table 8. Phenotypic correlations 57

Table 9. Trivariate Cholesky decomposition model-fitting results for set-shifting, inhibitory control, and school readiness 59

Table 10. Trivariate Cholesky decomposition model-fitting results for hyperactivity, externalizing behavior problems, and effortful control 63
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1.</td>
<td>Flanker trial sequence</td>
<td>30</td>
</tr>
<tr>
<td>Figure 2.</td>
<td>Incongruent Flanker trial</td>
<td>30</td>
</tr>
<tr>
<td>Figure 3.</td>
<td>DCCS trial sequence</td>
<td>32</td>
</tr>
<tr>
<td>Figure 4.</td>
<td>DCCS trial</td>
<td>32</td>
</tr>
<tr>
<td>Figure 5.</td>
<td>Univariate twin model</td>
<td>39</td>
</tr>
<tr>
<td>Figure 6.</td>
<td>Bivariate Cholesky decomposition model</td>
<td>40</td>
</tr>
<tr>
<td>Figure 7.</td>
<td>Best-fitting bivariate Cholesky decomposition model</td>
<td>52</td>
</tr>
<tr>
<td></td>
<td>for set-shifting and inhibitory control</td>
<td></td>
</tr>
<tr>
<td>Figure 8.</td>
<td>Best-fitting trivariate Cholesky decomposition model</td>
<td>56</td>
</tr>
<tr>
<td></td>
<td>for set-shifting, inhibitory control, and task orientation</td>
<td></td>
</tr>
<tr>
<td>Figure 9.</td>
<td>Best-fitting trivariate Cholesky decomposition model</td>
<td>60</td>
</tr>
<tr>
<td></td>
<td>for set-shifting, inhibitory control, and school readiness</td>
<td></td>
</tr>
<tr>
<td>Figure 10.</td>
<td>Best-fitting trivariate Cholesky decomposition model</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td>for hyperactivity, externalizing behavior problems, and effortful control</td>
<td></td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
<td></td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
<td></td>
</tr>
<tr>
<td>-2LL</td>
<td>Log-Likelihood</td>
<td></td>
</tr>
<tr>
<td>$\chi^2$</td>
<td>Chi-Square</td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>Additive Genetic Influences</td>
<td></td>
</tr>
<tr>
<td>ADHD</td>
<td>Attention Deficit/Hyperactivity Disorder</td>
<td></td>
</tr>
<tr>
<td>AIC</td>
<td>Akaike’s Information Criterion</td>
<td></td>
</tr>
<tr>
<td>BSRA</td>
<td>Bracken School Readiness Assessment-III</td>
<td></td>
</tr>
<tr>
<td>BUTP</td>
<td>Boston University Twin Project</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>Shared Environmental Influences</td>
<td></td>
</tr>
<tr>
<td>$e^2$</td>
<td>Shared Environmental Variance</td>
<td></td>
</tr>
<tr>
<td>CBCL</td>
<td>Child Behavior Checklist for Ages 1½-5</td>
<td></td>
</tr>
<tr>
<td>CBQ-SF</td>
<td>Child Behavior Questionnaire-Short Form</td>
<td></td>
</tr>
<tr>
<td>DCCS</td>
<td>Dimensional Change Card Sort</td>
<td></td>
</tr>
<tr>
<td>$df$</td>
<td>Degrees of Freedom</td>
<td></td>
</tr>
<tr>
<td>DNA</td>
<td>Deoxyribonucleic Acid</td>
<td></td>
</tr>
<tr>
<td>DZ</td>
<td>Dizygotic Twin Pairs</td>
<td></td>
</tr>
<tr>
<td>E</td>
<td>Nonshared Environmental Influences</td>
<td></td>
</tr>
<tr>
<td>$e^2$</td>
<td>Nonshared Environmental Variance</td>
<td></td>
</tr>
<tr>
<td>EF</td>
<td>Executive Functioning</td>
<td></td>
</tr>
<tr>
<td>Flanker</td>
<td>Flanker Inhibitory Control and Attention Test</td>
<td></td>
</tr>
<tr>
<td>GEE</td>
<td>Generalized Estimating Equations</td>
<td></td>
</tr>
<tr>
<td>GxE</td>
<td>Gene x Environment Interaction</td>
<td></td>
</tr>
</tbody>
</table>
\( h^2 \)  
Heritability

IBR  
Infant Behavior Record

Lab-TAB  
Laboratory Temperament Assessment Battery

MZ  
Monozygotic Twin Pairs

NIH Toolbox  
NIH Toolbox: Early Childhood Cognitive Battery

\( r_c \)  
Shared Environmental Correlation

\( r_e \)  
Nonshared Environmental Correlation

\( r_g \)  
Genetic Correlation

SAS  
Statistical Analysis System

\( SD \)  
Standard Deviation

SES  
Socioeconomic Status

TPVT  
Toolbox Picture Vocabulary Test
CHAPTER ONE

Introduction

Self-regulation involves the control of cognition, emotion, and behavior. Within the broader domain of self-regulation, there exists a set of cognitive control skills referred to as executive functioning (EF). These cognitive abilities regulate and coordinate the internal and transactional processes that enable individuals to guide goal-directed thought, action, and emotion (Anderson, 2002; Zelazo et al., 2013). EF facilitates a wide range of purposeful actions and allows us to fluidly approach novel behaviors and circumstances. Individual differences in EF play a significant role in adaptive functioning, as EF is necessary for higher-level mental and behavioral abilities across the lifespan (Garon, Bryson, & Smith, 2008).

There is substantial development of the basic skills required for EF across the first five years of life (Carlson, 2005). Developmental antecedents of EF emerge as simple behaviors such as regulating eye movements and searching for hidden objects in early infancy (Diamond, 1990; Johnson, 1995). More advanced skills, such as holding representations in mind, inhibiting responses based on a rule held in mind, and suppressing motivated motor responses build on these simple skills across the first three years of life (Garon et al., 2008). Entry to preschool marks a developmental transition out of toddlerhood and into schooling, where children are expected to regulate their behavior in accordance with external demands. For example, they are required to stop enjoyable activities, wait for meals, and clean up after themselves (e.g., Kochanska, Murray, & Coy, 1997). Because of their immature EF abilities, children often react negatively to
these day-to-day challenges. Even within the preschool years, there is a clear developmental shift in EF. Older preschoolers (i.e., 4-year-olds) consistently outperform younger preschoolers (i.e., 3-year-olds) on EF tasks.

The integration of these simpler antecedents lays the foundation for complex EF abilities to grow across early childhood. Further, the unification of attentional systems across the preschool period supports children’s ability to focus during structured tasks, allowing them to enhance target-relevant information and reduce target-irrelevant information (Igushi, Hoshi, Tanosaki, Taira, & Hashimoto, 2005). The development of EF forms a critical foundation for future cognitive capacities in adulthood (Garon et al., 2008).

**Defining Executive Functioning**

Although there is no universal conceptualization of EF (Friedman et al., 2008), it is often theorized as multiple processes that function together as a supervisory system that is important for planning, reasoning, and the integration of thought and action (Shallice & Burgess, 1996; Stuss & Alexander, 2000). The interactive functioning systems approach (Luria, 1973) proposes that there are three functional blocks of the brain. The first block regulates mental and waking states and is responsible for organizing goal-directed activity. The second receives, analyzes, and stores information. The final and most complex block controls and regulates voluntary activities. It includes: objective setting in accordance with motivated planned or executed activities, organizing a program for the best way to achieve a goal, monitoring the execution of activities and correcting insufficient actions, and comparing objectives with the results. This block was later called
EF. This theory proposed that EF is a multifaceted construct and laid the groundwork for evaluating the components of EF (i.e., set-shifting, inhibitory control, and working memory) and the interrelations among them. The present study will explore two facets of EF, set-shifting and inhibitory control, because they are well defined and can be assessed reliably in early childhood.

Set-Shifting

One component of EF, mental set-shifting, involves flexibly switching among multiple tasks to meet changing environmental demands. Most often, set-shifting tasks involve switching from a dominant, pre-formed stimulus-response association to a less-dominant response set to determine the rate of perseveration (i.e., inaccurate response repetition) to the initially presented (i.e., dominant) task and the automaticity of responding (e.g., switching from matching cards by color to matching by shape). In such tasks, participants experience frequent shifts between the rules governing the nature of the association that they must act in accordance with. Successful execution requires the participant to recognize the relevant goal of the task and subsequently activate the appropriate response set (i.e., identify the set of parameters related to the task rules, orient attention, and engage in response selection) to achieve the goal (Vandierendonck, Lefooghe, & Verbruggen, 2010). Set-shifting emerges at the end of the preschool period (Garon et al., 2008).

Inhibitory Control

Another component of EF, inhibitory control, involves the suppression or delay of a prepotent, salient response for one that is less dominant to achieve a goal. Inhibitory
control is often assessed in the laboratory under conditions of delay (e.g., bypassing a small reward for a larger reward that will be presented after a time delay), conflict (e.g., inhibiting responses to distractors flanking a target stimulus) and other challenges (Carlson & Moses, 2001). Inhibitory control emerges toward the end of the first year and undergoes substantial development across the toddler and preschool years (Diamond, 2002).

Theoretical Perspectives of Executive Functioning

Development of Set-Shifting

A number of theories claim to explain why young children have difficulties with EF. Most focus on one component of EF, set-shifting. One of the most widely used set-shifting tasks in developmental psychology research is the Dimensional Change Card Sort (DCCS; Zelazo, 2006). The DCCS requires individuals to match one of two alternative objects to a target object according to one dimension (e.g., shape) then by another dimension (e.g., color).

Some theorists suggest that there are qualitative changes in the way that younger children and older children approach set-shifting tasks. These perspectives propose that children are incapable of succeeding on tasks like the DCCS because they do not have the fundamental skills that are required to understand the task demands. The Cognitive Complexity and Control Theory (Zelazo & Frye, 1998; Zelazo, Müller, Frye, & Marcovitch, 2003) is an information processing theory proposing that children’s success on set-shifting tasks is dependent on their ability to represent an increasing number of levels embedded in a hierarchical structure of if-then rules (Zelazo et al., 2003) and age-
related increases in performance correspond with development of meta-cognition and reflection (Zelazo, Frye, & Rapus, 1996). According to this approach, 3-year-olds are only able to consider a pair of rules simultaneously (e.g., if red, match here; if blue, match here). It is not until approximately 5 years of age that children are able to nest a pair of rules under a higher-order rule that dictates which pair of incompatible rules they must select. For example, to match by color in the ‘color game’ and by shape in the ‘shape game’. The ability to select one of two incompatible rules is what enables children to succeed on this task.

Alternatively, the Re-Description Hypothesis posits that performance on set-shifting tasks is related to the understanding that the same object (e.g., a blue truck) has different features (e.g., shape and color) and can be described in more than one way and is driven by conceptual development (Kloo & Perner, 2005; Perner & Lang, 2002). Therefore, to flexibly change the dimension by which stimuli are matched, children must recognize that things can be described differently under diverse conditions and be able to re-describe the stimuli according to a new dimension (Kloo, Perner, Kerschuber, Dabernig, & Aichorn, 2008).

Another account (Perner, Stummer, & Lang, 1999) explains problems on the DCCS in terms of a failure to inhibit the initial matching schema in favor of the secondary schema at the level of behavioral responding (i.e., failures occur at the behavioral, rather than information processing, stage). However, this does not appear to be the case, because 3-year-olds have trouble on the DCCS not only when performing the task on their own, but also when they are judging the responses of a puppet (Jacques,
Moreover, they tend to perseverate when a verbal, rather than a manual response is required (Zelazo et al., 1996). Because young children perseverate, and thus, fail to exhibit cognitive flexibility under these diverse conditions, this suggests that these failures occur at the information processing stage rather than the level of behavioral responding.

Others propose that developmental change in set-shifting performance is due to quantitative growth of attentional skills. According to the Attentional Inertia Hypothesis, inertia is a force that facilities directed attention. To shift attention children must have adequate inhibitory abilities to override the previous attentional inertia, and these inhibitory skills develop over time (Kirkham, Cruess, & Diamond, 2003). Therefore, children perseverate on the first dimension that they match by in the DCCS because their attention remains stuck on the dimension that was initially presented (i.e., the dominant dimension). Likewise, an alternative theory maintains that a latent trace of the dimension that is initially attended to still exists in the attentional system. To successfully shift attention, children must actively maintain the current rule in memory without directly inhibiting the previous rule (Morton & Munakata, 2002).

The Dynamic Field Theory shares features with the later model (i.e., competition among memory traces) but incorporates novel features and successfully explains many of the empirical findings on the DCCS (Buss & Spencer, 2014). This theory proposes that children succeed on rule-use tasks, like the DCCS, when the neural mechanisms that underlie the dorsal (i.e., the ‘where’ stream) and ventral (i.e., the ‘what’ stream) visual pathways are integrated by a simple mechanism that modulates the resting level of neural
populations (Buss & Spencer, 2014). For younger children, neural populations do not reach full resting states between trials. Therefore, existing neural populations (i.e., lingering feature dimensions) influence the next trial, leading to perseverative responding. As children get older, neural resting state is achieved and children no longer perseverate.

**Contribution of Set-Shifting to Executive Functioning.** Set-shifting is the most complex EF component and naturally builds on attentional capacities and the other components of EF (i.e., inhibitory control and working memory). Nonetheless, set-shifting is not simply the coordination of the other EF components, rather it is an EF process that operates on the other EF processes (Garon et al., 2008). Given its complexity, set-shifting is the final facet of EF to develop.

**Development of Inhibitory Control**

The ability to suppress a dominant response emerges within the first year of life, but children become increasingly adept at inhibiting their behavior across early childhood (Garon et al., 2008). Simple types of response inhibition are displayed when young children stop an enjoyable behavior at the request of a caregiver. Whereas 8-month-olds are able to inhibit their behavior in response to external requests only 40% of the time (Kochanska, Tjebkes, & Forman, 1998), 22- and 33-month-olds are able to inhibit their behavior in these situations the majority of the time (i.e., 78% and 90%, respectively; Kochanska, 2002). Under conditions of delay (i.e., suppressing a desired response over a period of time), children from 24 to 56 months of age are able to progressively delay their behavior for longer periods (Carlson, 2005; Kochanska, Murray & Harlan, 2000;
Kochanska, Murray, Jacques, Koenig, & Vandecceest, 1996). Similar age-related improvements occur on Stroop-like tasks from 2 to 4 years of age (Carlson, Mandell, & Williams, 2004; Hughes, 2007).

There is disagreement surrounding the process by which cognitive inhibition occurs. Some researchers propose a ‘threshold’ model. According to this approach, at any given time we are engaging in multiple thoughts and behaviors. Consequently, any relevant action patterns underlying these thoughts and behaviors are at elevated thresholds for activation; we are prepared to perform a relevant action as soon as the appropriate triggering conditions occur (Norman & Shallice, 1986). Based on these competing activations, inhibitory control is simply a passive byproduct of selectively activating action patterns that are relevant to the present thought or behavior over those that are irrelevant (e.g., Munakata et al., 2011). Alternatively, some propose a more effortful threshold theory. This approach hypothesizes that inhibition involves both the activation of task-relevant information and the active suppression of information that is irrelevant to the task at hand (Posner & Cohen, 1984).

Others theorize that the inhibitory (i.e., “stop”) process is independent of the excitatory (i.e., “go”) process that initiates deliberate action (Logan & Cowan, 1984). In turn, successful inhibition is dependent on the winner of the race between these “stop” and “go” processes. If inhibition wins the race, the initiated action is stopped; however, if response execution wins, the “go” action functions as if the “stop” process was never initiated. Therefore, according to this model, inhibitory control is dependent on reaction time. Poor inhibitory capacities could result from responding too quickly to the “go”
signal or too slowly to the “stop” signal (Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Nonetheless, age-related improvements in inhibitory processes occur as we become more adept at activating the appropriate action under the correct conditions.

Contribution of Inhibitory Control to Executive Functioning. There remains controversy over the nature of the contribution of inhibitory control to the multidimensional construct of EF. Some researchers propose that inhibitory control is a central component that unifies EF (e.g., Barkley, 1997). Others suggest that inhibitory control depends on another facet of EF (i.e., working memory) and is not a separable component (e.g., Garon et al., 2008). More intermediate views theorize that inhibitory control is one of three interrelated but dissociable facets that reflect a unifying EF factor (e.g., Miyake et al., 2000). This theoretical disagreement raises questions about the structure of EF and the extent to which inhibitory control overlaps with other components of EF. The present study addresses this question by investigating the genetic and environmental overlap between inhibitory control and set-shifting. At a more global level, it has been suggested that there are two ways that inhibitory control can be related to thinking. First, that inhibitory control exerts control on the thinking process by eliminating competing inputs. Secondly, that inhibitory control itself is integral to the thinking process (Diamond, Balvin, & Diamond, 1963).

Measurement of Executive Functioning in Early Childhood

EF is increasingly recognized as a primary influence in key developmental outcomes (Zelazo, et al., 2013) ranging from behavior problems (Fahie & Symons, 2003) and autism (Luna, Doll, Hegedus, Minshew, & Sweeney, 2007) to socioeconomic status
and substance dependence (Moffit et al., 2011). Despite the developmental-significance of EF, methodological limitations of research on EF in early childhood have dampened progress in this area (Zelazo et al., 2013). Prior to the past decade, existing neuropsychological measures of EF were developmentally inappropriate for use with young children. Further, preschool children are low in attention span, linguistic competence, motivation levels and general background knowledge, complicating the assessment of EF during this developmental period. Therefore, tasks that effectively assess EF in preschoolers must minimize the complexity of verbal and manual responding and similar task demands by utilizing frameworks that are familiar to children, such as shapes and colors (Wiebe et al., 2011).

Consequently, although it is important to study EF as it comes online in early childhood, it is difficult to assess in young children. The recently-devised EF battery used in the present study, the NIH Toolbox: Early Childhood Cognitive Battery (Zelazo et al., 2013) is developmentally appropriate for use with young children. The assessments capitalize on children’s existing knowledge and abilities, and minimize the complexity of the explanation of the tasks. It is also critical to explore the structure of EF in young children to gain insight into the appropriate way to assess EF during this period.

The Structure of Executive Functioning

As previously noted, it is theorized that EF is a multidimensional construct including set-shifting, inhibitory control, and working memory. Research that focuses on the structure of EF is important for a more general understanding of the nature of cognitive development (Zelazo et al., 2013). It is reasonable to expect that different
components of EF rely on one another for successful execution of cognitive processes. For example, we need to attend to cues that signal a change in our environment, inhibit thoughts and behaviors that are not appropriate to the current situation, and shift to new cognitive or behavioral sets (Bari & Robbins, 2013).

To explore the structure of EF, researchers use structural equation modeling techniques to model the overlap between the facets of EF. Results from these analyses revealed that from middle childhood onward, individual differences in EF show both unity and diversity and EF is comprised of both dissociable but moderately intercorrelated factors, most often including set-shifting, inhibitory control, and working memory (Friedman & Miyake, 2004; Friedman et al., 2007, 2008; Huizinga, Dolan, & van der Molden, 2006; Lehto, Juujärvi, Kooistra, Pulkkinen, 2003; Miyake et al., 2000; Miyake & Friedman, 2012). Despite this, research on the factor structure of EF in younger participants is more consistent with a single-factor (Hughes, Ensor, Wilson, & Graham, 2010; Wiebe, Espy, & Charack, 2008; Wiebe et al., 2011). This suggests that EF is simpler in form in early childhood, and that the distinct factors that emerge later in development largely overlap during this period.

These findings call into question the notion that EF is distinguishable in early childhood (Wiebe, 2014). As development proceeds from global and diffuse to articulated and differentiated (Werner, 1948) it is possible that the structure of EF changes across development as a more unified structure in early childhood can give way to more dissociable components. This developmental pattern of EF is consistent with neurocognitive development in general, a process that involves the increasing
specialization of neural systems that begin relatively undifferentiated and become more specialized as development progresses (e.g., Johnson & Munakata, 2005; Tucker-Drob, 2009). Key milestones in brain development, including overproduction and pruning of synapses and increases in grey and white matter in the prefrontal cortex take place during the preschool years and continue into early adulthood (e.g., Huttenlocher & Dabholkar, 1997). Therefore, it is reasonable to expect age-related differences in factor structure, as behavioral gains in EF are subserved by development of the prefrontal cortex (Stuss & Knight, 2002). Nonetheless, EF is a developmentally-significant construct and a finer-grained understanding of the organization of EF will allow for more nuanced assessments of its biological correlates (e.g., candidate genes) and promote effective interventions for early EF deficits.

*Developmental Significance of Executive Functioning*

*Executive Functioning and Temperament*

As discussed earlier, EF falls under the umbrella of self-regulation. Self-regulation enables individuals to control and guide goal-directed cognition, emotion, and behavior over time and across changing contexts. Self-regulation has been implicated as an important protective factor for developmentally-significant outcomes, but sub-disciplines in the field of psychology conceptualize self-regulation from diverse frameworks (Bridgett, Oddi, Laake, Murdock, & Bachmann, 2013). Thus far, self-regulation has been theorized in a cognitive EF framework (e.g., Blair & Ursache, 2011; Gyurak et al., 2009). However, some researchers use a temperament framework to study self-regulation using measures of effortful control (Rothbart, Derryberry, & Posner,
Effortful control differs from EF because effortful control involves motivation and emotional drive (i.e., it is “hot”), whereas EF is emotionally neutral (i.e., it is “cold”).

There are clear commonalities between EF and effortful control. First, both share a common component: inhibition. Second, both share a common underlying process: the executive attention network (Zhou, Chen, & Main, 2012). Further, EF and effortful control are positively associated in preschoolers (Hongwanishkul, Happaney, Lee, & Zelazo, 2005). Nonetheless, most researchers agree that EF and effortful control are related, but distinct self-regulatory capacities (e.g., Blair & Ursache, 2011). Despite this, EF and effortful control are often used interchangeably in the literature.

More research is needed on the extent to which EF and effortful control are unique constructs to inform whether the call for an integrated conceptual model of self-regulation is supported empirically (Zhou et al., 2012). These findings have direct implications for developmental research on self-regulation (i.e., whether EF and effortful control can be used interchangeably as markers for maladaptive developmental outcomes). If EF and effortful control are related, the present study can explore the extent to which common genetic and environmental influences underlie the association between EF and effortful control as a direct test of the overlap between the constructs.

Further, there are mixed findings on the extent to which EF is related to other temperament dimensions. While some studies fail to find an association between the two constructs (e.g., Hongwanishkul et al., 2005) others have (e.g., Carlson, Mandell, &
Williams, 2004). The present study aims to resolve this conflict by identifying the origins of the associations between EF and a wide range of temperament dimensions.

Executive Functioning and School Readiness

The importance of EF to academic achievement is indisputable. Better EF skills are associated with higher academic performance across a wide range of studies (e.g., Espy et al., 2004; McClelland et al., 2007). It has been hypothesized that there are two avenues by which good EF skills support academic success. First, EF may facilitate academic success in a direct way by allowing children to meet the cognitive demands of mathematics and literacy activities. Good EF skills foster children’s capacity to quickly and efficiently inhibit previous perspectives and direct their attention to multiple dimensions of or perspectives on a problem (Diamond, Carlson, & Beck, 2005). For example, problem-solving, ordinality, transitivity, and pattern completion tasks require individuals to represent a problem in memory, flexibly switch attention to different elements of the problem, and to inhibit the tendency to respond to the most salient or available information in the problem in favor of an alternative based on context-dependent cues (Blair & Raver, 2015; Blair & Razza, 2007). EF is also required for literary activities such as flexibly maintaining letter sounds and symbols in memory (Fuhs, Farran, & Nesbitt, 2015) and identifying and manipulation of sound units within a word. For example, identifying a smaller word embedded within a longer word, like toothbrush (Blair & Raver, 2015).

The second way that EF may facilitate academic achievement is that it provides a foundation for behavioral success in the classroom. Children with more advanced EF
skills may benefit most from the academic environment because they are better able to pay attention, remember class rules, and engage in academic content (Fuhs, Farran, & Nesbitt, 2015). Educators echo this notion. Teachers report that the skills most required for success in the classroom are those regulated by EF—children’s capacities to sit still, pay attention, and follow rules (McClelland et al., 2007).

Many studies have demonstrated an association between EF and school readiness and academic achievement (Blair, 2002). Advanced EF skills place children at an advantage at school entry that is maintained throughout early schooling (Bull, Espy, & Wiebe, 2008). EF accounts for significant variance in preschool math scores (Espy et al., 2004) and longitudinally predicts mathematics and reading achievement in middle childhood (Bull et al., 2008; Clark, Pritchard & Woodward, 2010).

The association between EF and academic achievement is so robust that the relation persists after controlling for cognitive abilities (Espy et al., 2004), baseline academic capacities (McClelland et al., 2007), socio-emotional competence, effortful control (Blair & Razza, 2007) and maternal education (e.g., Espy et al., 2004). In fact, EF is so essential for academic achievement that research suggests that EF is often a better predictor of academic achievement than IQ (Blair & Raver, 2015; Blair & Razza, 2007). Despite accumulating evidence for the association between EF and school readiness, no studies have explored the genetic and environmental influences underlying this association in preschoolers. This is an important question, as uncovering the mechanisms connecting these constructs can help to inform targeted prevention and intervention
efforts. The present study aims to fill this critical gap in the literature by investigating the factors linking EF and school readiness in early childhood.

Executive Functioning and Behavior Problems

EF deficits are implicated in many clinical conditions with childhood onset (Pennington & Ozonoff, 1996). Impairments in EF are associated with internalizing problems, such as anxiety (Biederman, Rosenbaum, Chaloff, & Kagan, 1995), but are most notably observed in individuals with externalizing problems, such as Attention-Deficit/Hyperactivity Disorder (ADHD). At the behavioral level, ADHD is primarily manifested by excessive physical activity, fussiness, impulsivity, and inattention that exceed the age-appropriate standards (e.g., Glozman & Shevchenko, 2014). Impulsivity is the combined result of failed inhibitory processes and impulses that are triggered by situational factors (Hofmann et al., 2009). It is widely-accepted that the complex symptoms of ADHD arise from basic neuropsychological deficits (i.e., immature cognitive functions) that are related to EF, such as orientation in space and problem solving (e.g., Glozman & Shevchenko, 2014), that are presumed to emerge during the preschool period (Pauli-Pott & Becker, 2011). In fact, the link between these constructs is sufficient enough for EF to be thought of as an endophenotype for ADHD (Doyle et al., 2005).

The association between impaired EF and ADHD is not limited to diagnosis—dimensional measures of behavior problems show a similar pattern of results. There is evidence for impaired EF in children with ADHD and hyperactivity, as compared to controls, across development (e.g., Willcutt et al., 2001). EF deficits are present in many
neurodevelopmental disorders, but specific EF components appear to be implicated in externalizing problems. Response inhibition, a measure of behavioral control, and vigilance deficits are most commonly reported both in individuals with ADHD and hyperactivity (Berlin & Bohlin, 2002; Fahie & Symons, 2003; Geurts, Verte, Oosterlaan, Roeyers, & Sergeant, 2004; Schoemaker et al., 2012; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005; Willcutt et al., 2001).

Meta-analyses of the relation between EF performance and externalizing behavior problems (including hyperactive, aggressive, and disruptive symptoms in community samples and ADHD symptoms in clinical samples) in preschoolers revealed medium-to-large effect sizes (i.e., .22-.38) for overall EF, inhibition, and delay aversion (Pauli-Pott & Becker, 2011; Schoemaker, Mulder, Deković, & Matthys, 2013). These findings indicate that EF deficits emerge in populations with both dimensional and clinical levels of externalizing behavior problems and are a defining feature of these problems. Further evidence for this relation is demonstrated in brain imaging literature. Children with ADHD display with an impaired right-frontal response inhibition mechanism (Pliszka, Liotti, & Woldorff, 2000) and abnormalities in brain processes involved in motor inhibition and error detection (Overtoom et al., 2002). These findings provide additional support for the notion that EF deficits may function as an endophenotype for ADHD across different developmental periods.

ADHD is one of the most common reasons that children are referred to psychologists in childhood (e.g., Nigg, 2005), but despite the frequency of the disorder, it is often misdiagnosed, diagnosed late, or goes undetected (Glozman & Shevchenko,
2014). Due to the implication of EF impairments in ADHD and hyperactive behavior problems, assessment of the relation between these constructs in early childhood is essential for identifying preschoolers who are at risk for later maladjustment. Moreover, EF training programs for young children have led to increased EF performance (Diamond & Lee, 2011). Because EF is responsive to intervention in early childhood, if EF deficits are shown to co-occur with externalizing behavior problems, this could reveal potential targets for early prevention and intervention, as these children may benefit most from such interventions. The present study explores the extent to which EF and hyperactivity co-occur in early childhood and the factors that underlie the association and may inform these prevention and intervention efforts.

Etiology

Given the developmental significance of EF, it is important to understand why some children have better EF than others. Significant variation exists in individuals’ EF and this variation may be explained by genetic and environmental factors (Friedman et al., 2008). Behavioral genetic investigations in middle and late childhood and adulthood demonstrate genetic influences on individual differences in EF (e.g., Friedman et al., 2008; Polderman et al., 2007).

Multivariate analyses of all three domains of EF (i.e., set-shifting, inhibitory control and working memory) have uncovered considerable common variance among the facets of EF that is almost entirely due to common genetic effects (i.e., 99% heritable). Variance that is unique to each phenotype is due to both genetic and nonshared environmental factors (Friedman et al., 2008). EF deficits (i.e., parent ratings of
neuropsychological dysfunction) are also substantially heritable from middle childhood to adolescence, with genetic factors explaining 77% of the variance and nonshared environmental factors explaining the remainder of the variance (Coolidge, Thede, & Young, 2000). As discussed below, research exploring individual facets of EF also suggests genetic influences.

**Inhibitory Control and Attention**

Behavioral genetic studies from middle childhood to adolescence consistently reveal genetic influences on individual differences in inhibitory control and attention. Individual differences in sustained and selective attention in 5- and 12-year-olds were best explained by genetic (heritabilities 52%-63%) and nonshared environmental effects (Polderman et al., 2007). Heritabilities of attention *problems* in 6-year-olds were slightly higher in magnitude (77% for girls and 83% for boys). For sustained attention, however, it could not be determined whether familial resemblance was due to genetic or shared environmental influences (Groot, de Sonneville, Stins, & Boomsma, 2004).

Inhibitory control was moderately heritable (i.e., approximately 50%) in 9-12- and 18-year-old twin pairs (Polderman et al., 2007; 2009). In 17-year-old twin pairs, a common inhibitory control factor (i.e., a factor that reflects the variance common to three measures of inhibitory control) was highly heritable (i.e., 99%). Variance that was unique to each measure was a function of genetic and nonshared environmental effects (Friedman et al., 2008).

Only three prior behavioral genetic studies investigated genetic and environmental influences on one facet of EF, inhibitory control, in early childhood. A
parent-offspring design assessed inhibitory control with the Stroop test and demonstrated indirect genetic effects as mediated by the biological mother’s verbal intelligence (Leve et al., 2013). Stronger evidence for genetic influences on inhibitory control comes from two twin studies (Gagne & Goldsmith, 2011; Gagne & Saudino, 2010). Parent-rated inhibitory control was moderately heritable at ages 2 and 3, and although many of the same genetic influences were stable across age, novel genetic influences at age 3 also emerged. Genetic influences on observed inhibitory control were moderate at age 2 and nonsignificant at age 3. Novel shared environmental and nonshared environmental influences emerged at age 3 (Gagne & Saudino, 2010, 2016). These findings mirror prior work with three-year-olds (i.e., substantial heritability for parent, but not observer ratings) using the same measures (Gagne & Goldsmith, 2011). The authors note that it is unlikely that the genetic influences on observed inhibitory control disappear at age 3, because genetic influences are consistently found on observed inhibitory control at later ages (e.g., Lemery-Chalfant, 2008), but instead, these differential findings across age are likely due to ceiling effects (Gagne & Saudino, 2016). In these studies, however, inhibitory control was assessed as a temperament dimension (i.e., lab-based assessment of delay of gratification and parent ratings of temperamental inhibitory control), not as a cognitive process. The present study extends this body of literature by exploring the genetic and environmental influences on cognitive inhibitory control in early childhood.

**Set-Shifting**

Another facet of EF, set-shifting, is substantially heritable in adolescence and adulthood (i.e., heritabilities range from 50-80%), with the remainder of the variance due
to nonshared environmental influences (Anokhin, Heath, & Ralano, 2003; Friedman et al., 2008). No behavioral genetics studies have examined the genetic and environmental influences on set-shifting in early childhood. The present study will address this critical gap in the literature.

**Etiology of Executive Functioning in Early Childhood**

The current study uses cognitively-based measures of EF (i.e., set-shifting and inhibitory control) and assesses the genetic and environmental influences on these facets of EF in early childhood. It is important to understand the etiology of this EF earlier in development, as the factors that influence EF may change across age and studies on older children may not inform a younger population. For example, the relative importance of genetic influences on general cognitive ability has been shown to increase with age (i.e., the magnitude of heritability increases with age) and novel genetic effects emerge at later ages (Petrill et al., 2004). EF might show a similar pattern of lower heritability in early childhood and different genes may operate in early childhood. Further, despite prior evidence for genetic influences on EF in later childhood, the preschool period marks an important change in social and cognitive functioning, which may lead to differences in gene expression from those found later in development (Polderman et al., 2007).

Similarly, the impact of the environment may change across development. It is critical to gain a more comprehensive understanding of early EF, because understanding the factors that influence EF in preschool may help to identify children who are at genetic or environmental risk.
Genetic and Environmental Overlap

Further research is also required on the etiological structure of EF—that is, the extent to which different components of EF are influenced by common genetic and environmental factors. Only one study has explored the genetic and environmental overlap between the facets of EF in adolescence (Friedman et al., 2008). Assessments of the genetic and environmental influences on multiple components of EF in early childhood are lacking. This is an important question, as a more unified structure of EF in early childhood may show more genetic overlap than has been demonstrated with children at older ages when EF is more dissociable. Such empirical evidence on the genetic and environmental influences on the architecture of EF will allow for a more comprehensive understanding of the structure of EF as it develops in early childhood.

Executive Functioning and Developmental Outcomes

As indicated above, EF is intricately linked to important developmental outcomes, but little is known about the mechanisms linking EF to these outcomes. Temperament (e.g., Saudino, 2005), school readiness (Lemelin et al., 2007; Rhemtulla & Tucker-Drob, 2012), and behavior problems (e.g., Price, Simonoff, & Waldham, 2001; Saudino, Carter, Purper-Ouakil, & Gorwood, 2008) have been shown to be genetically influenced in early childhood, thus it is possible that links between EF and these outcomes are mediated genetically. There is evidence for genetic overlap between one measure of temperament, self-restraint, in toddlerhood and EF in adolescence (Friedman, Miyake, Robinson, & Hewitt, 2011), EF and reading ability (Christopher et al., 2016), and EF deficits and ADHD (Coolidge, Thede, & Young, 2000), but again, these associations have not been
assessed in early childhood. Thus, although we know that EF is associated with developmental outcomes in preschoolers, we do not know the mechanisms that underlie the associations.

Current Study

The preschool period is a time of substantial growth in EF, but relatively little is known about the etiology of EF and the links between early EF and key developmental outcomes during this period. The proposed study will address these gaps in the literature by studying the etiology of EF, conceptually-related aspects of temperament, school readiness and behavior problems in a sample of twins at age 4. This has important practical implications, as understanding the role of EF in school readiness and behavior problems will allow parents and educators to better understand the relation between these cognitive, socio-emotional, behavioral, and academic capacities and may help to identify children at risk for academic and behavioral difficulties and inform about early prevention and intervention.

Specific Aim 1: Explore genetic and environmental influences on EF at age 4

A) The genetic and environmental influences on the components of EF, such as set-shifting and inhibitory control are understudied young children. This multifaceted study of EF is the first to explore the etiology of EF using a validated computerized battery designed for use with young children. Quantitative genetic models were used to estimate genetic and environmental variance components of two facets of EF (i.e., set-shifting and inhibitory control). It was hypothesized that EF would be genetically-influenced in early childhood.
B) This study also examines whether set-shifting and inhibitory control are influenced by common genetic and environmental factors. This question is evaluated with multivariate quantitative genetic models to assess sources of covariance between domains. Due to the existing literature suggesting that EF has a unitary factor structure in early childhood (e.g., Wiebe et al., 2008), it was hypothesized that the facets of EF at this age are interrelated primarily due to genetic factors, with the remaining variance attributable to nonshared environmental influences.

**Specific Aim 2: Examine the links between EF and conceptually-related temperament dimensions**

EF and effortful control both fall under the umbrella of self-regulation, but little is known about the sources of covariance between the two. Further, there are inconsistent findings on the relation between EF and temperament in the literature. The present study utilizes multivariate quantitative genetic models to assess the genetic and environmental overlap between EF and temperament (i.e., effortful control and other measures) to examine the extent to which the same genetic and environmental factors operate across constructs. Given the conceptual overlap between EF and effortful control, it was hypothesized that there would be overlap of the genetic and environmental influences.

**Specific Aim 3: Explore the etiology of the association between EF and behavior problems and school readiness**

EF is implicated in academic failures (Blair & Razza, 2007). Further, studies with older children have shown that EF is most notably associated with externalizing behavior problems (Fahie & Symons, 2003). In addition to examining the phenotypic relations
between EF, effortful control and school readiness and EF, effortful control and behavior problems, multivariate quantitative genetic models were used to inform whether any observed associations in early childhood are due to common genetic and/or environmental influences. The present study is the first to explore the etiology of this association in preschoolers. It was predicted that EF would be phenotypically associated with school readiness and behavior problems and that these constructs are associated because of shared genetic influences.

EF deficits are considered an endophenotype for ADHD. The key criteria for establishing an endophenotype for a complex disorder are that the endophenotype: co-occurs with the disorder of interest; is a trait that can be measured reliably; should show evidence of heritability (Doyle et al., 2005). The present study addresses the extent to which EF can meet the criteria to be considered a ‘non-clinical’ endophenotype for dimensional measures of hyperactive behavior problems in early childhood.
CHAPTER TWO

Method

Sample

Two hundred nine (42 monozygotic male; 37 monozygotic female; 63 dizygotic male; 67 dizygotic female) four-year-old twin pairs participated in this study. Families were recruited from the Boston area based on birth records obtained through the Massachusetts Registry of Vital Records by the Boston University Twin Project (BUTP), a larger longitudinal study on temperament and related behaviors across the preschool period. Ethnicity of the sample was generally representative of the state of Massachusetts (89% Caucasian; 2% Black; 2% Asian; 7% Mixed). The reported ethnicity of the sample was 6% Hispanic or Latino and 94% non-Hispanic or Latino. Socioeconomic status (SES) of the sample was computed using the Hollingshead Four-Factor Index of Social Status (Hollingshead, 1975). The Hollingshead index comprises a composite of maternal and paternal education and occupation status. Both level of education and occupation status are assigned a score of 1-9 with 9 representing the highest level in both categories. The final SES index can be obtained by multiplying each parent’s education code by 3 and occupational code by 5, then summing the resultant scores and diving by 2. Using these criteria, the average level of SES for the present sample fell within the middle class range, with a mean Hollingshead index of 53.20 (SD=8.90, range=27-66).

Twins were screened to exclude any children who were not of normal birth weight (i.e., at least 1750 grams), gestational age (i.e., at least 34 weeks gestation), or who presented with possible developmental issues (e.g., chromosomal abnormalities) that
might affect their task performance. This is the standard participant screening procedure used in major developmental twin studies (e.g., MacArthur Longitudinal Twin study [Plomin et al., 1990]; Twins Early Development Study [Trouton, Spinath, & Plomin, 2002]) and ensures that the results are not skewed by data that are not representative of the greater population from which they were derived.

Zygosity was determined using DNA analyses obtained through cheek swab samples. In cases where DNA was not available ($n=3$), zygosity was determined using parent responses on physical similarity questionnaires, which have been shown to be more than 95% accurate when compared to DNA markers (Price, Freeman, Craig, Ebersole, & Plomin, 2000). In the present sample, the agreement as indicated by Cohen’s kappa between zygosity obtained through DNA analyses and parent questionnaire was .90 ($p<.01$). Moreover, 95% of twins identified via parent ratings were found to have been identified correctly when checked against DNA screening results.

**Procedure**

Twins completed one 2-hour visit at the BUTP laboratory within one month of their fourth birthday. Different testers individually assessed each member of the twin pair across four testing blocks (Table 1). Block 1 included the administration of the *National Institutes of Health Toolbox: Early Childhood Cognitive Battery* (NIH Toolbox; Zelazo, 2006) and the “*Surprise, It’s a Pop Up Snake*” exuberance episode from the *Laboratory Temperament Assessment Battery: Preschool Version* (Lab-TAB; Goldsmith, Reilly, Lemery, Longley, & Prescott, 1995). Block 2 comprised four Lab-TAB episodes (*Arc of Toys* [activity], *Imperfect Circles* [anger], *Corral of Balls* [activity], *Bead Sorting*...
[interest/persistence]), a sticker-sharing task, and measures of height and weight. Block 3 included administration of the *Bracken School Readiness Assessment-III* (Bracken, 2007), a measure of knowledge of verbs and nouns, and a parent-child interaction in which the dyad was asked to complete a difficult task. The final block contained a semi-structured parent-child free play and Lab-TAB episodes (*Stranger Approach* [fear], *Fidget Video* [activity], *Coffee Pot* [interest/persistence], and *Popping Bubbles* [exuberance]).

Each block took approximately 30 minutes to administer. Children alternated between cognitive testing in the small room and game-like activities in the playroom. One twin began in the small testing room (i.e., sequence: blocks 1, 2, 3, 4) and the other twin began in the playroom (i.e., sequence: blocks 2, 1, 4, 3). Order was counterbalanced across twins within families. All tasks were videotaped. At the conclusion of the visit, testers rated the temperament of the twins on the *Infant Behavior Record* (Bayley, 1969). Prior to visiting the lab, parents completed questionnaires about their children’s temperaments and behavior problems.

Table 1.

*BUTP protocol*

<table>
<thead>
<tr>
<th>Block 1 (small room)</th>
<th>Block 2 (playroom)</th>
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</thead>
<tbody>
<tr>
<td>NIH Toolbox</td>
<td>Arc of Toys (Activity)</td>
</tr>
<tr>
<td>Surprise! It’s a Pop Up Snake (Exuberance)</td>
<td>Imperfect Circles (Anger)</td>
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<tr>
<td></td>
<td>Corral of Balls (Activity)</td>
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<tr>
<td></td>
<td>Bead Sorting (Interest/Persistence)</td>
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<td></td>
<td>Sticker Sharing Task</td>
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<td></td>
<td>Height/Weight Measurement</td>
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<table>
<thead>
<tr>
<th>Block 3 (small room)</th>
<th>Block 4 (playroom)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bracken School Readiness Assessment</td>
<td>Stranger-Child Semi-Structured Free Play</td>
</tr>
<tr>
<td>Verbs/Nouns Assessment</td>
<td>Stranger Approach (Fear)</td>
</tr>
<tr>
<td>Parent-Child Etch-A-Sketch</td>
<td>Fidget Video (Activity)</td>
</tr>
<tr>
<td></td>
<td>Coffee Pot (Interest/Persistence)</td>
</tr>
<tr>
<td></td>
<td>Popping Bubbles (Exuberance)</td>
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</tbody>
</table>
Measures

Executive Functioning

Two measures of EF, inhibitory control and set-shifting, were assessed using the NIH Toolbox: Early Childhood Cognitive Battery (NIH Toolbox; Zelazo et al., 2013). The NIH Toolbox is a computerized battery of multidimensional measures normed for administration from ages 3-85 years. Both measures of EF on the NIH Toolbox have been shown to demonstrate excellent test-retest reliability (Zelazo et al., 2013). Further, both measures exhibit convergent validity with the WPPSI-III Block Design test, a measure of fluid cognition (Zelazo et al., 2013).

Inhibitory Control. The Flanker Inhibitory Control and Attention Test (Flanker; See Appendix A) assesses inhibitory control and attention by testing participants’ ability to attend to one visual stimulus while inhibiting attention to irrelevant stimuli flanking the target. In the traditional Flanker task, participants are asked to indicate the orientation of a central arrow while inhibiting attention to either congruent or incongruent arrows that flank it. In the NIH Toolbox version (adapted from the Attention Network Test; Rueda et al., 2004), the stimuli for ages 3-8 years are fish, which are larger and more engaging than arrows. For each trial, a fixation point was presented, followed by auditory and visual cues, ‘middle’, and the presentation of the Flanker stimuli (Figure 1). Children must attend to the target stimulus (the middle fish) and ignore the flanking fish, which are pointed either congruently or incongruently with the middle fish (Figure 2). Performance on incongruent trials provides a measure of inhibitory control in the context of visual selective attention (Zelazo, 2006). If children accurately respond on 90% or more of the
trials, they transition to the traditional Flanker task with 20 arrow trials.

NIH Toolbox-generated computed scores reflect performance on the Flanker. Computed scores are a combination of accuracy and reaction time. If accuracy levels are less than 80% the computed score is equal to the accuracy score. If accuracy levels reach or exceed 80%, then the reaction time and accuracy scores are combined to create the computed score. Reaction time scores are generated using individuals’ raw reaction time scores on correct, incongruent trials that were greater than or equal to 100ms and not more than 3 standard deviations away from the individual’s mean. The computed scores are converted to unadjusted scale scores that have a mean of 100 and a standard deviation of 15. Unadjusted scale scores compare the performance of the individual to the entire NIH Toolbox normative sample regardless of age or any other variable and are useful for assessing overall performance. Higher unadjusted scale scores are indicative of better EF. For 4-year-olds, the EF mean and standard deviation from the NIH Toolbox nationally-representative sample were 75.65 and 4.05, respectively (Slotkin et al., 2012).

*Set-Shifting*. Set-shifting, a measure of cognitive flexibility, was assessed with a modified version of the DCCS (see Appendix B). The DCCS includes four blocks:
practice, pre-switch, post-switch, and mixed. In the practice block, participants are presented with pictorial stimuli on a computer monitor and are instructed to match a centrally-located test stimulus to one of two lateralized target stimuli. Target stimuli are a brown rabbit and a white boat. Bivalent test stimuli include a white rabbit and a brown boat. Participants are required to match either by shape or color by pointing to the target stimulus that matches the test stimulus on the relevant dimension. As with in the Flanker task, following fixation and auditory and visual ‘middle’ cues (Figure 3), the test stimulus then appears on the screen and participants respond by pointing to one of two of the target stimuli, cancelling the test stimulus (Figure 4). In the practice block, children receive feedback on their responses. Children must get 3 out of 4 practice trials correct. If they fail, four practice trials are repeated up to three times. Once successfully completing 3 out of 4 of the practice trials, children proceed to practice trials for the other relevant dimension. Children who meet criterion on this dimension proceeded to test trials that are similar in structure but involve different shapes and colors (i.e., balls and trucks; yellow and blue).

Test trials begin with a pre-switch block that consists of five trials in which children sort by the last dimension used in the practice block. No feedback is provided during test trials. Children must correctly match 4 out of 5 trials to proceed to the next block, that consists of five trials in which children are instructed to sort by the other dimension. The transition between blocks is noted explicitly by instructions from the experimenter to switch (i.e., “Now we are going to play the color game. In the color game, we choose the picture on the bottom that is the same color as the picture in the
middle. If it [experimenter points to middle picture] is blue, we choose this picture [experimenter points to target stimulus], because they are both blue, they are the same color”). Children who correctly match on at least four trials in the post-switch block proceed to the mixed block. The mixed block consists of 30 trials of mixed shape and color matches.

Scoring for the DCCS task is identical to the Flanker task with the exception of the responses used to obtain reaction time. For the DCCS, reaction times are obtained using the child’s raw score from the non-dominant dimension (i.e., the dimension cued less frequently for sorting) median reaction time score. Again, higher unadjusted scale scores on the DCCS are indicative of better performance.

**Temperament**

*Parent-Rated Temperament.* Parent ratings of temperament were obtained via the *Child Behavior Questionnaire-Short Form* (CBQ-SF; Putnam & Rothbart, 2006). The CBQ-SF includes 94 questions that tap 15 subscales (Activity Level, Anger/Frustration, Approach, Attentional Focusing, Discomfort, Falling Reactivity and Soothability, Fear, High Intensity Pleasure, Impulsivity, Inhibition, Low Intensity Pleasure, Perceptual

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**Figure 3.** DCCS trial sequence

**Figure 4.** DCCS trial

Parents are asked to characterize their children’s reactions in a number of situations (e.g., “When drawing or coloring in a book, shows strong concentration”; “Can wait before entering into new activities if s/he is asked to”) on a scale from 1 (*extremely untrue of your child*) to 7 (*extremely true of your child*). Subscale scores represent the mean scores of all scale items with reverse-scored items corrected. Factor scores can range from 1-7 and are created by averaging the standard scale scores corresponding to the factors. The CBQ-SF demonstrates good internal consistency, criterion validity and exhibits reliability (Putnam & Rothbart, 2006). Reliability for parent ratings of temperament in the present sample as indicated by Cronbach’s alpha ranged from .55 for sadness to .87 for shyness.

*Observer-Rated Temperament.* At the conclusion of testing, examiners completed the *Infant Behavior Record* (IBR; Bayley, 1969). The IBR is a widely-used observer rating measure of temperament in behavior genetics research that utilizes 30 items to assess 3 broad behavioral dimensions: *Activity, Affect/Extraversion*, and *Task Orientation* (Matheny, 1983). The *Activity* factor assesses rate and vigor of movement and energy during the testing period; *Affect/Extraversion* taps social responsiveness, cooperativeness
and emotional tone in relation to the tester; and *Task Orientation* assesses attention span, persistence, goal directedness, and responsiveness to testing stimuli. Examiners rated the children’s behavior on 5- or 9-point rating scales. Factor scores were obtained by averaging the standardized items for each factor, thus possible scores range from -1 to 1.

The IBR demonstrates good reliability and validity for the age range of children in the present study (Nellis & Gridley, 1994). To evaluate inter-rater reliability for the IBR, a second rater completed IBR ratings for 33% of the children. Inter-rater reliability for *Activity, Affect/Extraversion, and Task Orientation* as indicated by intraclass correlations were .79, .76, and .81, respectively.

**Behavior Problems**

Behavior problems were assessed with parent ratings on the *Child Behavior Checklist for Ages 1½ -5* (CBCL; Achenbach & Rescorla, 2000). The CBCL includes 100 items that require parents to indicate whether specific behaviors have occurred within the last two months (e.g., “Temper tantrums or hot temper”; “Nervous, high-strung, or tense”) on a scale from 0 (*not true*) to 2 (*very true or often true*). In addition to Internalizing, Externalizing, and Total Behavior Problem scales (see Appendix C for ranges of possible scores), the CBCL includes 7 syndrome scale scores (Emotionally Reactive, Anxious/Depressed, Somatic Complaints, Withdrawn, Sleep Problems, Attention Problems, and Aggressive Behavior) and 5 DSM-oriented scales (Affective Problems, Anxiety Problems, Pervasive Developmental Problems, Attention Deficit/Hyperactivity Problems, and Oppositional Defiant Problems). The CBCL is widely-used in developmental and behavioral genetics research (Gjone & Stevenson,
1997) and is a valid, reliable assessment (Achenbach & Rescorla, 2000). Internal consistency in the present sample as demonstrated by Cronbach’s alpha ranged from .50 for somatic complaints to .92 for total behavior problems.

**School Readiness**

The *Bracken School Readiness Assessment-III* (BSRA; Panter & Bracken, 2009) provides information about children’s early academic capacities. The BSRA includes 88 items across five subtests (i.e., color, letters, number/counting, sizes/comparisons, shapes). Testers label a target item and the child selects one picture from 4 to 10 alternatives (e.g., “Look at all of the pictures, show me which animal is big”). A subtest is discontinued following 3 consecutive incorrect responses. Raw scores (i.e., total number correct) are converted into standard scores based on age (i.e., with a mean of 100 and a standard deviation of 15). The standard scores were used for all analyses. The BRSA is a good predictor of first grade readiness (Panter & Bracken, 2009) and demonstrates good reliability and validity (Bracken, 2007). To evaluate inter-rater reliability, a second rater completed BSRA ratings for 20% of the children. The intraclass correlation indicated that reliability was 99.9% \( (p<.01) \).

**Vocabulary**

To control for intelligence, the Toolbox Picture Vocabulary Test (TPVT), a subtest of the NIH Toolbox, was included as a possible covariate in the analyses. In this assessment, the computer generates a spoken cue and children must select its picture from 4 alternatives. This measure is similar to the Peabody Picture Vocabulary Test-4\textsuperscript{th} Edition (PPVT-IV), an assessment of verbal intelligence that correlates highly with full-scale
measures of intelligence (Hodapp & Gerken, 1999). The TPVT is administered in a computer-adaptive format. That is, the presentation of a question is dependent on the participant’s response on the previous question. Item response theory (IRT) is used to score the TPVT. A theta score is calculated for each participant. A theta score, like a z-score, has a mean of 0 and a SD of 1. The theta score is then converted to a computed score, with higher scores indicating better performance (Slotkin et al., 2013). The TPVT demonstrates convergent validity with the PPVT-IV (Dunn & Dunn, 2007) and high test-retest reliability (Gershon et al., 2013).

Data Analysis

The Twin Design: Overview

The twin design was used to investigate the genetic and environmental contributions to the observed variance in EF and the covariation between EF and outcome variables of interest. The twin method involves comparing monozygotic (MZ) twins, who share 100% of their genes with dizygotic (DZ) twins who share, on average, 50% of their segregating genes. Genetic influences are implied when degree of co-twin similarity on a trait covaries with genetic relatedness. Heritability \( (h^2) \) is the proportion of total variance in a phenotype that is attributable to genetic effects. Environmental factors comprise the remaining variance. Shared environments are family-wide experiences that act to make twins more similar. Nonshared environmental influences are experiences that are unique to the individual and make members of a family different.

Phenotypic Correlations

Phenotypic correlational analyses were used as a preliminary step to identify if
there were correlations between the components of EF and between EF and the outcome variables (e.g., school readiness). Where there was significant covariance to decompose (i.e., a correlation of .3 or greater), further behavioral genetic analyses were completed.

*Twin Intraclass and Cross Correlations*

Twin intraclass and cross correlations were calculated using a double entry procedure as an initial step in evaluating the genetic and environmental influences on the phenotypes. MZ correlations that exceed DZ correlations are indicative of additive genetic effects. If DZ twins are more similar than would be expected due to genetic similarity, (i.e., the DZ twin correlation is greater than one-half of the MZ twin correlation) this suggests the presence of shared environmental influences. MZ twin correlations that are less than one indicate the influences of nonshared environmental effects on a trait.

Cross-twin cross-trait correlations provide information about the covariance between measures. In these analyses, Twin 1’s score on a phenotype (e.g., inhibitory control) is correlated with Twin 2’s score on a different construct (e.g., school readiness) and vice versa. As with intraclass correlations, genetic contributions to the covariance between measures is suggested when the MZ cross-twin cross-trait correlation is greater than the DZ cross-twin cross-trait correlation.

*Model-Fitting Analyses*

Multivariate model-fitting analyses provided estimates of genetic and environmental effects on the domains. All models were fit to raw data using maximum-likelihood model-fitting procedures implemented in Mx structural equation modeling
software (Neale, Boker, Xie, & Maes, 2006). Goodness of model fit was assessed using likelihood-ratio chi-square ($\chi^2$) tests, calculated as the difference between the -2 log likelihood (-2LL) of the full model and that of a saturated model (i.e., a model in which the variance–covariance structure is not estimated and all variances and covariances for MZ and DZ twins are estimated). The difference in -2LL is asymptotically distributed as $\chi^2$ with degrees of freedom ($df$), which reflects the difference in the amount of estimated parameters between the full model and a saturated model. A series of sub-models were fit to the data to test the significance of parameters. The relative fit of the sub-models models were evaluated by the chi-square difference ($\Delta\chi^2$) between the full model and the sub-model and corresponding change in degrees of freedom ($\Delta df$). A nonsignificant change in $\chi^2$ between the full and reduced model indicates that the nonsignificant parameters can be dropped from the model without a significant decrement in overall model fit. Akaike’s Information Criterion (AIC; $AIC = \chi^2 - 2*\Delta df$) values were also computed, with lower AIC values indicating better fit of the model to the observed data.

**Univariate Models.** Univariate models (Figure 5) were used to evaluate the genetic and environmental influences on each facet of EF (e.g., inhibitory control) separately (Aim 1a). According to the univariate model, phenotypic variation (represented by rectangles) is due to three latent variables: additive genetic effects (A), shared environmental effects (C), and nonshared environmental effects (E). Curved, double-headed arrows indicate correlations between the variables that they connect. Because MZ twins share 100% of their genes and DZ twins share approximately 50% of their genes, the A factors correlate 1.0 for MZ twins and .5 for DZ twins. All twins in the
study were reared in the same home, so both MZ and DZ twins correlate 1.0 for shared environment. E influences are uncorrelated and depicted as residual variances for each twin. Using this model, estimates of heritability and shared and non-shared environmental variances and their 95% confidence intervals were estimated for each facet of EF.

![Univariate twin model](image)

Figure 5. Univariate twin model

**Multivariate Models.** Cholesky decomposition models were fit to the data to examine the genetic and environmental sources of covariance between the two facets of EF (Aim 1b) and between EF and the outcome measures (Aims 2-3). Multivariate models allow for examination of the degree of overlap of the genetic and environmental influences on the phenotypes of interest. For example, to explore the links between the two facets of EF, a bivariate Cholesky decomposition was fit to the data. A Cholesky decomposition (Figure 6, shown only for one twin) comprises two latent variables for each source of variance (i.e., genetic and environmental effects). The first factors (A₁, C₁, and E₁) load on both set-shifting and inhibitory control and the second factors (A₂, C₂, and E₂) load only on inhibitory control. All paths are standardized partial regressions
indicating the relative influence of the latent variables on the phenotypes. Paths $a_{11}, c_{11}, e_{11}$ estimate the genetic and environmental influences on set-shifting. Paths $a_{21}, c_{21},$ and $e_{21}$ estimate covariance (i.e., common effects) between the measures. Therefore, the variation in inhibitory control is due to both the genetic and environmental influences that overlap with set-shifting (i.e., paths $a_{21}, c_{21},$ and $e_{21}$) and the effects that are unique to inhibitory control (i.e., paths $a_{22}, c_{22}, e_{22}$). The squares of the path coefficients leading to a phenotype estimate the genetic and environmental variances for that phenotype. For example, the heritability for inhibitory control can be obtained by squaring paths $a_{21}, a_{22}$, summing the resultant values and multiplying by 100. Further, the percentage of total genetic variance attributed to one latent phenotype can be obtained by dividing the square of that path estimate by the total variance for that source of variance and multiplying by 100. As with the univariate models, sub-models were fit to determine the significance of parameters. These models will be described further in the methods section.

Figure 6. Bivariate Cholesky decomposition model
Data Transformations

Both set-shifting and inhibitory control were rank transformed to correct for negative skew. Hyperactive behavior problems and externalizing behavior problems were log-transformed to correct for positive skew. Further, because twin covariances can be inflated by variance due to sex, all scores were residualized for sex effects (McGue & Bouchard, 1984).
CHAPTER 3

Results

Descriptive Statistics

Table 2 lists the means and standard deviations for set-shifting, inhibitory control, effortful control, task orientation, school readiness, hyperactivity, and externalizing behavior problems by zygosity and sex. To account for the dependence of the data due to the fact that the sample comprised pairs of twins, the main effects of sex, zygosity, and the sex x zygosity interactions for all variables were estimated using generalized estimating equations (GEE) implemented with the SAS GENMOD procedure. GEE are extensions of the standard generalized linear models that allow for modeling of correlated data (Zeger & Liang, 1986). As can be seen in Table 2, there were no main effects for sex, zygosity, or sex x zygosity interactions for any of the variables.

Specific Aim 1: Explore genetic and environmental influences on EF at age 4

Aim 1a: Univariate Model-Fitting

Intraclass Correlations. Twin intraclass correlations (Table 3) were computed using the double-entry procedure in SAS. For both set-shifting and inhibitory control, the MZ twin correlations were significantly greater than the DZ correlations, suggesting the presence of genetic effects.

Model-Fitting Results. Univariate quantitative genetic models were used to obtain estimates of genetic, shared environmental, and nonshared environmental variances. Models were fit to the raw data using Mx. For each phenotype, in addition to the full ACE model, three reduced models were fit to the data: (1) a model that included only
Table 2.

*Means (standard deviations) by zygosity and sex.*

<table>
<thead>
<tr>
<th>Measures</th>
<th>MZ</th>
<th>DZ</th>
<th>Sex F (df)</th>
<th>Zygosity F (df)</th>
<th>Sex x Zygosity F (df)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
<td>Males</td>
<td>Females</td>
<td>-03</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(202)</td>
</tr>
<tr>
<td>Set-Shifting n pairs</td>
<td>70.84 (19.10)</td>
<td>76.14 (6.98)</td>
<td>73.87 (15.72)</td>
<td>76.12 (15.04)</td>
<td>- .03</td>
</tr>
<tr>
<td></td>
<td>42</td>
<td>37</td>
<td>62</td>
<td>64</td>
<td>(202)</td>
</tr>
<tr>
<td>Inhibitory Control n pairs</td>
<td>69.69 (18.43)</td>
<td>70.84 (18.09)</td>
<td>70.69 (18.07)</td>
<td>69.51 (20.97)</td>
<td>.36</td>
</tr>
<tr>
<td></td>
<td>42</td>
<td>37</td>
<td>62</td>
<td>64</td>
<td>(196)</td>
</tr>
<tr>
<td>Effortful Control n pairs</td>
<td>5.10 (.46)</td>
<td>5.49 (.46)</td>
<td>5.22 (.71)</td>
<td>5.50 (.46)</td>
<td>.01</td>
</tr>
<tr>
<td></td>
<td>33</td>
<td>33</td>
<td>56</td>
<td>61</td>
<td>(173)</td>
</tr>
<tr>
<td>Task Orientation n pairs</td>
<td>-.23 (.89)</td>
<td>.15 (.77)</td>
<td>-.07 (.95)</td>
<td>.12 (.79)</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>37</td>
<td>33</td>
<td>56</td>
<td>59</td>
<td>(179)</td>
</tr>
<tr>
<td>School Readiness n pairs</td>
<td>107.86 (17.58)</td>
<td>107.72 (13.00)</td>
<td>108.10 (14.11)</td>
<td>110.55 (13.74)</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>42</td>
<td>37</td>
<td>61</td>
<td>64</td>
<td>(199)</td>
</tr>
<tr>
<td>Hyperactivity n pairs</td>
<td>3.35 (2.56)</td>
<td>3.41 (2.68)</td>
<td>3.42 (2.56)</td>
<td>3.14 (2.44)</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>39</td>
<td>63</td>
<td>68</td>
<td>(203)</td>
</tr>
<tr>
<td>Externalizing Behavior Problems n pairs</td>
<td>8.92 (6.58)</td>
<td>9.14 (8.15)</td>
<td>8.84 (6.78)</td>
<td>8.88 (7.05)</td>
<td>.04</td>
</tr>
<tr>
<td></td>
<td>44</td>
<td>39</td>
<td>63</td>
<td>68</td>
<td>(203)</td>
</tr>
</tbody>
</table>
Table 3.

*Twin intraclass and cross-twin cross-trait correlations.*

<table>
<thead>
<tr>
<th></th>
<th>Set-Shifting</th>
<th>Inhibitory Control</th>
<th>Task Orientation</th>
<th>Effortful Control</th>
<th>School Readiness</th>
<th>Hyperactivity</th>
<th>Externalizing Problems</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MZ</strong></td>
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<td></td>
<td></td>
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<tr>
<td>Set-Shifting</td>
<td><strong>.40</strong>*</td>
<td></td>
<td>.40***</td>
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<td></td>
</tr>
<tr>
<td>Inhibitory Control</td>
<td><strong>.40</strong>*</td>
<td><strong>.48</strong>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task Orientation</td>
<td>.29*</td>
<td>.26*</td>
<td>.25*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effortful Control</td>
<td>.32**</td>
<td>.10</td>
<td>.27*</td>
<td><strong>.49</strong>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>School Readiness</td>
<td>.29**</td>
<td>.33**</td>
<td>.18</td>
<td>.17</td>
<td><strong>.85</strong>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperactivity</td>
<td>-.20</td>
<td>-.02</td>
<td>-.16</td>
<td>-.27*</td>
<td>-.14</td>
<td><strong>.67</strong>*</td>
<td></td>
</tr>
<tr>
<td>Externalizing Problems</td>
<td>-.18</td>
<td>-.04</td>
<td>-.13</td>
<td>-.28*</td>
<td>-.15</td>
<td><strong>.66</strong>*</td>
<td><strong>.83</strong>*</td>
</tr>
<tr>
<td><strong>DZ</strong></td>
<td><strong>.08</strong></td>
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</tr>
<tr>
<td>Set-Shifting</td>
<td>.14</td>
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<td></td>
</tr>
<tr>
<td>Inhibitory Control</td>
<td>.14</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Task Orientation</td>
<td>.22*</td>
<td>.05</td>
<td><strong>.14</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Effortful Control</td>
<td>-.07</td>
<td>.04</td>
<td>.03</td>
<td><strong>.33</strong>*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>School Readiness</td>
<td>.17</td>
<td>.09</td>
<td>.15</td>
<td>-.03</td>
<td><strong>.47</strong>*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperactivity</td>
<td>.02</td>
<td>-.08</td>
<td>.00</td>
<td>-.12</td>
<td>-.02</td>
<td><strong>.45</strong>*</td>
<td></td>
</tr>
<tr>
<td>Externalizing Problems</td>
<td>.01</td>
<td>-.04</td>
<td>.01</td>
<td>-.20*</td>
<td>.00</td>
<td><strong>.47</strong>*</td>
<td><strong>.56</strong>*</td>
</tr>
</tbody>
</table>

*p<.05, **p<.01, ***p<.001; Intraclass correlations are bolded.*
shared environmental and nonshared environmental effects (i.e., CE model); (2) a model that included only genetic effects and nonshared environmental effects (i.e., AE model); (3) a model that included only nonshared environmental effects (i.e., E model).

**Set-Shifting**

Table 4 presents the parameter estimates and fit statistics for the full ACE model and reduced models for set-shifting. It was not possible to drop genetic effects (i.e., CE model) without a significant decrement in overall model fit, indicating that additive genetic influences contribute to individual differences in set-shifting. The shared environmental effects (i.e., AE model) were estimated at 0 and dropping them did not result in a significant impact on model fit. Not surprisingly, the nonshared environmental influences only model provided the worst fit to the data. This makes sense, because a model that includes only nonshared environmental influences implies that there is no relation between twins, and as indicated in by the twin intraclass correlations, this is not the case in the present sample.

When evaluating model fit based on Akaike’s Information Criterion (AIC; $AIC = \chi^2 - 2*\Delta df$), the model with the lowest AIC is the most parsimonious model. The AE model had the lowest AIC (i.e., AE model $AIC = 6.99 - [2*4] = -1.01$), thus, a model that accounts for genetic and nonshared environmental effects best explains individual differences in set-shifting in the present sample. Indeed, set-shifting was moderately heritable ($h^2 = 36\% \ [17\% - 52\%]$), with the majority of the variance attributable to nonshared environmental influences ($e^2 = 64\% \ [48\% - 83\%]$).
Table 4.

Univariate model-fitting results for set-shifting.

<table>
<thead>
<tr>
<th>Model</th>
<th>Parameter estimates</th>
<th>Fit of model compared to saturated model$^a$</th>
<th>Difference in fit of models$^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A (95% CI)</td>
<td>C (95% CI)</td>
<td>E (95% CI)</td>
</tr>
<tr>
<td>Saturated Model: -2LL=1132.36, df=400</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>$\text{.36}$ (.06-.52)</td>
<td>$\text{.00}$ (.00-.19)</td>
<td>$\text{.64}$ (.48-.83)</td>
</tr>
<tr>
<td>Drop A (CE)</td>
<td>$\text{.20}$ (.06-.32)</td>
<td>$\text{.80}$ (.68-.94)</td>
<td></td>
</tr>
<tr>
<td>**Drop C (AE)</td>
<td><strong>$\text{.36}$ (.17-.52)</strong></td>
<td><strong>$\text{.64}$ (.48-.83)</strong></td>
<td></td>
</tr>
<tr>
<td>Drop AC (E)</td>
<td>1.0 (1.0-1.0)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note. A= additive genetic parameter; C= shared environmental parameter; E= nonshared environmental parameter; -2LL= log likelihood statistic; df= degrees of freedom; $\chi^2= $ chi-square fit statistic; AIC= Akaike’s information criterion.

$^a$ Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$ Relative fit of the model determined by the $\chi^2$ difference ($\Delta \chi^2$) between the full model and reduced models.
**Inhibitory Control**

A similar pattern emerged for inhibitory control. Table 5 presents the parameter estimates and model-fit statistics for the full ACE model and reduced models. The genetic effects (i.e., CE model) could not be dropped without a significant decrement in overall model fit. The shared environmental influences (i.e., AE model) were estimated at 0 and could be dropped. Additionally, the AE model had the lowest AIC (i.e., AE model \( AIC = 5.5 - [2 \times 4] = -7.45 \)). Again, the nonshared environmental influences only model provided the worst fit to the data. Therefore, a model that accounts for genetic and nonshared environmental effects provided the best fit. Approximately half of the variance in inhibitory control was due to genetic effects (\( h^2 = 46\% \) [30\%- 59\%]), with the other half attributable to nonshared environmental influences (\( e^2 = 54\% \) [41\%- 70\%]).

**Aim 1b: Bivariate Model Fitting**

**Phenotypic Correlational Analysis.** As a first step in evaluating the relation between set-shifting and inhibitory control, the phenotypic correlation between the two was examined. There was a significant positive correlation between the phenotypes (\( r = 0.30, p < 0.001 \)), indicating that there was sufficient variance to decompose with multivariate behavioral genetic analyses. Because the univariate model fitting analyses revealed that both set-shifting and inhibitory control are influenced by genetic effects, this begs the question of whether any of the same genetic influences are shared between the two (i.e., whether there is genetic covariance between set-shifting and inhibitory control).
Table 5.

*Univariate model-fitting results for inhibitory control.*

<table>
<thead>
<tr>
<th>Parameter estimates</th>
<th>Fit of model compared to saturated model$^a$</th>
<th>Difference in fit of models$^b$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Model</td>
<td>-2LL</td>
<td>df</td>
</tr>
<tr>
<td>Saturated Model: -2LL=1127.85, df=400</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACE</td>
<td>.46 (0.05-0.59)</td>
<td>.00 (0.00-0.31)</td>
</tr>
<tr>
<td>Drop A (CE)</td>
<td>.32 (0.19-0.43)</td>
<td>.68 (0.57-0.81)</td>
</tr>
<tr>
<td>Drop C (AE)</td>
<td>.46 (0.30-0.59)</td>
<td>.54 (0.41-0.70)</td>
</tr>
<tr>
<td>Drop AC (E)</td>
<td>1.0 (1.0-1.0)</td>
<td></td>
</tr>
</tbody>
</table>

Note. $A$= additive genetic parameter; $C$= shared environmental parameter; $E$= nonshared environmental parameter; -2LL= log likelihood statistic; $df$= degrees of freedom; $\chi^2$= chi-square fit statistic; AIC= Akaike’s information criterion.

$^a$Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$Relative fit of the model determined by the $\chi^2$ difference ($\Delta\chi^2$) between the full model and reduced models.
**Twin Cross-Twin Cross-Trait Correlations.** Cross-twin cross-trait correlations assess the relation between one twin’s performance on one measure with his or her co-twin’s performance on another, and were used to detect the presence of genetic and environmental covariance between variables. As with twin intraclass correlations, MZ cross-twin cross-trait correlations that exceed DZ twin cross-twin cross-trait correlations suggest the presence of genetic influences that contribute to the observed relation between two variables. As indicated in Table 3, the pattern of cross-twin cross-trait correlations between set-shifting and inhibitory control suggested the presence of genetic covariance, with the MZ cross-correlation exceeding the DZ cross-correlation.

**Multivariate Model-Fitting Results.** To decompose the relative contributions of common genetic and environmental factors to the observed covariance between set-shifting and inhibitory control, a bivariate Cholesky decomposition model was fit to the raw data in Mx. As previously noted, the bivariate Cholesky decomposition model examines the degree of overlap for genetic and environmental influences on set-shifting and inhibitory control. When inhibitory control is entered last in the model, the model also provides information about the extent to which genetic influences on inhibitory control operate independently from those contributing to the observed variation in set-shifting.

The fit statistics from the bivariate Cholesky decomposition model are presented in Table 6. Because the univariate model fitting procedures for set-shifting and inhibitory control revealed that, in both cases, the shared environmental parameters were estimated at 0 and could be dropped without a significant decrement in overall model fit, the base
Table 6.

**Bivariate Cholesky decomposition model-fitting results for set-shifting and inhibitory control.**

<table>
<thead>
<tr>
<th>Model</th>
<th>-2LL</th>
<th>df</th>
<th>$\chi^2$</th>
<th>$\Delta df$</th>
<th>p</th>
<th>AIC</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated</td>
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<td>792</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base$^c$</td>
<td>2222.41</td>
<td>806</td>
<td>10.97</td>
<td>14</td>
<td>.69</td>
<td>-17.03</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drop A Cov</td>
<td>2250.54</td>
<td>807</td>
<td>39.10</td>
<td>15</td>
<td>&lt;.001</td>
<td>9.10</td>
<td>28.13</td>
<td>1</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Drop E Cov</td>
<td><strong>2223.16</strong></td>
<td><strong>807</strong></td>
<td><strong>11.72</strong></td>
<td><strong>15</strong></td>
<td><strong>.70</strong></td>
<td><strong>-18.28</strong></td>
<td><strong>.75</strong></td>
<td>1</td>
<td><strong>.37</strong></td>
</tr>
</tbody>
</table>

*Note.* -2LL = log likelihood statistic; df = degrees of freedom; $\chi^2$ = chi-square fit statistic; AIC = Akaike’s information criterion; A = additive genetic influences; C = shared environmental influences; E = nonshared environmental influences.

$^a$ Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$ Relative fit of the model determined by the $\chi^2$ difference ($\Delta \chi^2$) between the full model and reduced models.

$^c$ A and E on both variables.
model used for the bivariate model fitting of set-shifting and inhibitory control included only additive genetic and nonshared environmental effects (i.e., an AE model). That is, because the shared environmental parameters did not contribute to the variance within each phenotype, it is impossible for shared environmental effects to contribute to the covariance between them. Therefore, two reduced models were fit to examine the significance of the genetic and nonshared environmental covariances, respectively. Each reduced model was compared to the base model to evaluate the significance of the dropped parameters. As seen in Table 6, the genetic covariance could not be dropped without a significant decrement in overall model fit (i.e., Drop A Cov model), but the nonshared environmental covariance (i.e., Drop E Cov model) could be dropped without a significant decrement in overall model fit. Therefore, the best-fitting model includes genetic effects common to both set-shifting and inhibitory control and variable-specific nonshared environmental influences.

The path diagram of the best-fitting model including the parameter estimates and their 95% confidence intervals is presented in Figure 7. An additive genetic factor (i.e., \( A_1 \)) common to both set-shifting and inhibitory control accounted for all of the overlap between the two phenotypes. The genetic factor unique to inhibitory control (i.e., \( A_2 \)) was not significant in the model, but was retained because these effects were nontrivial (i.e., accounted for 28% \([.35^2=.1225/.4361=.2809\times100=28.09\%]\) of the genetic variance in inhibitory control).

The genetic correlation \( (r_g) \) indicates the extent to which the genetic effects on set-shifting correlate with the genetic effects on inhibitory control independent of the
heritability of each. The genetic correlation between set-shifting and inhibitory control was high ($r_g = 0.85 \,[0.59-1.0]$) suggesting that many of the same genetic effects operate across both phenotypes.

![Diagram](image)

**Figure 7.** Best-fitting bivariate Cholesky decomposition model for set-shifting and inhibitory control. Dashed paths are nonsignificant. A= genetic factors, C= shared environmental factors, E=nonshared environmental factors.

**Specific Aim 2: Examine the links between EF and conceptually-related temperament dimensions**

**Phenotypic Correlational Analyses**

Set-shifting and inhibitory control were modestly associated with the parent-rated temperament dimension of effortful control ($r=.16, p<.05; r=.17, p<.05$, respectively). Although significant, with our sample there was not sufficient variance to decompose (i.e., a correlation in the range of .3), and no further behavioral genetic analyses were
pursued. In contrast, both set-shifting and inhibitory control were positively associated with the observer-rated temperament dimension of task orientation ($r=.30, p<.001; r=.31, p<.001$, respectively), and multivariate behavioral genetic analyses were undertaken to examine the extent to which the genetic and environmental influences on EF overlap with task orientation.

*Twin Intraclass and Cross-Twin Cross-Trait Correlations*

The MZ twin intraclass correlation for task orientation exceeded the DZ correlation, suggesting genetic influences on individual differences in task orientation (see Table 3). Similarly, the MZ cross-twin cross-trait correlations between set-shifting and task-orientation and inhibitory control and task orientation exceeded the DZ correlations, again, suggesting that genetic influences contribute to the covariation between EF and task orientation.

To decompose the relative contributions of common genetic and environmental factors to the observed covariance between set-shifting, inhibitory control and task orientation, a trivariate Cholesky decomposition model was fit to the raw data. As was the case with the bivariate model fitting of set-shifting and inhibitory control, the base model included all genetic and nonshared environmental parameters on all three variables and shared environmental influences on task orientation only.

Model-fit statistics are presented in Table 7. It was not possible to drop all genetic covariance (i.e., Drop A Cov model) from the base model without a significant decrement in overall fit. However, all nonshared environmental covariances (i.e., Drop E Cov model) could be dropped. Additionally, the genetic and shared environmental influences
Table 7.

*Trivariate Cholesky decomposition model-fitting results for set-shifting, inhibitory control, and task orientation.*

<table>
<thead>
<tr>
<th>Model</th>
<th>-2LL</th>
<th>df</th>
<th>$\chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
<th>AIC</th>
<th>$\Delta \chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturated</td>
<td>3073.99</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Base$^c$</td>
<td>3098.57</td>
<td>1166</td>
<td>24.58</td>
<td>29</td>
<td>.70</td>
<td>33.42</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drop A Cov</td>
<td>3137.54</td>
<td>1169</td>
<td>63.55</td>
<td>32</td>
<td>&lt;.001</td>
<td>.45</td>
<td>38.97</td>
<td>3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Drop E Cov</td>
<td>3101.85</td>
<td>1169</td>
<td>27.86</td>
<td>32</td>
<td>.68</td>
<td>36.14</td>
<td>3.28</td>
<td>3</td>
<td>.35</td>
</tr>
<tr>
<td>Drop E Cov, 0$^d$</td>
<td><strong>3102.02</strong></td>
<td>1172</td>
<td><strong>28.03</strong></td>
<td><strong>35</strong></td>
<td><strong>.79</strong></td>
<td><strong>-41.97</strong></td>
<td><strong>3.45</strong></td>
<td><strong>6</strong></td>
<td><strong>.75</strong></td>
</tr>
</tbody>
</table>

*Note.* -2LL = log likelihood statistic; $df$ = degrees of freedom; $\chi^2$ = chi-square fit statistic; AIC = Akaike’s information criterion; A = additive genetic influences; E = nonshared environmental influences.

$^a$ Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$ Relative fit of the model determined by the $\chi^2$ difference ($\Delta \chi^2$) between the full model and reduced models.

$^c$ A and E on all variables, C on task orientation.

$^d$ Drop all E covariance, A and C unique to task orientation.
unique to task orientation were estimated at or near 0 (i.e., less than .1 and accounting for less than 1% of the variance) and dropping them did not significantly impact model fit (i.e., Drop E Cov, 0 paths model). Therefore, a model that accounts for genetic covariance and variable-specific nonshared environmental influences best explained the relation between set-shifting, inhibitory control, and task orientation.

Figure 8 presents the best-fitting model for EF and task orientation. Individual differences in task orientation were a function of genetic factors ($h^2$=26% [15%-38%]), but the majority of the variance was due to nonshared environmental effects ($e^2$=74% [6%-85%]). A common genetic factor (i.e., A1) loaded on all three variables. All of the genetic variance in task orientation (i.e., .51$^2$=.2601/.260=1x100 =100%) was shared with set-shifting and inhibitory control. Variable-specific nonshared environmental influences emerged for each phenotype. The genetic correlations between set-shifting and task orientation ($r_g$=1.0 [1.0-1.0]) and inhibitory control and task orientation ($r_g$=.81 [.62-1.0]) were high, suggesting that almost all of the same genetic effects operate across EF and task orientation.
Figure 8. Best-fitting trivariate Cholesky decomposition model for set-shifting, inhibitory control, and task orientation. Dashed paths are nonsignificant. $A =$ genetic factors, $C =$ shared environmental factors, $E =$ nonshared environmental factors.
Specific Aim 3: Explore the etiology of the association between EF and developmental outcomes

Phenotypic Correlational Analyses

Table 8 presents the phenotypic correlations between set-shifting, inhibitory control, effortful control and school readiness, hyperactivity, externalizing behavior problems, and internalizing behavior problems. Children with better EF scored higher on the measure of school readiness. Effortful control showed a similar but more modest relation with school readiness. Neither facet of EF was associated with behavior problems. In contrast, parent-rated effortful control was inversely correlated with hyperactivity and externalizing behavior problems. That is, children who had better effortful control had fewer behavior problems. Two separate trivariate Cholesky decomposition models were fit to explore the sources of genetic and environmental covariance between i) set-shifting, inhibitory control and school readiness; and ii) effortful control, hyperactivity, and externalizing behavior problems.

<table>
<thead>
<tr>
<th></th>
<th>Set-Shifting</th>
<th>Inhibitory Control</th>
<th>Effortful Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>School Readiness</td>
<td>.33***</td>
<td>.34***</td>
<td>.16*</td>
</tr>
<tr>
<td>Hyperactivity</td>
<td>-.09</td>
<td>-.16</td>
<td>-.46***</td>
</tr>
<tr>
<td>Externalizing Problems</td>
<td>-.10</td>
<td>-.14</td>
<td>-.41***</td>
</tr>
<tr>
<td>Internalizing Problems</td>
<td>-.07</td>
<td>-.04</td>
<td>-.01</td>
</tr>
</tbody>
</table>

*p<.05, **p<.01, ***p<.001
Twin Intraclass and Cross-Twin Cross-Trait Correlations.

As indicated in Table 3, in all instances, the MZ twin intraclass and cross-twin cross-trait correlations exceeded the DZ twin correlations, suggesting the presence of genetic effects on effortful control, school readiness, hyperactivity, and externalizing behavior problems. For hyperactivity and externalizing behavior problems, the DZ twin intraclass and cross-twin cross-trait correlations were greater than half of the MZ twin intraclass correlations, suggesting that shared environmental influences contribute to individual differences in these phenotypes and the covariation between them.

Set-Shifting, Inhibitory Control, and School Readiness

Multivariate Model-Fitting Results. Model-fitting results for set-shifting, inhibitory control, and school readiness are presented in Table 9. The base trivariate Cholesky decomposition model included all genetic and nonshared environmental effects on all variables and allowed shared environmental influences on school readiness. It was not possible to drop all genetic covariance (i.e., Drop A Cov model) without a significant decrement in overall model fit, but all nonshared environmental covariance (i.e., Drop E Cov model) could be dropped. The genetic and shared environmental influences unique to school readiness were estimated at or near 0, and once again, dropping them did not significantly impact model fit. Therefore, a model that accounts for genetic covariance between set-shifting and school readiness and inhibitory control and school readiness and variable-specific nonshared environmental influences best explained the relation between set-shifting, inhibitory control, and school readiness (see Figure 9).
Table 9.

Trivariate Cholesky decomposition model-fitting results for set-shifting, inhibitory control, and school readiness.

<table>
<thead>
<tr>
<th>Model</th>
<th>-2LL</th>
<th>df</th>
<th>$\chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
<th>AIC</th>
<th>$\Delta\chi^2$</th>
<th>$\Delta df$</th>
<th>$p$</th>
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<td></td>
<td></td>
</tr>
<tr>
<td>Base$^c$</td>
<td>5376.13</td>
<td>1204</td>
<td>26.53</td>
<td>29</td>
<td>.65</td>
<td>-33.47</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drop A Cov</td>
<td>5418.78</td>
<td>1207</td>
<td>69.18</td>
<td>32</td>
<td>&lt;.001</td>
<td>5.18</td>
<td>42.65</td>
<td>3</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Drop E Cov</td>
<td>5385.63</td>
<td>1207</td>
<td>36.03</td>
<td>32</td>
<td>.29</td>
<td>-27.97</td>
<td>9.50</td>
<td>3</td>
<td>&lt;.05</td>
</tr>
<tr>
<td>Drop E Cov, 0 paths$^d$</td>
<td>5385.63</td>
<td>1209</td>
<td>36.03</td>
<td>34</td>
<td>.37</td>
<td>-31.97</td>
<td>9.50</td>
<td>5</td>
<td>.09</td>
</tr>
</tbody>
</table>

Note. -2LL= log likelihood statistic; df= degrees of freedom; $\chi^2$ = chi-square fit statistic; AIC= Akaike’s information criterion; A= additive genetic influences; E= nonshared environmental influences.

$^a$ Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$ Relative fit of the model determined by the $\chi^2$ difference ($\Delta\chi^2$) between the full model and reduced models.

$^c$ A and E on all variables, C on school readiness.

$^d$ Drop all E covariance, A and C unique to school readiness.
Figure 9. Best-fitting trivariate Cholesky decomposition model for set-shifting, inhibitory control, and school readiness. Dashed paths are nonsignificant. $A =$ genetic factors, $C =$ shared environmental factors, $E =$ nonshared environmental factors.
School readiness was highly heritable ($h^2=82\% \ [77\%-88\%]$) with the remainder of the variance attributable to nonshared environmental effects ($e^2=17\% \ [13\%-24\%]$). A common genetic factor (i.e., $A_1$) linked EF and school readiness. Approximately half of the genetic variance in school readiness was shared with set-shifting and inhibitory control (i.e., $A_1^2=.3721/ .8210=.4532\times100=45.32\%$). The genetic covariance path between inhibitory control and school readiness was significant, but because the path leading from that latent factor (i.e., $A_2$) to inhibitory control was nonsignificant, this suggests that these genetic effects are largely specific to school readiness. These influences accounted for approximately half of the genetic variance in school readiness (i.e., $A_1^2=.4489/ .8210=.5468\times100=54.68\%$). Nonshared environmental influences were unique to each phenotype. The genetic correlations between set-shifting and school readiness (i.e., $r_g=.67 \ [.47-.85]$) and inhibitory control and school readiness (i.e., $r_g=.52 \ [.37-.69]$) were moderate, suggesting that although some of the genetic effects on EF and school readiness are common, there are also effects that are unique to each.

*Hyperactivity, Externalizing Behavior Problems, and Effortful Control*

*Multivariate Model Fitting Results.* To inform what parameters should be included in the base trivariate Cholesky decomposition model between hyperactivity, externalizing behavior problems, and effortful control, univariate models were first fit to explore the genetic and environmental influences on each phenotype individually. Hyperactivity was influenced by genetic, shared environmental, and nonshared environmental effects (i.e., $A=48\% \ [16\%-76\%]$, $C=20\% \ [0\%-46\%]$, and $E=32\% \ [23\%-44\%]$). Genetic, shared environmental, and nonshared environmental effects also
emerged for externalizing behavior problems (i.e., \( A=53\% \) [31\%-79\%], \( C=30\% \) [4\%-50\%], and \( E=17\% \) [12\%-25\%]). Effortful control was influenced by genetic and nonshared environmental effects (i.e., \( A=66\% \) [28\%-76\%] and \( E=34\% \) [23\%-52\%], respectively). Shared environmental influences on effortful control were estimated at 0 and were therefore not included in the multivariate base model.

Model-fitting results are presented in Table 10. It was not possible to drop all genetic covariance (i.e., Drop A Cov model), shared environmental covariance (i.e., Drop C Cov model) or all nonshared environmental covariance (i.e., Drop E Cov model) without a significant decrement in overall model fit. The nonshared environmental covariance path from externalizing behavior problems to effortful control was estimated near 0, and could be dropped (i.e., Drop 0 path model). Therefore, a model that accounts for genetic, shared environmental, and nonshared environmental covariance between hyperactivity and externalizing behavior problems and genetic and nonshared environmental overlap between behavior problems and effortful control best fit the data.

As can be seen in Figure 10, common genetic (i.e., \( A_1 \)) and nonshared environmental (i.e., \( E_1 \)) factors linked hyperactivity, externalizing behavior problems, and effortful control. Although nonsignificant, there was also modest genetic overlap between externalizing behavior problems and effortful control that was unique from hyperactivity. Fifty-percent (i.e., \( .57^2=.3249/.6499=.4999 \times 100=49.99\% \)) of the genetic variance for effortful control was common with hyperactivity and externalizing behavior problems, 7\% (i.e., \( .21^2=.0441/.6499=.0679 \times 100=6.79\% \)) was shared with externalizing behavior problems above and beyond that common to all three, and 43\% (i.e., \( .53^2=.2809/.6499= "))
Table 10.

**Trivariate Cholesky decomposition model-fitting results for hyperactivity, externalizing behavior problems, and effortful control.**

<table>
<thead>
<tr>
<th>Model</th>
<th>Fit of model compared to saturated model</th>
<th>Difference in fit of models</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-2LL</td>
<td>df</td>
</tr>
<tr>
<td>Saturated</td>
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<td>1149</td>
</tr>
<tr>
<td>Base$^c$</td>
<td>416.21</td>
<td>1176</td>
</tr>
<tr>
<td>Drop A Cov</td>
<td>449.14</td>
<td>1179</td>
</tr>
<tr>
<td>Drop C Cov</td>
<td>424.34</td>
<td>1177</td>
</tr>
<tr>
<td>Drop E Cov</td>
<td>500.15</td>
<td>1177</td>
</tr>
<tr>
<td>Drop 0 path$^d$</td>
<td>416.22</td>
<td>1177</td>
</tr>
</tbody>
</table>

*Note.* -2LL = log likelihood statistic; df = degrees of freedom; $\chi^2$ = chi-square fit statistic; AIC = Akaike’s information criterion; A = additive genetic influences; E = nonshared environmental influences.

$^a$Overall fit of the model determined by the difference in -2LL of the model and that of a saturated model.

$^b$Relative fit of the model determined by the $\chi^2$ difference ($\Delta\chi^2$) between the full model and reduced models.

$^c$A, C, and E on hyperactivity and externalizing behavior problems, A and E on effortful control.

$^d$Drop E covariance between externalizing and effortful control.
Figure 10. Best-fitting trivariate Cholesky decomposition model for hyperactivity, externalizing behavior problems, and effortful control. Dashed paths are nonsignificant. A= genetic factors, C= shared environmental factors, E= nonshared environmental factors.
.4322\times 100=43.22\% ) was unique to effortful control. Of the total nonshared environmental variance in effortful control, 22\% (i.e., .28^2=.0784/.3488=.2248\times 100=22.48\% ) was shared with hyperactivity and externalizing behavior problems and 78\% (i.e., .52^2=.2704/.3488=.7752\times 100=77.52\% ) was unique to effortful control. The genetic correlations between hyperactivity and effortful control (i.e., r_g=-.71 [-.97--.46]) and externalizing behavior problems and effortful control (i.e., r_g=-.59 [-.77--.42]) indicate that although some of the genetic influences on behavior problems overlap with effortful control, there are also effects that are unique to each. The nonshared environmental correlation between hyperactivity and effortful control was high (i.e., r_e=-.91 [-1.0--.65]) suggesting substantial overlap in the nonshared environmental influences on the constructs.
CHAPTER FOUR

Discussion

EF is integral to adaptive functioning and there is substantial variability in EF in early childhood, but why? Despite the developmental significance of EF, prior to this study, relatively little was known about the relative genetic and environmental influences on individual differences in EF in early childhood. The present study makes unique contributions to the existing literature as the first study of the genetic etiology and developmental-significance of two facets of EF in early childhood, the overlap between them, and the mechanisms underlying the relations between EF and other key aspects of early childhood development. The findings indicate that variability in set-shifting and inhibitory control is due to both genetic and nonshared environmental factors. Genetic influences underlie the association between set-shifting and inhibitory control in early childhood, and these same genetic influences also underlie their relations with task orientation and school readiness.

Etiology of Executive Functioning in Early Childhood

It is integral to isolate what sources (i.e., genetic and environmental) are responsible for variability in early EF to identify children who are at genetic or environmental risk for EF deficits and inform how EF can be most effectively targeted through intervention. Prior behavioral genetic studies of temperamental inhibitory control in early childhood using lab-based assessments have yielded mixed results. In the same sample, observed inhibitory control was influenced by genetic factors at age 2, whereas at age 3, shared environmental effects accounted for familial resemblance (Gagne &
Goldsmith, 2011; Gagne & Saudino, 2010, 2016). The authors noted that the emergence of shared environmental influences at age 3 was likely due to ceiling effects (Gagne & Saudino, 2016), as research with older samples (e.g., Lemery-Chalfant et al., 2008) consistently finds genetic influences on inhibitory control. The present results add resolution to the mixed findings. When inhibitory control is tested with a measure that is suitable for a wide range of ages (i.e., the NIH Toolbox), no shared environmental influences were present. This provides support for the notion of ceiling effects and underscores the importance of using developmentally-appropriate assessments of EF.

The finding that individual differences in set-shifting were due to genetic and nonshared environmental factors mirrors research with adolescence and adults (Anokhin, Heath, & Ralano, 2003; Friedman et al., 2008), however, compared to older populations, the heritability estimate for set-shifting at age 4 was lower (i.e., 36% at age 4 and 50-80% in adolescence and adulthood). That said, the confidence intervals around the genetic and nonshared environmental variance estimates in the present study were wide. In fact, the confidence intervals overlap with the heritability estimates found in older populations, indicating that it is possible that the heritability of set-shifting may be higher than estimated in the present study and more consistent with the heritabilities of set-shifting at older ages. Replication with a larger sample is needed to ascertain precise estimates and to determine whether the heritability magnitude for set-shifting does indeed increase with age. When heritabilities change with age, they tend to increase rather than decrease (e.g., Petrill et al., 2004), so it is reasonable to expect that the heritability of set-shifting would be lower in early childhood, but more research is needed.
Both EF domains were largely influenced by the same genetic factors, although there were some modest genetic effects unique to each phenotype, as indicated by the fact that the genetic correlation was less than 1. Phenotypic literature has identified that the facets of EF are largely overlapping in early childhood (e.g., Wiebe et al., 2008), but prior to this study, it was unclear what sources (i.e., genetic, environmental) accounted for that overlap. The present results revealed that the overlap is entirely a function of common genetic effects. This provides further evidence for a developmental pattern of EF that is consistent with neurocognitive development. That is, a pattern that proceeds from diffuse to articulated at the level of genetic etiology.

The current analytical approach does not allow us to identify which particular genetic influences are important. That is, we cannot determine that, for example, common genetic variance is, in part, capturing variance common to speed of processing, an unmeasured third variable (e.g., sustained attention), or that the genetic influences have a bidirectional relation (Christopher et al., 2016). That said, one possible explanation for the high genetic correlation between the facets of EF is the “generalist genes” hypothesis; an approach proposing that genes have general, rather than specific effects in the brain (Kovacs & Plomin, 2006). Through pleiotropy (i.e., manifold effects of genes), a gene influences several areas of the brain, and each area, in turn, effects several abilities. That is, generalist genes operate pleiotropically throughout the brain and create genetic correlations among brain processes (Kovacs & Plomin, 2006). Molecular genetics methods can be used to identify DNA variants associated with these processes and
determine if the same genes underlie both phenotypes. If they do, this would provide further support for the generalist genes hypothesis of EF.

In adults, for sustained attention, inhibitory control, and error processing, there is overlap of the DNA variation within the dopaminergic (e.g., DAT1, DRD4), serotonergic (e.g., TPH2, 5-HTTLRP), and noradrenaline (e.g., -1,021 C/T SNP in DBH gene) systems, suggesting that there may be common variants across the facets of EF (see Barnes, Dean, Nandam, O’Connell, & Bellgrove, 2011 for a review). Less is known about genetic variants underlying EF in childhood. Preliminary results from a candidate gene study suggest that the one of the same genes that influence EF in adults also influences EF in childhood (Sherman, Hodel, Markant, & Thomas, 2015). In children, DNA variations of an enzyme (i.e., Catechol-O-methyltransferase) that degrades catecholamines (e.g., norepinephrine, serotonin, dopamine) is associated with working memory tasks that require sustained attention. Although these results are preliminary and should be interpreted with caution, it suggests that the monoaminergic system may also be implicated in child EF. Further molecular genetics work is needed on more components of EF to determine what genes overlap across the facets in childhood and if they are the same genes that overlap in adulthood. This is an important line of work, as linking specific genes to EF will make it possible to identify individuals who are at genetic risk for EF deficits and identify biological pathways for the development of interventions.

One clear limitation of molecular genetics research is that when DNA variants are identified, the effects tend to be small and rarely replicate (Barnes et al., 2011). A
A promising approach to identifying genes common to the facets of EF is to test the aggregate effects of multiple genes, because when combined, they have larger effects (Miyake & Friedman, 2012). Such a model can simulate the effects of multiple genetic variants acting jointly or independently, and can provide a theoretical framework with which to develop hypotheses regarding the genetic factors influencing the structure of EF (Miyake & Friedman, 2012).

Another possibility is that the genetic overlap observed between set-shifting and inhibitory control is a result of common neurological underpinnings. Research with adolescents and adults has demonstrated that there are overlapping brain regions across both set-shifting and inhibitory control, including pre-frontal areas (i.e., mid-dorsolateral and mid-ventrolateral regions, anterior cingulate gyrus, right inferior prefrontal cortex, orbitofrontal cortex), the dorsolateral anterior cingulate cortex, and the posterior parietal cortex (see Alvarez & Emory, 2006 for a review). There is evidence for developmental change in patterns of activation of the brain areas that underlie EF. Although children activate many of the same brain areas during EF tasks as adults, there is a significantly higher volume of prefrontal activation in adolescents than adults—an effect that is most pronounced in the dorsolateral prefrontal cortex extending into the cingulate. By contrast, adults showed more activation in the ventral region of the prefrontal cortex (Blakemore & Choudhury, 2006).

One explanation for the differential patterns of activation across development is that task performance increases with age and better performance is indexed by activation in different areas of the brain. For example, the dorsolateral prefrontal cortex is
negatively correlated with behavioral performance on EF tasks (i.e., more activation in the dorsolateral prefrontal cortex is associated with worse performance), whereas activation in the ventral region of the prefrontal cortex is associated with increased performance on EF tasks. Adolescents, who perform more poorly on EF tasks than adults, show greater activation in brain areas associated with poorer behavioral performance. Adults show the reverse pattern. Additionally, adolescents show more diffuse patterns of activation across the brain during EF tasks, whereas activation in adults is more focal (Blakemore & Choudhury, 2006). Taken together, these findings suggest that the brain networks that are recruited for EF tasks are modified with development (i.e., they become more efficient with time) and that behavioral gains on EF tasks are subserved by brain development.

Much less is known about the neurological underpinnings of EF in early childhood—a period characterized by both rapid brain development (i.e., pruning of synapses, synaptic proliferation, myelination, increases in grey and white matter in the prefrontal cortex) and substantial behavioral gains in EF. Evidence suggests that the anterior cingulate and frontal areas are also implicated in early EF (Bell & Deater-Deckard, 2007) and, as is observed in older samples, there are individual differences in the magnitude of brain activation relative to task performance. As indexed by the brain’s N2 response, an indicator of conflict monitoring, preschoolers who succeed on EF tasks (i.e., do not perseverate on the DCCS and succeed on incongruent Flanker trials) are better able to detect conflict across stimuli by recruiting the lateral prefrontal cortex (Buss, Dennis, Brooker, & Sippel, 2011; Espinet, Anderson, & Zelazo, 2012).
These findings raise the question of what individual differences in the common neurological functioning across set-shifting and inhibitory control may be genetically influenced. It is possible that individual differences in activation of a given region may contribute to the genetic covariance between the facets of EF. At the phenotypic level, there is evidence for individual differences in brain activation in adults related to EF task performance, with more activation in the prefrontal cortex and anterior cingulate cortex for subjects with higher working memory capacity (Osaka et al., 2004). There may also be genetic influences on individual differences in functionality. For example, the connectivity between brain regions and the strength of these connections may also be heritable and account for some of the genetic covariation between set-shifting and inhibitory control.

Another consideration is that EF is an emerging capacity in preschoolers. Therefore, from a brain development perspective, it is possible that the genetic correlation between set-shifting and inhibitory control results from genetically-influenced developmental timing of the maturation of brain regions that are important for EF and other developmental processes that occur in the brain across early childhood. For example, synaptic pruning aids EF (e.g., Selemon, 2013) and synaptic pruning is genetically-influenced (Toga, Thompson, & Sowell, 2006), suggesting that individual differences in synaptic pruning may also, in part, explain the genetic correlation. Importantly, these genetic effects would likely be specific to young children. This underscores the need for longitudinal studies of the genetic and environmental influences on EF that span key developmental periods to determine if the same genetic influences
underlie EF across time. An alternative explanation for the high genetic correlation is that, because both set-shifting and inhibitory control tap attention (e.g., Garon et al., 2008), it is possible that the EF assessments measure a common attentional phenotype and that these individual differences contribute to the genetic correlation.

A combination of neuroimaging techniques and genetics (i.e., imaging genomics) allows for the identification of phenotypes in the brain related to functional polymorphisms in genes that are important for complex behaviors, like EF (Hariri & Weinberger, 2003). Research suggests that individual differences at the level of genetic variation are associated with differential brain activation during EF tasks. For example, carriers of a less-active allele for the serotonergic MAOA gene (i.e., one that degrades noradrenaline and serotonin more slowly) demonstrate higher cortical activity in the bilateral extrastriate cortex and right superior parietal cortex, but less activation in the right ventrolateral prefrontal cortex than carriers of the more active allele for inhibitory control (Passamonti et al., 2006). At question is whether genetic variation is associated with the same brain activation across multiple facets of EF—thereby contributing to the genetic correlation among them. And, again, whether these findings can be replicated with younger samples.

In addition to genetic influences, variation in both facets of EF was, in part, due to nonshared environmental factors, but these effects did not covary. That is, both set-shifting and inhibitory control were influenced by nonshared environmental factors, but these effects were not the same for both phenotypes. This raises the question of what nonshared environments, beyond measurement error, independently influence these
variables. This has important implications, because identifying nonshared environmental influences on these constructs may help to identify children who are at environmental risk and inform specific avenues for intervention.

It is unlikely that features of the testing situation contributed substantially to the differential nonshared environmental influences on the facets of EF, as the NIH Toolbox tasks are scripted and there is little tester variation in administration. Consequently, it is more likely that variability in child-specific skills and experiences differentially promote set-shifting and inhibitory control. That is, children may have more or less experience outside of the testing environment that aids their performance on one task over the other. For instance, children may be variable in their exposure to activities, such as matching games, that could influence performance on the DCCS. That is, even within a family, one child may have more exposure to matching games than their twin because they seek out opportunities to play these games. Further, children may be variable in the extent to which they experience sleep, exercise, and differential parent or teacher scaffolding that may aid only one component of EF. Nonetheless, researchers interested in environmental influences on EF will need to consider the fact that different environmental experiences likely operate for each facet.

Executive Functioning and Temperament

Both EF and effortful control are distinct high-level self-regulatory capacities. Although they share some similarities (i.e., a common component [inhibition] and process [executive attention]; Zhou et al., 2012), EF is “cold” (i.e., cognitive, intentional, and emotionally-neutral), whereas effortful control is “hot” (i.e., an emotional
temperamental behavioral disposition that is less overtly intentional; Blair & Razza, 2007; Zelazo & Müller, 2002). Based on the conceptual overlap between EF and effortful control, and the fact that set-shifting and effortful control have been found to be positively associated in preschoolers (Hongwanishkul et al., 2005), it was predicted that EF and effortful control would be related. In our sample, the observed correlation between EF and effortful control was only modest. Although there was a significant correlation between EF and effortful control in the prior study (Hongwanishkul et al., 2005), the correlation was also modest, and only slightly higher than the observed correlation in the present study (i.e., .21 vs. .16 and .17). These findings suggest that EF and effortful control are largely separable in early childhood. In further support for the distinction between the two constructs, in the present study, EF and effortful control were differentially-associated with developmental outcomes, as will be discussed later in more detail. Therefore, despite the fact that EF and effortful control share some commonalities, these findings provide evidence that they are largely distinct, and underscore the importance of obtaining a comprehensive assessment of self-regulation by measuring both EF and effortful control, as each may provide unique information about child behavior.

EF was not related to most aspects of temperament at either the level of lower-order dimensions (e.g., anger, sadness, shyness, smiling/laugher) or higher-order factors (e.g., activity, affect/extraversion, surgency, negative affectivity). There are mixed findings on the relation between EF and temperament in the literature. While some studies fail to find an association between the two constructs (e.g., Hongwanishkul et al.,
others have (e.g., Carlson, Mandell, & Williams, 2004). For example, children who performed better on an EF composite also had better temperamental inhibitory control, attentional focusing, and perceptual sensitivity (Carlson, Mandell, & Williams, 2004). One potential reason for the discrepant finding is that in the present study cold, rather than hot, EF was assessed. The EF composite used by Carlson and colleagues included both hot (i.e., bear/dragon, tower building) and cold (i.e., snack delay, gift delay) measures of EF. It is possible that the relation between the temperament dimensions and the EF composite emerged because emotionality is common to temperament and hot EF. In fact, most studies that find an association between temperament and self-regulation assess self-regulation with hot measures (i.e., using measures of effortful control; e.g., Rothbart, Ziaie, & O’Boyle, 1992; Valiente, Lemery-Chalfant, & Swanson, 2010). The current findings shed light a larger issue in the field concerning how to differentiate the constructs that fall under the umbrella of self-regulation and echo the call for an integrated approach to the study of self-regulation (Zhou et al., 2012).

One significant relation between EF and temperament did emerge—children who had better EF also had better task orientation (i.e., a dimension of temperament reflecting attention span, persistence, goal directedness, and responsiveness to testing stimuli). Like EF, task orientation is genetically-influenced and it is overlapping genetic effects that explain the association between EF and task orientation. Because there is genetic overlap, this begs the question of what genetic influences are common to both. A possible explanation for the observed genetic overlap between EF and task orientation is that pleiotropic effects may underlie both EF and task orientation. Like EF, polymorphisms in
the dopaminergic system are also associated with temperament (Auerbach, Faroy, Ebstein, Kahana, & Levine, 2001), indicating potential pleiotropic effects, but no studies have addressed this question directly. Another explanation is that EF (Garon et al., 2008) and task orientation both index an attentional phenotype and that individual differences in attentional skills may underlie this association.

These findings have implications for intervention. Because there is overlap at the level of genetic etiology, it is possible that an intervention on one of these constructs will also yield positive outcomes on the other. In fact, a school-based intervention yielded positive effects for both set-shifting and task orientation (Bierman, Nix, Greenberg, Blair, & Domitrovich, 2008), suggesting that these constructs are malleable and interact in early childhood. Additionally, because there are common genetic effects among EF and task orientation, this indicates that there might be a “risky” temperament that signifies that children are at genetic risk for EF deficits.

Executive Functioning and School Readiness

Children with better EF also had better school readiness. Effortful control showed a similar but more modest relation with school readiness. This is consistent with prior research that finds an association between objectively-rated effortful control (i.e., obtained through teacher ratings) and school readiness (Blair & Razza, 2007). EF and school readiness shared common genetic effects, although as indicated by the genetic correlation (i.e., a correlation less than 1), there were also genetic influences unique to each. These findings suggest that genetics, rather than differences in one’s environment (e.g., parents, preschool, peers) drive the relation between EF and school readiness.
One possible explanation for the genetic overlap between EF and school readiness is that EF serves as a foundation for learning. That is, some of the genetic influences on school readiness may reflect underlying EF. Cognitive abilities in early childhood longitudinally predict reading and spelling abilities in first and fourth grades, and the correlations were primarily a function of genetic influences, suggesting causal pathways from cognitive abilities to learning to read (Christopher et al., 2015). Although directionality cannot be assessed in the present sample because the data is not longitudinal, it is reasonable to expect that there may be a directionality from EF to school readiness, as EF longitudinally predicts academic success phenotypically (e.g., Bull et al., 2008). Further, this has yet to be tested in a behavioral genetics framework, but it is possible that genetic effects on EF lay the foundation for later school readiness and academic success, as is the case with cognitive abilities. If so, it may be possible to identify children who are at genetic risk for poor academic outcomes by identifying those with early EF deficits.

Another explanation may for the genetic correlation between EF and school readiness may, again, be a result of pleiotropy. In fact, there is evidence for genome-wide pleiotropy between general cognitive ability and language and reading, and cognitive ability and mathematics (Trzaskowski et al., 2013). If it is the case that pleiotropy operates across EF and school readiness, this can inform research using molecular genetics methods in the search for identifying genetic variants associated. That is, assuming that pleiotropy operates on EF and school readiness, identifying genetic variants in one of these areas may inform the other.
The behavioral genetics analysis of school readiness produced an intriguing result with regard to environmental effects. Although prior research has found substantial shared environmental influences for school readiness (Lemelin et al., 2007; Rhemtulla & Tucker-Drob, 2012), this was not the case in the current study; individual differences in school readiness were due to genetic and nonshared environmental influences. At the phenotypic level, school readiness as assessed with the BSRA is associated with familial factors including SES and parental education and employment (e.g., Tunçeli & Akman, 2013), which makes the current findings more puzzling.

One reason why shared environmental influences did not emerge may be a result of differences in the measures of school readiness across studies. The measure of school readiness utilized in the present study assesses basic factual knowledge (e.g., knowledge of colors, shapes, letters), whereas the measures of school readiness used in the prior studies tapped higher-level, complex skills (e.g., numerical operations, patterns, letter sounds, word matching, writing). Shared environmental influences comprise between-family variation. Because school readiness in the current study reflected such basic academic skills, it is unlikely that there is much variation between families in the present sample. That is, because most families teach their children colors, numbers, and letters early on, shared environmental influences might not have emerged on the BSRA in the present study because families are homogeneous in the extent to which they teach their children these concepts. Conversely, in higher SES samples, shared familial factors (e.g., level of parental education, intelligence) may be more important for complex school readiness skills and contribute to variance between families, thereby explaining why
shared environmental influences have been observed previously. These findings suggest that it may be most fruitful to target shared familial factors for high-level complex school readiness skills, as most children in families such as those included in the current study already receive the input needed for basic school readiness skills.

Further, there is evidence of a Gene x Environment (GxE) interaction between mathematics skill and SES, with genetic influences more pronounced at higher levels of SES. This suggests that genes for early mathematic capacities are more strongly expressed in children from higher SES families (Rhemtulla & Tucker-Drob, 2012). Given the fact that the present sample was relatively high in SES, it is possible that the lack of shared environmental influences reflects a GxE interaction between school readiness and SES. Research with a more diverse sample would add resolution to this question.

**Executive Functioning and Behavior Problems**

Neither set-shifting nor inhibitory control was related to behavior problems, whereas effortful control was inversely associated with hyperactivity and externalizing behavior problems. The null finding between EF and behavior problems was particularly surprising as EF deficits are often considered a defining feature of externalizing behavior problems and ADHD (e.g., Doyle et al., 2005). Sampling and methodological differences may explain the differential finding in the present study. A meta-analysis of 22 studies of EF and externalizing behavior problems in preschoolers (i.e., 4- to 6-years-old) revealed main effects of set-shifting, inhibitory control and working memory on externalizing behavior problems, with more pronounced effects in older preschoolers (Schoemaker et al., 2013). The children in the present study were notably younger than many of those
included in the meta-analysis—only 6 of these studies included children at or younger than 4-years-old. Therefore, a significant association between EF and hyperactivity and externalizing behavior problems may not have emerged because the children in the present sample were younger than those assessed in the studies reviewed by the meta-analysis. Additionally, of these 6 studies, some samples were clinically-referred, suggesting more extreme levels of externalizing behavior problems. The present study may not have replicated these findings because the levels of hyperactivity and externalizing behavior problems were relatively low. Further, multiple methods (e.g., questionnaires, interviews) and informants (e.g., teachers, parents, research assistants) were utilized in the studies included in the meta-analysis. Different raters may provide unique information about children and different methods may also tap different capacities of the child. Prior research has found that teacher, but not parent, ratings of externalizing behavior problems were associated with EF deficits (e.g., Oosterlaan, Scheres, & Sergeant, 2005). In fact, the present results are consistent with the notion that teachers, rather than parents are the ideal informants on child behavior problems (e.g., Loeber, Green, Lahey, & Stouthamer-Loeber, 1990), but as teacher ratings of behavior problems were not obtained in the present study, this cannot be tested. The present study included only one rater of behavior problems (i.e., parents) using one method (i.e., questionnaires) and EF was only assessed via a lab-based computerized battery. It is possible that if ratings of behavior problems and EF were obtained via multiple-methods and informants that these associations would have emerged. Future work should aim to clarify at what age this association emerges, as it appears that there is a developmental shift in the
association between EF and externalizing behavior problems from 4 to 5 years of age, and tease-apart rater and methodological effects.

Conversely, the temperament dimension effortful control was related to hyperactivity and externalizing behavior problems. EF and effortful control were only moderately related, so it is not surprising that they differentially predict developmental outcomes, but this raises the question of why effortful control was related to behavior problems but EF was not. One potential explanation for the differential finding is methodological; parents rated both effortful control and behavior problems. It is possible that these associations emerged as a result of shared method variance. Another explanation is that differences in the global concepts of EF and effortful control drive the differential associations. Evidence suggests that the emotional and motivational components common to effortful control and externalizing behavior problems underlie their association (e.g., Pinsonneault, Parent, Castellanos-Ryan, & Seguin, 2015). It is reasonable to expect that effortful control would be related to maladjustment, as both involve modulation of emotion (i.e., emotion regulation) and motivation (Eisenberg et al., 2009), and that EF would not, as emotionality and motivation are not tapped with cold EF measures. Emotional dysregulation and motivation deficits are prevalent in individuals with ADHD (Shaw, Stingaris, Nigg, & Leibenluft, 2014; Volkow et al., 2010). Further, children with externalizing problems typically experience breakdowns in their self-regulation in contexts with high emotional and motivational significance. When researchers parse self-regulation tasks by the requirement of emotionality (i.e., into hot and cold tasks), emotional tasks appear to be stronger predictors of externalizing behavior
problems (Pinsonneault et al., 2015). For example, deficits in effortful control (e.g., inability to delay gratification in tasks that involve compelling and easily accessible rewards) predict behavior problems in elementary school (Eisenberg et al., 2009; Kim, Nordling, Yoon, Boldt, & Kochanska, 2013). Additionally, children with externalizing behavior problems show better EF performance in cold versus hot EF tasks (Woltering, Lishak, Hodgson, Granic, & Zelazo, 2016). Further, physiological data suggests that children with externalizing problems struggle to regulate their emotional state during emotional tasks (Woltering et al., 2016).

A clear directionality is implied in these findings. That is, that effortful control deficits precede behavior problems. Transactional mechanisms of influence across these domains may act to strengthen their co-occurrence (Pinsonneault et al., 2015). An important next step is to use longitudinal designs to tease apart the directions of effect between effortful control deficits and behavior problems to determine if they mutually exacerbate one another across time and identify whether genetic or environmental influences drive these effects.

These findings suggest that EF may be less effective than effortful control in capturing behavioral impairments in children (Woltering et al., 2016). Further, interventions that target young children’s effortful control (e.g., through behavioral therapy, play, or games) may reduce their risk for developing behavior problems (Kim et al., 2013). But again, this assumes a unidirectional effect from effortful control to behavior problems. If the reverse effect is also true, behavioral interventions may also yield positive effects on effortful control.
Because effortful control was related to behavior problems, but EF was not, this raises an important question about the nature of self-regulation deficits in behavior problems. It may indeed be the case that EF deficits are associated with externalizing behavior problems but that this pattern did not emerge in the present study for methodological or sampling reasons. An alternative explanation is that effortful control is simply a better predictor of externalizing behavior problems in a nonclinical sample in early childhood. Nonetheless, the present results support the notion that deficits in self-regulation are related to externalizing behavior problems, but emphasize the importance of a nuanced assessment of self-regulation.

Effortful control and behavior problems were linked by common genetic and nonshared environmental effects. Although no prior behavioral genetic studies have explored the sources genetic and environmental covariance between hyperactivity, externalizing behavior problems and effortful control in early childhood, the present findings are consistent with prior research that found genetic and nonshared environmental overlap between one component of effortful control, inhibitory control, and externalizing behavior problems in preschoolers (Gagne, Saudino, & Asherson, 2011). Interestingly, in middle childhood, genetic influences alone account for the covariation between the two phenotypes (Lemery-Chalfant et al., 2008). This developmental shift has implications for intervention in samples similar to that of the present study. Because there is nonshared environmental covariance between effortful control and externalizing behavior problems in early, but not middle childhood, it
suggests that there may be an optimal window for nonshared environmental interventions on effortful control and behavior problems.

Possible sources of common nonshared environmental effects that may contribute to the shared etiology between effortful control and behavior problems include differential parental treatment or child-specific experiences. For example, within a family, if a child has better effortful control, they may elicit more positive parenting that buffers against behavior problems, whereas a child who has worse effortful control may elicit more negative parenting that promotes behavior problems. Further, mechanisms outside of the family (e.g., differential daycare experiences) are partly responsible for the covariation between cognitive deficits and externalizing behavior problems (Pinsonneault et al., 2015) and may explain some of the observed nonshared environmental covariance. Another potential explanation is methodological. Measures of temperament, like effortful control, to some extent, may tap psychopathology and measures of symptoms may tap temperament (Lemery-Chalfant et al., 2008).

Common genetic effects also underlie effortful control and behavior problems. As discussed earlier, it is possible that common neurological underpinnings are responsible for the genetic overlap between the constructs. Individual differences in effortful control are subserved by functioning of the executive attention network (Rothbart, Derryberry, & Posner, 1994), which is thought to involve the anterior cingulate cortex (Vijayakumar et al., 2014). In adolescence, changes in effortful control over time mediate the relation between thinning of the left anterior cingulate cortex and externalizing behavior problems, indicating that effortful control mediates the relation between neurobiological
development and mental health during adolescence (Vijayakumar et al., 2014). It is unknown whether this is also the case in early childhood, but it suggests that neurological development is integral to the relation between effortful control and behavior problems. Further, molecular genetics research indicates that genetic variation in the dopamine system is associated with both self-regulation (Rothbart & Posner, 2005) and ADHD (Acosta, Arcos-Burgos, & Muenke, 2004), suggesting that pleiotropic effects may be in operation, but this is an open empirical question.

**Implications**

These findings have implications for developmental research on the predictors of key outcomes. EF and effortful control are largely discrete self-regulatory capacities that should not be used interchangeably. Instead, they should be measured in tandem, as each provides unique developmental information. Consistent with the literature, EF is more predictive of academic than behavioral functioning (Willoughby Kupersmidt, & Voegler-Lee, 2012), whereas for effortful control, the reverse is true (e.g., Woltering et al., 2016).

There was evidence for genetic effects on EF, effortful control, task orientation, school readiness and behavior problems. There is a mistaken popular view that ‘genetic’ means ‘immutable’ (i.e., that the genetic blueprint is deterministic). Genetically-influenced traits can be modified by environmental interventions, as has been demonstrated with EF (e.g., Diamond & Lee, 2011). Further, prior to this study, results on the heritability of EF in early childhood was patchy. These findings add resolution by reporting the genetic and environmental influences on two facets of cognitive EF during a key developmental period.
The results of the present study have direct implications for prevention and intervention efforts geared toward populations similar to the sample utilized in the present study. First, because set-shifting and inhibitory control were influenced by unique nonshared environmental effects, it suggests a one-size-fits-all environmental intervention may not aid both domains. Second, because shared environmental influences did not emerge, interventions on EF that target the shared environment are not likely to be fruitful. Third, if through replication, it can be confirmed that the heritability estimates for EF are lower in early childhood than later in development, it would suggest that this is the ideal developmental window for environmental interventions, as EF is likely to be more malleable. Fourth, because EF exhibits genetic overlap with key developmental outcomes, possibly as a result of generalist genes, it suggests that it may be possible to identify children who are at genetic or environmental risk for maladaptive development.

Limitations

The results of the present study should be considered in light of some limitations. One notable limitation of the present study is that working memory was not assessed. Multidimensional studies of EF typically include all facets of EF: set-shifting, inhibitory control, and working memory. Although it would be informative to study all facets of EF, the NIH Toolbox: Early Childhood Cognitive Battery includes set-shifting and inhibitory control as measures of EF, as they can be assessed reliably in preschool. Nonetheless, without measuring working memory we were not able to obtain a holistic picture of the structure of all facets of early EF. That said, this is the first behavioral genetics study of
multiple components of EF during the preschool period and contributes unique information to the existing literature.

Characteristics of the sample should also be noted. The modest sample size led to wide confidence intervals for some estimates and there was not sufficient power to test for sex differences at the level of etiology. Additionally, the sample included only twins, and it is important that results from twin studies generalize to singleton samples. Finally, the present sample was relatively homogeneous in terms of SES, limiting generalizability. The relative contributions of genetic and environmental influences on individual differences in traits are tied to the population at study. Therefore, it is important to note that this pattern of findings may not generalize to more diverse samples. For example, it is reasonable to expect that shared or nonshared environmental influences that did not emerge in the present study due to a lack of variability in such environments across individuals or families may emerge in other samples that are more variable in such environments. Consequently, the same prevention and intervention efforts that may be effective for high SES samples may not generalize to low income, high-risk samples—those that are typically in need of intervention.

**Future Directions**

A future goal of the current project is to explore the developmental pattern of EF across early childhood. The present sample will be assessed longitudinally at ages 3, 4, and 5. By including multiple time points, it will be possible to assess the extent to which the same genetic and environmental influences operate on EF across age, irrespective of relative heritabilities. This will inform sources (i.e., genetic, environmental) of continuity
and change across development. Further, longitudinal models will allow for the assessment of covariance between variables over time.

Further, in addition to main effects of genetics, heritability estimates may encompass Gene x Environment (GxE) interactions, whereby the magnitude of genetic influences on a phenotype differs as a function of the environment. Further work is needed to examine whether GxE interactions operate on EF. For example, to determine whether genetic influences on EF are expressed equally across a range of socioeconomic strata (Engelhardt, Briley, Mann, Harden, & Tucker-Drob, 2015). To obtain a complete picture of how genes and environments interact, it will be necessary to measure EF in children across a wide range of ages and contexts to identify specific aspects of the environment (e.g., parent education, income) that serve to promote the expression of genes for EF. This question has clear relevance to public policy and intervention (Rhemtulla & Tucker-Drob, 2012).

**Conclusion**

EF at age 4 was influenced by genetic and nonshared environmental effects, mirroring results from studies of older populations. Two facets of EF, set-shifting and inhibitory control, share common genetic influences, suggesting that these constructs overlap in early childhood. EF was related to task orientation and school readiness and the same genetic effects thank link set-shifting and inhibitory control also underlie their relations to these outcomes. A related construct to EF, effortful control, was associated with hyperactive and externalizing behavior problems. Genetic and nonshared environmental factors link hyperactivity, externalizing behavior problems, and effortful
control. These findings suggest that it may be possible to identify children who are at genetic risk for maladaptive developmental outcomes by identifying those with EF deficits.
APPENDICES

Appendix A. NIH Toolbox Flanker Administration Instructions

Appendix B. NIH Toolbox DCCS Administration Instructions

Appendix C. Possible Ranges for CBCL Scores
<table>
<thead>
<tr>
<th>Practice- intro</th>
<th>Child’s Screen Written Content</th>
<th>Examiner (E) Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Here’s a fish! This is the tail → [fish] ← this is the mouth. The fish is pointing this way, the same way the fish is swimming.</td>
<td>Points to child’s screen, E clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>Here the MIDDLE fish is circled. Can you point to the MIDDLE fish?</td>
<td>Child clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>Where’s the MIDDLE fish here?</td>
<td>Child clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>Look at all of the fish!!! The fish in the MIDDLE is hungry.</td>
<td>E clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>To feed the MIDDLE fish, choose the button that matches the way the MIDDLE fish is pointing.</td>
<td>Demonstrates by pointing to child’s screen.</td>
</tr>
<tr>
<td></td>
<td>If the middle fish is pointing this way, choose this button.</td>
<td>Demonstrates by pointing to child’s screen.</td>
</tr>
<tr>
<td></td>
<td>If the middle fish is pointing this way, choose this button.</td>
<td>Demonstrates by pointing to child’s screen.</td>
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<tr>
<td></td>
<td>Sometimes all the fish point the same way. Sometimes the MIDDLE fish points a different way from his friends, like this: [picture of incongruent fish]. You should always choose the button that matches the way the MIDDLE fish is pointing. You will see and hear the word MIDDLE to remind you.</td>
<td>Points; chooses button.</td>
</tr>
<tr>
<td></td>
<td>Here the MIDDLE fish is pointing this way, so I’ll choose this button.</td>
<td>Points; chooses button.</td>
</tr>
<tr>
<td></td>
<td>Here the MIDDLE fish is pointing this way, so I’ll choose this button.</td>
<td>Points; chooses button.</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; then clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>4 practice items.</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed</td>
<td>Let’s practice some more. If the MIDDLE fish is pointing this way, choose this button.</td>
<td>Points; chooses button.</td>
</tr>
<tr>
<td>-------------------------</td>
<td>----------------------------------------------------------------------------------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Transition to more practice items</td>
<td>If the middle fish is pointing this way, choose this button.</td>
<td>Demonstrates by pointing to child’s screen.</td>
</tr>
<tr>
<td>More practice, if needed</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Transition</td>
<td>4 practice items</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed</td>
<td>Let’s practice some more. If the MIDDLE fish is pointing this way, choose this button.</td>
<td>Points; chooses button.</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Transition</td>
<td>4 practice items</td>
<td></td>
</tr>
<tr>
<td>Test items transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>More test items if 90% success on fish</td>
<td>Now you will do the same thing, but you will see arrows instead of fish. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; then clicks NEXT on E’s screen</td>
</tr>
<tr>
<td></td>
<td>20 test items (arrows)</td>
<td></td>
</tr>
</tbody>
</table>
# APPENDIX B

NIH Toolbox DCCS Administration Instructions

<table>
<thead>
<tr>
<th>Practice-intro</th>
<th>Child’s Screen Written Content</th>
<th>Examiner (E) Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>We’re going to play a matching game with colors and shapes.</td>
<td>Reads screen; then clicks NEXT on E’s screen.</td>
<td></td>
</tr>
<tr>
<td>SHAPE intro</td>
<td>We’ll play the SHAPE game first. In the SHAPE game, choose the picture that’s the same SHAPE as the picture in the middle of the screen. If it’s a BOAT choose this picture.</td>
<td>Points to BOAT; demonstrates use of index finger</td>
</tr>
<tr>
<td></td>
<td>If it’s a RABBIT, choose that picture.</td>
<td>Points to RABBIT; demonstrates use of index finger</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Shape practice</td>
<td>4 items sorted by shape.</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed.</td>
<td>Let’s practice some more. In the SHAPE game, choose the picture that’s the same SHAPE as the picture in the middle of the screen. If it’s a BOAT, choose this picture.</td>
<td>Chooses BOAT</td>
</tr>
<tr>
<td></td>
<td>If it’s a RABBIT, choose that picture.</td>
<td>Chooses RABBIT</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Shape practice again</td>
<td>4 items sorted by SHAPE.</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed.</td>
<td>Let’s practice some more. In the SHAPE game, choose the picture that’s the same SHAPE as the picture in the middle of the screen. If it’s a BOAT, choose this picture.</td>
<td>Chooses BOAT</td>
</tr>
<tr>
<td></td>
<td>If it’s a RABBIT, choose that picture.</td>
<td>Chooses RABBIT</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Shape practice again</td>
<td>4 items sorted by SHAPE.</td>
<td></td>
</tr>
<tr>
<td>----------------------</td>
<td>--------------------------</td>
<td></td>
</tr>
<tr>
<td>COLOR intro</td>
<td>We can also match by COLOR. In the COLOR game, choose the picture that’s the same COLOR as the picture in the middle of the screen. If it’s BROWN, choose this picture.</td>
<td>Points to; chooses BROWN picture</td>
</tr>
<tr>
<td>Transition</td>
<td>If it’s WHITE, choose that picture</td>
<td>Points to; chooses WHITE picture</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Color practice</td>
<td>4 items sorted by color.</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed</td>
<td>Let’s practice some more, in the COLOR game, choose the picture that is the same COLOR as the picture in the middle of the screen. If it’s WHITE, choose this picture.</td>
<td>Chooses WHITE picture</td>
</tr>
<tr>
<td>Transition</td>
<td>If it’s BROWN, choose that picture.</td>
<td>Chooses BROWN picture</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Color practice</td>
<td>4 items sorted by COLOR</td>
<td></td>
</tr>
<tr>
<td>More practice, if needed</td>
<td>Let’s practice some more, in the COLOR game, choose the picture that is the same COLOR as the picture in the middle of the screen. If it’s WHITE, choose this picture.</td>
<td>Chooses WHITE picture</td>
</tr>
<tr>
<td>Transition</td>
<td>If it’s BROWN, choose that picture.</td>
<td>Chooses BROWN picture</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Color practice</td>
<td>4 items sorted by COLOR</td>
<td></td>
</tr>
<tr>
<td>COLOR and SHAPE</td>
<td>Now we’re going to play with some different SHAPES and COLORS. This time, we’ll use</td>
<td></td>
</tr>
<tr>
<td>intro</td>
<td>BALLS and TRUCKS that are YELLOW and BLUE.</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Pre-switch intro</td>
<td>Let’s start with the COLOR game. Remember the COLOR game? In the COLOR game choose the picture that’s the same color as the picture in the middle of the screen. If it’s a BLUE one, choose this picture.</td>
<td>Chooses BLUE picture</td>
</tr>
<tr>
<td></td>
<td>And if it’s a YELLOW one, choose that picture.</td>
<td>Chooses YELLOW picture</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Color items</td>
<td>5 items sorted by color (if child does not get 4 of 5 correct, test terminates)</td>
<td></td>
</tr>
<tr>
<td>Post-switch intro</td>
<td>Now we’re going to play the SHAPE game. Remember the SHAPE game? In the SHAPE game, choose the picture that’s the same SHAPE as the picture in the middle of the screen. If it’s a TRUCK, choose this picture.</td>
<td>Chooses TRUCK</td>
</tr>
<tr>
<td></td>
<td>If it’s a BALL, choose that picture.</td>
<td>Chooses BALL</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; clicks NEXT on E’s screen.</td>
</tr>
<tr>
<td>Intro mixed items</td>
<td>We can also play both games together. Remember, when you see or hear the word SHAPE, choose the picture that’s the same SHAPE as the picture in the middle of the screen. If it’s a TRUCK, choose this picture.</td>
<td>Chooses TRUCK</td>
</tr>
<tr>
<td></td>
<td>If it’s a BALL, choose that picture.</td>
<td>Chooses BALL</td>
</tr>
<tr>
<td></td>
<td>When you see or hear the word COLOR, choose the picture that’s the same COLOR as the picture in the middle of the screen. If it’s a BLUE one, choose this picture.</td>
<td>Chooses BLUE picture</td>
</tr>
<tr>
<td></td>
<td>If it’s a YELLOW one, choose this picture.</td>
<td>Chooses YELLOW picture</td>
</tr>
<tr>
<td>Transition</td>
<td>Now you try. Keep your eyes on the star. Answer as fast as you can without making mistakes. If you make a mistake, just keep going.</td>
<td>Reads screen; then clicks NEXT on E’s screen</td>
</tr>
<tr>
<td>Test items</td>
<td>30 mixed items</td>
<td></td>
</tr>
</tbody>
</table>
### APPENDIX C

Possible Ranges for CBCL Scores

<table>
<thead>
<tr>
<th>Scale</th>
<th>Number of Questions</th>
<th>Possible Range of Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Internalizing Behavior Problems</td>
<td>36</td>
<td>0-72</td>
</tr>
<tr>
<td>Externalizing Behavior Problems</td>
<td>24</td>
<td>0-48</td>
</tr>
<tr>
<td>Total Behavior Problems</td>
<td>99</td>
<td>0-198</td>
</tr>
<tr>
<td>Emotionally Reactive</td>
<td>9</td>
<td>0-18</td>
</tr>
<tr>
<td>Anxious/ Depressed</td>
<td>8</td>
<td>0-16</td>
</tr>
<tr>
<td>Somatic Complaints</td>
<td>11</td>
<td>0-22</td>
</tr>
<tr>
<td>Withdrawn</td>
<td>8</td>
<td>0-16</td>
</tr>
<tr>
<td>Sleep Problems</td>
<td>7</td>
<td>0-14</td>
</tr>
<tr>
<td>Attention Problems</td>
<td>5</td>
<td>0-10</td>
</tr>
<tr>
<td>Aggressive Behavior</td>
<td>18</td>
<td>0-36</td>
</tr>
<tr>
<td>Affective Problems</td>
<td>10</td>
<td>0-20</td>
</tr>
<tr>
<td>Anxiety Problems</td>
<td>10</td>
<td>0-20</td>
</tr>
<tr>
<td>Pervasive Developmental Problems</td>
<td>13</td>
<td>0-26</td>
</tr>
<tr>
<td>ADHD Problems</td>
<td>6</td>
<td>0-12</td>
</tr>
<tr>
<td>Oppositional Defiant Problems</td>
<td>6</td>
<td>0-12</td>
</tr>
</tbody>
</table>
References


Hollingshead, A. B. (1975). *The four-factor index of social status*. Unpublished manuscript, Yale University, New Haven, CT.


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