A Behavioral Genetic Study of the Links Between Working Memory and Aspects of Attention in Middle Childhood

Zhe Wang

Thesis submitted to the faculty of the
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

Master of Science

In

Psychology

Kirby Deater-Deckard, Chair
Martha Ann Bell
Bradley White

November 5, 2010

Blacksburg, Virginia

Keywords: working memory, attention, behavioral genetic, middle childhood

A Behavioral Genetic Study of the Links Between Working Memory and Aspects of Attention in Middle Childhood

Zhe Wang

ABSTRACT

The purpose of the current study was to explore the genetic and environmental association between working memory and different behavioral aspects of the attention network (i.e., executive attention, alerting attention, and orienting attention), using a twin design. Data were from 131 monozygotic (39% male) and 173 same-sex dizygotic (44% male) twins. Individual differences in working memory performance and behavioral measures of executive attention, alerting attention, and orienting attention were found to be moderately heritable. A modest nonshared environmental effect was found for all variables. Individual differences in working memory were significantly correlated with variability in executive and alerting attention, but not orienting attention. All of the association between working memory and executive as well as alerting attention was statistically mediated by genetic influences, indicating a common genetic mechanism or mechanisms underlying the links between working memory and certain behavioral indicators of attention.

Table of Contents

List of Tables

List of Figures

1.0 - Introduction

Children's self-regulation has been linked with their academic achievement and social-emotional competence. Children with better self-regulation are seen as more competent academically (Bramlett, Scott, & Rowell, 2000; Ponitz, McClelIand, Matthews, & Morrison, 2009; Smith, Borkowski, & Whitman, 2008) and more successful in social relations and social-emotional adjustment (Barkley, 1997; Buckner, Mezzacappa, & Beardslee, 2009; Eisenberg et al., 1997), but the causes of these connections are not well understood. Thus, it is important to understand the mechanisms underlying self-regulation, as this will inform interventions for improving children's cognitive and social-emotional outcomes. Self-regulation is a multi-faceted concept, including emotion self-regulation and cognitive self-regulation, both of which are multi-faceted concepts themselves. As important as it is, it is also difficult to examine all aspects of self-regulation in depth at the same time. Therefore, in the current study, effort was mainly focused on two specific aspects of cognitive self-regulation: working memory and attention.

As described in more depth below, individual differences in working memory and attention have been shown by theoretical and empirical studies in the last two decades to be intricately linked to each other. However, there are debates about the etiology of the links between the two. Some researchers have argued that working memory and attention are positively linked with each other, with variation in attention leading to variation in working memory performance. Within this group of researchers, there is a secondary debate about whether it is attention scope or attention control that influences working memory. Other researchers hold the view that working memory and attention are essentially parts of the same construct with different names (Diamond, 2002). These debates about the association between working memory and different aspects of attention naturally lead to the following questions: what is the etiology of the links between individual differences in working memory and aspects of attention? Do the links represent a common underlying genetic mechanism or are they results of environmental effects? These questions make further exploration into these links using different methods important and necessary. If working memory and aspects of attention reflect genetically identical constructs, instead of doing redundant work by studying them separately, researchers can simply operationalize them in the same way to represent a single cognitive self-regulation mechanism. In the meantime, instead of being limited to cognitive tasks, methods

such as behavioral observations and questionnaires can be developed to capture the underlying common mechanism, because behaviors reflecting attention are observable in everyday life. This will also guide future molecular genetic studies when trying to find out which set of candidate genes can explain the etiology of the links. Therefore, the goal of the current study was to quantify the genetic and non-genetic (i.e. environmental) variance in, and covariance between, working memory and multiple aspects of attention in middle childhood, using a twin design.

1.1 - Hypothesis 1: The link between working memory and attention

Working memory is the set of mental processes holding limited information in a temporarily accessible state in service of cognition (Cowan et al., 2005).The construct of working memory is first proposed by Baddeley to replace the unitary short-term memory system (Baddeley & Hitch, 1974). According to Baddeley, there are four components in working memory: the phonological loop, the visuospatial sketchpad, the episode buffer, and the central executive (an attention control system). Their respective responsibilities are: speech-based information processing, visual and spatial information processing, manipulating episodic information such as a story or a movie scene, and strategy selection and information integration (Baddeley, 2000). According to Baddeley, working memory is a limited resource in terms of its capacity, is time limited due to memory decay, and is also limited by the speed and the efficiency of mnemonic strategies (Baddeley, 1986). Therefore, depending on the specific processing demands in a certain task, working memory capacity can be allocated to processing functions, storage functions or both. As to how attention works as a central executive in working memory, Baddeley referred to Norman and Shallice's supervisory attentional system (SAS; Norman & Shallice, 1980) model, in which SAS was responsible for maintaining goals and overriding existing activities in order to achieve longer-term aims.

After Baddeley's working memory model specified attention control as a central executive function, many studies were conducted to examine how attention influenced the functioning of working memory. Some researchers believe that attention is linked with the executive processing of working memory. For example, Engle and colleagues have proposed that people with better attention control have higher working memory capacity. In their theory, the domain-specific short-term storage component and the domain-free controlled-attention processing component together make a hierarchical working memory construct. In their research, they have used tasks in different modalities, such as a visual-orienting task (i.e. visual modality;

Kane, Bleckley, Conway, & Engle, 2001), a reading span task, and an operation span task (i.e. verbal modality; Turner & Engle, 1989). With these tasks, they have found that regardless of modality, people with higher working memory capacity perform better in tasks requiring attention control (e.g., antisaccade task; Engle & Kane, 2004). On the other hand, they also found that the performance in the higher working memory capacity group degrades substantially when multiple tasks are conducted at the same time (i.e., when attention control strategies do not apply due to the distraction by multiple concurrent tasks; Engle & Kane, 2004). Based on this evidence, Engle and Kane have argued that some people perform better in working memory tasks because they control their attention better to keep the task goal in a highly active state and to resist interference of irrelevant information. They believe that working memory is a capability limited in its capacity because people are limited in their ability to control attention.

In contrast, other researchers believe that it is the scope of attention—not the control of attention— that matters in individual differences in performance in working memory tasks. Cowan has argued that the storage component of working memory is partially attention-demanding (e.g., phonological loop) and the storage capacity is largely determined by the scope of attention (up to four or five items; Cowan et al., 2005). He has disagreed with methods combining the processing and storage tasks together to assess working memory capacity. Instead, he has suggested pure attention-capacity measures as a substitute, such as memory for ignored speech, running memory tasks and visual-array comparison tasks. Unlike previously used working memory tasks, in these pure attention-capacity measures, people are unable to use strategies (e.g., rehearsal and chunking) because the information they receive is overwhelming and produces too much interference. However, Cowan did not suggest considering the scope of attention and the control of attention completely separately. Although the neural substrates underlying the scope and the control of attention are different (Cowan et al, 2005), coordinated cooperation between these neural substrates is required to accomplish complex cognitive tasks. In his "adjustable attention" view of working memory, attention functions like a camera, focusing to hold on to a task goal in the face of interference (a zoom-in camera with high resolution) and zooming out to apprehend more independent items (up to four or five) at a time (a zoom-out camera with larger scope; Cowan et al., 2005). Therefore, those who have better control over their attention are more likely to have larger attention scope as well.

Neurological bases of different working memory components have also been examined by previous research. Baddeley (2003) summarized the neuroimaging and other neuropsychological evidence in both normal subjects and patients with lesions in their brains and concluded that three basic components of working memory (excluding the episodic buffer) were localized in different brain regions. The inferior parietal cortex is involved in the storage process and the premotor area is involved in the rehearsal process, with the left and right hemisphere responsible for verbal and spatial information processing, respectively. The dorsolateral prefrontal cortex (DLPFC) appears to mediate the executive process of working memory. Kane and Engle (2002) also summarized a series of brain imaging and neuropsychological studies on attention and working memory and claimed the role of the DLPFC in the functioning of the two, in support of their attention-controlled view of working memory. They have proposed that the DLPFC, a critical structure in the attention control network, has a unique executive-attention role in actively maintaining access to stimulus representations and goals in working memory in interference-rich contexts. This implicates an anatomical association between controlled attention and executive working memory.

An important reason why different researchers have held different views on the association between working memory and attention is that, similar to working memory, attention also is a multi-faceted construct itself. When examining the role of attention in the functioning of working memory, different studies have focused on different facets of attention, leading to diverse views that may not be compatible.

Based on behavioral and neuropsychological evidence, Posner and Petersen (1990) have proposed an attention network with three independent subsystems: the alerting network, the orienting network and the executive control network. The respective functions of the three networks are: maintaining an alert state and readiness, selecting targets and orienting to sensory events, and detecting signals and resolving conflicts among possible actions for focal processing.

In the examination of the locus of attention, researchers have found that the three subsystems of the attention network are located in different brain areas. Neuroimaging studies have shown activity in the frontal and parietal cortex of the right hemisphere in tasks involving the alerting network. The orienting network depends largely on activation in the posterior parietal lobe. Activation in the DLPFC and anterior cingulate has been found to be involved in the executive attention network (Posner & Petersen, 1990).

In accordance with the idea that attention can be divided into different subsystems, and that these sub-divisions inform theory about working memory, Osaka and colleagues conducted a series of studies of the neural bases of executive attention and attention focusing in two groups of people with different working memory capacities (Osaka, Komori, Morishita, & Osaka, 2007; Osaka et al., 2003; Osaka, et al, 2004). They found significant activation in the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC) in executive attention tasks. Maintaining attention was associated with activity in DLPFC, and inhibitory control was associated with activity in ACC. They also found the neural bases for attention focusing in the left superior parietal lobule (SPL). In all these studies, high working memory-span subjects were significantly different from low-span subjects in activation in DLPFC, ACC and the left SPL, suggesting a link between working memory and executive attention, as well as a link between working memory and attention focusing. Similar results have been found regarding sustained attention and working memory performance in research in schizophrenia (Silver & Feldman, 2005)

In summary, one can see evidence of overlap between different subsystems of attention and working memory in terms of their functions and anatomical localizations. Some of the neuronal mechanisms for working memory and attention are so intertwined that one may question whether they are even distinguishable (Desimone, 1998). For example, located in DLPFC, working memory and the executive attention are both voluntary control processes that deal with detecting and integrating conflict information. The frontal lobe is involved in both the alerting and the rehearsal process of working memory, with the alerting potentially providing maintained attention states to complete the rehearsal process. However, the locus of the orienting network (posterior parietal lobe) does not overlap with the voluntary processing of working memory. It also shows independence with the other two aspects of the attention network (Fan, McCandliss, Sommer, Raz, & Posner, 2002).

However, all the evidence for solid associations between working memory and aspects of attention has been based on studies of adults. Are these associations already built in infancy? If not, how do they develop across childhood? Behavioral and neuropsychological studies have indicated that different aspects of attention and working memory have different developmental patterns. Working memory and executive attention are believed to undergo one of the longest periods of development of all mental capacities, because the underlying neural network (i.e. PFC)

that supports them takes over twenty years to fully develop in humans (Sowell et al., 1999). The development of these "PFC functions" begins late in the first year (i.e. signs of inhibition in A-not-B task and error detection), and major improvement can be observed at 3-4 years and 7-11 years via age appropriate tasks (e.g., day-night task at 3-4 years and anti-saccade task at 7-11 years; Diamond, 2002). Orienting attention, on the other hand, undergoes relatively fast development compared to working memory and executive attention. Its development is associated with the fast maturation of the parietal lobe at 3-4 months (Johnson, Posner, & Rothbart, 1991), and is approaching stable state toward the end of the first year (Posner& Rothbart, 2006). The alerting attention network is the least studied among all the attention networks, but evidence of its developmental course can be found in other lines of studies that name this attention network differently (i.e. sustained attention; they all refer to the initiation and the maintenance of the vigilance state on some stimuli; Richards, 2004). Rapid development of sustained attention can be observed between 3 to 6 months, measured by increased heart rate responsiveness (Richards, 1995). Yet, sustained attention also has been associated with the development of the prefrontal cortex (Reynolds & Richards, 2003), which implies a potentially prolonged development of sustained attention, like that of executive attention.

Although following different developmental paths, some studies have shown behavioral evidence of positive correlations between individual differences in working memory, executive attention and alerting attention, but no associations with orienting attention in early childhood (Wolfe & Bell, 2004)—a pattern that is similar to findings in the adult studies. Therefore, based on the evidence of previous research, the first hypothesis of the current study concerning the phenotypic (i.e., directly observed and measured) links between working memory and aspects of attention is that working memory will be positively associated with executive and alerting attention, but not orienting attention.

Measurement of working memory and attention. Before turning to the other study hypotheses, consideration of measurement issues is worthwhile. Working memory is often measured with cognitive tasks in different modalities, such as verbal modality (e.g., digit span tasks and word recall tasks), and visual-spatial modality (e.g., picture memory and spatial tracking tasks). Verbal modality is the most often used because the phonological loop is the best

understood among the three slave systems of working memory (Baddeley & Hitch, 1994). Therefore, in the current study, we used cognitive tasks in verbal modality—digit span tasks—to measure working memory.

However, there have been debates on how the two different digit span tasks—forward digit span and backward digit span— measure working memory. Some studies have shown that forward digit span task and backward digit span task capture different processes of working memory (Hayslip & Kennelly, 1982). According to this view, the forward digit span task is a measure of the storage function of working memory while the backward digit span measures both the storage function and the central executive. The logic of this argument is that in a backward digit span task, individuals suppress the predominant response of repeating the digit string in its original order, but carry out a subdominant response to report the string in the reverse order, which reflects the ability of central executive. However, other studies have not found any evidence differentiating these two tasks (Wiegersma & Meertse, 1990; Verhaegen, Marcoen, & Goossens, 1993). Although there is doubt about using forward digit span as a working memory measure, in the current study, working memory score was represented using a combination of the two tasks, in order to have a score that was reliable and maximally variable.

Similar to working memory, the attention network can be measured by cognitive tasks. For example, based on the attention network theory, Fan et al. (2002) developed an Attention Network Test (ANT) to examine the three different aspects of attention separately. However, unlike working memory, attention is easy to see by observers (e.g., parents of children) in everyday behavior. Therefore, it can be measured via behavior observation and questionnaires as well. One of the higher order factors in Rothbart's temperament theory and measure concerning self-regulation is effortful control, defined as the ability to inhibit a dominant response in order to activate a subdominant response to plan and to detect errors (Rothbart, Ellis, Rueda, & Posner, 2003). Posner and Rothbart (2006) have viewed this as a self-regulation mechanism that is an outcome of the development of the attention network (i.e., the neural substrate of effortful control). Three of the four components of effortful control (i.e., inhibitory control, attention focusing, and perceptual sensitivity) correspond with the three aspects of the attention network, conceptually and functionally. Inhibitory control, corresponding with the executive network, is defined as the capacity to plan and to suppress inappropriate approach responses under instructions or in novel or uncertain situations (Rothbart, Ahadi, Hershey, & Fisher, 2001).

Attention focusing reflects the alerting network because it is defined as the ability to maintain attention focus upon task-related channels. Perceptual sensitivity is defined as the amount of detection of slight, low intensity stimuli from the external environment and is related to orienting (Posner & Raichle, 1994). Rothbart et al. (2001) developed the Children's Behavior Questionnaire (CBQ) to assess children's temperament, including the above three aspects which reflect the underlying attention network.

Compared to cognitive assessment, a questionnaire approach has some advantage. The ANT is a one-session 20-minutes task. The results of the task may be influenced by many factors such as the children's moods on the day of the task or the unfamiliarity of the new environment. There has been evidence of low test-retest reliability in children's version of the ANT (Rueda, et al., 2004). However, the parent-report CBQ is a summary of a variety of child behaviors over a long period of time across many situations. Comparatively, CBQ may be a more reliable measurement of school-age children's behavior. Thus, in the current study, we used the CBQ to measure the three aspects of attention. The other advantage of using a questionnaire-based measure of attention is that it allows for different methods of assessment for the two major constructs (attention and working memory). This removes any effects of method (e.g. task, questionnaire) that, if used for both constructs in the same way, would artificially inflate correlations between the two constructs.

A measure of verbal ability also will be included in the analyses for the following reason. In working memory task, the understanding of the question and the production of the answer require a certain level of verbal ability. Also, children with higher verbal ability are more likely to exhibit better executive control and attentive behavior (Joseph, McGrath, & Tager-Flusberg, 2005). Taken together, the potential links between working memory and certain aspects of attention may be accounted for by expressive verbal ability, or more generally, verbal intelligence. Therefore, verbal ability will be included in the analyses to test for this possibility.

1.2 - Hypotheses 2 and 3: Genetic etiology of the link between working memory and attention

The corresponding functions and anatomical localizations between working memory and different subsystems of attention network that provide the foundation for the first hypothesis, lead to the following questions: what explains the association between working memory and different aspects of attention? Are there common sets of genes or shared environment factors

such as cognitive training that explain the corresponding functions of the two? The behavioral genetic design provides a tool to examine the genetic and environmental covariance between different aspects of attention and working memory, and implicates whether these sub-components of attention and working memory represent genetically similar or genetically distinct underlying mechanisms. However, to our knowledge, the links between working memory and different aspects of attention have not been examined by any study at the behavioral genetic level; traditionally, they have been studied separately.

Ando, Ono and Wright (2001) were the first to investigate individual differences in both the storage and executive functions in working memory using a behavioral genetic approach. Using a Japanese twin sample of 16-29 year olds, they found significant heritability estimates (i.e., the proportion of phenotypic variance accounted for by genetic differences among individuals) in individual differences in working memory in both spatial and verbal modalities (43%-49%) as well as significant non-shared environment estimates (i.e., the proportion of phenotypic variance explained by the environment factors that do not lead to similarity among family members; 35%-57%).

Kremen et al. (2008) examined both the storage and executive functions of working memory at the behavioral genetic level with a sample of 663 middle-aged males. They found that the storage (digit span) and the executive functions (digit transformation) of working memory were influenced by the same set of genes. There was an additive genetic influence accounting for 92% of the total covariance and a non-shared environment influence accounting for the other 7% of covariance between storage and executive working memory.

Note that although Ando et al. (2001) were the first to investigate individual differences in sub-components of working memory with behavioral genetic analyses, behavioral genetic studies of short-term memory (the storage function of working memory) have been conducted for decades. For example, in the examination of the origins of individual differences in short-term memory among elderly, Finkel and McGue (1993) used four different short-term memory tasks (word recall, immediate and delayed text recall and figure memory) and found that 56%-64% of the total variance in short-term memory performance was attributed to genetic factors and that the remaining 36%-44% was due to non-shared environment factors. Another study, using a Swedish twin sample, found that 32%-44% and 56%-68% of the total variance in performance in

short-term memory tasks could be attributed to genetic and non-shared environment influences, respectively (3 types of short-term memory tasks were used: digit span, names and faces memory and picture memory; Pedersen, Plomin, Nesselroade, & McCleam, 1992).

As for behavioral genetic studies of attention, Fan and colleagues estimated genetic variance in the three aspects of the attention network and found that the executive network was significantly heritable (Fan, Wu, Fossella, & Posner, 2001). Seventy-two percent of the variance in individual differences in the executive network was explained by genetic influence and the final 28% was explained by non-shared environmental influence. Only a small amount of variance in the alerting network was explained by heritability (18%) and all variance in the orienting network was explained by environmental factors. Yet, due to the small sample size used (26 pairs of twins in total) the study was underpowered to detect significant heritability.

Some other recent genetic studies on attention in children have focused on Attention Deficit Hyperactivity Disorder (ADHD). By looking into the studies in one subtype of ADHD, the predominantly inattentive type, one can get insight into potential genetic and environmental influences on attention. Sherman and colleagues found an average of 0.54 genetic effects, 0.19 shared environmental effects (i.e., the proportion of phenotypic variance explained by the environmental factors leading to the family similarity) and 0.27 non-shared environmental effects in individual differences in inattention in a sample of teenage boys (Sherman, Iacono, & McGue, 1997). Neuman et al. (1999) extended this finding in a sample of adolescent twin girls.

Some studies also have shown that genetic influences explained the stability of individual differences in attention across time. A previous analysis of the first two annual waves of data from the current study found a 0.39 bivariate heritability estimate in the longitudinal stability of attention span/task persistence (Deater-Deckard, Petrill, Thompson, & DeThorne , 2006) and another study found that 45-90% of the longitudinally stable total genetic variance in inattention was explained by stable genetic influences (Larsson, Lichtenstein, & Larsson, 2006).

Furthermore, at a molecular genetic level, genes for a dopamine transporter (DAT1), dopamine receptor D4 (DRD4), and dopamine receptor D5 (DRD5) have been found to be replicated candidate genes relating to attention in several studies, but with small effect sizes indicating weak associations (Faraone, Doyle, Mick, & Biederman, 2001; Li, Sham, Owen, & He, 2006). There have not been studies that have replicated the effect of any particular gene on working memory, let alone the association between working memory and attention. Therefore,

considering the limited progress in the molecular genetic studies on the two constructs, a behavioral genetic level of analysis seemed to be appropriate and informative, and will be used in the current study.

As summarized above, working memory and attention are both substantially influenced by genetic factors. However, we do not know much about the heritability of the different subsystems of attention. To our knowledge, the only behavioral genetic study that examined those three subsystems separately was underpowered (Fan et al., 2001). Therefore, one contribution of the current study is to estimate the genetic and non-genetic variance in different subsystems of attention. Based on prior behavioral genetic studies, the second hypothesis is that the variability in working memory, executive network, alerting network and orienting network of attention will be attributed to moderate sized heritable and nonshared environmental effects.

The most novel contribution of the current study is its examination of the etiology of the links between working memory and different aspects of attention. The third hypothesis is that there will be genetic covariance between working memory and executive attention, and between working memory and alerting attention. In contrast, genetic covariance between working memory and orienting is not expected, because as stated in the first hypothesis, the phenotypic correlation between working memory and orienting is expected to be zero.

In summary, as past studies have shown, the development of working memory and attention networks both are linked to children's intelligence, academic achievement and other social-emotional adjustment outcomes. Therefore, apart from the theoretical insight into the links between the two multi-faceted constructs, practically, this study will improve our understanding of early child education with respect to children whose academic performance and other social skills are impaired due to deficits in working memory and attention.

The three hypotheses of the current study are:

Hypothesis 1: Working memory will be positively correlated with both inhibitory control (executive network) and attention focusing (alerting network). Working memory will not be correlated with perceptual sensitivity (orienting network).

Hypothesis 2: Variability in working memory, inhibitory control, attention focusing, and perceptual sensitivity will be attributed to both heritable and nonshared environmental sources. Shared environmental variance will be negligible. There will be moderate genetic variance in all the above four constructs.

Hypothesis 3: There will be genetic covariance between working memory and inhibitory control, and between working memory and attention focusing. There will not be any genetic covariance between working memory and perceptual sensitivity.

2.0 - Method

2.1 - Participants

The data were from the second and third waves of the Western Reserve Reading Project (WRRP, a longitudinal twin study). On average, two consecutive waves were one year apart. Families were mainly recruited from the Cleveland and Cincinnati metropolitan areas. There were 131 monozygotic (MZ, 39% male) twin pairs and 173 same-sex dizygotic (DZ, 44% male) twin pairs in total. The average age in the first wave was 6.09 (SD = .69, range = 4.33-7.92). Ninety-two percent of the sample was Caucasian and 5% was black. There was a wide range of parental educational level: 12-17% high school or less, 23-27% some college, 31% bachelor's degree, and 25% some post-graduate education or degree. In the current study, three aspects of the attention network were assessed once (between wave2 and wave3), and working memory and verbal ability were assessed two times. There were 93 and 114 pairs of MZ and DZ twins that had complete data on attention, and 120 and 161 pairs of MZ and DZ twins that had complete data on working memory and verbal ability. There were 93 and 114 pairs of MZ and DZ twins that had complete data on all study variables.

2.2 - Procedure

In each of the two annual assessment waves, data were collected during a three-hour home visit. Before the assessments, informed consent was obtained and parents were informed that they would receive $100 for their participation. During the visits, parents and twins completed a series of tests including both behavioral and cognitive assessments. Parents' were invited to complete a supplemental temperamental questionnaire following the first annual home visit by mail.

2.3 - Measures

Attention

Children's Behavior Questionnaire (CBQ; Rothbart, 1981). The CBQ is a parent-report temperament measurement designed for children from three to seven-years old. It is rated on a 7-point Likert scale (l=extremely untrue of your child; 4=neither true nor false of your child; 7=extremely true of your child). The choice "not applicable" is provided in case that the children

are not observed in the situations described. The CBQ assesses fifteen dimensions, among which, we chose the inhibitory control, attention focusing and perceptual sensitivity dimensions in the current study. The CBQ has been reported in a number of studies to be an effective tool for assessing children's temperament, with reported Cronbach's αs from .67 to .94 (Ahadi, Rothbart, & Ye, 1993; Kochanska, Devet, Goldman, Murray, & Putnam, 1994). The Cronbach's αs in the current sample are from .60 to .87, similar to those found in previous studies. One sample item from the inhibitory control subscale is "can lower his/her voice when asked to do so". The attention focusing subscale contains items such as "has a hard time concentrating on an activity when there are distracting noises". One sample item from the perceptual sensitivity subscale is "notices the smoothness or roughness of objects s(he) touches." The mother and father reports were averaged to gain a more reliable score.

Working Memory & Verbal Ability

To test children's working memory and verbal ability, children completed the Stanford-Binet Intelligence Scale IV (SBIV; Thorndike, Hagen, & Sattler, 1986). SBIV is a standardized test that measures intelligence and cognitive abilities in children and adults. The internal consistency reliabilities range from 0.73 to 0.94 and the test-retest reliabilities from 0.56 to 0.78 for different subtests of SBIV (Thorndike et al., 1986). The forward digit span and the backward digit span sub-tests of the SBIV were averaged to measure children's working memory. The correlation was .60 between working memory scores across two waves. To yield a more reliable composite measure, working memory scores were averaged and then standardized for each child across two waves to yield a single working memory performance z-score. The expressive vocabulary sub-test was used to measure children's verbal ability. The correlation between age-standardized verbal scores across two waves was .46. To yield a more reliable composite score, they were averaged across two waves and standardized to derive a single vocabulary performance z-score.

2.4 - Analytic Strategy

Several behavioral genetic models were applied to examine the genetic and nongenetic (i.e. environment) variance of and covariance between working memory, inhibitory control, attention focusing, perceptual sensitivity, and verbal ability. In these models, the observed phenotypic variance and covariance were decomposed into latent genetic, shared environmental, and nonshared environmental variance and covariance. All the models were fit to twin

variance/covariance matrices using maximum likelihood model-fitting procedures in Mx (Neale, 1997). Root Means Square Error of Approximation (RMSEA; Steiger, 1990), Akaike's Information Criterion (AIC; Burnham & Anderson, 1992), and χ^2 difference test were used to select the best fitting model among all the competing models. A RMSEA value below .06 indicates a close fit, and between .06 and .10 indicates an adequate fit. The lower the AIC value, the better the model fits. When two models were nested, we used the χ^2 difference test, to balance the loss in fit with the difference in degrees of freedom.

A univariate behavioral genetic model was used to examine the genetic, shared, and nonshared environmental variance of the five variables (*Figure1*). The variance of each of the five observed variables were decomposed into latent additive genetic (A), shared environmental (C) and nonshared environmental (E) variance (ACE model). When there was potential nonadditive genetic effect (DZ twin correlations approach 0 or negative value), models that examined dominant genetic effect (ADE model) were tested. Due to the fact that monozygotic (MZ) twins share 100% of their genes and dizygotic (DZ) twins share 50% of their genes on average, the correlation for additive genetic similarity between twin 1 and twin 2 was set as 1 and 0.5 for MZ and DZ twins, respectively. The correlation for dominant genetic similarity between twin 1 and twin 2 was set as 1 and 0.25 for MZ and DZ twins, respectively. The correlation for shared environment (i.e. environmental factors that lead to twin similarity) between twin 1 and twin 2 was set as 1 for both MZ and DZ twins. The correlation for nonshared environment (i.e., environmental factors that do not lead to twin similarity) between twin 1 and twin 2 was set as 0 for both MZ and DZ twins.

To examine the genetic and environmental covariance between working memory, inhibitory control, attention focusing, perceptual sensitivity, and verbal ability, a series of bivariate and trivariate Cholesky decomposition models were applied (*Figure2 & Figure3*). Generally, in Cholesky Models, the variance of and covariance between variables were decomposed into additive genetic (A_i), shared environmental (C_i), and nonshared environmental covariance including error (E_i), as well as residual genetic (a), shared environmental (c), and nonshared environmental variance (e) (in the case of dominant genetic effect (d), they are A_i, D_i, E_i, a, d & e). The pathways between genetic, shared environmental, and nonshared environmental variance and covariance across twins were set in exactly the same manner as in the univariate models.

3.0 - Results

Descriptive statistics of and correlations between the study variables are shown in Table 1. Descriptive statistics of age standard scores for working memory and verbal ability are shown although the composite Z scores were used for other analyses. Scores were widely distributed, with only modest skewness (-.48 to.64) and kurtosis (-.15 to 1.08). We further explored the potential correlations between the study variables and child sex (0 = female, 1 = male), age, and zygosity (1 = MZ, 2 = DZ). For child sex, the only consistent correlation found across twin 1 and twin 2 was with perceptual sensitivity: $r = -.31$, $p<.001$ for twin 1 and $r = -.36$, $p<.001$ for twin 2, suggesting higher perceptual sensitivity scores for girls. None of the other variables showed consistently (across twins) significant correlations with child sex ($r = -.12$ to .02 for twin 1, $r = -.23$ to .02 for twin 2). For child age, there were no significant correlations between age and any of the five study variables consistently across twins ($r = -.18$ to .08 for twin 1, $r = -.11$ to .08 for twin 2). No significant correlation was found between study variables and zygosity ($r = -.03$ to .08 for twin 1, $r = -.13$ to .09 for twin 2).

3.1 - Hypothesis 1: Phenotypic Correlations

Correlations between study variables were also examined, separately for twin 1 and 2 sub-samples; ranges of correlations spanning both twins are presented. As shown in Table 1, correlations between the three attention scores were modest to moderate ($r = .27$ to .61). Consistent with the first hypothesis, working memory was positively correlated with attention focusing ($r = .27$ to .29) and inhibitory control ($r = .17$ to .21), but not with perceptual sensitivity ($r = -.10$ to .08). Similarly, verbal scores were positively correlated with attention focusing ($r = .14$ to .21) and inhibitory control ($r = .14$ to .18), but not with perceptual sensitivity ($r = -.06$ to .02). Finally, working memory was positively correlated with verbal scores ($r = .26$ to .30).

To control for the potential influence of verbal ability on the phenotypic correlations between working memory and different aspects of attention, partial correlations between study variables were also tested, controlling for verbal scores. These correlations are presented in Table 1. After controlling for verbal ability scores, working memory was still positively correlated with both inhibitory control ($r = .13$ to .17), and attention focusing ($r = .24$ to .25), but not with perceptual sensitivity ($r = -.08$ to .08), for both twin 1 and twin 2.

3.2 - Hypothesis 2: Genetic and Nonshared Environmental Variance

To test the second hypothesis, twin intra-class correlations for the study variables were estimated (Table 2). Correlations between MZ twin scores were all higher than DZ twins, suggesting that there was genetic variance in all five variables. However, the correlation between DZ twins on attention focusing was -.02, suggesting a potential sibling contrast effect (i.e. reporter contrasts one twin's behavior with the other twin and inflates sibling dissimilarity). Therefore, to minimize this problem, cases that had a likely sibling contrast effect were removed from further analysis, resulting in 7 pairs of DZ twins removed. This was done by computing an absolute difference score for each pair of twins on each parent-report variable and taking out the outliers from the difference score distributions. There was evidence of twin-difference outliers for several variables that were greater than 3.5 standard deviations above the mean difference score.

In order to estimate the genetic and environmental variance of the five study variables, univariate behavioral genetic models (ACE & ADE) were applied (*Figure 1*). Nested models were also tested by fixing certain paths at 0 (e.g., the C path was fixed at 0 in AE model, meaning that the shared environmental effect is negligible). The best fitting models were chosen to be presented for each variable, with model fit indices and variance estimates shown in Table 3. All of the best fitting models fit at least adequately (RMSEA = .000 to .071; AIC = -5.568 to -1.889). Consistent with the second hypothesis, there was moderate to substantial genetic variance in all five variables (h^2 = .55 to .78). Genetic variance appeared to be additive with only one exception: there was significant genetic dominance for attention focusing (d^2 = .55). As predicted in hypothesis 2, shared environmental variance was negligible for all variables, and there was modest to moderate nonshared environmental variance (e^2 = .22 to .45) in all study variables.

Statistical power. Power analysis was conducted using MX based on previous studies (Neale & Cardon, 1992; Schmitz, Cherny, & Fulker, 1998). At alpha = .05, given the sample sizes and the computed genetic effect sizes, the power estimates to detect additive genetic variance for inhibitory control, perceptual sensitivity, working memory, and verbal ability were: 1.00, .84, .94, and .64, respectively. Given the computed shared environment effect sizes, the power to detect shared environmental variance for the four variables was: .09, .50, .14, and .19, respectively. For attention focusing, the power to detect dominant genetic variance was .98; the

power to detect shared environmental variance was .10. Therefore, power was substantial (i.e., above .80) to detect genetic variance in all variables except verbal ability, but was very low to detect small shared environmental variance in all variables.

3.3 - Hypothesis 3: Genetic Covariance Patterns

To test the third hypothesis, a series of bivariate and trivariate Cholesky Decomposition models (*Figure 2 & Figure 3*) were estimated to probe the structure of the phenotypic correlations between the study variables. Shared environmental variance was not included in these analyses because it was negligible in all five variables in univariate analyses. For attention focusing, the DE model was the best fitting model, so it did not converge with the other four variables for which the AE model was the best fitting model. To address the discrepancy, a univariate AE model was examined for attention focusing, and it fit adequately: χ^2 (df, p) = 4.05 (3, .26), AIC = -1.950, RMSEA = .074. Therefore, the bivariate AE model was chosen to examine genetic and environmental correlations between each pair of the 5 variables.

Model fit indices, genetic and nonshared environmental correlations were shown in Table 4. All but one (i.e. PS-WM) of the bivariate models fit at least adequately (RMSEA = .000 to .093; AIC = -17.983 to -3.096). The poor fit for the PS-WM model (RMSEA = .106; AIC = -5.460) was expected because there was no covariance between perceptual sensitivity and working memory. Moderate genetic correlations were found between the three attention scales (r = .38 to .66). As predicted in hypothesis 3, inhibitory control and attention focusing, but not perceptual sensitivity, had genetic correlations with working memory (r = .25 to .45). The only significant nonshared environmental correlations found were between inhibitory control and attention focusing, and between inhibitory control and verbal ability.

Power analysis was again conducted in regard to detection of genetic correlations between each pair of variables. At alpha = .05, given the sample sizes, genetic variance found in univariate analysis, and the computed genetic correlation effect sizes, power to detect genetic correlations was sufficient (i.e. > .80) to detect most of the genetic correlations except for the one between inhibitory control and verbal ability. These bivariate (i.e., genetic correlation) power estimates are shown in the last column in Table 4.

In the final set of models in which the genetic and nonshared environmental correlations were estimated between inhibitory control, attention focusing, and working memory simultaneously, three trivariate Cholesky models were estimated, in which the order of the three

variables was iterated. Thus, depending on the analysis, the order of variables differed; as shown in *Figure 3*, phenotype 1, phenotype 2, and phenotype 3 were: inhibitory control, attention focusing, and working memory in model 1; attention focusing, working memory, and inhibitory control in model 2; working memory, inhibitory control, and attention focusing in model 3. Note also that verbal ability had a significant genetic correlation with working memory but none of the attention variables. Therefore, to remove any potential confounds with verbal ability, the verbal IQ score was regressed out of the working memory score, and the standardized residual of the working memory score was used.

Path estimates and model fit indices are shown in Table 5. All three models fit adequately (RMSEA = .052 to .067; AIC = -23.846 to -25.639). There was overlapping genetic variance across all three variables (i.e., A1 paths for IC, AF, and WM were all significant). After controlling for the genetic variance that was shared by all three variables, there was still genetic covariance remaining between attention focusing and working memory (i.e. A2 paths for AF and WM were significant in model 1), and between inhibitory control and attention focusing (i.e. A2 paths for IC and AF were both significant in model 3), but not between inhibitory control and working memory (i.e. A2 path for IC was not significant in model 2). After controlling for all the genetic covariances, there was residual genetic variance in all three variables (i.e., a3 paths in three models were all significant). Nonshared environmental covariance was only found between attention focusing and inhibitory control, as already reported above in regard to the bivariate analyses.

4.0 - Discussion

Attention and memory have been two of the most heavily investigated topics since the establishment of psychology as a scientific discipline (Ebbinghaus, 1964; James, 1890), but the rising interest in their biological foundations and the notion of taking them as inter-related aspects of cognitive self-regulation are very recent (Bell & Deater-Deckard, 2007). In the past three decades, evidence from both behavioral and neurological studies has indicated potential common mechanisms that are shared by working memory and some aspects of attention (Engle & Kane, 2004; Osaka, et al., 2007). Yet, one cannot infer, from the phenotypic correlations and the common neuronal anatomy, whether working memory and certain aspects of attention are the same construct or two separate constructs working conjointly, in terms of their genetic etiology. In the current study, an attempt was made to answer this question via a behavioral genetic

approach. The aim was to clarify the genetic as well as the environmental sources of overlapping and distinct influences for working memory and attention, and to further shed light on the theoretical organization of different aspects of cognitive self-regulation.

4.1 - Hypothesis 1: Phenotypic correlations

Three hypotheses concerning the links between different components of attention network and working memory were tested in the current study. The first hypothesis was that at the phenotypic level, working memory is positively correlated with both inhibitory control and attention focusing, but not perceptual sensitivity. The results supported this hypothesis: for both twin1 and twin 2, significant positive correlations were found between inhibitory control and working memory (.17 and .21), and between attention focusing and working memory (.29 and .27), but not between perceptual sensitivity and working memory (-.10 and .08). The pattern of the phenotypic correlations between working memory and three aspects of attention remained the same after removing the variance in verbal scores from the correlations. The phenotypic correlations above did not vary as a function of children's age: the three attention scores measured by the CBQ were not correlated with children's age; the working memory and verbal scores were age standardized scores, with the variance associated with age being removed. Previous studies on a group of children have found similar results, but with slightly larger effect sizes for correlations between inhibitory control and working memory, and between attention focusing and working memory (Wolfe & Bell, 2004; Wolfe & Bell, 2007; average correlations in .30 range).

One potential explanation for the smaller phenotypic effect sizes in the current study is the use of a digit span task (forward plus backward) in the current study as the sole working memory measure—an important caveat that is discussed in more detail below.

4.2 - Hypothesis 2: Genetic and nonshared environmental variance

The second hypothesis was concerning the genetic and environmental variability in working memory, inhibitory control, attention focusing and perceptual sensitivity. The result from univariate behavioral genetic analysis supported the second hypothesis: moderate genetic (.55 to .78) variance was found for all variables. Furthermore, the effect of shared environment was negligible with all environmental variance being nonshared (.22 to .45). The patterns of

variance decomposition and the effect sizes were much like previous studies (Goldsmith, Buss, & Lemery, 1997), which highlighted the importance of genetic influence on both working memory and three aspects of attention.

The genetic effect appeared to be additive for all variables except attention focusing. For attention focusing, although the dominant genetic model fit adequately, the possibility of potential sibling contrast effects could not be ruled out. In fact, previous studies have shown that the low DZ correlation found in temperament measures is in part due to sibling contrast effect(Saudino, Cherny, & Plomin, 2000), and in part due to genetic dominance effects (Mullineaux, Deater-Deckard, Petrill, Thompson, & DeThorne, 2009). Therefore, in the following multivariate analyses, the additive genetic model, which also fit adequately, was used for attention focusing.

4.3 - Hypothesis 3: Genetic covariance patterns

Though informative, phenotypic correlation and univariate behavioral genetic analysis do not address how genetic and environmental factors influence the associations between working memory and different components of attention network. The third hypothesis was that there would be genetic covariance between working memory and inhibitory control, and between working memory and attention focusing. In contrast, genetic covariance between working memory and perceptual sensitivity was not expected.

Bivariate and trivariate behavioral genetic analyses were applied to obtain genetic and environmental covariance between working memory and attention. The bivariate behavioral genetic analyses showed that the correlations between working memory and inhibitory control as well as between working memory and attention focusing were all through genetic influence (i.e., their nonshared environmental correlations approached 0). This result supported the third hypothesis of the current study and suggested a common genetic mechanism or mechanisms on working memory and executive attention, as well as working memory and focusing attention. The simplest explanation for these results is that there is a common underlying set of genetic factors that account for the overlap between attention and working memory performance.

Could it be that the genetic covariance between working memory and certain aspects of attention simply represents the underlying genetic mechanisms on verbal ability, or more generally, intelligence? This potential explanation was examined in the current study. The results did not support this explanation: neither the genetic correlation between executive attention and

verbal ability, nor the one between attention focusing and verbal ability, was significant. Therefore, the common genetic mechanism found between working memory and aspects of attention probably is not attributable to genetic influences on verbal ability or "intelligence" more generally.

However, verbal ability was correlated with working memory completely through overlapping genetic influences (i.e., through genetic correlation). This suggests that the genetic variance in working memory as measured by a digit span task, also accounts for genetic variance in expressive verbal ability. To control for this verbal ability confound and to test for a more "generalizable relation" between working memory and attention, verbal ability was regressed out of working memory scores for the trivariate analyses between inhibitory control, attention focusing, and working memory.

The trivariate behavioral genetic analyses showed that there was common genetic variance shared by working memory, inhibitory control and attention focusing. This result supports the theory that working memory and certain aspects of attention are partially the same construct (Baddeley & Hitch, 1974). As this theory states, effortful attention (i.e. executive attention and maintenance of alerting state) is an essential part of working memory, responsible for keeping an active state of central information and monitoring potential interference, be it verbal or spatial.

However, these three aspects (working memory, inhibitory control, and attention focusing) are not "just" the same thing, at the behavioral genetic level of analysis. After controlling for the genetic covariance between them, there were still moderate amounts of genetic variance remaining that was specific to each of the three constructs. This evidence for "unique" genetic variance may indicate that individual differences in working memory, executive attention, and attention focusing—although partially arising from overlapping genetic influences—may include other genetic components that are independent of each other.

The unique genetic influences that are specific to working memory, executive attention, and attention focusing might also be age-specific, and as a result, may change across the lifespan. Among all cognitive abilities, the development of executive abilities such as working memory and executive attention undergoes the longest period of time to reach full maturation (Kostovic, Skavic, Strinovic, 1988). During this prolonged period of time, the brain undergoes a great deal of change and rearrangement of connections between cortexes (Diamond, 2002). By 20 years of

age, the underlying genetic mechanisms of these fully developed executive abilities and the links between them probably have changed, and may even be different from the ones at 6 or 7-years of age. Studies have supported this explanation by showing a strong developmental shift from lower to higher levels of genetic influence on attention from 4 to 8 years (Deater-Deckard, Petrill, Thompson, & DeThorne, 2005; Deater-Deckard & Wang, in press). In fact, it has been shown that genetic influence on cognitive abilities grows in magnitude over time (McCartney, Harris, Bernieri, 1990; Plomin, Fulker, Corley, & Defries, 1997). At the same time, the phenotypic correlation between working memory and executive attention has been found to be slightly stronger in adults than in school age children ($r = .24$ in school-age children vs. $r = .35$ in adults; Redick & Engle, 2006). These studies, although not sufficiently on their own, provide preliminary support for the possibility of a developmental increase in the genetic associations between working memory and aspects of attention. These semi-independent abilities may be better integrated by the age of 20, and perhaps even develop into one substantially genetic-dependent master mechanism.

4.4 - Limitations and future directions

The current study examined the genetic and environmental variance links between working memory and different aspects of the attention network. However, the measures used in the current study, due to the limitation of secondary data analysis, were not ideal. As previously mentioned, forward digit span task mostly measures short-term memory (i.e. the storage component of working memory, Engle & Kane, 2004); it may not capture the executive component of working memory, leading to potential deflation in the phenotypic correlation with attention. In addition, performance on backward digit span, although improving in school-age children compared to early childhood, is relatively poor (i.e. about 3 digits on average); performance does not reach adult levels until about age 13 (Diamond, 2002). Therefore, backward digit span task may not be an age-appropriate measurement for executive memory in school-age children. In fact, in the current study, the correlation between forward and backward digit span task is .26, whereas in adults (from age 16-79), the correlation is in the .5 to .6 range (Gregoire & Van der Linden, 1997). The difference in correlations between forward and backward digit span in school-age children and adults provides a support for the above explanation, and implies the drawback of using backward digit span to measure working memory

in school-age children. The limitation in performance in backward digit span task may have resulted in reduced variance in working memory scores, and thus a reduction in the covariance between working memory and attention.

Another concern is that working memory, inhibitory control, and attention focusing were all positively correlated with verbal ability for both twin1 and twin 2. These positive correlations suggested that parent-report child's attentive behaviors and performance in working memory task measured by digit span may rely on expressive verbal ability. The links between working memory and certain aspects of attention may simply be a reflection of this common influence of verbal ability. The correlation between working memory and verbal ability could also be artificial— a shared method effect—since the verbal and digit span tasks were measured using Stanford-Binet test. Further analyses at the behavioral genetic level examined these possibilities, with the logic being that any method effect should function the same way for MZ and DZ twins and emerge as shared environmental variance and covariance. No such shared environment variance or covariance effects were detected, thus ruling out method bias as a likely alternative explanation for the results.

As to the measurement of attention, compared to cognitive tasks, they may not be direct measures of the cognitive functions and underlying neural activities involved (Posner & Rothbart, 2006). This limitation in measurement may have an impact in the acquired effect sizes of the correlations; other studies with more direct and age-appropriate measurement of attention and working memory have found stronger correlations between working memory, executive attention, and focusing attention in children (Wolfe & Bell, 2007) than were found in the current study. However, the use of questionnaire removes any effects of method (e.g. task, questionnaire) that, if used for both constructs in the same way, would artificially inflate correlations between the two constructs. Therefore, in future studies, age-appropriate cognitive task and questionnaire measures of attention should be used together to examine the above measurement issues.

Another limitation is that the sole use of a twin design is underpowered for detecting small shared environmental effects. This limitation makes it difficult to tell, for sure, whether or not shared environmental effects contribute to the association between working memory and aspects of attention. It would be a stronger design if other sibling pairs with additional genetic relatedness information, such as adoptive sibling pairs, were included (Plomin, DeFries, McClearn, & McGuffin, 2008). With more power for detecting shared environment afforded by

the inclusion of adoptive, step- and half-sibling pairs in future studies, it will be exciting to see whether or not the associations between working memory and aspects of attention are mediated through shared environmental effects as well as genetic effects.

It is also a pity that there was not enough data to examine the links between working memory, executive attention, and attention focusing longitudinally, especially during the age of 7-15 years. During this transition to adolescence, the brain structures and brain activities (i.e. prefrontal cortex) that are thought to relate to these cognitive abilities are undergoing rapid development (Huttenlocher & Dabholkar, 1997; Sowell et al, 1999). Based on the evidence of increasing genetic influence on cognitive abilities such as executive functions, and the increasing phenotypic correlations between them, it would be intriguing to answer the question of how the overlapping genetic mechanisms influence the integration of different aspects of cognitive self-regulation developmentally.

Another exciting future direction would be to examine potential candidate genes explaining the common mechanisms between working memory, executive attention and attention focusing. Behavioral genetic studies are informative in terms of exploring potential genetic relations between different aspects of human behavior, but they do not provide information on what exact underlying genetic mechanisms support these relations. Ultimately, it is molecular genetic studies that will answer these questions regarding which specific sets of genes are responsible for linking different aspects of cognitive self-regulation together. It is our goal to eventually map these variances and covariances onto the structural variations in DNA that account for the variances and covariances. To this end, dopamine and serotonin transporter and receptor genes would be good candidates. Variation in dopamine receptor 4 gene (DRD4) has been associated with attention behaviors across many studies (Faraone et al, 2001; Li et al, 2006), and may also help explain the development increase in heritable variance in the transition to middle childhood and beyond (Deater-Decard & Wang, in press). In regard to the serotonin neurotransmitter system, studies have established the important role that it plays in self-regulation processes (Canli, et al, 2005; Krakowski, 2003; Reuter, Ott, Vaitl, & Hennig, 2007). In particular, variation in serotonin transporter gene 5-HTT and its synthesis rate-regulating enzyme tryptophan hydroxylase (TPH) gene has been associated with attention regulation.

5.0 - Conclusions

Consider the question: “Can attention and working memory really be distinguished from one another?” Diamond (2002, pp. 492) gave us a hint:

> The difference is, in part, merely semantic—one can say that information is held in working memory for several seconds or that focused attention on the information is sustained for several seconds, and they mean the same thing. The same PFC system that enables us to selectively keep our mind focused on the information we want to hold in mind also helps us to selectively attend to stimuli in our environment (turning out irrelevant stimuli).

The current study partially supported this argument: in middle childhood, parts of working memory, executive attention and attention focusing shared a common source of underlying genetic covariance that accounts for all the associations between them. More behavioral genetic studies are needed to replicate and extend the findings in various age groups. Future molecular genetic studies are needed to map the associations on measurable structural variations in DNA, and to explain how, through genetic influence, different brain areas and activities are organized to integrate different aspects of cognitive self-regulation. The current study’s results would suggest that when they are identified, many of the genetic influences that operate on working memory will also operate on some but not all aspects of the attention network. Furthermore, there will be ample variance arising from non-genetic influences, but these will not explain any of the within-family similarity in these cognitive self-regulation skills.

References

Ahadi, S. A., Rothbart, M. K., & Ye, R. M. (1993). Children's temperament in the US and China: Similarities and differences. *European Journal of Personality, 7,* 359-377.

Ando, J., Ono, Y., & Wright, M. J. (2001). Genetic structure of spatial and verbal working memory. *Behavior Genetics, 31,* 615-624.

Baddeley, A. D. (1986). *Working memory.* Oxford, UK: Clarendon Press.

Baddeley, A. D. (2000). The episodic buffer: A new component of working memory? *Trends in Cognitive Sciences, 4,* 417-423.

Baddeley, A. D. (2003). Working memory: Looking back and looking forward. *Nature Reviews Neuroscience, 4,* 829-839.

Baddeley, A. D., & Hitch, G. J. (1974). Working memory. In G. Bower (Ed.), *The psychology of learning and motivation* (Vol.8, pp.47-90). San Diego, CA: Academic Press.

Baddeley, A. D., & Hitch, G. J. (1994). Developments in the concept of working memory. *Neuropsychology, 8,* 485-493.

Barkley, R. (1997). *ADHD and the nature of self-control.* New York: Guilford.

Bell, M. A., & Deater-Deckard, K. (2007). Biological systems and the development of self-regulation: Integrating behavior, genetics, and psychophysiology. *Journal of Developmental and Behavioral Pediatrics, 28(5),* 409-420.

Bramlett, R. K., Scott, P. L., & Rowell, R. K. (2000). A comparison of temperament and social skills in predicting academic performance in first graders. *Special Services in Schools, 16,* 147-158.

Buckner, J. C., Mezzacappa, E., & Beardslee, W. R. (2009). Self-regulation and its relations to adaptive functioning in low income youths. *American Journal of Orthopsychiatry, 79,* 19-30.

Burnham, K. P., & Anderson, D. R. (1992). Data-based selection of an appropriate biological model: The key to modern data analysis. In D. R. McCullough & R. H. Barrett (Eds.), *Wildlife 2001: Population* (pp. 16-30). London: Elsevier.

Canli, T., Omura, K., Haas, B. W., Fallgatter, A., Todd, R., Constable, R. T., et al, (2005). Beyond affect: A role for genetic variation of the serotonin transporter in neural activattion during a cognitive attention task. *Proceedings of the National Academy of Sciences, 102,* 12224-12229.

Cowan, N., Elliott, E. M., Saults, J. S., Morey, C. C., Mattox, S., Hismjatullina, A., et al. (2005). On the capacity of attention: Its estimation and its role in working memory and cognitive aptitudes. *Cognitive Psychology, 51,* 42-100.

Deater-Deckard, K., Petrill, S. A., Thompson, L. A., & DeThorne, L. S. (2006). A longitudinal behavioral genetic analysis of task persistence. *Developmental Science, 9,* 498-504.

Deater-Deckard, K., & Wang, Z. (in press). Development of temperament and attention: Behavioral genetic approaches. Chapter to appear in M. Posner (Ed.), *Cognitive neuroscience of attention.* New York: Guildford.

Desimone, R. (1998). Visual attention mediated by biased competition in extrastriate visual cortex. *Philosophical Transactions of the Royal Society B: Biological Sciences, 353,* 1245-1255.

Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function*. Oxford: University Press.

Ebbinghaus, H. (1964). *Memory: A contribution to experimental psychology* (H. A. ruger & C. E., Bussenius, Trans.). New York: Dover Publications, Inc. (Originally published, 1885).

Eisenberg, N., Fabes, R. A., Shepard, S. A., Murphy, B. C., Guthrie, I. K., Jones, S., et al. (1997). Contemporaneous and longitudinal prediction of children's social functioning from regulation and emotionality. *Child Development, 68,* 642-664.

Engle, R. W., & Kane, M. J. (2004). Executive attention, working memory capacity, and a two-factor theory of cognitive control. *The Psychology of Learning and Motivation, 44,* 145-199.

Fan, J., Wu., Y., Fossella, J. A., & Posner, M. I. (2001). Assessing the heritability of attentional networks. *BMC Neuroscience, 2,* 14.

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of CognitiveNeuroscience, 14,* 340-347.

Faraone, S. V., Doyle, A. E., Mick, E., & Biederman, J. (2001). Meta-analysis of the association between the 7-repeat allele of the dopamine d(4) receptor gene and attention deficit hyperactivity disorder. *American Journal of Psychiatry, 158,* 1052-1057.

Finkel, D., & McGue, M. (1993). The origins of individual differences in memory among the elderly: A behavior genetic analysis. *Psychology and Aging, 8,* 527-537.

Goldsmith, H. H., Buss, K. A., & Lemery, K. S. (1997). Toddler and childhood temperament: Expanded content, stronger genetic evidence, new evidence for the importance of environment. *Developmental Psychology, 33(6),* 891-905.

Gregoire, J., & Van der Linden, M. (1997). Effect of age on forward and backward digit spans. *Aging, Neuropsychology, and Cognition, 4(2),* 140-149.

Hayslip, B., & Kennelly, K. J. (1982). Short-term memory and crystallized-fluid intelligence in adulthood. *Research on Aging, 4,* 314-332.

Huttenlocher, P. R., & Dabholkar, A. S. (1997). Regional differences in synaptogenesis in human cerebral cortex. *The Journal of Comparative Neurology, 387(2),* 167-178.

James, W. (1890). *Principles of Psychology, Vol. 1.* New York: Holt.

Johnson, M. H., Posner, M. I., & Rothbart, M. K. (1991). Components of visual orienting in early infancy: Contingency learning, anticipatory looking and disengaging. *Journal of Cognitive Neuroscience, 3,* 335-344.

Joseph, R. M., McGrath, L. M., & Tager-Flusberg, H. (2005). Executive dysfunction and its relation to language ability in verbal school-age children with autism. *Developmental Neuropsychology, 27(3),* 361-378.

Kane, M. J., Bleckley, M. K., Conway, A. R. A., & Engle, R.W. (2001). A controlled- attention view of working memory capacity. *Journal of Experimental Psychology, 130,* 169-183.

Kane, M. J., & Engle, R. W. (2002). The role of prefrontal cortex in working-memory capacity, executive attention, and general fluid intelligence: An individual-differences perspective. *Psychonomic Bulletin & Review, 9,* 637-671.

Kochanska, G., Devet, K., Goldman, M., Murray, M., & Putnam, S. (1994). Maternal reports of conscience, development, and temperament in young children. *Child Development, 65,* 852-868.

Kostovic, I., Skavic, J., & Strinovic, D. (1988). Acetylcholinesterase in the human frontal associative cortex during the period of cognitive development: Early laminar shifts and later innervations of pyramidal neurons. *Neuroscience Letters, 90,* 107-112.

Krakowski, M. (2003). Violence and serotonin: Influence of impulse control, affect regulation, and social functioning. *The Journal of Neuropsychiatry and Clinical Neurosciences, 15(3),* 294-305.

Kremen, W. S., Xian, H., Jacobson, K. C., Eaves, L. J., Franz, C. E., Panizzon, M. S., et al. (2008). Storage and executive components of working memory: Integrating cognitive psychology and behavior genetics in the study of aging. *Journal of Gerontology, 63B,* 84-91.

Larsson, H., Lichtenstein, P., & Larsson, J. (2006). Genetic Contributions to the development of ADHD subtypes from childhood to adolescence. *Journal of American Academy of Child and Adolescent Psychiatry, 45,* 973-981.

Li, D., Sham, P. C., Owen, M. J., & He, L. (2006). Meta-analysis shows significant association between dopamine system genes and attention deficit hyperactivity disorder (ADHD). *Human Molecular Genetics, 15,* 2276-2284.

McCartney, K., Harris, M. J., & Bernieri, F. (1990). Growing up and growing apart: A developmental meta-analysis of twin studies. *Psychological Bulletin, 107,* 226-237.

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

Neal, M. C. (1997). *Mx: Statistical modeling*. Richmond, VA: Department of Psychiatry, Medical College of Virginia.

Neale, M. C., & Cardon, L. R. (1992). *Methodology for genetic studies of twins and families.* Dordrecht, Netherlands: Kluwer Academic Publishers.

Neuman, R. J., Todd, R. D., Heath, A. C., Reich, W., Hudziak, J. J., Bucholz, K. K., et al. (1999). Evaluation of ADHD typology in three contrasting samples: A latent class approach. *Journal of the American Academy of Adolescent Psychiatry, 38,* 25-33.

Norman, D. A., & Shallice, T. (1980). *Attention to action: Willed and automatic control of behavior.* University of California San Diego CHIP Report 99.

Osaka, M., Komori, M., Morishita, M., & Osaka, N. (2007). Neural bases of focusing attention in working memory: An fMRI study based on group differences. *Cognitive, Affective, & Behavioral Neuroscience, 7,* 130-139.

Osaka, M., Osaka, N., Kondo, H., Morishita, M., Fukuyama, H., Aso, T., et al. (2003). The neural basis of individual differences in working memory capacity: An fMRI study. *NeuroImage, 18,* 789-797.

Osaka, N., Osaka, M., Kondo,H., Morishita, M., Fukuyama, H., & Shibasaki, H. (2004). The neural basis of executive function in working memory: An fMRI study based on individual differences. *NeuroImage, 21,* 623-631.

Pedersen, N. L., Plomin, R., Nesselroade, J. R., & McClearn, G. E. (1992). A quantitative genetic analysis of cognitive abilities during the second half of the life span. *Psychological Science, 3,* 346-353.

Plomin, R., DeFries, J. C., McClearn, G. E., & McGuffin, P. M. (2008). *Behavioral genetics*. NY: Worth Publishers.

Plomin, R., Fulker, D. W., Corley, R., & DeFries, J. C. (1997). Nature, nurture and cognitive development from 1 to 16 years: A parent-offspring adoption study. *Psychological Science, 8,* 442-447.

Ponitz, C. C., McClelIand, M. M., Matthews, S., & Morrison, F. K. (2009). A structured observation of behavioral self-regulation and its contribution to kindergarten outcomes. *Developmental Psychology, 45,* 605-619.

Posner, M. I., & Petersen, S. E. (1990). The attention systems of the human brain. *Annual Review of Neuroscience, 13,* 25-42.

Posner, M. I., & Raichle, M. E. (1994). *Images of the mind*. New York: Scientific American Library.

Posner, M. I., & Rothbart, M. K. (2006). *Educating the human brain.* Washington, DC: American Psychological Association.

Redick, T. S., & Engle, R. W. (2006). Working memory capacity and attention network test performance. *Applied Cognitive Psychology, 20,* 713-721.

Rueda, M. R., Fan, J., McCandliss, B. D., Halparin, J. D., Gruber, D. B., Lercari, L., P., et al. (2004). Development of attentional networks in childhood. *Neuropsychologia, 42,* 1029-1040.

Reuter, M., Ott, U., Vaitl, D., & Hennig, J. (2007). Impaired executive control is associated with a variation in the promoter region of the tryptophan hydroxylase 2 gene. *Journal of Cognitive Neuroscience, 19,* 401-408.

Reynolds, M., & Richards, J. E. (2003). *Infant attention and cortical sources of ERP for recognition of briefly presented visual stimuli*. Poster presented at the biennial meeting of the Society for Research in Child Development, Tampa, FL.

Richards, J. E. (1995). Infant cognitive psychophysiology: Normal development and implications for abnormal developmental outcomes. In T. H. Ollendick & R. J. Prinz (Eds.), *Advances in clinical child psychology* (Vol. 17, pp. 77-107). New York: Plenum Press.

Richards, J. E. (2004). The development of sustained attention in infants. In: M. I. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 342-356). New York: Guildford Press.

Rothbart, M. K. (1981). Measurement of temperament in infancy. *Child Development, 52,* 569-578.

Rothbart, M. K., Ahadi, S. A., Hershey, K. L., & Fisher, P. (2001). Investigation of temperament at three to seven years: The children's behavior questionnaire. *Child Development, 72,* 1394-1408.

Rothbart, M. K., Ellis, L. K., Rueda, M. R., & Posner, M. I. (2003). Developing Mechanisms of temperamental effortful control. *Journal of Personality, 71,* 1113-1144.

Saudino, K. J., Cherny, S., & Plomin, R. (2000). Parent ratings of temperament in twins: Explaining the 'too low' DZ correlations. *Twin Research, 3(4),* 224-233.

Schmitz, S., Cherny, S. S., & Fulker, D. W. (1998). Increase in power through multivariate analysis. *Behavior Genetics, 28,* 357-363.

Sherman, D. K., Iacono, W. G., & McGue, M. K. (1997). Attention-Deficit Hyperactivity Disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 36,* 745-753.

Silver, H., & Feldman, P. (2005). Evidence for sustained attention and working memory in schizophrenia sharing a common mechanism. *The Journal of Neuropsychiatry and Clinical Neuroscience, 17,* 391-398.

Smith, L. E., Borkowski, J. G., & Whitman, T. L. (2008). From reading readiness to reading competence: The role of self-regulation in at-risk children. *Scientific Studies of Reading, 12,* 131-152.

Sowell, E. R., Thompson, P. M., Holmes, C. J., Batth, R., Jernigan, T. L., & Toga, A. W. (1999). Localizing age-related changes in brain structure between childhood and adolescence using statistical parametric mapping. *NeuroImage, 9,* 587-597.

Steiger, J. H. (1990). Structural model evaluation and modification: An interval estimation approach. *Multivariate Behavioral Research, 20,* 173-180.

Thorndike, R. L., Hagen, E. P., & Sattler, J. M. (1986). *Guide for administering and scoring the fourth edition: Stanford-Binet Intelligence Scale*. Chicago, IL: Riverside.

Turner, M. L., & Engle, R. W. (1989). Is working memory capacity task dependent? *Journal of Memory and Language, 28,* 127-154.

Verhaegen, P., Marcoen, A., & Goossens, L. (1993). Facts and fiction about memory aging: A quantitative integration of research findings. *Journal of Gerontology, 48(4),* 157-171.

Wiergersman, S., & Meerste, K. (1990). Subjective ordering, working memory, and aging. *Experimental Aging Research, 16,* 73-77.

Wolfe, C. D., & Bell, M. A. (2004). Working memory and inhibitory control in early childhood: Contributions from physiology, temperament, and language. *Developmental Psychobiology, 44,* 68-83.

Wolfe, C., & Bell, M. A. (2007). Sources of variability in working memory in early childhood: A consideration of age, temperament, language, and brain electrical activity. *Cognitive Development, 22,* 431-455.

Table 1. *Descriptive statistics and correlations*

	Inhibitory control (IC)	Attention focusing(AF)	Perceptual sensitivity (PS)	Working memory (WM)	Verbal ability (VB)
Twin1 IC	5.07(.81)				
Twin1 AF	.61***/.60***	5.02(.86)			
Twin1 PS	.30***/.31***	.27***/.29***	5.41(.79)		
Twin1 WM	.17*/.13*	.29***/.24***	-.10/-.08	52.57 (5. 10)	
Twin1 VB	.15*	.22**	-.06	.30***	47.58(5. 50)
Twin2 IC	5.03 (.86)				
Twin2 AF	.57***/.56***	4.97 (.97)			
Twin2 PS	.40***/.41***	.31***/.31***	5.33 (.84)		
Twin2 WM	.21**/.17*	.27***/.25***	.08/.08	52.54 (4.87)	
Twin2 VB	.18*	.14*	.02	.26***	47.37 (5.22)

Note: Numbers on-diagonal are means (standard deviations) of the studied variables. Numbers off-diagonal are correlations between study variables/ partial correlations between study variables controlling for verbal ability. N = 207. * $p < .05$. ** $p < .01$. *** $p < .001$.

Table 2. *Intra-class correlations for MZ and DZ twins with and without sibling contrast outliers*

		MZ	DZ
IC	(full sample)	.76***(n=93)	.18** (n=114)
	(outliers removed)	n/a	.33***(n=110)
AF	(full sample)	.56***(n=93)	-.02 (n=114)
	(outliers removed)	n/a	.08 (n=112)
PS	(full sample)	.75***(n=93)	.52***(n=114)
	(outliers removed)	n/a	.55***(n=113)
WM	(full sample)	.66***(n=120)	.39***(n=161)
	(outliers removed)	n/a	n/a
VB	(full sample)	.56***(n=120)	.36*** (n=161)
	(outliers removed)	n/a	n/a

Note: correlations are shown with/without contrasted siblings; n represents number of twin pairs ** $p<.01$. *** $p<.001$, n/a = not applicable. See Table 1 for variable abbreviations.

Table 3. *Univariate models: genetic, shared environmental, and nonshared environmental variance estimates (with 95% confidence intervals), best fitting model, and model fit indices*

	a^2	d^2	c^2	e^2	Model	χ^2 (df,p)	AIC	RMSEA
IC	.74 (.65-.81)	–	–	.26 (.19-.35)	AE	.53 (3, .91)	-5.469	.000
AF	–	.55 (.40-.66)	–	.45 (.34-.60)	DE	.43 (3, .93)	-5.568	.000
PS	.78(.70-.84)	–	–	.22 (.16-.30)	AE	4.11 (3, .25)	-1.889	.071
WM	.65 (.55-.72)	–	–	.35 (.28-.45)	AE	3.49 (3, .32)	-2.514	.070
VB	.57 (.46-.66)	–	–	.43 (.34-.54)	AE	1.53 (3, .68)	-4.472	.021

Note: a^2: additive genetic variance; d^2: dominant genetic variance; c^2: shared environmental variance; e^2: nonshared environmental variance. See Table 1 for variable abbreviations.

Table 4. *Bivariate models: genetic and nonshared environmental correlations and model fit indices*

	r	r_a	r_e	χ^2 (df,p)	AIC	RMSEA	Power for r_a
IC-AF	.60*	.66*	.54*	7.37 (10, .69)	-12.634	.033	1.00
IC-PS	.34*	.38*	.14	14.23 (10, .09)	-5.767	.087	1.00
AF-PS	.30*	.44*	.05	16.90 (10, .08)	-3.096	.093	1.00
IC-WM	.17*	.25*	.00	8.85 (10, .55)	-11.148	.065	.99
AF-WM	.28*	.45*	.07	10.25 (10, .42)	-9.749	.077	1.00
PS-WM	.01	.02	.00	14.54 (10, .15)	-5.460	.106	.96
IC-VB	.17*	.15	.19*	2.02 (10, 1.00)	-17.983	.000	.58
AF-VB	.18*	.20	.14	8.82 (10, .55)	-11.180	.050	.93
PS-VB	-.03	.00	.09	8.42 (10, .59)	-11.585	.053	.83
WM-VB	.27*	.46*	.00	10.24 (10, .42)	-9.765	.071	1.00

Note: r: phenotypic correlation; r_a: genetic correlation; r_e: nonshared environment correlation; * $p < .05$. See Table 1 for variable abbreviations.

Table 5. *Path estimates (with 95% confidence intervals) and model fit indices for trivariate Cholesky Decompostion*

Variable order	A1	A2	a3	E1	E2	e3
IC	**.68 (.61-.76)**	–	–	**.41 (.36-.47)**	–	–
AF	**.40 (.28-.52)**	**.47 (.35-.57)**	–	**.36 (.24-.48)**	**.54 (.47-.62)**	–
WM	**.15 (.03-.27)**	**.25 (.07-.44)**	**.63 (.48-.74)**	.00 (.00-.08)	.05 (.00-.17)	**.67 (.59-.76)**
Model 1: χ^2 (*df*, *p*) = 16.36 (21, .75); AIC = -25.639; RMSEA = .052.						
AF	**.63 (.51-.74)**	–	–	**.64 (.56-.73)**	–	–
WM	**.28 (.13-.44)**	**.63 (.50-.74)**	–	.02 (.00-.14)	**.67 (.59-.76)**	–
IC	**.43 (.31-.55)**	.00 (.00-.08)	**.52 (.45-.60)**	**.23 (.16-.31)**	.00 (.00-.05)	**.34 (.30-.39)**
Model 2: χ^2 (*df*, *p*) = 18.15 (21, .64); AIC = -23.846; RMSEA = .067.						
WM	**.69 (.58-.80)**	–	–	**.67 (.59-.76)**	–	–
IC	**.15 (.03-.27)**	**.67 (.59-.75)**	–	.00 (.00-.05)	**.41 (.36-.47)**	–
AF	**.26 (.12-.40)**	**.36 (.23-.48)**	**.43 (.29-.54)**	.04 (.00-.14)	**.36 (.24-.48)**	**.54 (.47-.62)**
Model 3: χ^2 (*df*, *p*) = 16.36 (21, .75); AIC = -25.639; RMSEA = .052.						

Note: significant paths (p<.05) are shown in bold. See Table 1 for variable abbreviations.

Figure 1. *Univariate Behavioral Genetic Models*

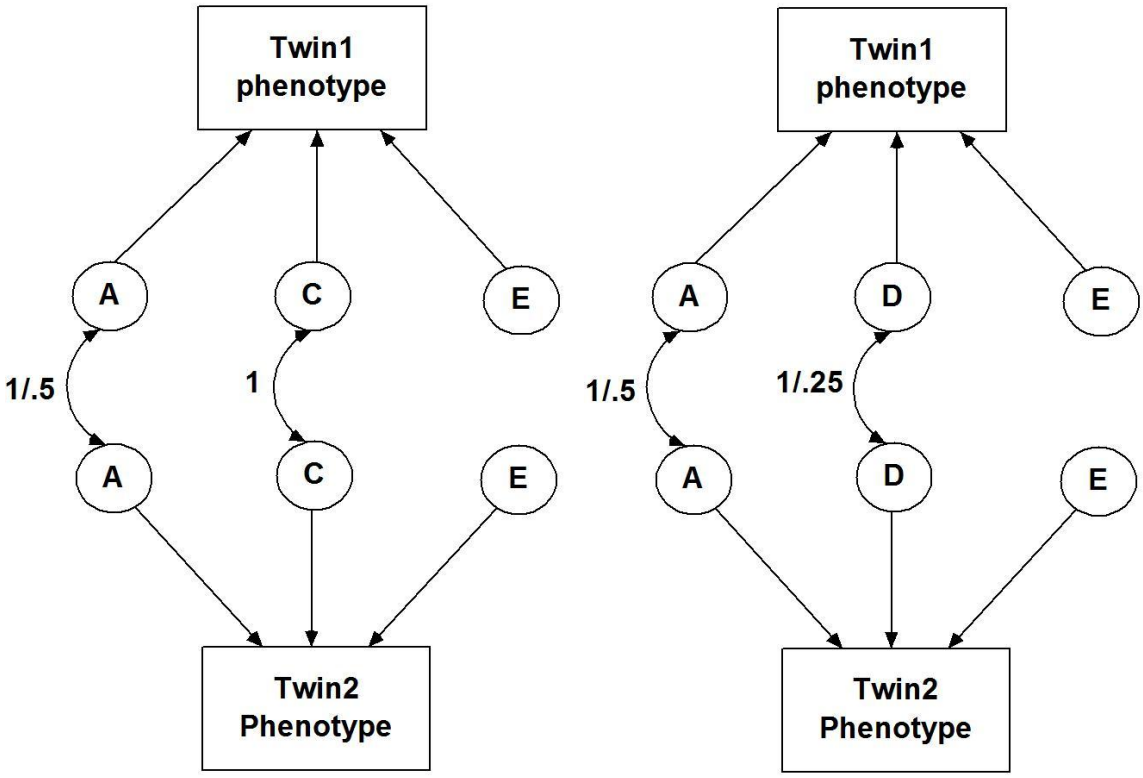

Figure 1. Univariate behavioral genetic models were used to examine the genetic and environemental variance in inhibitory control, attention focusing, perceptual sensitivity, working memory and verbal ability. Latent variables represent additive genetic variance (A), dominant genetic effect (D), shared environmental variance (C), and nonshared environmental variance including error (E). The correlations between latent variables representing additive genetic variance across twins were set at 1 for monozygotic (MZ) twins and .5 for dizygotic (DZ) twins. The correlations between dominant genetic variance across twins were set at 1 for MZ twins and .25 for DZ twins. The correlations for shared environmental variance across twins were set at 1 for both MZ and DZ twins, whereas the correlations for nonshared environmental variance across twins were set at 0 for both MZ and DZ twins.

Figure 2. *Bivariate Cholesky Decomposition*

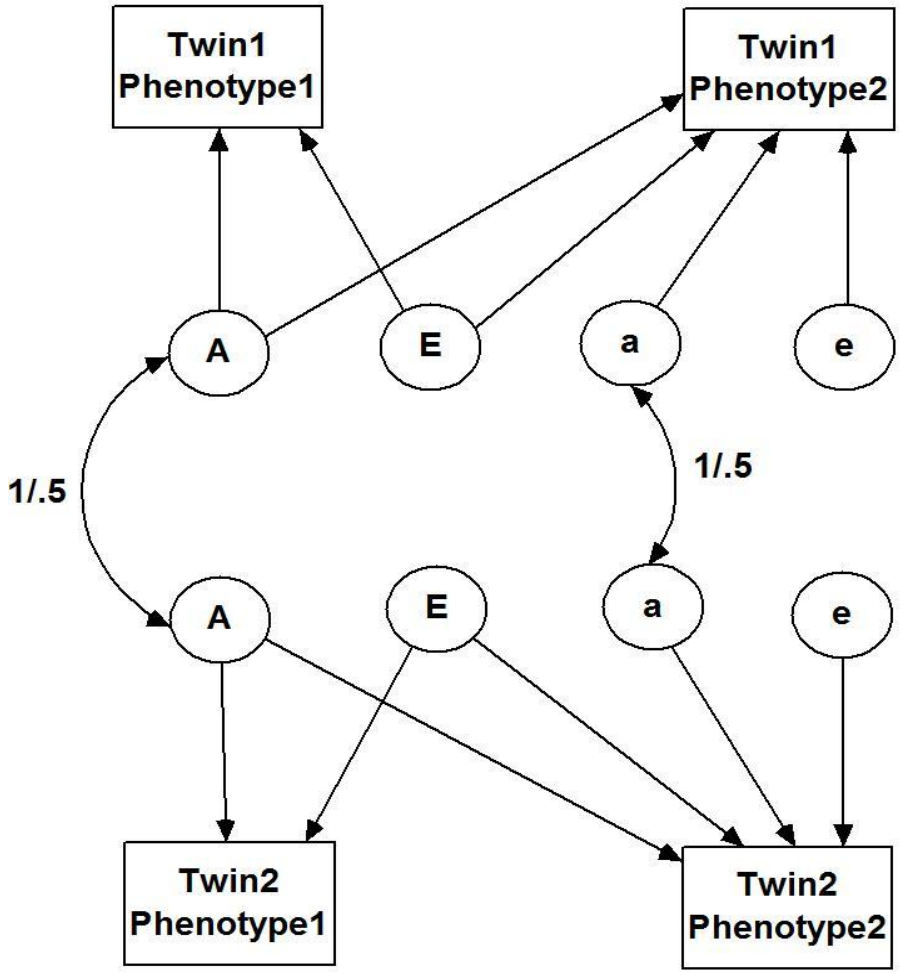

Figure 2. Bivariate Cholesky decomposition was used to partition the variance of, and covariance between each pair of inhibitory control, attention focusing, perceptual sensitivity, working memory, and verbal ability into genetic and non-genetic components. Latent variables represent overlapping additive genetic (A) and nonshared environment covariance including error (E), as well as residual genetic (a) and nonshared environmental (e) variance. The correlations between latent genetic variance and covariance across twins were set at 1 for MZ twins and .5 for DZ twins. For both MZ and DZ twins, the correlations for nonshared environmental variance and covariance across twins were set at 0.

Figure 3. *Trivariate Cholesky Decomposition*

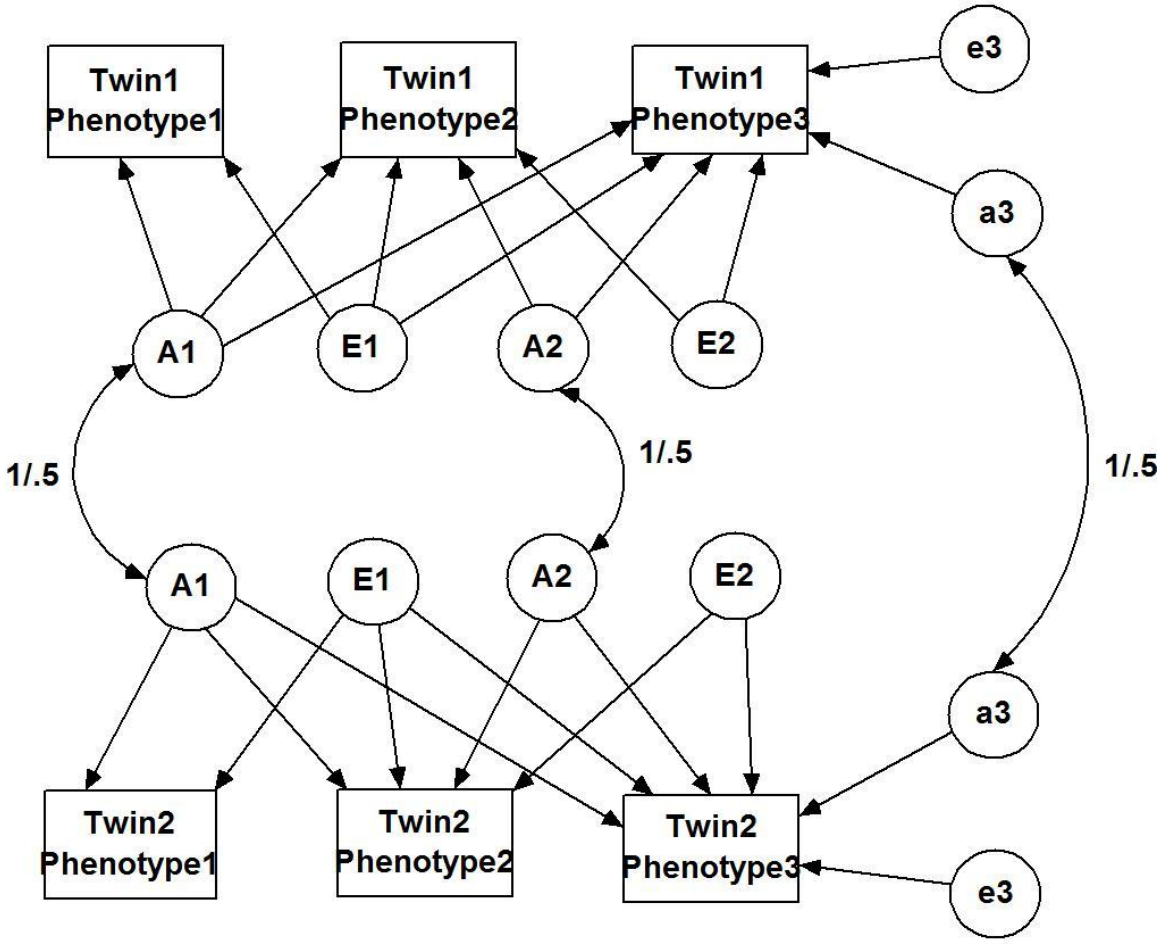

Figure 3. Trivariate Cholesky Decomposition was used to partition the variance of, and covariance among inhibitory control, inattention, and working memory into genetic and non-genetic components. Latent variables represent overlapping additive genetic (A) and nonshared environment (E) covariance (A1/E1-genetic/nonshared environment covariance among phenotype 1 2 &3; A2/E2-genetic/nonshared environment covariance between phenotype 2 & 3 controlling for A1/E1), as well as residual genetic (a3) and nonshared environmental (e3) variance. The correlations between latent genetic variance and covariance across twins were set at 1 for MZ twins and .5 for DZ twins. For both MZ and DZ twins, the correlations for nonshared environmental variance and covariance across twins were set at 0.