DEVELOPMENTAL ASPECTS OF TASK PERSISTENCE AND ITS RELATIONSHIP WITH READING SKILLS

A Dissertation in
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by

Saradha Ramesh

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The thesis of Saradha Ramesh was reviewed and approved* by the following:

Gerald E. McClearn  
Evan Pugh Professor of Health and Human Development and  
Biobehavioral Health  
Chair of Committee

Stephen A. Petrill  
Professor of Human Development and Family Science  
The Ohio State University  
Special Member  
Thesis Advisor

George P. Vogler  
Professor of Biobehavioral Health

David J. Vandenergh  
Associate Professor of Biobehavioral Health  
Research Associate, The Center for Development and Health Genetics  
Neuroscience Faculty, The Neuroscience Institute, The Huck Institutes for the Life Sciences

Susan McHale  
Professor of Human Development and Family studies

Byron C. Jones  
Professor of Biobehavioral Health  
Professor in Charge of the Graduate Program

*Signatures are on file in the Graduate School
ABSTRACT

In this study, the etiology of stability and change in task persistence and also the relationship of task persistence with reading related skills were examined using a twin sample (N=367 pairs) from a longitudinal Western Reserve Reading project. Twins were in kindergarten or first grade during the first assessment, and they were followed every year for cognitive and behavioral assessments. This study was based on three waves of assessments. A task persistence composite was formed from teacher and tester ratings of the children’s behavior observed at classroom and at home. Multivariate developmental genetic models were used to examine the genetic and non-genetic contributions to stability and change in task persistence. The findings suggested that genetic factors contributed to stability, and change was substantially due to nonshared environmental influences unique to individuals along with age-specific genetic influences at an earlier occasion. Quantitative differences in the genetic and environmental influences on task persistence were observed between boys and girls. In addition, relationships between task persistence, reading-related skills and general cognitive ability were modest. The relationships were mainly through genetic links and to some extent through nonshared environmental effects.
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Chapter 1

INTRODUCTION

Although there are considerable individual differences in the way preschool children approach tasks, it is thought that children who persist at their work are likely to learn more and perform better in school than children who do not (Karnes, Johnson, Cohen & Shwedel, 1985). Task persistence is an aspect of self-regulation, cognition, and behavior (Deater-Deckard, Petrill, Thompson, & DeThorne, 2005). It seems to have significant influence on child competence, from self-regulation to cognitive performance (McCartney & Berry, 2005).

Psychological aspects of task persistence

Task persistence, an aspect of self-regulation, includes the related constructs of attentional orientation, concentration, continuance of difficult tasks, and sustained task effort (Charak, 2000). Task persistence is conceptually similar to Rothbart’s effortful control (1989), which is an aspect of temperament. Effortful control is the ability to perform a subdominant response while ignoring a dominant response (Rothbart & Bates, 1998). This control is achieved by attention regulation (or attentional control) and behavioral regulation (or inhibitory control). This capacity enables children to improve their skills by deploying more attention if needed and planning more efficient strategies for coping, thus regulating their behavior voluntarily (Ruff & Rothbart, 1996). Therefore,
effortful control can be viewed as a part of executive attention systems and the self-regulation aspect of temperament as well. Also, one of the components of effortful control, executive attention, is functionally related to a broad system of executive functions (Reuda, Posner, & Rothbart, 2005), which encompass a number of cognitive processes such as working memory, inhibitory control etc. These are central to self-regulation and goal directed activities (Blair, Zelazo, & Greenberg, 2005). Deficits in executive functioning especially in temperamental regulation or effortful control are reported in children with attention deficit hyperactive disorder (Barkley, 1997) and also in other behavioral disorders (Kochanska, 1991; Rothbart & Bates, 1998) such as the externalizing of problems (Valiente et al., 2003).

**Psychobiological aspects of Effortful control**

**Developmental course**

Effortful control emerges between 6-12 months of age along with the maturation of attention mechanisms linked to the anterior attention networks (Rothbart, Derryberry, & Posner, 1994). Effortful control continues to develop during toddler and preschool period. At this stage of maturation, attention abilities can be seen as the ability to inhibit forbidden behaviors (Rothbart & Putnam, 2002). In a recent study (2000), Kochanska, Murray & Harlan assessed effortful control in children at 22 and 33 months. They examined all effortful control functions such as delaying, slowing down motor activity, suppressing initiating activity to a signal, employing effortful attention, and lowering
voice. Results of the study suggested that effortful control considerably improved
between 22 and 33 months. Beginning at age two and a half, effortful control continues to
develop throughout the preschool period and later becomes stable (Rothbart & Putnam,
2002).

**Gender**

Gender differences in effortful control are another interesting feature. In the
above-mentioned Kochanska, Murray & Harlan study (2000), gender differences in
behavioral tasks that test effortful control functions were also examined. Results
suggested gender differences in effortful control, as girls outperformed boys in many
behavioral tasks. Similar results were documented in a meta-analytic review (Else-Quest,
Hyde, Goldsmith, & Van Hulle, 2006). The review has reported gender difference in
temperament. Effortful control showed a significantly larger difference, favoring girls.
The differences noticed in the effortful control behavior were manifested as differences in
their learning approach. These differences in their learning approach explain about two-
thirds of female advantage in literacy learning (Ready, LoGerfo, Burkam, & Lee, 2005).
However, the etiology of gender difference in effortful control is not known.

**Neuroscience**

Effortful control is related to the activation of the midline frontal brain (Posner &
Raichle, 1994; Gusnard, Akbudak, Sulman, & Raichle, 2002). Brain research has been
able to identify critical brain regions whose activity and development directly relates to the performance and development of effortful control (Bush, Luu & Posner, 2000).

Neuroimaging studies clearly identified the anterior cingulate cortex (ACC) as one of the main nodes of executive attention networks. In particular, the dorsal ACC is involved in cognitive conflict tasks (Fan, McCandliss, Sommer, Raz, & Posner, 2002) and the rostral ACC is involved in affect-related tasks (Pizzagalli, Peccoralo, Davidson, & Cohen, 2006). Although all human beings share similar anatomical attention networks, differences in the efficiency of networks lead to individual differences in effortful control. These individual differences arise from complex gene-environment processes.

Molecular Genetics

Quantitative genetics studies have established high genetic influence (h²=.58) on effortful control (Goldsmith, Buss & Lemery, 1997). Information about types of attention networks, brain regions involved in attention requiring tasks, and neurotransmitters that modulate the activity of those areas and high heritability of the trait guided the search for candidate genes of executive attention and its function (Fan, Wu, Fossella, & Posner, 2001). A Fossella et al. (2002) study in a sample of adults aged 18-50 years old has shown that variations in executive attention are related to genetic polymorphisms in candidate genes that influence the dopaminergic neurotransmitter system. Another study (Reuda, Rothbart, McCandliss, Saccomanno & Posner, 2005) conducted in a sample of 4-year-old and 6-year old children also confirmed the association of long form of DAT1 gene with better effortful control. In addition, the efficiency of attention networks was
examined by comparing a control group with an experimental group. Children in the experimental group received attention training for 5 days, after which improvement in performance was clearly evident. The experimental group improved in reaction time in the solving of conflict tasks, and electrophysiological data. EEG recordings showed a more mature pattern, similar to adults, when children participated in the same conflict tasks. This study is evidence for the fact that environmental factors can bring changes in a trait that appears to be highly genetically influenced.

Taken together, neurobiological findings suggest that effortful control undergoes developmental changes, such as changes within organisms across age (McCall, 1981) where there are individual differences in development. However, the etiological factors underlying such developmental changes are unknown.

Task persistence: developmental quantitative genetics perspective

The main focus of developmental theory is to elucidate the mechanisms by which person-environment transactions influence individual development (e.g. Baltes & Smith, 2004). Such developmental processes are associated with quantitative and qualitative changes that lead to differences in the individual’s behavior. There is now a general consensus in the field of developmental psychology that nature (genetics) as well as nurture (environment) and interactions between them shape behavioral development (Plomin, 2004; Partridge, 2005). Quantitative genetics is the study of genetic and environmental factors that create behavioral differences among individuals (Plomin, 1990). Adoption and twin designs are commonly used methods for studying human
behavior, used to assess the extent of genetic and environmental factors influencing behavior, which is an initial step to understand the etiology of individual differences. The theory and methods of quantitative genetics can be applied to an interdisciplinary field, developmental behavioral genetics to address developmental questions such as causes of stability versus change in a behavior, the extent to which genetic and environmental factors are common to two or more traits, and the interplay between genes and environment (Plomin, 2004).

In a recent study of 3.5 year old twins from a population-based British sample, Petrill & Deater-Deckard (2004) formed a task persistence composite from four variables measuring task orientation: (1) A number of completed trials of simple-choice reaction time by a twin in the lab reported as a measure of task orientation; (2) Two tester rated items measuring attention and persistence of a twin using the Bayley Infant Behavior record during a home visit; and (3) An observer ratings of the child’s attention and persistence using the Parent-Child Interaction system of global ratings (Deater-Deckard, 2000) after watching the videotape of mother-child interactions from the home visit. Exploratory factor analysis of task persistence composite variables yielded a single factor with high correlations between the variables. The composite score was standardized before conducting quantitative genetics analyses. The findings of this study demonstrated modest genetic ($h^2=.37$) and shared environmental influences ($c^2=.25$) on task persistence. However, genetic and environmental sources as well as the magnitude of these influences may vary at different ages. Age related changes in task persistence may occur as a result of many developmental processes. In general, an individual’s task persistence development trajectory may be associated with normative brain maturation,
formal schooling, and aspects of family socialization. Further, since these processes tend
to be unique for each individual, task persistence development is not only associated with
these processes, but also with individual differences. It is therefore important to study
both overall trends in development as well as individual developmental trajectories of
task persistence. Moreover, it is necessary to integrate the contributions of nature and
nurture to study developmental change as well as stability in individual differences in
behavior.

Task persistence: Stability and change

As was mentioned earlier, task persistence is conceptually and functionally
similar to a psychological construct, effortful control. The similarities between task
persistence and effortful control are evident in the following ways: (1) The abilities to
focus, to sustain attention and to regulate behavioral impulses to persist on challenging
tasks, have been studied as indexes of effortful control (Zhou, Hofer, Eisenberg, Reiser,
Spinrad, & Fabes, 2007). Similarly, those indices have been used to measure task
persistence (Petrill & Deater-Deckard, 2004). (2) The ability of conflict regulation in
cognitive tasks is related to effortful control as measured by parent report (Rothbart,
Ellis, Reuda & Posner, 2003). The measurement of task persistence is based on the
behavioral reports by teachers, testers, and observers, which is within the theoretical
perspective of effortful control. (3) Similar to effortful control, task persistence brings
about changes in many domains of functioning including cognition, temperament, and
behavior (McCartney & Berry, 2005). (4) Rothbart’s model shows the developmental
changes in effortful control and integrates them with an individual difference approach (Kochanska, Murray & Harlan, 2000). Developmental changes as well as individual differences were observed in task persistence, as demonstrated in the Deater-Deckard et al. (2006) study. (5) Heredity and experience play an important role in effortful control as well as in task persistence (Borkenau, Riemann, Angleiter, & Spinath, 2001; Petrill & Deater-Deckard, 2004; Deater-Deckard et al., 2005). Due to the similarities between effortful control and task persistence, extensive phenotypic research in effortful control was used as a basis for further behavior genetic studies on task persistence.

Phenotypic studies demonstrate that the substrate for effortful control emerges at 6-12 months of age and develops significantly over the toddler and preschool years (Rothbart et al, 2003). This trait continues to develop up to age 7-8 and then maintains stability until adulthood. It is not possible to identify the factors underlying the changes in development solely on the basis of non-behavioral genetic studies. Because of this, developmental behavior genetic studies are required. Developmental behavior genetics considers two types of developmental change: heritability changes and age-to-age genetic change and continuity (Plomin, 1986). Heritability, a measure of genetic variance, can change as genetic and environmental systems come into play during development. Age-to-age genetic correlations refer to the extent to which genetic factors overlap over time. Genetic change is implicated when the genetic effects at two ages does not covary. The meaning of stability and change and how those changes are assessed in quantitative genetics will now be explained.

The biological origin and early appearance of a trait do not imply that a trait will be stable (Buss & Plomin, 1984). Trait stability is usually indexed by correlations
between trait scores across two points in time (rank-order stability), which reflect the degree to which the relative ordering of individuals on a given trait is maintained. Maturational or historical processes shared by a population can cause changes in a trait. Over time, these processes could cause change in the overall mean level of specific traits. Measurement of mean-level differences over time will indicate whether the sample as a whole is increasing or decreasing on a trait (Caspi, Roberts & Shiner, 2005). Generally, stability (same rank order over time) is conceptually independent of change over time (changes in mean level).

The periodic activation and inactivation of regulatory genes and the environmental differences in experience are sources of stability as well as change in development. If the genes that affect a trait during childhood overlap with those genes that influence the same trait during adulthood, then there will be stability from childhood and into adulthood. Conversely, if the set of genes influencing a trait at one age differs from the set of genes affecting the same trait at another age either qualitatively or quantitatively, then the difference will contribute to the instability of a trait. Similarly, environmental influences contribute to the stability or instability of a trait over time. The genetic correlation, which measures the extent of the overlap between genes, and environmental correlation, which measures the extent of overlap between environments that affect a trait over time, help to determine the stability of a trait (Plomin & DeFries, 1981). Such assessments are only possible if longitudinal quantitative genetic data are available.

Deater-Deckard et al. (2006) examined task persistence applying this correlation method to assess the stability and change in the individual differences of task persistence
using the available two wave longitudinal data from the Western Reserve Reading project (WRRP). Using the twin sample from WRRP, Deater-Deckard et al., (2005) formed a task persistence construct similar to the construct created in Petrill et al.’s 2004 study. They used assessments from teachers, testers, and observers on attention and persistence, which demonstrated not only a developmental shift in task persistence from non-genetic to genetic variance, but also showed an increase in genetic variance in 4-8 year-old-twins.

In a subsequent study by Deater-Deckard et al. (2006), it was shown that genetic influences accounted for the stability while nonshared environment, which indicates twin dissimilarity, accounted for the change observed in task persistence across the two waves of measurement. Several other quantitative genetic studies also have shown stability in individual differences in attention and persistence over time (Gagne, Saudino & Cherny, 2003; Goldsmith, Buss & Lemery, 1997). In Price et al. (2005) study of stability of ADHD symptoms in preschool children with ADHD, genetic factors accounted for about 91% of the stable variance in ADHD symptoms.

Neither traditional mean-level analyses nor stability coefficients have been able to capture individual patterns of change. However, either recent multilevel modeling or individual growth curve modeling (Bryk & Raudenbush, 1987) can be used to examine individual patterns of change over time. Using this approach, Halverson & Deal (2001) examined individual change in persistence in children aged 4 to 7 years and demonstrated an increase in persistence in individual children over time. In addition, their study shows that domains of family context along with changes in other dimensions of temperament explain a significant portion of individual change in persistence.
In summary, quantitative genetic research has shown that stability in task persistence is due to genetic factors. However, little is known about the continuance of this stability beyond early elementary school age and the etiological factors underlying individual growth trajectories of task persistence.

**Relationship between task persistence and reading skills**

Based on the Vygotsky work (1962), David H. Rose and Nicole Strangman (2007) identified three essential components of learning underlying the Universal Design of Learning, which is an innovative approach to education. The three components are recognition, strategy, and affect, and their respective neural substrates are recognition networks, strategic networks, and affective networks. Recognition networks are important for recognizing patterns such as patterns of letters and numbers; these networks play a key role in academic content areas. The differences in structure and function of these networks as well as the differences in the training and practice explain individual differences in recognition. Learning is not only dependent on this component but also on strategic networks. Strategic networks are responsible for the abilities to ignore irrelevant stimuli, plan, coordinate, self-monitor, and execute actions. Affective networks process and manipulate the function of recognition and strategic networks. Although these networks are anatomically and psychologically distinct, they exert influence on every act of cognition. For example, the act of reading involves not only decoding skills (recognition), but also understanding the reading material by self-monitoring (strategy,) and continuation of the act depends on interest or motivation (affect). One of the goals of
this study is to examine the association between recognition (reading) and strategic (task persistence) pattern and to test the underpinnings of the association.

Reading is a learned skill, which involves the recognition and comprehension of written text. The emergence of this skill occurs in multiple stages and develops as a collaborative process of biologically rooted language skills, cognitive ability etc. and appropriate nourishing social environment. Chall (1983) described the development of reading ability in six stages: Stage 0 Prereading; Stage 1 Emergent Literacy; Stage 2 Confirmations and fluency; Stage 3 Reading for Learning “The New”; Stage 4 Multiple View Points; Stage 5 Construction and Reconstruction. In Chall’s model, each stage builds on skills mastered in the previous stages of literacy. The first four stages were more relevant for the present study as the study consisted of elementary school age twins.

Early reading acquisition helps children to learn in other academic areas (Fyrsten, Nurmi, & Lyytinen, 2006). Extensive research has been done on the development of skills required for reading. Early reading depends on knowledge of alphabetical principle, which is the ability to associate sounds with letters and use these sounds to form words. Phonological awareness is a prerequisite skill for the development of alphabetical principle.

There is a substantial body of evidence indicating that phonological processing, especially phonological awareness, is one of the best predictors of reading acquisition (Adams & Bruck, 1995; Stanovich, Cunningham & Cramer, 1984; National Reading Panel, 2000). Phonological awareness refers to the ability to perceive and manipulate the sounds of spoken words (Goswami & Bryant, 1990) and is a good predictor of word and non-word reading, and reading comprehension (Torgesen, Wagner & Rashotte, 1997;
Ehri, Nunes, Willows, Schuster, Yaghoub-Zadeh, & Shanahan, 2001). Deficit of phonological processes impedes the acquisition of word reading skills, which in turn slows down the development of reading fluency (Torgesen, Wagner & Rashotte, 1994; Wagner, Torgesen, & Rashotte, 1994; Lyon 1995; Catts, 1996). Overall, phonological awareness, alphabet knowledge and oral language are the three areas that are important at the emergent stage of literacy.

The next stage is the confirmation and fluency. At this stage, children decode words fluently and accurately and integrate the syntactic and semantic information in text to identify words. These word identification processes serve as a foundation for reading comprehension, which is the next stage of learning the new. This stage occurs at ages 8 to 14 years, and the focus changes from "learning to read" to "reading to learn." As word identification becomes an automatic process, attention and working memory resources can be diverted for integrating text propositions and constructing meaning, which are the ultimate purposes of reading.

The reading process demands attention from the individual especially in beginning readers (Hidi, 1995). In particular, attention may modulate the rate and the extent of activation of word processing routes such as lexical and non-lexical routes (Herdman, 1992). Hence, individuals are responsible for regulating their internal processes and directing their attention to the reading task while ignoring other demands on their attention. Previous research has shown that attention regulation, commonly referred to as persistence or task orientation, is linked to academic achievement. Generally, low task persistence is associated with lower academic performance (Martin, 1989; Bramlett, Scott, & Rowell, 2000).
Though it is a behavior related to academic achievement, task persistence influences the development of cognitive skills. In current models of cognition, there is considerable overlap between the theoretical constructs of task persistence and general cognitive ability (Anderson, 2001). Many studies of normally developing infants and toddlers have shown that task persistence accounts for significant variance in general cognitive ability (Kelley & Jennings, 2003; Banerjee & Tamis-LeMonda, 2006). It has been shown that task persistence with its characteristic of ignoring irrelevant stimuli enhances the development of general cognitive ability (Dempster, 1991). Functional imaging studies have observed prefrontal lobe activation in children during attention tasks (Casey et al., 1997). This brain region has been implicated in developmental disorders such as Attention Deficit Hyperactive disorder (ADHD), and reading disabilities (Lazar & Frank, 1998).

We know that the correlation between reading and processes associated with reading acquisition, such as general cognitive ability (Lyon, 1989; Naglieri, 2001) and task persistence (Lundberg & Gorel, 2006), as well as the correlation between reading related processes such as phonological awareness, decoding, and word recognition, are typically high in the range of 0.30-0.80, both in normal and reading-disabled children. To increase our understanding further, examination at the etiological level reveals whether genetic or environmental factors influence these cognitive skills and also the commonalities/uniqueness of these influential factors. Quantitative genetics assess the effects of genes and environment and also quantify the changes in both the specific and overlapping effects of these influences, either as consequences of development or
variations in genes and environment. In addition, the genetic and environmental covariance among the traits can also be estimated.

Quantitative genetic research has suggested that genetic influences were predominant in individual differences in reading (Olson, 2007). For example, Byrne et al. (2002) study showed predominant additive genetic influences and moderate shared environmental influences on phonological awareness in preschool children enrolled in International Longitudinal Twin Study (ILTS) of early reading development. They showed significant genetic influences on reading at the end of kindergarten. Results of a follow up study (Byrne et al., 2005) of the same twins when they were at the end of first grade suggested that a single genetic factor accounted for all the genetic variance of reading and spelling and there was no influence from shared environment, as the project included samples from Australia, Scandanavia, and the U.S. Similarly, another independent study (Harlaar, Spinath, Dale & Plomin, 2005) of twins in the U.K. at the age of 7 estimated high genetic influences on both group deficits and individual differences in word and non-word reading within the same sample. The Petrill, Deater-Deckard, Thompson, Schatschneider, DeThorne, & Vandenergh (2006) study on the early development of reading and related skills in an Ohio twin sample, reported both genetic and environmental influences on early reading skills in twins who were in kindergarten or first grade during the first assessment and in first or second grade during the second assessment.

Also, in the assessment of the correlation between reading and general cognitive ability (Harlaar, Hayiou-Thomas, & Plomin, 2005), genetic influences largely accounted for the covariation between them. The genetic and shared environmental correlations
were also high. With respect to task persistence, the Deater-Deckard et al. (2005) study has also demonstrated a moderate association between task persistence and cognitive performance or achievement.

Overall, quantitative genetics research has shown that there is an association between general cognitive ability and reading related skills as well as between general cognitive ability and task persistence. However, little is known about whether the genetic and non-genetic effects that influence task persistence also influence general cognitive ability and reading skills.

Many studies have examined etiology of reading disabilities (RD) and Attention Deficit Hyperactivity Disorder (ADHD) in school-age children (e.g. Willcutt et al., 2001; Rucklidge & Tannock, 2002; Willcutt, Pennington, Chhabildas, Olson, & Hulslander, 2005). Among three different types of ADHD, inattentive, hyperactive/impulsive, and combined type, (Barkley, 1998), the inattentive type has stronger phenotypic relationship with reading disability. Nearly 95% of this relationship can be attributed to common genetic influences (Willcutt et al., 2000). Overall, quantitative genetic studies have provided strong support for the common genetic etiology hypothesis suggesting that comorbidity between RD and ADHD in school-age children is due to common genetic influences (Light, Pennington, Gilger, & DeFries, 1995; Willcutt, Pennington, & DeFries, 2000b). A genetic linkage analysis study in sibling pairs diagnosed with ADHD, along with RD in at least one individual of a pair (Willcutt, Pennington, Smith, Cardon, Gayan, Knopik, Olson, & DeFries, 2002), suggested a quantitative trait locus (QTL) for ADHD on chromosome 6, which is a well replicated QTL region for RD as well. Further bivariate linkage analysis in the same sample of individuals having both ADHD and RD
suggested that comorbidity might be partly due to pleiotrophic effects of a QTL on chromosome 6p.

Quantitative genetic research suggests that reading disability is the quantitative extreme of the same genetic and environmental factors responsible for normal variation in reading ability (Meaburn, Harlaar, Craig, Schalkwyk, & Plomin, 2007). Similar distribution can be expected for another quantitative trait, task persistence. Since quantitative genetic studies have established the links between RD and ADHD, which are the disorders at the extremes of quantitative distribution, it is possible to find the relationships in normal variations of reading and task persistence. Little research has been done in finding the contributions of genetic and environmental factors to the relationship between reading skills and task persistence along a continuum.

**Statement of problem**

Past research has shown that several factors influence task persistence in complex ways, and that the sources of influence at different ages may be quite different. For example, parental warmth has shown to have influence on task persistence behavior in young children (Deater-Deckard et al., 2005) whereas self-efficacy beliefs determine task persistence behavior in adults (Pajares, 1996). From an etiological perspective, different sets of genes have their maximum influence at different times during the lifespan. Similarly, particular environments can influence individuals at different times during their lives. There is therefore a need to investigate the growth and change in task persistence systematically as the child grows and matures. There are few longitudinal
studies that have examined the development of task persistence, and still fewer that have used quantitative genetic designs to study the etiology of the stability and change within task persistence. This study addresses this gap by using common genetic factor modeling and genetic simplex modeling to test the hypothesis of genetic continuity in task persistence in twins. The sample was from an ongoing longitudinal twin study. Twins were assessed initially when they were in kindergarten or first grade and were followed at yearly intervals with cognitive and behavioral assessments. Analyses were done for this study using data from the available three waves of measurements.

Very limited information about change can be derived from the analysis of correlation or from covariance matrices. In particular, such analysis cannot provide information about either individual stability or change. To the best of my knowledge, none of the quantitative genetic studies were designed to describe the individual growth trajectories of task persistence. This study assesses individual change using a latent growth curve modeling.

No genetically informed study has examined the association between task persistence and basic reading skills in a population-based sample. Many studies have examined the association of general cognitive ability and reading skills (Harlaar et al. 2005) in a population based sample. Moreover, the association of reading skills with inattention/impulsivity measures was examined in a sample of preschool children (Willcutt et al. 2007). A recent study (Zumberge, Baker, & Manis, 2007) of 9- to 10-year-old twins examined the etiology of variation in reading and its association with inattention. However, studies of relations between normal variation of reading related
skills and normal variation of task persistence behavior using longitudinal data have not been done.

Objectives of the Study

The primary purpose of this study is to examine stability and change in task persistence using a longitudinal twin study known as Western Reserve Reading project. This study also seeks to gain an understanding of the factors that may influence the association between task persistence and reading related skills. The guiding research questions are: (1) what are the etiological factors that account for the stability and change in the development of task persistence? (2) What are the factors underlying the relationship between task persistence and reading related skills?

The specific objectives and hypotheses of this study are:

Aim 1: To examine the etiology of individual differences in task persistence.

Hypothesis 1: Genetic influences will increase and shared environmental influences will decrease as the child grows older. In addition, there will be unique nonshared environmental influences.

Aim 2: To assess the stability and change in task persistence across three waves of assessments.

Hypothesis 2: Genetic factors will explain the stability and nonshared environmental influences will account for the change.

Aim 3: To describe and analyze the development of task persistence across three measurement occasions using an individual growth modeling method.
Hypothesis 3: Given the development of task persistence as well as exposure to structured environment such as a school setting, recreation centre etc., occur during early elementary school, there will be an increase in task persistence across measurement occasions. Genetic influences are primary in terms of variation in intercept whereas variation in slope should have a larger environmental component.

**Aim 4**: To identify the quantitative differences in task persistence due to sex effects.

Hypothesis 4: As shown in previous research, there will be a sex difference in task persistence which will manifest as differences in the magnitude of genetic and environmental influences.

**Aim 5**: To assess the association between task persistence and reading related skills.

Hypothesis 5: There will be moderate correlations between task persistence and reading related skills through both genetic and environmental pathways.

**Significance of the study**

Task persistence, a behavior related to academic achievement, influences the development of cognitive skills. A Rothbart, Ellis, Rueda, & Posner (2003) study provided evidence for the view that maturation of effortful control occurs predominantly during toddler and preschool years. There is a need to study the etiological factors influencing the development of task persistence during early childhood. Very few past studies have looked at task persistence with a longitudinal perspective.
This study is significant in that it attempts to examine task persistence as a dynamic process. Another significance of this study is that it seeks to use a modern analytical tool, namely, individual growth modeling, to study the development of task persistence using more than two waves of measurement occasions.

Measurement of heritability provides guidance in searching for candidate genes. An estimation of environmental effects assesses the extent of influence of specific environments associated with the development of task persistence. Moreover, understanding the developmental trajectories of task persistence during early childhood provides insights into normal as well as developmental pathologies. This would enable educators, parents, and policymakers to adopt measures or develop interventions tailored to meet the specific needs of students, thus helping them to enhance their learning skills.

This study examines the association of task persistence with reading related skills. This helps us to increase our understanding of the complexities of reading development and reading disorders. Such quantitative genetic studies in addition to molecular genetic research hold the promise of linking genetic and environmental influences to specific genes, brain functions, and behaviors in individuals (Olson, 2007). These provide guidance in the designing of interventions to avoid reading failure.
Chapter 2

METHODS

Sample and procedure

The Western Reserve Reading project (WRRP) is an ongoing cohort-sequential study involving 367 pairs of monozygotic and same sex dizygotic twins, designed to evaluate the genetic and environmental influences on development of early literacy skills. Participants were recruited mainly through school nominations. 273 schools in the State of Ohio participated in recruiting families with twins who have been enrolled in kindergarten but have not finished first grade. Additional families who fit in the selection criteria were recruited through birth records, mothers of twin clubs, and media advertisements. These twins live mainly in the Cleveland, Columbus, and Cincinnati metropolitan areas, as well as other areas of Ohio and Western Pennsylvania. Most of the twins live with both parents. Zygosity of the twins was determined by genotyping for DNA markers (Price, Freeman, Craig, Petrill & Plomin, 2000). Few parents (n=41) declined consent for DNA collection from their twins. In such cases, zygosity was determined based on physical similarity ratings of twins by their parents (Goldsmith, 1991). Ninety-two percent of the twins' parents were white; about 24% of them hold a post-graduate degree, 30% had a bachelor’s degree, 18% had some college education, and 12% had high school education or less.

As an initial part of the larger study, recruited families received an information package which included a letter and brochure explaining about the study as well as a stamped postcard indicating interest in the study. Families who returned their post card
were contacted by phone and were given questionnaires in order to collect demographic information, pre/post natal information about twins, parental attitudes and home environment. After the completed questionnaires were received from families, trained examiners scheduled and conducted a home visit. During the home visit, separate examiners tested each twin individually to avoid rater bias. Home visits were given mainly to conduct cognitive assessments in both twins and parents. During each home visit, twins completed a 90- minute battery of cognitive and reading related assessments. In addition, examiners assessed twin-twin interactions and parent-twin interactions during structured and unstructured play sessions. Following the first assessment, subsequent home visits were conducted within a year of previous assessment as part of an ongoing study. Three reading assessments and one mathematics assessment have been conducted so far.

The current study is based on twins pairs who have completed Wave 1 (n=120 MZ, 167 DZ), Wave 2 (n=108 MZ, 154 DZ), and Wave 3 (n=84 MZ, 115 DZ) assessments to date. In Wave 1, the mean age of the twins was M= 6.08 (SD=.69, range 4.3 to 7.9 years) and mean Stanford-Binet was M=100.00 (SD=13.0, range 61-139). In Wave 2, the mean age of the twins was M=7.2 (SD=.66, range 6.0 to 8.8 years) and the mean Stanford-Binet was M=101.25 (SD=12.4, range 66-142). In Wave 3, mean age of the twins was M=8.3 (SD=.73, range 6.9 to 10 years) and mean Stanford-Binet was M=103.32 (SD=13.5, range 71-154). Approximately 57% of the sample participants were girls; the remaining 43% were boys.
Measures

Task persistence (TP)

Studies have shown that task persistence behavior measured in experimental tasks was found comparable to tester ratings of persistence during a Bayley examination (Yarrow, Morgan, Jennings, Harmon, & Gaiter, 1982) and also to teacher ratings (Jennings, Conners, Sankaranarayan, & Katz, 1982). For the current study, a task persistence composite was formed from items measuring attention and persistence. Those items were teachers' and testers' ratings of a child’s behavior observed in the classroom and during home visits, correspondingly. During the home visit, two 8-minute segments of interaction between each twin and a parent were videotaped including an unstructured ‘free play’ session using each twin’s favorite toy and a structured session using an Etch-A-sketch toy and a tilting maze box. Both games required joint effort and attention to the tasks. Testers completed the Bayley Behavior Record (Bayley, 1969) after the home visit. Two tester-rated items relevant to persistence (1- consistently lacks persistence; 2- typically not persistent; one or two instances of persistence; 3- lacks persistence half the time; 4- typically persistent; lacks persistence in one or two instance; 5- consistently persistent) and attention/distractibility (1- constantly off task; does not attend; 2- typically off task; attends in one or two instances; 3- off task half the time; 4- typically attends ; attention wanders in one or two instances; 5- constantly attends) were used.

A Teacher Report Form (TRF; Achenbach, 1991) was sent to each twin’s teacher after obtaining the parental consent. The form was then collected through mail. Teachers reported on a 3-point likert scale (0=not true; 1-somewhat or sometimes true; 2-always
true). From the TRF, 3 items that evaluate task persistence (4: fails to finish things; 8: cannot concentrate or pay attention for long; 78: inattentive, easily distracted) were used for analysis. The items were reverse coded.

Table 2-1 shows descriptive statistics of the items in the task persistence composite. The mean and standard deviations of teacher rated items 4, 8 and 78 were consistent across all three waves. The distribution can be considered approximately normal for items 8 and 78. Item 4 was positively skewed and the kurtosis value was high. In the inferential statistical analysis, these items were reverse scored so that higher values indicate better attention.
The mean and standard deviations of tester-rated items J & K were similar in Wave 1 and Wave 2. In Wave 3, the mean value of these items was high and standard deviation was low, implying a lesser amount of variance. The distributions were slightly negatively skewed, implying that many observations were higher than the mean value. The kurtosis values were within normal limits (+/- 1.94) except for in Wave 2, where the value was slightly high. There were no outliers.
Pearson correlations among teacher and tester rated items were moderate ($r > .20$) in all three waves except for in Wave 3, the correlations between tester rated item K and other teacher rated items were not significant. Since the correlation values were moderate, factor analyses were done as a next step.

The factor loadings on task persistence in Wave 1 and 2 were both high and similar (See Table 2-2). In Wave 3, factor loadings on teacher rated items were high and those of tester rated items were less than .50, which could be due to skewed data. Skewness causes restriction of variance and affects the item's ability to distinguish varying levels of characteristics (Tabachnick & Fidell, 1996). Skewness, less variance, and low standard deviations in Wave 3 could be due to restrictions in the range of item responses.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fail to finish things (reverse scored)</td>
<td>.74</td>
<td>.73</td>
<td>.80</td>
</tr>
<tr>
<td>Cannot concentrate (reverse scored)</td>
<td>.82</td>
<td>.81</td>
<td>.90</td>
</tr>
<tr>
<td>Inattentive, distracted (reverse scored)</td>
<td>.81</td>
<td>.78</td>
<td>.88</td>
</tr>
<tr>
<td>Attends well</td>
<td>.64</td>
<td>.66</td>
<td>.28</td>
</tr>
<tr>
<td>Persists in task</td>
<td>.53</td>
<td>.54</td>
<td>.19</td>
</tr>
</tbody>
</table>

Internal consistency of the items of the task persistence composite was also measured for all three waves of measurement occasions; Cronbach’s value was $\alpha > .70$ (Wave 1 $\alpha = .77$, Wave 2 $\alpha = .77$, Wave 3 $\alpha = .72$). Since the composite has good internal
consistency and the predictive validity is greater when more than one informant method is used, all five items were retained to form the task persistence composite score.

A single composite score of task persistence (TP) was derived across these methods and reports. In all the items, higher score represents “better” task persistence. Each score was converted to a z-score, then averaged and standardized again, so that the final task composite score was in standard deviation units (Deater-Deckard, Petrill, Thompson, & DeThorne, 2005). For the present study, all five items for forming task persistence composite score are available in Wave 1, Wave 2, and Wave 3.

General cognitive ability (IQ)

General cognitive ability (IQ) was assessed using a short form of the Stanford-Binet Intelligence Scale (Thorndike, Hagen & Sattler, 1986) that included tests of verbal reasoning (Vocabulary), abstract/visual reasoning (pattern analysis), quantitative reasoning, and short-term memory (memory for sentences & memory for digits). These subtest scores were summed and standardized to form the Composite Summary of Area Score (SAS). This measure has a mean of 100 and standard deviation of 15.

Reading-related skills

Reading outcomes were assessed using the Woodcock Reading Mastery Test (WRMT-R; Woodcock, 1987). The subtests that measure Letter Identification (LID),
Word Identification (WID), Word Attack (WATT), and Reading Comprehension (RCOMP) were used to assess skills required for beginning readers. In the LID subtest, the task is to identify printed upper or lower case letters, whereas in WID, the task is to read words. These subtests were used to assess letter knowledge and word knowledge, respectively. The subtest WATT was used to assess phonological decoding skills, in which children were asked to read nonsense words or words that are used less frequently in English language, until they reached an error criterion. The subtest passage comprehension was used to measure RCOMP, in which children were asked to read a short passage and identify a key word missing from the passage. All subtest scores were residualized for age and sex using a regression method. In this study, each measure was used as an independent variable.

Phonological awareness (PA) was measured using the Phonological Awareness Test (Robertson & Salter, 1997) including six subtests that measure rhyming (discrimination and production), phoneme isolation (initial), phonemic segmentation (whole word), and phonemic deletion (syllabic deletion and phoneme deletion). In Wave 1 all six subtests and in Wave 2 & 3, subtests other than rhyming and phonemic isolation were used. These subtests were summed and this score was residualized for age and sex, also using a regression procedure (see Petrill et al., 2006a).

Expressive vocabulary was assessed using test scores from the Boston Naming Test (Kaplan, Goodglass & Weintraub, 1983), which consists of 60 pictures, ordered from easiest to most difficult items. This test requires children to name the pictured item and total score was based on the number of correct responses. The raw scores were then corrected for age and sex by regression procedure.
Analytic methods

Twin design

The classical twin design is the most commonly used design in quantitative genetics. The existence of monozygotic and dizygotic twins provides a natural experiment for disentangling genetic from environmental factors. The twin method has two major advantages. First, the degree of difference in genetic relatedness between monozygotic and dizygotic twins is greater than between any other relatives, as monozygotic twins (MZ) share 100% of genetic materials and dizygotic twins (DZ) share 50% of genetic materials. Second, both types of twins share the same womb, are born at the same time and live in the same family (Plomin, 1990).

In quantitative genetic research, an intraclass correlation refers to the extent of similarity of individuals within pairs. The value ranges from 0, indicating no similarity and 1.0, indicating perfect resemblance. The essence of the twin method is the comparison of the correlations for the two types of twins with an assumption of equal environment. If heredity is important for a trait, then monozygotic twins resemble more than dizygotic twins. If there is not much difference among monozygotic and dizygotic twins, then environment is important. Moreover, twin design allows for partitioning the variation of a trait or covariation between multiple traits into genetic, shared environmental and nonshared environmental components. Heritability refers to the extent to which variability in a trait is influenced by genetic variations within a population. Shared environment refers to environmental influences that contribute to resemblance among siblings beyond genetic relatedness and nonshared environment refers to
environmental influences that are unique to a twin and tend to make the individuals in a twin pair different from each other.

Heritability is calculated as twice the difference in intraclass correlation between MZ and DZ twins: $h^2 = 2(r_{mz} - r_{dz})$. Shared environment is the difference between MZ intraclass correlation and heritability: $c^2 = r_{mz} - h^2$, and nonshared environment is one minus heritability and shared environment: $e^2 = 1 - h^2 + c^2$. Overall, the intraclass correlations provide an estimate of genetic and environmental effects, but this method does not simultaneously estimate these effects nor do they provide inferential tests of significance.

**Quantitative genetic models**

*Longitudinal genetic models to assess stability and change*

The aim of longitudinal analysis is to examine how genetic and environmental influences contribute to the development of traits over time (Boomsma & Molenaar, 1987; McArdle 1986). Few basic models are used for longitudinal data analyses in quantitative genetics research: (1) The Cholesky model; (2) Common factor independent pathway model; (3) Simplex model; and (4) Latent growth curve model.

Multivariate analysis focuses on the covariance within the same measure across different measurement occasions. Just like the univariate analysis, multivariate genetic analysis decomposes the phenotypic variance of a trait into genetic and environmental
components of covariance between measurement occasions, and also specific genetic and environmental components of variance for each measurement occasion.

As a first step to multivariate analysis, it is important to establish a saturated model, so that hypothesis-driven restricted models can be compared against a saturated model (Neale, Boker, Bergeman, & Maes, 2006). This comparison will provide an index of fit for a model with genetic and environmental components (Neale, Boker, Bergeman, & Maes, 2006). The Cholesky model (See Fig. 1) can serve as a saturated model to compare against restricted models because this model has the same number of parameters as there are covariances - m (m+1)/2 for m variables and will always fit perfectly (Neale & McArdle, 2000). This model has a limited use, however, in longitudinal data analysis because the causation is unidirectional through time (Boomsma, Martin, & Molenaar, 1989).

The common factor model and the simplex model are frequently used statistical models that test two different developmental mechanisms (Lemery et al, 1999). A common factor independent-pathway or biometric model (See Fig. 2) predicts that the same genes and environment have different magnitude of effects on the covariance over time. This model represents a developmental mechanism which suggests that one stable factor brings about the behavioral change at each age. In this model, developmental changes can be assessed from the change in the factor loadings over time (Van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). In a simplex model (See Fig. 3), genetic and environmental latent variables at a particular time are related to the immediately preceding variables. This model then suggests that stability of a trait is maintained by prior experience and influences that are transmitted to subsequent ages and
change in a trait may be brought by new influences that enter at each age (Van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003).

In addition, the above mentioned developmental models can be distinguished based on their correlation patterns. The correlation coefficients of the simplex model are highest between measures taken closer in time. It decreases as the measurement occasions are spaced further apart. On the contrary, the common factor model assumes no specific pattern of correlation as the time varies (Van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003).

However, the common factor and simplex models cannot address within-individual differences in change in task persistence over time, as those models cannot predict mean differences between MZ and DZ twins (Neale & McArdle, 2000). Latent growth curve models can predict changes in mean, variance, and covariance over time. Examination of within-individual variability as opposed to covariance models of stability may provide a different view in understanding the etiology (Reynolds, Finkel, Gatz & Pedersen, 2002). For instance, genetic influences operating at a particular point in time may be different from those affecting the rate of change over time (Reynolds, Finkel, Gatz & Pedersen, 2002).

In the current study, the Cholesky decomposition, the common factor independent pathway, simplex and latent growth curve models were used to examine the etiological factors underlying task persistence as well as the changes in those factors that occur as the time varies.
**Analysis with reading related skills**

A Cholesky model was also used in this study to examine the association between task persistence and reading related skills. As opposed to the previous analysis, where the focus is on the covariance between the same measures over time, here the interest is on the covariance between two different traits: task persistence and reading related skills. Multivariate genetic analysis decomposes the phenotypic covariance into genetic and environmental sources of covariance (Plomin et al., 2001).

**Statistical models**

**Cholesky model**

The Cholesky decomposition is the most commonly used model in twin design for doing multivariate analysis. The idea of using the Cholesky model is to describe the variation within, and the covariation between the observed variables and latent factors. Factor loadings indicate the relationship between a latent factor and an observed variable and factor correlations represent the relationships between the hypothesized latent factors. In the Cholesky model (See Fig. 1), the first variable is assumed to be caused by a latent factor that also influences other variables. The second variable is assumed to be caused by a second latent factor that can explain variance in all variables except for the previous one. This pattern continues until the final variable, which is influenced by a latent factor that is specific only to that variable. A1, A2, A3 are latent factors representing genetic effects. These factors are assumed to be uncorrelated within the
siblings. Each genetic factor is viewed as a non-overlapping set of genes. Similarly, the model was used for shared (C1, C2, C3) and nonshared environmental factors (E1, E2, E3) and these factors also support the same concept similar to genetic factors. Each genetic factor is correlated with the corresponding genetic factor of the other sibling based on genetic relatedness. Since MZ twins share 100% of genes, the correlation value is 1.0. DZ twins share 50% of genes, so the value is 0.5. The value is 1.0 for the correlation among shared environmental factors, since these factors are assumed to affect the siblings in the same way. But, for nonshared environmental factors the value is 0, because these factors account for the twin dissimilarity. This full model was estimated using the Mx program (Neale, Boker, Xie, & Maes, 1999).

Figure 1: Cholesky decomposition model for one twin

- A1 A2 A3 = latent genetic factors in ACE model
- C1 C2 C3 = latent shared environmental factors in ACE model
- E1 E2 E3 = latent nonshared environmental factors in ACE model
Independent pathway model

A common-factor independent pathway model (See Fig. 2) allows for common genetic, shared and nonshared environmental influences \((A_C, C_C, E_C)\) on the latent phenotype across all occasions as well as specific genetic and environmental influences \((a_s, c_s, e_s)\) at each occasion. Later influences are occasion-specific, which means they are not correlated across waves. So, in the full model, variance is partitioned into genetic and environmental components that are shared in common across occasions, and also genetic and environmental variances specific to each occasion. The loadings on the

![Figure 2: Common factor independent pathway model for one twin in a pair](image)

\(A_c, C_c, & E_c = \) latent common genetic, shared and nonshared environmental factors 
\(a_s, c_s, & e_s = \) latent specific genetic, shared and nonshared environmental residual factors
common factors will reveal the degree to which the trait shares genetic and environmental factors across occasions.

**Simplex model**

A simplex model includes two types of causal pathways: transmission pathways and innovation pathways (See Fig. 3). Transmission pathways include pathways connecting genetic factors at adjacent occasions. These pathways denote that genetic factors at one occasion influence genetic factors at the next occasion. Innovation pathways represent new inputs at each occasion, which are not influenced by previous occasions. The figure illustrates the relationships between genetic (A), shared environmental (C), and nonshared environmental (E) factors across three waves (Wave 1, Wave 2, Wave 3).

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**Figure 3: Simplex model**

A, C, & E = latent genetic, shared and nonshared environmental factors
Horizontal arrows = regression of the latent factor on the previous latent factor
Vertical arrows = new input at time i; e = measurement error.
factors at a particular occasion are influenced by factors operating during previous occasion(s). Similarly, the model includes transmission pathways that connect environmental factors. Such relationships between latent factors are termed autoregressive. In addition, the model includes innovation pathways, which represent new genetic and environmental influences that operate at each specific occasion. At the first occasion, the first latent factors cannot be explained by factors associated with an earlier occasion; therefore this factor itself is regarded as an innovation (Van Beijsterveldt, Bartels, Hudziak, & Boomsma, 2003). The total variance is the sum of the occasion-specific innovation effects and the occasion-occasion transmission effects.

*Latent growth model*

The latent growth curve model is illustrated in Fig. 4. In this model, individual scores at any time are a linear function of a latent intercept (I), slope (S), and random error. Two latent factors denoted as I and S represent the average level of task persistence at the initial status and rate of change or slope respectively. Factor loadings on an intercept are fixed to 1, and on slope, they are allowed to vary so that they capture the systematic changes in task persistence over time. The factor means and variances can also be estimated using this model. To investigate genetic and environmental influences on intercept and slope, the variance of the intercept and slope can be decomposed into genetic and environmental components.
A, C, & E = latent genetic, shared and nonshared environmental factors

One of the purposes of the current study is to evaluate change in task persistence across three measurement occasions and to assess the significance of genetic and environmental influences on growth model parameters. A biometric latent growth curve model was used for this purpose (Neale & McArdle, 2000). Based on recommendations by Willett, Singer and Martin (1998), the data set was created with variables that were not standardized. In this longitudinal analysis, the age and sex of the individuals were used as covariates, and the age variable was centered. Centering refers to subtracting the
mean age of the sample from the individual observed age so that the transformed value indicates how much each individual deviates from the mean. This centering was done to make the interpretation of the intercept more meaningful, as the variable lacks a natural zero point. In the present case, the mode of the age variable in Wave 1 (6.5 years) was subtracted from the age of each individual, so that the average score of the intercept was for a 6.5 year-old respondent. The random errors were fixed at the same value in all three measurement occasions.

Sex difference analyses

Analyses were done to test whether there are differences in task persistence scores due to sex effects. As a first step, analysis of variance (ANOVA) was used to test the hypothesis that the means of task persistence scores were equal for boys and girls. SAS program was used to conduct ANOVA and F statistic was used to assess the significance of main effect.

Twin correlations by sex and zygosity provide information about the sex differences in etiology. Quantitative sex difference is implicated when resemblance within male and female same-sex twin pairs differs as a function of zygosity. Qualitative sex difference is implicated when the same-sex DZ twin pairs resemble more than the opposite-sex DZ twin pairs.

Furthermore, to understand more about the etiological patterns, structural equation models were applied using Mx program. Three models namely saturated model, heterogeneity and homogeneity models were used to examine sex differences in the
etiology of task persistence. In the saturated model, the means and variances were set to vary across groups. This model was used for model fitting analysis. Heterogeneity (See Fig. 5) and homogeneity models were formed to test whether the variances differ as a function of sex. In the heterogeneity model, genetic and environmental parameters are allowed to vary. The heterogeneity model was used to assess the differences in the magnitude of genetic and environmental influences that affect males and females. In the homogeneity model, the genetic and environmental parameters are set to be equal with an

Figure 5: Heterogeneity model for monozygotic females
(Source: Colorado Twin workshop)

A, C, & E: Genetic, Shared and Nonshared environmental latent factors respectively. T₁ and T₂ represents twin 1 and twin 2 task persistence score. m₁ & m₂ are mean values of twins. aᵣ, cᵣ, eᵣ: genetic, shared and nonshared environmental path coefficients.
assumption that the same genetic and environmental influences affect males and females to the same extent.

Model fitting

Model fitting analyses were done using the Mx software program (Neale, 1994). A common factor independent pathway model and a simplex model were both compared to the Cholesky model, which was used as a saturated model in this study. These models were compared by chi-square tests. The chi-square statistic was computed by subtracting -2LL for the saturated model from that of reduced models. Akaike information criterion (AIC) values were also obtained. The model fits the observed data well if the AIC value is low. Although the common factor and simplex models do not form a nested pair of models, they were compared in terms of parsimony (lower AIC values) and goodness of fit because those models represent alternative sets of constraints on the saturated model (Neale & Cardon, 1992). However, in the present study, our interest is in understanding the developmental mechanisms.

The same criteria (Chi-square statistic and AIC) were applied to find out the significance of A, C, and E parameters in models used for finding sex differences in task persistence. Various sub-models were formed by equating means across zygosity and within zygosity leaving other parameters free. In addition, sub-models were formed by dropping parameters of interest to test the significance of A, C, and E parameters. Fit statistics for the sub-models were compared with those for the saturated model. This resulted in chi-square and AIC values that were comparable for the different sub-models. Based on these results, the significance of the parameters was assessed.
Apart from model fitting, the significance of the genetic and environmental parameters was assessed using confidence intervals of point estimates. Confidence intervals provide an interval of values and the end points of the interval give an estimate of size of the error associated with the point estimate.
Chapter 3

RESULTS

Descriptive Statistics

The descriptive statistics of the task persistence composite score are shown in
Table 3-1. The distributions were slightly skewed (< 1.75) and kurtosis values were
slightly high (< 3.6). Multivariate analysis of variance on task persistence (TP) scores
was conducted to examine the extent to which TP scores at each wave differ as a function
of zygosity. The results revealed that on an average, MZ twins scored higher than the DZ
twins on most occasions but the main effect for zygosity was not significant, F(6, 396) =

<table>
<thead>
<tr>
<th>Task persistence</th>
<th>n</th>
<th>MZ Mean</th>
<th>SD</th>
<th>n</th>
<th>DZ Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave 1</td>
<td>201</td>
<td>-.02</td>
<td>.74</td>
<td>264</td>
<td>.04</td>
<td>.69</td>
</tr>
<tr>
<td>Wave 2</td>
<td>154</td>
<td>.10</td>
<td>.68</td>
<td>226</td>
<td>-.10</td>
<td>.76</td>
</tr>
<tr>
<td>Wave 3</td>
<td>104</td>
<td>.05</td>
<td>.68</td>
<td>157</td>
<td>-.04</td>
<td>.67</td>
</tr>
</tbody>
</table>

1.61, p=.14 and the effect size of zygosity on TP scores at each wave was minimal
($\eta^2=.01$). Similarly, analysis of variance of TP scores was performed to examine the
extent to which the TP scores differ between boys and girls at each wave. The mean and
standard deviation of TP scores by sex is presented in Table 3-2, and the analysis
revealed that there is a main effect for sex ($\eta^2 = .04$) and on average, girls scored significantly higher on TP scores than boys at each wave.

Tab. 3-2: Descriptive Statistics of Task Persistence Scores by Sex.

<table>
<thead>
<tr>
<th></th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>By sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>202</td>
<td>159</td>
<td>118</td>
</tr>
<tr>
<td>M</td>
<td>-.20</td>
<td>-.25</td>
<td>-.23</td>
</tr>
<tr>
<td>SD</td>
<td>1.06</td>
<td>1.19</td>
<td>1.18</td>
</tr>
<tr>
<td>Females</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>n</td>
<td>274</td>
<td>213</td>
<td>145</td>
</tr>
<tr>
<td>M</td>
<td>.17</td>
<td>.16</td>
<td>.19</td>
</tr>
<tr>
<td>SD</td>
<td>.88</td>
<td>.80</td>
<td>.77</td>
</tr>
</tbody>
</table>

In Wave 1, 2, and 3, the phenotypic correlations of task persistence were moderate: $r = .52$ between Wave 1 and 2; $r = .49$ between Wave 1 and 3; $r = .51$ between Wave 2 and 3.

Genetic analyses

In Wave 1 and 3, the intraclass correlation (ICC) (See Table 3-3) values suggest genetic influences on task persistence as the MZ correlation was twice that of the DZ correlation. In Wave 2, the correlations value suggests both genetic and environmental influences on the trait as the values were nearly the same. Other than within-wave correlations, cross-wave twin correlations (See Table 3-3) can be used to decompose the
Tab. 3-3: Within-Wave and Cross-Wave Twin correlations of Task Persistence Scores

<table>
<thead>
<tr>
<th></th>
<th>Within-wave correlations</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>By zygosity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZ</td>
<td>.79*</td>
<td>.55*</td>
<td>.59*</td>
<td></td>
</tr>
<tr>
<td>DZ</td>
<td>.30*</td>
<td>.48*</td>
<td>.25*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>By sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MZF</td>
<td>.69*</td>
<td>.14</td>
<td>.79*</td>
<td></td>
</tr>
<tr>
<td>MZM</td>
<td>.87*</td>
<td>.59*</td>
<td>.37</td>
<td></td>
</tr>
<tr>
<td>DZF</td>
<td>.50*</td>
<td>.30*</td>
<td>.32*</td>
<td></td>
</tr>
<tr>
<td>DZM</td>
<td>.22</td>
<td>.49*</td>
<td>.18</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Cross-wave correlations</th>
<th>Wave 1-2</th>
<th>Wave 1-3</th>
<th>Wave 2-3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>By zygosity</td>
<td>Wave 1-2</td>
<td>Wave 1-3</td>
<td>Wave 2-3</td>
</tr>
<tr>
<td>MZ</td>
<td>.50*</td>
<td>.55*</td>
<td>.59*</td>
<td></td>
</tr>
<tr>
<td>DZ</td>
<td>.30*</td>
<td>.29*</td>
<td>.24*</td>
<td></td>
</tr>
<tr>
<td></td>
<td>By sex</td>
<td>Wave 1-2</td>
<td>Wave 1-3</td>
<td>Wave 2-3</td>
</tr>
<tr>
<td>MZF</td>
<td>.46*</td>
<td>.66*</td>
<td>.78*</td>
<td></td>
</tr>
<tr>
<td>MZM</td>
<td>.63*</td>
<td>.56*</td>
<td>.37</td>
<td></td>
</tr>
<tr>
<td>DZF</td>
<td>.30*</td>
<td>.44*</td>
<td>.33*</td>
<td></td>
</tr>
<tr>
<td>DZM</td>
<td>.37*</td>
<td>.14</td>
<td>.15</td>
<td></td>
</tr>
</tbody>
</table>

Note: *p<.05.
covariance between task persistence scores across occasions into genetic and environmental influences. The cross-wave MZ correlations were greater than the DZ cross-wave correlations and specifically, the MZ correlation was twice that of DZ correlation between wave 2 & 3. These values suggest that mainly genetic influences account for the stability over time.

As previously mentioned, quantitative sex differences are suggested when the resemblance within male and female same-sex pairs differs as a function of zygosity. Within-wave correlations were not similar for male and female groups, implicating differences in the extent of genetic and environmental contributions to phenotypic variance. In addition, MZ cross-wave correlations were greater than DZ cross-wave correlations, suggesting genetic factors contribution to phenotypic stability were significant.

*Saturated Model: Cholesky Decomposition*

The maximum likelihood value for the Cholesky model was 2758.554. Degrees of freedom were 1075, and Akaike’s information criteria (AIC) value was 608.554. In this model, parameter estimates such as heritability ($h^2$), shared ($c^2$) and nonshared ($e^2$) environments can be obtained by summing the squared path coefficients (See Table 3-4) from the latent factors on that measure at each occasion. For example, squaring path coefficients from A1 to Wave 2 ($0.48^2 = 0.23$) as well as from A2 to Wave 2 ($0.38^2 = 0.14$) and then summing up all the squared coefficients ($0.23 + 0.14 = 0.37$) will give the heritability estimate of task persistence at Wave 2.
Tab. 3-4: Standardized Path Coefficients from Cholesky model of Task Persistence

<table>
<thead>
<tr>
<th>Task persistence</th>
<th>Genetic path coefficients</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A1</td>
<td>A2</td>
</tr>
<tr>
<td>Wave1</td>
<td>.85 [.72, .96]</td>
<td></td>
</tr>
<tr>
<td>Wave2</td>
<td>.48 [.27, .69]</td>
<td>.38 [.04, .60]</td>
</tr>
<tr>
<td>Wave3</td>
<td>.49 [.22, .70]</td>
<td>.56 [.11, .76]</td>
</tr>
</tbody>
</table>

Shared Environment path coefficients

<table>
<thead>
<tr>
<th></th>
<th>C1</th>
<th>C2</th>
<th>C3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave1</td>
<td>.23 [.00, .49]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave2</td>
<td>.55 [.00, .75]</td>
<td>.00 [.00, .54]</td>
<td></td>
</tr>
<tr>
<td>Wave3</td>
<td>.32 [.00, .60]</td>
<td>.00 [.00, .47]</td>
<td>.00 [.00, .33]</td>
</tr>
</tbody>
</table>

Nonshared Environment path coefficients

<table>
<thead>
<tr>
<th></th>
<th>E1</th>
<th>E2</th>
<th>E3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave1</td>
<td>.48 [.41, .55]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wave2</td>
<td>.08 [.00, .22]</td>
<td>.60 [.53, .68]</td>
<td></td>
</tr>
<tr>
<td>Wave3</td>
<td>.09 [.00, .35]</td>
<td>.00 [.00, .11]</td>
<td>.63 [.51, .77]</td>
</tr>
</tbody>
</table>
The parameter estimates are presented in Table 3-5. Genetic influences on task persistence were predominant in Wave 1 and 3, whereas in Wave 2, both genetic and shared environmental influences were seen. Furthermore, the contributions of the genetic and environmental factors to the phenotypic correlations between Waves 1, 2 & 3 task persistence scores can be estimated by summing the products of the path coefficients from the latent factors that load on a pair of occasions, and then dividing by the phenotypic correlation between the scores measured at two different occasions. For example, the sum of the products of path coefficients from genetic factors A1 to Wave 2 and Wave 3 (.48*.49 =.24) and from A2 to Wave 2 and Wave 3 (.38*.56=.21) was .45, which indicates the genetic contributions to the phenotypic correlation between Wave 2 and 3 task persistence scores. Similarly, shared and nonshared environmental contributions were calculated; they were .18 and .01, correspondingly. The phenotypic correlation between two measurement occasions is the sum of all genetic and environmental contributions. So, the phenotypic correlation between Wave 2 and 3 measures was .64 (.45+.18+.01). The extent of contribution of genetic influences to the phenotypic correlation calculated by dividing the genetic contribution (.45) by the phenotypic correlation (.64), indicates that 70% of the phenotypic correlation is mediated genetically. Similarly, genetic influences contributed about 72% to the phenotypic correlation of task persistence scores between Wave 1 and 2, and 79% between Wave 1 and 3, respectively. Shared environmental contributions (13 to 28%) were moderate and nonshared environmental influences contributions (2 to 7%) were minimal.
Information about the genetic and environmental correlations can be obtained further from the Cholesky decomposition (Table 3-5). Genetic correlations explain the extent to which individual differences on task persistence at two different time points share the same genetic influences. Environmental correlations indicate the extent to which individual differences reflect the same shared or nonshared environmental influences. The correlation values can range between -1 and +1. Also, the genetic and environmental correlation values are independent of the extent of genetic and environmental influences on task persistence at each occasion (Harlaar et al., 2007). Note
that the genetic overlap among task persistence measures across three waves was high and significant. Also, the genetic correlations between closer time points were higher than those that were farther apart. Correlations between shared and nonshared environmental influences were insignificant.

*Longitudinal analyses*

To understand the mechanisms underlying the development of task persistence, the independent pathway and simplex models were employed. The independent pathway model is shown in Fig. 2. The common nonshared environmental pathway was fixed to zero to identify the model. The maximum likelihood value for the independent pathway model was 2773.90, degrees of freedom were 1087, and the AIC value was 599.90. As explained earlier, this model decomposes the variance and covariance among measures of task persistence across three waves into common and specific genetic and environmental effects. For example, .69, a genetic path estimate is a standardized partial regression coefficient of Wave 1 task persistence, which by squaring will give an estimate of the genetic variance due to a common factor. Similarly, genetic and environmental variances due to common and specific effects can be obtained by squaring the path coefficients.

The path coefficients of the model are presented in Table 3-6. Genetic effects accounted for 73% (.69^2 + .51^2) of variance in the Wave 1, 42% (.65^2 + .00) of the variance in Wave 2 and 46% (.67^2 + .14^2) of the variance in Wave 3. The common genetic factor accounted for 64% (.47/.74) of the genetic influence on Wave 1 task persistence and 100% (.42/.42) in Wave 2 and 98% in Wave 3 (.45/.46). Shared
environmental influences were due to common factors, but were not significant. However, significant unique nonshared environmental effects were seen in Wave 1 ($0.48^2=.23$), Wave 2 ($0.57^2=.32$) and in Wave 3 ($0.68^2=.46$).

| Tab.3-6: Path Coefficients of Independent Pathway Model with Confidence Intervals |
|---|---|---|---|
| Task persistence variance | Wave 1 | Wave 2 | Wave 3 |
| Genetic General | 0.69[0.49, 0.89] | 0.65[0.45, 0.82] | 0.67[0.43, 0.83] |
| Genetic Unique | 0.51[0.35, 0.64] | 0.00[0.35, 0.35] | 0.14[0.48, 0.48] |
| Shared Environment General | 0.19[0.44, 0.44] | 0.50[0.67, 0.67] | 0.30[0.57, 0.57] |
| Shared Environment Unique | 0.00[0.37, 0.37] | 0.00[0.37, 0.37] | 0.00[0.33, 0.33] |
| Nonshared Environment General | 0 | 0 | 0 |
| Nonshared Environment Unique | 0.48[0.41, 0.55] | 0.57[0.50, 0.66] | 0.68[0.52, 0.78] |

In summary, common A, C and E factors accounted for nearly 60% of the total variance. Note that the genetic factor loadings were significant on all three measurement occasions and similar in magnitude.

*Simplex model*

The simplex model decomposes the variance into genetic and environmental components, which were further classified into innovation and transmission effects. Innovation means new influences that act at a specific age. Transmitted influences are influences from a previous age that are still operating. The non-standardized estimates
can be converted to standardized coefficients by dividing the squared innovation path estimate by the total variance (sum of all genetic and environmental components and error variances); and dividing the product of the squared transmission path estimate and the genetic variance of the previous age by the total variance. The standardized path coefficients of the simplex model are presented in Table 3-7. Genetic variance due to innovation effect is calculated by squaring the genetic innovation path estimate. For example, genetic variance due to innovation effects in Wave 2 is .25(.50²). Genetic variance due to transmission effects are obtained from the product of squared transmission path estimate and genetic variance from previous occasion. For example, genetic variance due to transmission effects in Wave 2 is .38 (.71²*.77), where .77(.88²) is the genetic variance of task persistence in Wave 1. Similarly, environmental variances were also calculated. To identify the model, the transmission pathways of shared and nonshared environmental effects were fixed to zero.

<table>
<thead>
<tr>
<th>Variance</th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Genetic</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Innovation</td>
<td>.88[.77, .98]</td>
<td>.50[.33, .65]</td>
<td>.17[.00, .50]</td>
</tr>
<tr>
<td>Transmission</td>
<td>.71[.58, .92]</td>
<td>.95[.74,1.2]</td>
<td></td>
</tr>
<tr>
<td><strong>Shared Environment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Innovation</td>
<td>.00[.00, .36]</td>
<td>.27[.00, .45]</td>
<td>.00[.00, .34]</td>
</tr>
<tr>
<td>Transmission</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td><strong>Non-shared Environment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Innovation</td>
<td>.32[.00, .54]</td>
<td>.47[.09, .66]</td>
<td>.56[.17, .77]</td>
</tr>
<tr>
<td>Transmission</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Tab. 3-7: Genetic, shared and nonshared environmental path coefficients of a Simplex model with confidence intervals in brackets.
The maximum likelihood value for the full model was 2768.04, degrees of freedom were 1087, and the AIC value was 594.05. Genetic effects predominated in all three waves. The proportion of genetic variance contributed by innovation effects decreased from Wave 1 to Wave 3 from 100% to 5%, whereas contribution from transmission effects increased from 60% to 95%. Genetic innovation effects and transmission effects except the innovation effect in Wave 3 were significant. Shared environmental innovation effects in all three waves were not significant. Innovation effects contributed to nonshared environmental variance were significant. Error variance was about 0.12. Overall, the contribution of genetic factors was large, significant and stable across waves.

Approximately 66% of the phenotypic correlations observed between measurement occasions can be attributed to genetic factors and 20% can be attributed to nonshared environmental factors. The extent to which same genes or environmental factors contribute to the phenotypic correlation was denoted by genetic and environmental correlations. The correlation values were same as those values obtained from a Cholesky model. The genetic correlation between Wave 1 and Wave 2 as well as between Wave 1 and Wave 3 was high (~70 %) and stable, implying that most of the genes that operated in Wave 1 also act in Wave 2 and Wave 3. Also, the genetic correlation between Wave 2 and Wave 3 was 99%, reflecting the simplex pattern where the correlation between the measures will be high when the two time points are closer. The nonshared environmental correlations were low, indicating that nonshared environmental effects were mostly age-specific.
**Latent Growth curve model**

One of the purposes of the current study is to describe and analyze the development of task persistence across three measurement occasions and to assess the significance of genetic and environmental influences on growth model parameters. The model is presented in Fig. 4.

The model fitting results are shown in Table 3-8. The maximum likelihood value for the ACE model was 946.65, the degrees of freedom were 882, and AIC value was -817. The significance of parameters was tested by dropping them from the model and comparing the fit of the nested model to that of the full model using the chi-square critical value (-2ll) for the number of degrees of freedom gained in the nested model and AIC values. In this analysis, dropping the covariance parameter between intercept and slope caused a significant loss of model fit, implying that there is a significant relationship between the average performance and change in performance of a twin over time. Additionally, dropping the genetic factor that acts on an intercept led to a decrease comparing the fit of the nested model to that of the full model using the chi-square critical value (-2ll) for the number of degrees of freedom gained in the nested model and AIC values. In this analysis, dropping the covariance parameter between intercept and slope caused a significant loss of model fit, implying that there is a significant relationship between the average performance and change in performance of a twin over time. Additionally, dropping the genetic factor that acts on an intercept led to a decrease

<table>
<thead>
<tr>
<th>Task persistence</th>
<th>-2ll</th>
<th>df</th>
<th>Δχ²</th>
<th>Δdf</th>
<th>P</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE model</td>
<td>946.65</td>
<td>882</td>
<td></td>
<td></td>
<td></td>
<td>-817.34</td>
</tr>
<tr>
<td>No covariance - I &amp; S</td>
<td>972.66</td>
<td>885</td>
<td>26.00</td>
<td>3</td>
<td>&lt;.001</td>
<td>-797.34</td>
</tr>
<tr>
<td>No h² on Intercept</td>
<td>967.07</td>
<td>884</td>
<td>20.41</td>
<td>2</td>
<td>&lt;.001</td>
<td>-800.93</td>
</tr>
<tr>
<td>No h² on Slope</td>
<td>949.19</td>
<td>884</td>
<td>2.54</td>
<td>2</td>
<td>.28</td>
<td>-818.80</td>
</tr>
<tr>
<td>No c² on Intercept</td>
<td>946.73</td>
<td>884</td>
<td>.085</td>
<td>2</td>
<td>.95</td>
<td>-821.26</td>
</tr>
<tr>
<td>No c² on Slope</td>
<td>946.73</td>
<td>884</td>
<td>.085</td>
<td>2</td>
<td>.95</td>
<td>-821.26</td>
</tr>
</tbody>
</table>

Note: I-intercept; S-Slope; h²-Heritability; c²-Common shared environment.
in model fit, implying that genetic effects are central for the average performance of a twin.

The mean values as well as the genetic and environmental sources of variance of intercept and slope are presented in Table 3-9. Genetic influences predominated on both average performance (Intercept) and change (slope) in performance over time; however, the magnitude was less on slope. Shared environmental influences were minimal and not significant. Nonshared environmental contributions increased over time.

---

Tab. 3-9: Genetic and environmental sources of variance from latent growth model with age and sex as covariates

<table>
<thead>
<tr>
<th>Model</th>
<th>(h^2)</th>
<th>(c^2)</th>
<th>(e^2)</th>
<th>(h^2)</th>
<th>(c^2)</th>
<th>(e^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full</td>
<td>.92 [.48, 1.0]</td>
<td>.07 [.00, .49]</td>
<td>.01 [.00, .10]</td>
<td>.65 [.00, .99]</td>
<td>.05 [.00, .75]</td>
<td>.29 [.00, .73]</td>
</tr>
<tr>
<td>Age</td>
<td>.98 [.51, 1.0]</td>
<td>.01 [.00, .46]</td>
<td>.01 [.00, .10]</td>
<td>.69 [.00, .99]</td>
<td>.01 [.00, .74]</td>
<td>.30 [.00, .74]</td>
</tr>
<tr>
<td>Sex</td>
<td>.95 [.50, 1.0]</td>
<td>.05 [.00-.48]</td>
<td>.00 [.00, .10]</td>
<td>.66 [.00, .99]</td>
<td>.04 [.00, .76]</td>
<td>.30 [.00, .73]</td>
</tr>
</tbody>
</table>

Note: \(h^2\)-Heritability; \(c^2\)-Common shared environment; \(e^2\)-Nonshared environment.

The magnitude of genetic influences slightly increased when age was added as a covariate in the model. Age has a significant effect on task persistence only in Wave 2 (.08, CI: .02, .15). Increase in the mean value from 1.54[1.48, 1.60] to 1.62[1.5, 1.69] was noticed when sex was added as a covariate to the model. There were no changes in the mean values of slope. The effect of sex at Wave 1 (-.15[-.28, -.03]), Wave 2 (-.14[-.24, -.03]), and Wave 3(-.16[-.28, -.04]), was significant.
**Heterogeneity**

To find out the sex differences in task persistence heterogeneity and homogeneity models were used. Model fitting analyses were done using Mx (Neale, 1999) and twice the negative log-likelihood (-2LL) value for each model was obtained. The model fit was then assessed by comparing the -2LL of the saturated model with a sub-model. The difference in -2LL is distributed as chi-square. If the chi-square difference relative to its degrees of freedom was low, then that model fit was considered good. Model fitting results are presented in Table 3-10. Tests for homogeneity of variance were significant in

<table>
<thead>
<tr>
<th>Task persistence</th>
<th>-2ll</th>
<th>df</th>
<th>Δdf</th>
<th>Δχ²</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wave 1</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saturated</td>
<td>1157.52</td>
<td>439</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity</td>
<td>1172.48</td>
<td>447</td>
<td>8</td>
<td>14.9</td>
<td>.06</td>
</tr>
<tr>
<td>Homogeneity</td>
<td>1178.48</td>
<td>449</td>
<td>10</td>
<td>20.9</td>
<td>.02</td>
</tr>
<tr>
<td><strong>Wave 2</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saturated</td>
<td>977.38</td>
<td>356</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity</td>
<td>984.81</td>
<td>364</td>
<td>8</td>
<td>7.41</td>
<td>.49</td>
</tr>
<tr>
<td>Homogeneity</td>
<td>1006.40</td>
<td>366</td>
<td>10</td>
<td>29.0</td>
<td>.00</td>
</tr>
<tr>
<td><strong>Wave 3</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Saturated</td>
<td>636.83</td>
<td>235</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heterogeneity</td>
<td>651.47</td>
<td>243</td>
<td>8</td>
<td>14.6</td>
<td>.06</td>
</tr>
<tr>
<td>Homogeneity</td>
<td>679.16</td>
<td>245</td>
<td>10</td>
<td>42.3</td>
<td>.00</td>
</tr>
</tbody>
</table>

-2ll: maximum loglikelihood value; df: degrees of freedom; AIC: Akaike Information Criteria; Δdf- change in the degrees of freedom; Δχ²-Chi-square statistic; EQ- equal.
Wave 1, Wave 2 and Wave 3, suggesting that the magnitude of genetic and environmental influences is different for males and females. In all occasions, the AE model was the best fitting model for both males and females.

In addition to model fitting analysis, parameter estimates were obtained for males and females from the AE model, which split the phenotypic variance into genetic, and nonshared environmental variances for males and females. In Wave 1, no significant differences were noticed in the parameter estimates (See Table 3-11). In Wave 2, genetic influences were predominant in males whereas nonshared environmental influences were important for females. This was changed in Wave 3 where we can see that genetic influences predominate in both males and females, but the magnitude of the effects differed slightly for both groups. In Wave 3, the magnitude of nonshared influences increased in males whereas genetic effects increased in females.

<table>
<thead>
<tr>
<th>Model</th>
<th>$h^2$</th>
<th>Male $c^2$</th>
<th>Female $c^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wave 1</td>
<td>.75 [.59, .84]</td>
<td>.00</td>
<td>.25 [.15, .41]</td>
</tr>
<tr>
<td>Wave 2</td>
<td>.76 [.59, .85]</td>
<td>.00</td>
<td>.24 [.14, .41]</td>
</tr>
<tr>
<td>Wave 3</td>
<td>.65 [.13, .84]</td>
<td>.00</td>
<td>.35 [.16, .87]</td>
</tr>
</tbody>
</table>

Note: *p<.05.
In summary, the results suggested that there were quantitative differences in the genetic and environmental effects between males and females. An independent analysis to test whether the same genes and environmental factors that influence one sex also affect the opposite sex was not done because the sample has only same sex twins.

*Analyses with reading related skills*

The Pearson correlation between task persistence and Letter identification, Word identification, Word attack, Expressive vocabulary, Phonological awareness, General cognitive ability, and Reading comprehension, on average, was moderate (r range = .12 to .28).

The Cholesky model can be used to estimate genetic, shared environmental and nonshared environmental variances and also the correlations underlying the phenotypic correlation between traits. First, this model was used to estimate heritability (h²), shared environment (c²), and nonshared environment (e²) for each measure. Heritability and environmental estimates were obtained by squaring the genetic (a), shared (c) and nonshared environmental (e) paths, respectively. Estimates of genetic, shared and nonshared environmental contributions to Task Persistence (TP), General Cognitive Ability (IQ), Phonological Awareness (PA), Letter Identification (LID), Word Identification (WID), Word Attack (WATT), Expressive Vocabulary (BNT), and Reading Comprehension (RCOMP) are presented in Table 3-12. The statistical significance of each estimated parameter was determined by measuring the confidence intervals using the Mx program. Second, the contributions of genetic and environmental
Tab. 3-12: Heritability, shared environmental and nonshared environmental estimates of task persistence and reading related skills.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wave1</th>
<th>Wave2</th>
<th>Wave3</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>h²</td>
<td>c²</td>
<td>h²</td>
</tr>
<tr>
<td>TP</td>
<td>.77*</td>
<td>.00</td>
<td>.23*</td>
</tr>
<tr>
<td>LID</td>
<td>.28*</td>
<td>.45*</td>
<td>.28*</td>
</tr>
<tr>
<td>WID</td>
<td>.54*</td>
<td>.34*</td>
<td>.12*</td>
</tr>
<tr>
<td>WATT</td>
<td>.46*</td>
<td>.33*</td>
<td>.21*</td>
</tr>
<tr>
<td>PA</td>
<td>.65*</td>
<td>.18</td>
<td>.16*</td>
</tr>
<tr>
<td>BNT</td>
<td>.29</td>
<td>.46</td>
<td>.25*</td>
</tr>
<tr>
<td>IQ</td>
<td>.33*</td>
<td>.41*</td>
<td>.26*</td>
</tr>
<tr>
<td>RCOMP</td>
<td>.46*</td>
<td>.31*</td>
<td>.23*</td>
</tr>
</tbody>
</table>

Note: *p< .05. TP-Task persistence; LID- Letter Identification; WID-Word identification; WATT-Word attack; PA- Phonological Awareness; BNT-Expressive vocabulary; IQ-General cognitive ability; RCOMP-Reading comprehension.

Factors to the phenotypic correlations were estimated. As mentioned earlier, the total phenotypic correlation was obtained by summing up all standardized genetic, shared and nonshared environmental pathways from the model. The genetic and environmental covariances are presented in Table 3-13. The proportion of phenotypic correlation attributed to genetic influences was estimated by dividing the estimate obtained from genetic pathways by the total phenotypic correlation. Similarly, the extent of contribution of environmental influences to phenotypic correlation can be estimated. Lastly, information about the genetic and environmental correlations, which explains the extent to which the traits share the common genetic and environmental factors, was reported (See Table 3-14).
Tab. 3-13: Genetic, shared and environmental covariances of task persistence with reading related skill with confidence intervals in brackets

<table>
<thead>
<tr>
<th></th>
<th>Wave 1</th>
<th>Wave 2</th>
<th>Wave 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>TP with LID</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>.12 [.00, .29]</td>
<td>.01 [.00, .21]</td>
<td>.12 [.00, .29]</td>
</tr>
<tr>
<td>Shared Env.</td>
<td>.07 [.00, .25]</td>
<td>.13 [.00, .27]</td>
<td>.00 [.00, .13]</td>
</tr>
<tr>
<td>Non Shared Env.</td>
<td>.05 [.00, .11]</td>
<td>.04 [.00, .10]</td>
<td>.05 [.00, .14]</td>
</tr>
<tr>
<td><strong>TP with WATT</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>.10 [.00, .23]</td>
<td>.01 [.00, .21]</td>
<td>.14 [.00, .33]</td>
</tr>
<tr>
<td>Shared Env.</td>
<td>.03 [.00, .14]</td>
<td>.16 [.00, .30]</td>
<td>.03 [.00, .22]</td>
</tr>
<tr>
<td>Non Shared Env.</td>
<td>.03 [.00, .08]</td>
<td>.07 [.01, .12]</td>
<td>.08 [.02, .09]</td>
</tr>
<tr>
<td><strong>TP with PA</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>.03 [.00, .20]</td>
<td>.00 [.00, .23]</td>
<td>.11 [.00, .27]</td>
</tr>
<tr>
<td>Shared Env.</td>
<td>.08 [.00, .21]</td>
<td>.14 [.00, .26]</td>
<td>.00 [.00, .18]</td>
</tr>
<tr>
<td>Non Shared Env.</td>
<td>.05 [.01, .09]</td>
<td>.09 [.01, .15]</td>
<td>.10 [.01, .17]</td>
</tr>
<tr>
<td><strong>TP with IQ</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>.25 [.12, .38]</td>
<td>.08 [.00, .25]</td>
<td>.17 [.00, .34]</td>
</tr>
<tr>
<td>Shared Env.</td>
<td>.02 [.00, .12]</td>
<td>.05 [.00, .21]</td>
<td>.00 [.00, .23]</td>
</tr>
<tr>
<td>Non Shared Env.</td>
<td>.02 [.00, .06]</td>
<td>.00 [.00, .10]</td>
<td>.05 [.00, .16]</td>
</tr>
<tr>
<td><strong>TP with RCOMP</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Genetic</td>
<td>.22 [.00, .33]</td>
<td>.13 [.00, .26]</td>
<td>.22 [.02, .39]</td>
</tr>
<tr>
<td>Shared Env.</td>
<td>.00 [.00, .25]</td>
<td>.00 [.00, .18]</td>
<td>.00 [.00, .00]</td>
</tr>
<tr>
<td>Non Shared Env.</td>
<td>.02 [.01, .07]</td>
<td>.12 [.05, .20]</td>
<td>.08 [.01, .17]</td>
</tr>
</tbody>
</table>

Task persistence and letter identification

Quantitative genetic analyses results suggest that there were substantial environmental influences on LID (e.g. $c^2 = 0.49$; $e^2 = 0.22$ in Wave 3). Of an observed phenotypic correlation between TP and LID, 50% (.12/.24) in Wave 1 and 57% (.12/.21)
in Wave 3 was explained by genetic influences. There were no significant genetic and environmental correlations between TP and LID in any of the three waves.

Task persistence and Word Identification

In Wave 1 and Wave 2, both genetic and environmental influences were seen in WID. In Wave 3, the influences were mainly through genetic effects. Fifty-nine percent (.10/.17) of the correlation between TP and WID in Wave 1 and 56% (.14/.25) in Wave 3 was explained by genetic effects. The genetic correlation between TP and WID was not significant, but the nonshared environmental correlation was moderate in Wave 2 and in Wave 3, indicating that the influences on TP were shared with WID to some extent.

Task persistence and Word Attack

Both genetic and environmental influences were seen on WATT in all three measurement occasions. In Wave 1 and Wave 2, approximately 60 % of the phenotypic correlation between TP and WATT was due to shared environment effects and the rest of it was due to nonshared environmental factors. In Wave 3, both genetic and nonshared environmental influences contributed equally to the phenotypic correlation. It is interesting to note that significant nonshared environmental correlations between TP and WATT were consistent across all three waves (See Table 3-14).
Tab. 3-14: Genetic, shared environmental and nonshared environmental correlations of task persistence with reading related variables in Wave 1, 2 and 3

<table>
<thead>
<tr>
<th>Variable</th>
<th>Wave 1 $r_g$</th>
<th>$r_e$</th>
<th>$r_c$</th>
<th>Wave 2 $r_g$</th>
<th>$r_e$</th>
<th>$r_c$</th>
<th>Wave 3 $r_g$</th>
<th>$r_e$</th>
<th>$r_c$</th>
</tr>
</thead>
<tbody>
<tr>
<td>LID</td>
<td>.26</td>
<td>.99</td>
<td>.23</td>
<td>.13</td>
<td>.28</td>
<td>.13</td>
<td>.28</td>
<td>.00</td>
<td>.19</td>
</tr>
<tr>
<td>WID</td>
<td>.15</td>
<td>.44</td>
<td>.23</td>
<td>.04</td>
<td>.48</td>
<td>.33*</td>
<td>.21</td>
<td>.76</td>
<td>.39*</td>
</tr>
<tr>
<td>WAITT</td>
<td>.05</td>
<td>.99</td>
<td>.23*</td>
<td>.00</td>
<td>.59</td>
<td>.26*</td>
<td>.17</td>
<td>.00</td>
<td>.37*</td>
</tr>
<tr>
<td>PA</td>
<td>.35*</td>
<td>.37</td>
<td>.11</td>
<td>.22</td>
<td>.14</td>
<td>.10</td>
<td>.46</td>
<td>.65</td>
<td>.12</td>
</tr>
<tr>
<td>BNT</td>
<td>.00</td>
<td>.21</td>
<td>.16</td>
<td>.13</td>
<td>.00</td>
<td>.16</td>
<td>.29</td>
<td>.00</td>
<td>.04</td>
</tr>
<tr>
<td>IQ</td>
<td>.45</td>
<td>.00</td>
<td>.11</td>
<td>.34</td>
<td>.00</td>
<td>.39*</td>
<td>.37*</td>
<td>.00</td>
<td>.31</td>
</tr>
<tr>
<td>RCOMP</td>
<td>.31</td>
<td>.00</td>
<td>.00</td>
<td>.34</td>
<td>.38</td>
<td>.21</td>
<td>.48*</td>
<td>.00</td>
<td>.19</td>
</tr>
</tbody>
</table>

Note: *P < .05; $r_g$ - genetic correlation; $r_e$ - shared environmental correlation; $r_c$ - nonshared environmental correlation; LID - Letter Identification; WID - Word identification; WATT - Word attack; PA - Phonological Awareness; BNT - Expressive vocabulary; IQ - General cognitive ability; RCOMP - Reading comprehension.

Task persistence and Phonological Awareness

Univariate genetic and nonshared environmental estimates of PA were significant in all three measurement occasions. Shared environmental influences were seen in Wave 2 and Wave 3. The contribution of genetic effects to phenotypic correlation decreased from 85% to 50% from Wave 1 to Wave 2, and then increased to 80% in Wave 3. There was a significant genetic correlation between TP and PA in Wave 1 only. The environmental correlations between TP and PA were not significant.

Task persistence and Expressive vocabulary

Substantial shared environmental influences on BNT were seen on all three measurement occasions. There were no significant genetic or environmental correlations between TP and BNT in all three waves.
Task persistence and General cognitive ability

Approximately equal genetic and environmental effects were seen on general cognitive ability in all three waves. Of an observed phenotypic correlation between TP and IQ in Wave 1, nearly 90% (.22/.24) was explained by genetic influences, and in other two occasions, the genetic contribution was still significant. No significant genetic or environmental correlations were observed between TP and IQ in Wave 1. In Wave 2, moderate nonshared environmental correlation and in Wave 3, moderate genetic and nonshared environmental correlations were seen.

Task persistence and Reading comprehension

A substantial amount of genetic influences and moderate shared environmental influences were seen on RCOMP. Genetic influences explained most (~85%) of the correlation observed between TP and RCOMP in Wave 1 and Wave 3, while shared influences contributed moderately (~25%) in Wave 2. It is interesting to note a moderate (.49) genetic correlation between TP and RCOMP in Wave 3. Environmental correlations were not significant.

In summary, the major source of variance contributing to the relationship between task persistence and across all reading related skills in Wave 1 and in Wave 3 was genetic. In Wave 2, the role of shared environmental influences was evident. Nonshared environmental influences were significant in all three waves; however, this also includes measurement error. Regarding the genetic and environmental correlations, significant nonshared environmental correlations were observed between TP and WID as well as between TP and WATT in Wave 2 and 3. In Wave 3, there was evidence that the genes that exert influence on TP also affect higher cognitive skills such as IQ and RCOMP.
Chapter 4

DISCUSSION

Stability and change

The longitudinal quantitative genetic study extended previous research on task persistence by exploring genetic and non-genetic etiologies of stability and change over three measurement occasions. In the current study, four models were used to assess the stability and change in task persistence. The Cholesky model was used as a saturated model, and variance and covariance values provided information about change and stability, respectively. In addition, a simplex model and a common factor independent pathway model were used to distinguish between a transmission process and a common factor process underlying the genetic and environmental influences on task persistence throughout the early elementary school years. The simplex model assumes that behavior at subsequent points in time is causally linked and earlier genetic factors or experience influence the present behavior. The common factor independent pathway related continuity of a behavior to stable underlying genetic and/or environmental factors. A biometric latent growth curve model was applied to assess the change.

The results suggest that the stability of task persistence across three measurement occasions appears to be due to genetic influences. Additionally, nonshared environmental influences along with some age-specific genetic influences accounted for the change in Wave 1. Shared environmental contributions were not significant.
In longitudinal studies, information about stability and change can be gained from three estimates: stability coefficients, heritability, and genetic and environmental correlations. The stability coefficient is simply the correlation of the same variable between two points in time. In this study, the stability coefficients of task persistence across Wave 1, Wave 2, and Wave 3 measurement occasions were .52, .51 and .49, respectively. These values indicate moderate correlation of task persistence across the three waves. Also, the coefficients’ values are around .50 (less than a perfect correlation of 1.0), which indicates that there are individual differences in change. However, change patterns do not vary randomly across individuals, in which case stability coefficients can be zero (Hultsch, 1998).

Individual differences focus on specific differences between individuals rather than the average differences between groups of people. Developmental behavioral genetics is limited to the study of genetic effects on individual differences in behavior (Plomin, 1986). Changes in genetic influences occur during development. Genetic changes can be viewed as changes in the magnitude of genetic variance at each point in time, as well as changes in the genetic covariance across different points in time. In this study, genetic effects were predominant in all three waves, but differences in the magnitude of genetic effects were still seen. In Wave 1, the heritability of task persistence was .72, which decreased to .37 in Wave 2 and then increased to .52 in Wave 3. The decrease in the magnitude of heritability in Wave 2 and Wave 3 was observed as the environmental effects increased on those two occasions. In Wave 2, shared environmental effects on task persistence were moderate and significant. Shared environmental influences, which account for twin similarity beyond genetic effects, were
This is perhaps because the twins were either exposed to similar learning environments, or were exposed to environments, which enforce their self-regulation, such as structured playground activities, which demand more attention and regulation of their behavior to achieve goals. It is interesting to note the increase in nonshared environmental influences on task persistence. Nonshared environmental influences are relevant environmental influences that are specific to each child (Dunn & Plomin, 1990). It also includes idiosyncratic experiences such as those resulting from chance (Rowe & Plomin, 1981). Changes in the magnitude of environmental influences are expected in normal development, since prevailing environmental conditions influence biological development. As children age, they tend to seek (gene-environment interactions) and have different experiences (passive and provocative gene-environment correlations). Measurement error is also included in this component, however.

The results of this study corroborated previous research on task persistence. For example, a quantitative longitudinal study conducted by Goldsmith and Gottesman (1981) in a sample of 350 twin pairs demonstrated that genetic factors play a substantial role in the variability in task persistence observed in infants and young children. In the same study, they observed a decrease in the magnitude of genetic influences between 4 and 7 years, which is the age of school entry. The authors explained that the variability in the magnitude of influences could be due to new experiences in school, which may cause changes in the influences that affect the trait. A review of quantitative genetics longitudinal studies (Goldsmith, 1983) also documented the genetic bases for stability in certain personality dimensions which also include persistence.
In addition to change in heritability, the second type of genetic change in development is age-to-age change and continuity (Plomin, 1990). The changes may not reflect the molecular mechanisms underlying the genetic changes. For example, heritability measured at one point in time may be due to genes that are still operating from a previous point in time, due to new genetic influences, or due to a single genetic factor. These different operating mechanisms were distinguished based on the simplex and common factor models. In this study, the simplex model showed that genetic influences substantially accounted for the stability of task persistence. Additive genetic transmission factors accounted for 32% of the stability. Changes in task persistence at each occasion were brought on by innovation factors. In Wave 1, genetic factors were responsible for the change, whereas nonshared environmental factors played a role in all three occasions. A similar pattern was observed in a common factor independent pathway model, where common genetic factors influence task persistence in all three occasions. Unique nonshared environmental influences were seen in Wave 2 and Wave 3.

Continuity and change are often indexed by correlations of the same variable scores across two points in time (Caspi, Roberts & Shiner, 2005). The genetic correlations between closer points in time in Wave 1 and Wave 2, and in Wave 2 and Wave 3 were .79 and .98, respectively. The genetic correlation between distant time-points, as between Wave 1 and Wave 3 was .66. This corresponds to a transmission pattern in which the correlations between scores of same variable measured at closer time intervals are greater than those measured at distant time intervals.

Overall, genetic change measured as change in the magnitude of heritability and high correlations across three waves indicated that task persistence is a stable trait.
Change in task persistence was due to nonshared environmental influences along with some genetic influences in the earlier occasion.

Change in mean-level also occurs during development; this refers to changes in the average trait level of a population (Caspi, Roberts, & Shiner, 2005). In a recent study by Qhou et al. (2007), three developmental trajectories were identified for persistence. The majority of children showed a high level of stable persistence from ages 5 to 10 years, and a subgroup of children with low persistence at the beginning showed an increase along with a change in maturity level. The authors explained that the differences observed in the stability of persistence in subgroups could be due to individual differences. Task persistence, being a multifaceted behavior, could both influence and be influenced by many factors, such as intelligence and parental warmth. (Deater-Deckard et al., 2005). Differences in the genes as well as the experiences contributing to the individual differences could affect age-related changes in the population means and within population differences in their developmental trajectories (Zhou et al., 2007). The findings of latent growth curve analysis in the present study suggested that genetic influences affect the mean level of task persistence behavior. In addition, inclusions of age and sex as covariates in the model revealed that age and sex have significant effects on task persistence. The addition of age as a covariate in the model decreased the magnitude of shared environmental effects. Effects of age on task persistence was significant only in Wave 2, whereas adding sex as a covariate increased the mean value of the intercept, and significant effects of sex were seen in all three measurement occasions.
In summary, genetic influences were predominant on intercept and slope, but the effect is more significant on intercept than on slope. The effects of nonshared environmental influences on slope were not significant. Moreover, age and sex effects were seen on intercept through genetic influences. The outcomes of the growth curve analysis required further analyses exploring the effects of sex differences on task persistence to be done.

**Sex differences**

From quantitative genetic methods, the etiology of the mean differences between the sexes cannot be assessed, but contribution of the genetic and environmental effects to group differences can be estimated (Dolan, Molenaar, & Boomsma, 1992). Tests were conducted to examine the sex differences in means as well as variances. These tests were mainly conducted to assess the quantitative difference i.e. differences in the magnitude of genetic and environmental influences, though the genetic and environmental influences were assumed to be the same. In the present study, the results suggested that there were differences in means of task persistence scores between sex groups. In addition, tests for homogeneity were significant, implying that the magnitude of genetic and environmental influences on task persistence was different for males and females. Genetic influences were predominant for both sexes in all three waves except in Wave 2, where nonshared environmental influences were predominant in females. Though the genetic and environmental estimates seem to be similar, there may be differences in the phenotypic
variance. Sex differences may be due to differences in gene expression (Turner, Cardon, Hewitt, 1995).

In summary, there are sex differences in task persistence, which are evident from the differences in means as well as from the differences in the extent of genetic and environmental influences on task persistence.

Relationship between task persistence and reading related skills

Current theories posit distinct but interrelated influences on learning from recognition and strategic networks (Rose & Strangman, 2007). To test the correlation, multivariate genetic analyses were conducted to examine the sources of variance and covariance between task persistence and reading related skills, such as phonological awareness, letter identification, word identification, word attack, reading comprehension and general cognitive ability. Multivariate analyses were conducted for each wave separately.

Shared environmental influences were dominant on letter identification in all three waves and genetic and nonshared influences were the second source of influences. In contrast, genetic influences were predominant on phonological awareness in Wave 1. The magnitude of influences decreased in later occasions, whereas shared and nonshared influences increased across waves.

Both genetic and environmental influences were seen on Word Identification (Word ID) and Non-word Identification (Word Attack) in all three waves. In the Byrne et al. (2007) study, heritability and environmental estimates of word and non-word reading
were compared with the results of Petrill et al. study (2007) using a WRRP sample, which was used in the present study as well. The comparison revealed some discrepancies in sample estimates. Differences in the measures as well as the differential effects of genes on reading accuracy and reading efficiency were accounted as the reasons for the discrepancies (Byrne et al., 2007). This comparison is applicable only to Wave 1 of the present study, since quantitative genetic studies of reading skills beyond second grade are limited.

Almost equal contributions from genetic and environmental influences were observed on general cognitive ability (IQ) in all three occasions. In reading comprehension, both genetic and shared environmental influences were seen in the first occasion. Later on, the magnitude of genetic influences increased and shared environmental influences decreased.

One of the main purposes of the study was to examine the covariance between task persistence and reading related skills. The amount of contribution to phenotypic correlation between task persistence and reading related skills was mostly from genetic influences, except in the correlation between task persistence and word attack, where shared environmental influences played a major role. In addition, an estimation of genetic, shared, and nonshared environmental correlations was done to examine whether there was evidence of overlapping genetic or environmental influences across task persistence and reading related skills. There were moderate (.33, .39) nonshared environmental correlations between task persistence and word identification in Wave 2 and Wave 3. Similarly, moderate (.23-.37) nonshared correlations were observed between task persistence and word attack; also, there is a crescendo pattern across all three waves.
There was a moderate (.35) genetic correlation between task persistence and phonological awareness only in Wave 1. In subsequent waves, there were moderate to high genetic and shared environmental correlations, but these were not significant. Though genetic and nonshared environmental correlations between task persistence and general cognitive ability were seen in all occasions, the nonshared environment correlation (.39) became significant only in Wave 2, and the genetic correlation (.37) in Wave 3. There was a high (.48) genetic correlation between task persistence and reading comprehension in Wave 3.

In summary, the extent of overlap of genes and environmental factors between task persistence and reading related skills was moderate. If we classify the reading related skills hierarchically, the relationship between task persistence and lower cognitive skills such as letter identification, phonological awareness, word attack, and word identification were fairly small whereas the relationship was moderate between task persistence and higher cognitive skills, such as reading comprehension and general cognitive ability.
Chapter 5

CONCLUSIONS

Limitations

Apart from general methodological limitations of the twin design (Plomin et al., 2001), a few limitations of the present study should be noted. The present study is currently limited to three measurement occasions. The development of components of task persistence, such as attentional and inhibitory control, continues to show age-related improvement throughout childhood (Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999; Williams, Ponesse, Schachar, Logan, & Tannock, 1999). Moreover, it is possible to notice age-related changes beyond early childhood. Task persistence is a complex process, certain components, especially inhibitory control, continues to grow throughout childhood and adolescence (Murphy et. al, 1999; Williams, 1999). Research on normative development of effortful control, a construct with structural and functional overlap with task persistence, identified different patterns of trajectories in children from 5 to 10 years old (Zhou et al., 2007). Since the standard latent growth model, which relies on the assumption that individual change is similar for all individuals, was used in this study, different trajectory patterns were not observed. Further longitudinal studies to detect nonlinear changes, as well as different patterns of change, are needed to elucidate genetic and environmental contributions to intra-individual change, as well as individual
differences in task persistence. These goals could be achieved by using longitudinal data from more than three occasions, and by using latent growth mixture models.

The sample contains boys and girls in approximately equal proportions. Therefore, it seems reasonable to set up all models used in the study separately for boys and girls. In the present study, this was not possible due to lack of power. The quantitative differences in genetic and environmental effects on boys and girls were estimated in this study. But, qualitative differences, such as whether the same set or different sets of genes or environments influencing task persistence in both sexes, were not tested. To examine the qualitative differences, opposite-sex twin pairs should be included in the model.

Another caveat is that the measurements were based only on teacher and tester ratings. Since different teachers reported on twins’ behaviors at different ages, the chances of introducing bias in their reports were less. Moreover, the validity and reliability of self-reported measures on task persistence, an aspect of self-regulation, by early and middle- elementary school aged twins are questionable (Murphy, Eisenberg, Fabes, Shepard, & Guthrie, 1999). Reports from teachers and testers can be used, at least in the measurement of task persistence, because the measures of the construct relied on observable behaviors rather than self-report of internally experienced feelings.

Another limitation of this study is related to generalizability of the results. Beyond the limitation in generalizing the results of twin studies to singletons, issues in operational definition of task persistence could pose problems. Studies may vary in their definition of task persistence or differ in the methods of measuring it. These discrepancies may cause differences in the estimates of heritability and environmental
effects. In addition, since the behavioral measures are mostly based on the observational
reports, perceptions, and standards set by the raters by which are used to judge the
behavior as normal or abnormal. The context in which the behavior occurs could also
affect the ratings (Silberg et al., 1994). This variability in behavioral reports by different
informants may cause discrepancies in the genetic and environmental influence
estimations. However, in this study, consistency of the task persistence behavior was
established across situations and informants.

Another caveat is that this study was limited to estimation of the main effects.
The models used in this study to estimate genetic and environmental effects are based on
the assumption that these influences are independent. This excludes the possibility of
gene-environment interactions and correlations that give specific explanations for finding
individual differences. Generally, the interactions between genetic and nonshared
influences tend to increase the proportion of nonshared influence, and the correlation
between genetic and nonshared influences acts to increase the proportion of genetic
influence (Purcell, 2002). The chances of gene-environment interactions in task
persistence cannot be excluded since the proportion of nonshared environmental
influences on task persistence increased across all three measurement occasions of this
study. It is thus crucial for future studies to assess gene-environment interactions in the
presence of gene-environment correlations and include environmental measures to
enhance the detection of such interactions.
Implications

The contributions from genetic and environmental effects to phenotypic variations can be assessed from cross-sectional studies, but it is not possible to understand the developmental nature of the trait. Longitudinal studies examine the extent to which genetic and environmental factors are transmitted from age-to-age, changes in the influences, or specific influences operating at specific age or a combination thereof. The clear understanding of the developmental pattern provides guidelines for designing interventions. In this study, it is clear that task persistence is a stable trait and genetic factors accounted for the stability, while nonshared influences accounted for the change. The fact that genes have a powerful influence on task persistence does not mean that the trait is unchangeable. Identifying the mechanisms through which genes influence behavior will allow us to design effective interventions. One possible mechanism, by which genes affecting task persistence behavior could be through the environment of a child, may be through the structures of environment that was imposed on a child or through the structures that the child actively selects from the environment. Nonshared environment may bring out traits that are latent and could thus amplify the effect of genes (Steen, 1996).

Additional findings of this study are that the moderate level of nonshared influences with no transmission of effects across ages supports interventions that address issues pertinent to environmental factors specific to individuals. For example, for children with low task persistence, instead of encouraging group activity, encouraging the playing of interactive games on personal computers could motivate them to self-monitor
and regulate their attention. Players with attention deficit disorder, when they engaged in interactive games that detect brain waves, improved in the ability to sustain attention (Pope & Bogart, 1996). Early interventions that address the changes due to development could have long-term beneficial impacts.

Importantly, the results showed that task persistence has a modest but significant genetic correlation with reading related skills, especially letter identification, word identification, phonological awareness, and reading comprehension, as well as with general cognitive ability. It suggests that the genes of small effect size, which affect task persistence, may have influence on reading skills. This finding has implications for studies searching for common factors between behavior and cognitive skills, as well as for studies searching for the genes responsible for Attention Deficit Hyperactive disorder (ADHD) and co-morbid reading disability (RD). The results were more in favor of the shared common etiology hypothesis (Willcutt et al., 2001) of ADHD and RD. In children diagnosed as ADHD with RD, Stevenson et al. (2005) noticed that these disorders are associated with the alpha 2A adrenergic receptor polymorphism with the G allele. Further molecular genetic studies are warranted to do a systematic search for other polymorphisms associated with these disorders.

Another implication of this study is the development of intervention strategies for improving reading ability through its association with task persistence. It is well known that gene expression can be altered by changes in the microenvironment of corresponding activated brain regions. Although task persistence, a self-regulatory process, is biologically based, it is influenced by experience as well as the environment wherein child is placed (Blair, 2003). If this situation-specific influence on this component of
temperament (Teglasi, Cohn, Meshbesher, 2004), is acknowledged, interventions could be directed at this component to improve reading ability. However, intervention studies are needed to support the idea of using psychological concepts as a base for education.
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VITA

Saradha Ramesh

Office Address: Center for Developmental and Health Genetics
101 Amy Gardner House
The Pennsylvania State University
University Park, PA 16802
Phone: (814) 865-4668
E-mail: saradha196@yahoo.com

Home Address: 800 Bulfinch Drive
Apt. 314
Andover, MA 01810
Phone: (978) 208-8659

Education:
1986-1992 M.B.B.S.
Thanjavur Medical College, Thanjavur, India

Selected Professional Experiences at The Pennsylvania State University:

2004-2007 Graduate Research Assistant; Center for Developmental and Health Genetics

2007 Course Instructor; BBH 119, Behavior, Health & Disease; Department of Biobehavioral Health; Summer Session #1

2004-2007 Graduate Teaching Assistant; Department of Biobehavioral Health.

Honors and Special Awards:
November 2005: Hintz Family Fellowship for Graduate Excellence

Additional Relevant Training: