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Processing Speed and Longitudinal Trajectories of Change for Cognitive Abilities: The Swedish Adoption/Twin Study of Aging

Deborah Finkel[1] and Nancy L. Pedersen[2,3]
[1]School of Social Sciences, Indiana University Southeast, New Albany, IN, USA,
[2]Department of Medical Epidemiology and Biostatistics, Karolinska Institute, Stockholm, Sweden,
and [3]Department of Psychology, University of Southern California, Los Angeles, CA, USA

ABSTRACT

In this manuscript, The Swedish Adoption/Twin Study of Aging (SATSA) is described. Although the study is a multidisciplinary program in gerontological genetics, the summary of findings focuses on the cognitive measures. In the second part of the manuscript, we investigate the role played by measures of processing speed in explaining the longitudinal trajectories of change for cognitive abilities and the genetic and environmental influences on those trajectories. When processing speed was regressed out of cognitive measures representing three cognitive domains (crystallized, fluid, and memory) and a general cognitive factor, the trajectory of decline was less severe. Quantitative genetic analyses indicated that environmental variance increases in late adulthood. A substantial portion of the genetic variance for these cognitive abilities was accounted for by genetic variance for speed. With increasing age, genetic variance associated with processing speed becomes a more prominent component of genetic variance for fluid abilities and for the general cognitive factor.

PART 1. THE SWEDISH ADOPTION/ TWIN STUDY OF AGING (SATSA): DESIGN AND RESULTS CONCERNING COGNITIVE ABILITIES

Historical Overview

The Swedish Adoption/Twin Study of Aging (SATSA) is a longitudinal program of research in gerontological genetics, the purpose of which is to study genetic and environmental sources of individual differences. The origin of the project dates to 1978 when it was observed that an appreciable number of twins in the Swedish Twin Registry (Lichtenstein et al., 2002; Pedersen, Lichtenstein, & Svedberg, 2002) indicated that they had been separated at some time during early rearing. Most of these twins were born before the Second World War and separated for reasons of economic hardship, death of one or both parents, etc., and thus were on average in their early 60's in the 1980's. As a result of the age distribution of the twins and the adoptive twin design, SATSA represents an inimitable opportunity to characterize genetic and environmental sources of individual differences during aging.

Initially, SATSA data collection was based on a 3-year interval between measurement occasions (in IPT5 the testing interval was 7 years). During

Address correspondence to: Nancy L. Pedersen, Department of Medical Epidemiology and Biostatistics, Karolinska Institute, Box 281, SE 171 77 Stockholm, Sweden. Fax: +46-8-31-49-75. E-mail: nancy.pedersen@mep.ki.se

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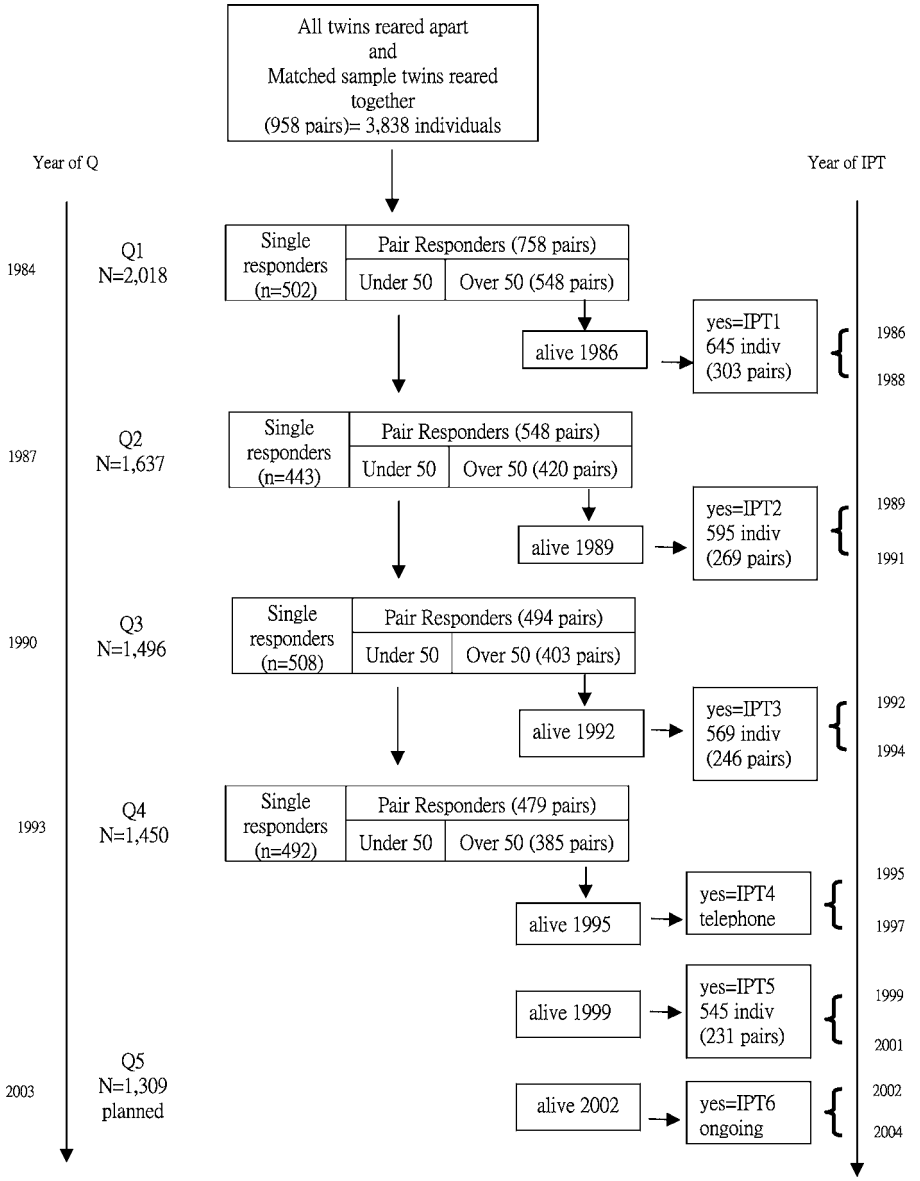

Fig. 1. SATSA testing design. Q = questionnaire phase, IPT = in person testing.

each wave of testing, both mail-out questionnaire (Q) and in-person testing (IPT) components are included (Fig. 1). The base population is comprised of all pairs of twins from the Swedish Twin Registry who indicated that they had been separated before the age of 11 and reared apart, and a sample of twins reared together matched on the basis of gender, date and county of birth (Pedersen, Friberg, Floderus-Myrhed, McClearn, & Plomin, 1984). Description of the SATSA sample is particularly complex in that the study includes not only questionnaire and in person testing forms of data collection, but also twins who can be treated either as individuals or as members of pairs. At the onset of SATSA we chose to contact all surviving individuals from pairs identified as reared apart and the matched sample of twins reared together (Pedersen et al.,

1991). Thus, the following description of the sample will in part treat the sample as individuals, and in part as pairs.

Questionnaire Phases

The first questionnaire (Q1) was sent in 1984 to all (2,845) surviving individuals from the base sample; 2,018 responded. Q1 contained chapters concerning Health, Personality, Rearing Environment, Working Environment, and Current Environment. Measures were chosen that represented both gerontological and quantitative genetic issues. Care was taken to select scales used in other studies of aging that were available at that time (e.g., the OARS, (Fillenbaum & Smyer, 1981) and H-70 (Rinder, Roupe, Steen, & Svanborg, 1975)).

Q2 was sent in 1987, 3 years after Q1. The chapters concerning working environment and early rearing environment were not included in Q2. Some scales were shortened whereas others were added. Q3 was sent in 1990 and Q4 was sent in 1993. Q2–Q4 were sent to all individuals alive at the time of the mailing, regardless of response to previous questionnaires (see Fig. 1). The same questionnaire as that sent at Q2 and Q3 was sent to the twins participating in in-person testing waves 2 and 3 (IPT2 and IPT3, see below) 1 week prior to in-person testing. A questionnaire was also sent to participants in IPT5. Thus, for a total of 394 individuals there is contemporaneous collection of information about physical health, health-related behaviors, personality, and social support from seven measurement occasions thus far.

In-Person Testing (IPT)

In-person testing (IPT) involved an interview, administration of cognitive tests and a health examination of a subset of the SATSA twins. In order to be contacted for participation in IPT1, subjects had to be members of pairs in which both responded to Q1 and were above the age of 50. The first wave of IPT was conducted between 1986 and 1988; 645 individuals in 303 complete pairs were tested. All individuals participating in IPT1, regardless of the survival status of their twin partner, were contacted for testing in IPT2. In addition, the subsample of pairs responding to Q1 who turned 50 years of age between 1986 and 1990 were also contacted for IPT2 testing. A total of 595 individuals participated in IPT2, which was conducted between 1989 and 1991. All individuals who had participated in either IPT1 or IPT2, regardless of the status of their twin partner, were contacted for testing in IPT3. Again, a subsample of pairs responding to Q1 who turned 50 years of age between 1990 and 1993 were also contacted for IPT3 testing. Five hundred sixty-nine individuals participated in IPT3, which was conducted between 1992 and 1994. Due to funding considerations, the fourth ''IPT'' became a telephone based brief cognitive screening test rather than an in-person cognitive battery. All individuals who had participated in any IPT, regardless of the status of their twin partner, were contacted for testing in IPT5. Again, pairs responding to Q1 who turned 50 years of age between 1993 and 1999 were also contacted for IPT5 testing. IPT5 was conducted between 1999 and 2001, and 545 individuals participated.

The number of participants assessed at each IPT and Q phase of SATSA is presented in Figure 1. The zygosity and rearing status of individuals participating at each testing phase is presented in Table 1, along with information about the extent of longitudinal data available. The number of individuals who have participated in IPT1, IPT2, IPT3, or IPT5 by zygosity and rearing status, is presented in the left half of Table 1(a). Data for the questionnaire assessments are presented in Table 1(b). The number includes participants from both complete and incomplete pairs. The extent of longitudinal data available for participants (i.e., the number of measurement occasions individuals have participated in) is presented in the right half of the table.

IPT Measures

IPT consists of two major components: biomedical and cognitive assessment. The choice of measures was directed in part by concerns for maintaining comparability with other aging studies (notably Swedish studies: H-70 and OCTO, and American studies such as the Older American's Resources and Services, the MacArthur Research Network on Successful Aging field studies), for minimizing time

Table 1. Number of Individuals Participating in In-Person Testing (a) and Questionnaire (b) Phases of SATSA, by Zygosity and Rearing Status.

Group	Participation in IPTs				Longitudinal participation			
	IPT1	IPT2	IPT3	IPT5	Only 1 IPT	Any 2 IPTs	Any 3 IPTs	All 4 IPTs
(a) In-Person Testing								
MZA	101	84	81	77	33	14	38	63
MZT	140	132	126	122	46	30	52	95
DZA	216	204	191	189	48	51	65	133
DZT	184	170	166	154	39	37	69	102
XZA	2	3	1	0	2	2	0	0
XZT	2	2	4	3	1	3	0	1
Total	645	595	569	545	169	137	224	394
Group	Participation in Qs				Longitudinal participation			
	Q1	Q2	Q3	Q4	Only 1 Q	Any 2 Qs	Any 3 Qs	All 4 Qs
(b) Questionnaire								
MZA	260	216	198	186	49	47	55	138
MZT	386	320	295	270	68	56	65	224
DZA	664	553	490	495	120	106	118	379
DZT	566	441	410	392	111	90	102	303
XZA	51	40	37	40	19	18	11	20
XZT	92	67	66	67	27	24	19	40
Total	2019	1637	1496	1450	394	341	370	1104

Note. MZ = identical, DZ = fraternal, XZ = undetermined zygosity, A = apart, T = together.

demands on the subjects, and for ease in administration in remote sites. The biomedical portion of the battery was designed to assess general health status, with particular attention to measures which are sensitive to age changes and might therefore be useful biomarkers of aging (Reff & Schneider, 1982). For this reason, measures of lung function (vital capacity and forced expiratory volume), grip strength, and physical performance measures for upper and lower body functional capacity were included. Many of the performance measures were chosen on the basis of their ecological validity for measuring activities of daily living (e.g., turn key in lock, pour glass of water). The extensive nutritional survey allows computation of quantity and frequency measures of several nutrients. At IPT3, information about occupational history and environmental exposures (particularly those which may influence cognitive decline) was included in a structured interview. Fasting blood samples were drawn for analysis of a wide range of clinical chemistries including lipids and for confirmation of zygosity diagnosis.

Cognitive Battery

The cognitive battery was designed to represent the domains of crystallized and fluid intelligence and memory (Nesselroade, Pedersen, McClearn, Plomin, & Bergeman, 1988) and the battery (Table 2) included all of the tests used in the H-70 study of aging in Gothenburg, Sweden (Berg, 1980). Additional measures of spatial ability and perceptual speed were included so that specific cognitive abilities, commonly analyzed in quantitative genetic research, would be represented. The Swedish version of Folstein's Mini Mental State Examination (MMSE) (Folstein, Folstein,

Table 2. Cognitive Tests and Domains in SATSA.

Test	Domain	Specific cognitive ability
Information	Crystallized	Verbal
Synonyms[a]	Crystallized	Verbal
Analogies	Fluid and Crystallized	Verbal, reasoning
Figure Logic[a]	Fluid	Spatial, reasoning
Block Design[a]	Fluid	Spatial
Card Rotations		Spatial
Digit Span (F & B)		Memory
Picture Memory[a]		Memory
Names and Faces		Memory
CERAD Word List[b]		Verbal Memory
Symbol Digit		Perceptual Speed
Figure identification[a]		Perceptual Speed

Note. [a]Test included in H-70 study of aging in Sweden.
[b]Test added at IPT5.

& McHugh, 1975) was included as a screening instrument for possible cognitive impairment. At IPT5, the Word List task from the CERAD (Storandt, Botwinick, Danziger, Berg, & Hughes, 1984) was added to the cognitive battery.

SUMMARY OF RESULTS CONCERNING COGNITIVE ABILITIES

Individual Differences in the Elderly

SATSA provides some of the first evidence for the relative importance of genetic and environmental influences on cognitive abilities in adults over 50 years of age. Eighty percent of the differences seen for general cognitive ability are due to genetic differences (Pedersen, Plomin, Nesselroade, & McClearn, 1992). These results are based on the IPT1 data, and treat the sample as one age group, with a range from 50 to 85 years. However, a further exploration into age differences in heritability estimates during the last half of the lifespan found some differences across age groups (Finkel, Pedersen, McGue, & McClearn, 1995). Older Swedish twins (over 65 years) demonstrated a significantly lower heritability for general cognitive abilities, suggesting a possible inverted L-shaped function for the relationship between heritability and IQ later in life. These results suggest that environmental influences become increasingly important for individual differences in cognitive ability late in life. Indeed, findings from the OCTO-Twin study (McClearn et al., 1997) confirm the trend to slightly lower heritabilities for general cognitive abilities in the oldest old. Heritability for *g* in this sample of twin 80 years and older was 62%. Results from the Longitudinal Study of Aging Danish Twins (McGue & Christensen, 2001) also confirm the trend to somewhat lower heritabilities for general cognitive abilities in twins aged 75 and older.

Analyses of the measures of specific cognitive abilities demonstrated that heritabilities for memory measures in SATSA were somewhat lower than verbal, spatial or perceptual speed measures (Pedersen et al., 1992). Further exploration of the memory data indicated that heritabilities for memory in SATSA are somewhat lower than those found in Minnesota Twins Study of Adult Development and Aging, (Finkel, Pedersen, & McGue, 1995) most likely reflecting the considerable differences in the types of memory measures included in the two studies.

Initial Longitudinal Findings

Initial analyses from SATSA were based on cognitive data from two time points with a three year interval (Plomin, Pedersen, Lichtenstein, & McClearn, 1994). As might be expected for this relatively short period of time, the phenotypic stability was quite high: $r = .93$, and there was

little change in total variance. The heritabilities at both time points were similar and substantial (∼.80). Further, genetic influences at time 1 were more highly correlated with genetic influences at time 2 than was the case for environmental influences. In other words, there was greater genetic than environmental stability across a 3-year period. Because heritabilities were substantial at both time points and the genetic correlation between occasions (i.e., genetic stability) was substantial (.83), phenotypic stability predominantly reflected genetic influences.

Cohort-Sequential Analyses

There were two limitations of these early analyses: first, only 2 time points were included and second, in order to be included in the analyses, both members of the pairs had to participate at both time points. Developments in various structural equation model-fitting programs allow for missing values for some participants and some variables. The three occasion SATSA cognitive data were analyzed using such a technique, and, at the same time, issues concerning cohort versus longitudinal trends in means, variances, and components of variance were examined (Finkel, Pedersen, McClearn, Plomin, & Berg, 1996; Finkel, Pedersen, Plomin, & McClearn, 1998). Cohort-sequential analyses combining cross-sectional (cohort) and longitudinal information confirm a general decline in mean levels of cognitive performance, however, no change in total variance for general cognitive abilities. Thus, earlier predictions that individual differences should increase with advancing age were once again not supported. More interesting were the analyses of the genetic and environmental components of variance. Inspection of the longitudinal trends for the separate cohorts clearly demonstrates that there is a longitudinal *decrease* in genetic variance for the oldest cohorts. Heritability is relatively stable longitudinally at approximately 80% in the younger cohorts. In the older cohorts, heritability decreases from approximately 80% at time 1 to 60% at time 3. Again, this is the same heritability as that reported for 80+ year old twins in OCTO-Twin. We may be observing the effect of terminal decline in cognitive abilities. It may be that twin similarity for cognitive abilities decreases as members of a twin pair begin to decline at slightly different times or the rate of decline differs for the members of the pair. Thus, it appears as though environmental factors are important for the timing of entry into or the trajectory of terminal decline.

Aging Trajectories for Specific Cognitive Abilities

Latent growth models incorporating two linear slopes were recently applied to cognitive data in order to address several phenotypic issues (Finkel, Reynolds, McArdle, Gatz, & Pedersen, in press). Results indicated stability or even improvement up to age 70 for measures of crystallized ability, followed by significant decline. Linear age changes were found for many cognitive abilities. For those measures with a large speed component, a growth curve model incorporating two separate slopes was indicated, suggesting a significant acceleration in linear decline after age 65. Accelerating decline in cognitive performance at age 65 may reflect true aging changes, or it may be a consequence of the transition from an active (presumably stimulating) work life to retirement. Furthermore, it is possible that a more accurate model of cognitive aging includes not one but two transition periods when the rate of decline changes. Five measurement occasions are required to test higher-level growth curves.

Longitudinal Changes in Heritability of Specific Cognitive Abilities

We have extended the latent growth curve models to include twin data to examine genetic and environmental influences on mean level of cognitive ability as well as rates of change over a 6-year span. Following the results of Finkel et al. (in press) five of the cognitive measures were fit with a linear latent growth model, and five were fit with the two slope model that estimated the change in rate of decline with age (Reynolds, Finkel, Gatz, & Pedersen, 2002). Genetic and environmental influences on both the mean level and the rate of decline were estimated. As expected from cross-sectional data, significant heritabilities were found for variation in the mean level of performance on all 10 cognitive abilities.

In contrast, the impact of genetic and environmental influences on rate of change varied measure by measure. Significant genetic influences on the slope were found for Information, Symbol Digit, Block Design, and Analogies. For the remaining six cognitive measures, variance in the rate of decline was explained entirely by environmental factors. These results serve to emphasize that heritability is not a static number. The relative influence of genetic factors will change as the dynamic combination of genetic and environmental factors changes over the lifespan. Furthermore, each component of cognitive aging needs to be examined independently; we will not find a single aging trajectory, nor will we find a single explanation for cognitive aging. It is clear that cross-sectional investigations of genetic and environmental influences on aging that can estimate the contributions to the mean level of performance are failing to capture the dynamic process of aging. Only by applying methods such as latent growth curve analysis to longitudinal twin data will we be able to isolate the genetic and environmental influences on the dynamic processes of aging.

ASSOCIATIONS BETWEEN COGNITIVE AND PHYSICAL FUNCTIONING

Perceptual and Motor Speed

Given the consistent evidence for significant genetic influences on cognitive ability and perceptual speed throughout the adult lifespan, Finkel and Pedersen (Finkel & Pedersen, 2000) conducted a bivariate quantitative genetic analysis of the relationship between perceptual speed and cognitive aging. Results indicated that 70% of the genetic variance in the cognitive factor was shared with perceptual speed. Thus, the heritability of cognitive ability in adulthood results, at least in part, from genetic influences associated with perceptual speed instead of genes for cognitive functioning, per se. A multivariate quantitative genetic analysis incorporating measures of perceptual speed, motor speed, and cognitive ability was conducted on the middle-aged and young-old cohorts of SATSA (Finkel, Pedersen, & Harris, 2000), to examine possible age differences in the relationship between speed and cognition. Results indicated qualitative differences between cohorts in the nature of the genetic and environmental variance common to motor speed, perceptual speed, and cognitive abilities. The genetic variance in cognitive functioning in the middle-aged cohort was defined primarily by motor speed, whereas genetic variance in the older cohort was defined by perceptual speed.

Cognition and Olfactory Functioning

Individuals differ in their ability to detect odors and to identify or label odors. Furthermore, there are significant age differences in these abilities. Phenotypic analysis of olfactory functioning and the relationship between cognitive and olfactory functioning in the SATSA sample revealed strong positive correlations between odor identification (the ability to identify an odor) and several measures of verbal ability (Larsson, Finkel, & Pedersen, 2000). Quantitative genetic analysis of four measures of olfactory functioning indicated moderate heritability for odor identification (29%) and perceived intensity (25%). Bivariate genetic analyses revealed that the relationship between odor identification and measures of verbal ability identified by Larsson et al. (Larsson et al., 2000) was primarily genetically mediated (Finkel, Pedersen, & Larsson, 2001). Indeed, all of the genetic influences on odor identification could be attributed to genetic influences on verbal ability. The strong genetic mediation of the correlation between odor identification and verbal ability provides additional support for the hypothesis that odor identification and verbal ability in general tap the same cognitive domain (Larsson, 1997). In addition, the results provide some insight into the nature of age differences in olfactory functioning. Declines in the cognitive component of performance on some olfactory measures may reflect genetically influenced cognitive decline.

Cognitive and Biological Markers of Aging

There has been considerable attention paid to the utility of cognitive, performance, and physiological measures as markers of the aging process.

Combined analyses of SATSA and OCTO-Twin identified four factors that serve as markers of aging: general knowledge, fluid abilities, physiological functioning, and health. Analysis of twin similarity for these components of aging suggested the relative influence of genetic and environmental factors varies greatly for different components of functional aging (Finkel, Pedersen, Berg, & Johansson, 2000). Recent analyses of SATSA data have focused on the aging trajectory for variables that serve as markers of aging, and the genetic and environmental influences on the rates of decline. Growth curve analysis of five markers of aging indicate a steeper rate of decline for men than women in forced expiratory volume (FEV1) and grip strength and a moderate rate of increase in mean arterial pressure for both men and women (Finkel et al., 2003). For two variables, motor functioning and well-being, growth curve analysis identified a turning point (age 70) at which functioning changed from stability to decline. Quantitative genetic growth curve analysis indicated genetic influences on the mean level of performance on all five markers of aging (Finkel et al., 2003). The same was not true for the rates of change with age. Genetic influences on the slope were found for three of the variables: Motor Functioning, Mean Arterial Pressure, and Forced Expiratory Volume. Attempts to address the issue of the etiology of the aging process will depend on whether the focus is static performance or dynamic change.

OTHER MULTIVARIATE ANALYSES

Cognition and Personality

An analysis by Wetherell and colleagues demonstrated how the SATSA dataset can fruitfully be analyzed to test hypotheses concerning phenotypic associations across behavioral domains. Although this analysis did not evaluate genetic and environmental mediation of associations, it did address issues of cross-sectional and longitudinal relationships. Wetherell (Wetherell, Reynolds, Gatz, & Pedersen, 2002) used random effects models to analyze cross-sectional relationships between cognitive performance and state anxiety and longitudinal relationships between cognitive change and neuroticism, after controlling for sex, age, and education. Cross-sectionally, higher state anxiety was associated with poorer performance on all cognitive tests. In longitudinal models, the main effects for neuroticism were significant for all tests, but there were no significant interactions between neuroticism and sex, neuroticism and time, and neuroticism by sex by time. These results suggest that anxiety is related to poorer performance across multiple cognitive domains in older adults, but provide no support for anxiety as a risk factor for cognitive decline.

NORMAL AGING AND DEMENTIA

There is considerable synergy between SATSA and the Study of Dementia in Swedish Twins (Gatz et al., 1997), which focused initially on SATSA twins who have developed dementia. A recent investigation compared preclinical cognitive functioning in identical twins from SATSA and OCTO-Twin who were discordant for dementia diagnosis (Andel et al., 2001). In contrast to their nondemented cotwin partners, twins who later developed dementia showed poorer performance on tests of memory and attention, visuospatial/reasoning skills, perceptual speed, and MMSE. Further comparisons demonstrated that the nondemented cotwins scored lower than matched controls on several measures of cognitive ability. Thus, nondemented cotwin partners of demented twins show a cognitive profile similar to the premorbid changes that presage dementia.

Other analyses of the SATSA data demonstrate how associations relevant to dementia and cognitive decline can be addressed. Low educational attainment has been identified as a risk factor for both Alzheimer's disease and dementia in general (Mortimer, 1994), and there is a well-documented association between scores on the Mini-Mental State Examination (MMSE) and educational attainment (Magaziner, Bassett, & Hebel, 1987). The etiology of the association between MMSE scores, education, and general cognitive ability has been addressed in SATSA (Pedersen, Reynolds, & Gatz, 1996). For men, the association between

MMSE and education is primarily due to a common genetic factor influencing both education and MMSE performance. Furthermore, the genetic influences on MMSE performance almost entirely reflect genetic influences on general cognitive abilities, supporting the hypothesis that the association between education and MMSE performance predominantly reflects cerebral capacity. However, for women further investigation in the SATSA sample (Gatz et al., 2001) found that the role of education as a risk factor was weaker than many published reports. Although speculative, this outcome is compatible with the prediction of a weaker education–dementia relationship in a population or cohort where education was less directly reflective of intellectual capacities.

PART 2. THE ROLE OF PROCESSING SPEED IN EXPLAINING THE LONGITUDINAL TRAJECTORIES OF CHANGE FOR COGNITIVE ABILITIES

The processing-speed theory (Birren, 1964; Salthouse, 1996) posits that cognitive aging results from generalized slowing of perceptual and cognitive processes. Evidence from cross-sectional studies indicates that up to 79% of age-related variance in many cognitive abilities can be explained by age-related variance in measures of processing speed (Verhaeghen & Salthouse, 1997). Previous investigations of cross-sectional data available from SATSA reported that 70% of the genetic variance in the cognitive factor was shared with perceptual speed (Finkel & Pedersen, 2000). However, Sliwinski and Hofer (Sliwinski & Hofer, 1999) expressed concern that investigations of mediating factors used primarily cross-sectional data, whereas longitudinal data allow for analysis of within-person variance and may produce different results. Several longitudinal studies have reported that only 5% to 30% of the intraindividual age changes in cognitive abilities are explained by intraindividual age changes in processing speed (Hultsch, Hertzog, Small, McDonald-Miszczak, & Dixon, 1992; MacDonald, Hultsch, Strauss, & Dixon, 2003; Sliwinski & Buschke, 1999; Taylor, Miller, & Tinklenberg, 1992; Zimprich, 2002; Zimprich & Martin, 2002). Fuller understanding of the nature of the relationship between processing speed and cognitive aging requires analysis of the effects of speed mediation on longitudinal changes in genetic and environmental influences on cognitive ability. For example, there is evidence that the genetic factors influencing level of performance may or may not be associated with the genetic factors influencing the rate of decline (Reynolds, Finkel, et al., 2002).

Longitudinal twin designs allow for the estimation not only of longitudinal trends in performance, but also the estimation of longitudinal trends in genetic and environmental influences on performance. In a genetically informative sample, latent growth curve analyses can be used to investigate genetic and environmental influences on the mean level of performance and on rate of change. Genetic and environmental influences on static (intercept) and dynamic (slope) measures of aging may differ both in magnitude and nature. In addition, changes with age in genetic and environmental components of variance can be calculated from the latent growth curve parameters. In other words, both the heritability of change and the change in heritability can be estimated.

Our goal in the present analysis was to investigate the role played by measures of processing speed in explaining the longitudinal trajectories of change for cognitive abilities and the genetic and environmental influences on those trajectories. Four measures of cognitive ability were used, reflecting the domains assessed by the SATSA cognitive battery: Information (crystallized ability), Block Design (fluid ability), Thurstone's Picture Memory (memory), and a general cognitive factor ("*g*"). We predict that when the cognitive measures are corrected for the effects of processing speed, they will demonstrate a milder decline trajectory. In the quantitative genetic analysis, removing the variance associated with processing speed will result in a decrease in overall genetic variance for the cognitive measures. However, it is difficult to predict the effect correction for processing speed will have on estimates of genetic influences on the rate of decline with age.

METHOD

Participants

Ascertainment procedures for SATSA were described above. The sample for the current analyses included individuals from both complete and incomplete twin pairs. In total, 798 nondemented individuals had available cognitive data across 13 years. Dementia status was determined by clinical diagnosis based on current diagnostic criteria (Gatz et al., 1997). Age at time of measurement ranged from 50 to 96 years. The minimum requirement for inclusion in the current analyses was cognitive data at one testing occasion. Thirty-seven percent of the sample participated in all four testing occasions; 76% of the participants had data from at least two occasions. As expected from population demographics in this age range, 60% of the sample was female.

The goals of the present analyses include both phenotypic and twin applications of latent growth curve models. In the phenotypic analyses, all models were fit to a sample that included a randomly selected member of each twin pair (hereafter, twin A). Phenotypic analyses were then replicated in a sample consisting of the other member of each twin pair (twin B). Individuals from incomplete pairs or of unknown zygosity were randomly assigned to either the twin A or twin B sample. Data from both complete and incomplete pairs with known zygosity were used in the twin analyses.

Measures

As described above, the SATSA cognitive test battery includes 12 cognitive measures drawn from various sources and chosen to assess four areas of cognitive ability: crystallized abilities, fluid abilities, memory, and perceptual speed. Reliabilities for these tests range from .82 to .96 (Pedersen et al., 1992). For the current analyses, one representative measure was chosen from each domain. Information was chosen to assess crystallized abilities and Block Design to represent fluid abilities. Perceptual speed was measured by the Symbol Digit task. WAIS Digit Symbol involves writing unusual symbols by an array of digits (Wechsler, 1972). In contrast, the Digit Symbol task used in SATSA involves oral report of the appropriate digit for each symbol (Pedersen et al., 1992). All mechanical components of the task have been eliminated; therefore, this version of Digit Symbol may serve as a purer measure of perceptual speed than the conventional version. For ease of presentation, these cognitive measures were recoded to percent correct of the total possible points for each respective test.

A principal components analysis was used to create a measure of general cognitive ability, or "*g*". Individuals' scores on the first principal component of all the cognitive measures were obtained at IPT1 (as in Pedersen et al., 1992). With the exception of Forward Digit Span, all measures loaded higher than .50 on the first principal component, which accounted for 45% of the total variance. Our goal was to avoid adding any error to the latent growth curve model by using a principal component that varied in definition at each time point. Therefore, scores on each cognitive measure were standardized using the means and variances observed at IPT1, creating an identical metric for each cognitive measure at all three time points. Next, a general cognitive factor was created for each testing occasion by combining the now standardized cognitive scores using the factor loadings from the principal components analysis conducted at IPT1. This procedure ensured that the definition of the general cognitive factor remained invariant across testing occasions. Finally, all scores were multiplied by 10 and a constant of 30 was added to ensure that parameters would not be near zero, given that factor score distributions are standardized to mean of zero and unit variance.

Statistical Method

Speed Correction

Although there are several possible methods for investigating the influence of processing speed on latent growth curve models of cognitive aging, in the current analysis we chose to correct the measures of cognitive ability for variance associated with processing speed. Latent growth curve estimates for corrected and uncorrected cognitive measures were then compared. To that end, Symbol Digit scores from each individual's first time of measurement were regressed out of the scores of the cognitive measures from occasion 1, 2, 3, and 5. To ensure comparability, the speed-corrected scores were then standardized to have the same mean and variance as the uncorrected scores from the same time of measurement.

Phenotypic Model

A latent growth curve model was used to examine age changes in general cognitive ability (for details see McArdle, Prescott, Hamagami, & Horn, 1998). The model provides estimation of fixed effects, that is fixed population parameters as estimated by the average growth model of the entire sample, and random effects, that is individual variation in growth model parameters. Latent growth curve models take into account missing data by giving more weight to individuals with the most time points. In addition, individuals with only one time of measurement contribute to the estimation of the intercept, but not to the estimation of the slope(s). The model in Figure 2 is an intercept and slope model of change that incorporates two separate slopes (Bryk & Raudenbush, 1992): one slope for middle age (e.g.,

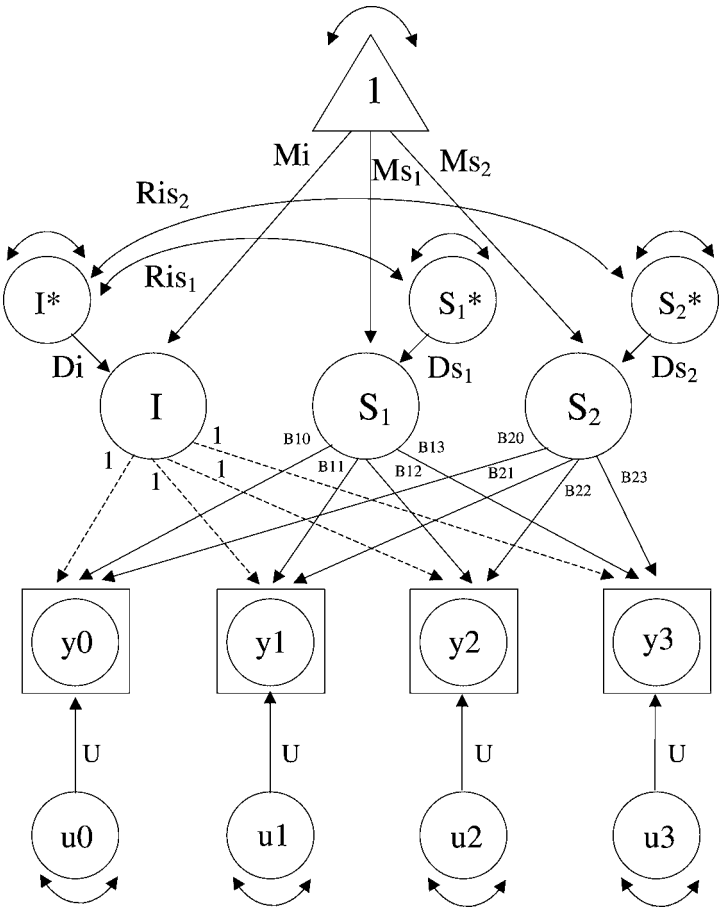

Fig. 2. Latent growth model of change incorporating two slopes: S_1 (age < 65) and S_2 (age > 65). Observed data are denoted by y_0 through y_3. Mi = mean intercept; Ms_1 = mean slope 1; Ms_2 = mean slope 2; U_0 through U_3 indicate random error. I^*, $S_1{}^*$, and $S_2{}^*$ refer to the standardized scores of I, S_1, and S_2. Di denotes deviations from the group intercept and Ds_1 and Ds_2 denote deviations from the group slopes. The relationship between initial intercept and the slopes is represented by the correlations between intercept and slope (Ris_1 and Ris_2). The paths from the latent slopes to the observed scores are the age basis coefficients, B1(t) and B2(t).

age < 65) and a separate slope for young-old age (e.g., age > 65). Based on results from longitudinal studies and growth curve analyses (e.g., Caskie, Schaie, & Willis, 1999; Finkel et al., 1998; Schaie, 1996) we chose to focus on the transition from young-old to old-old age. Previous analyses testing various centering ages indicated that centering at age 65 provided the best fit to the data for all measures except Information, which required a centering age of 70 (Finkel, Reynolds et al., 2003). Testing for the optimal centering age was conducted in the current analysis as well.

According to the model employed in these analyses, individual scores at any one time are a linear function of a latent intercept (I), slope (S_1 or S_2), and random error ($U_0 - U_3$). S_1 refers to the slope up to age 65 and S_2 indicates the slope after age 65. I^*, $S_1{}^*$, and $S_2{}^*$ refer to the standardized scores of I, S_1, and S_2. The model fitting procedure entails fitting individual growth models to all available data; repeated measurements on the markers of aging are indicated by the y_0 through y_3 variables. The paths from the latent slope factors to the observed scores are the age basis coefficients, B1(t) and B2(t). The age basis serves as a marker for the age of the subject at each time of measurement, adjusted for the centering age. Values of B1(t) were set to zero for any age greater than 65, thereby defining S_1 as the rate of change up to age 65. Similarly, values of B2(t) were set to zero for any age less than 65, defining S_2 as the rate of

change after age 65. Individuals who were assessed both before and after age 65 contributed data to both S_1 and S_2.

The random errors or uniquenesses ($U_0 - U_3$) represent unaccounted variation from fitting the growth model to the biobehavioral measures; note that they are by definition fixed at the same value 'u' at all time points. The means (Mi = mean intercept; Ms_1 = mean slope 1; Ms_2 = mean slope 2) are the estimates of the growth model for the entire sample, centered at a particular age (in this case, 65 years). That is, the path estimates of the average growth model for the entire sample indicate average performance (Mi) on general cognitive ability *at age 65*, the average rate of change in performance up to age 65 (Ms_1), and the average rate of change in performance after age 65 (Ms_2). Deviations from the group shape are captured by parameters reflecting deviations from the group intercept (Di) and slopes (Ds_1 and Ds_2) (for details, see Finkel et al., 2003). The model described assesses systematic variance over age, as opposed to systematic variance over time (cf. Reynolds, Gatz, et al., 2002). Therefore, the slope deviation reflects only that variance in the slope that is systematic with age. Finally, the relationship between initial intercept and the slopes is represented by the correlations between intercept and the respective slopes (Ris_1 and Ris_2). The random and fixed effects parameter estimates were obtained using PROC Mixed in SAS 8.0 (SAS Institute, 1999).

Quantitative Genetic Model

The variance in latent growth curve parameters can be divided into four separate components: additive genetic effects (*Va*), correlated environmental effects or general cultural effects that can be shared by twins regardless of rearing status (*Vc*), shared rearing environmental effects that serve to make the members of a family more similar (*Vs*), and nonshared environmental effects including error (*Vns*). The phenotypic covariance between twins, assuming the four components of variance are uncorrelated, can be expressed as in the following equations for monozygotic twins reared together (MZT), monozygotic twin reared apart (MZA), dizygotic twins reared together (DZT), and dizygotic twin reared apart (DZA). The abbreviation covMZT represents the covariance between MZT twins.

$$\begin{aligned}\text{covMZT} &= Va + Vc + Vs,\\ \text{covMZA} &= Va + Vc,\\ \text{covDZT} &= \tfrac{1}{2}Va + Vc + Vs,\\ \text{covDZA} &= \tfrac{1}{2}Va + Vc.\end{aligned}$$

By fitting structural models to the observed MZA, MZT, DZA, and DZT covariance matrices, we can estimate the proportion of total phenotypic variance accounted for by the variance in genetic factors, shared rearing environment factors, correlated environment factors, and nonshared environment factors. Quantitative genetic latent growth curve models were fit with the structural equation modeling program Mx version 1.50d (Neale, Boker, Xie, & Maes, 1999). Intercept variance, slope variance, and the intercept-slope correlations were decomposed into genetic and environmental components. The unexplained variance of the model, Du, was not decomposed into genetic and environmental components. The raw maximum likelihood estimation procedure was used throughout. We tested the significance of components of variance using a difference chi-square test (i.e., subtracting the $-2\log$ likelihoods of the models being compared).

RESULTS

Latent Growth Curve Model

The first step of the analysis was to determine whether a one- or two-slope latent growth curve model provided the best fit to the speed-corrected cognitive measures. Three latent growth curve models were fit to the data to test hypotheses regarding growth and change over time. First, a ''no growth'' or intercept-only model was fit by estimating only intercepts. Second, rate of change was considered by adding a single slope effect that included all ages. The two nested models were compared using the difference chi-square test. Third, the two-slope model was fit to the data. If the two-slope model provided a better fit than the single slope model, then we concluded that there was a significant change in the rate of decline in the cognitive measure during late adulthood. Previous analyses indicted that a two-slope model provided the best fit to the uncorrected cognitive measures (Finkel, Reynolds et al., 2003). The current analysis indicted that the two-slope model also provided the best fit to the data for the speed-corrected cognitive measures. Parameter estimates from the analysis in the Twin A sample are presented in Table 3. Comparison of models with various centering ages indicated that model fit was optimized when the centering age was 65 years for all measures except information. In the current analyses including four measurement occasions, the optimal centering age for Information was 75.

Although aging trajectories for both original and speed-corrected cognitive measures described

Table 3. Parameter Estimates from the Latent Growth Curve Models for Both Original and Speed-Corrected Cognitive Measures.

Variable	Cage[a]	Parameter estimates								
		Fixed effect			Random effects[b]					
		Intercept	Slope 1	Slope 2	varI	$varS_1$	$varS_2$	Ris_1	Ris_2	Residual
Information	75									
Original		74.48**	0.01	−1.37**	298.81	.27**	1.87**	.51**	.02	38.18
Speed-corrected		76.80**	0.26**	−1.21**	294.29	.41**	2.13**	.49**	−.16	50.34
Block Design	65									
Original		47.89**	−0.57**	−1.12**	211.35	.01	.40**	1.0	−.73**	67.18
Speed-corrected		48.28**	−0.15	−0.85**	201.19	.02	.52**	−1.0	−.74**	111.62
Thurstone's Memory	65									
Original		79.03**	−0.08	−0.78**	252.71	.12*	2.02**	1.0**	−.57**	87.48
Speed-corrected		78.40**	0.13	−0.51**	258.62	.16*	2.37**	.88*	−.63**	113.18
General Cognition	65									
Original		33.69**	−0.21**	−0.58**	99.79	.10**	.19**	.43*	−.51**	6.59
Speed-corrected		34.19**	−0.05	−0.45**	110.60	.18*	.34**	.24	−.70**	17.39

Note. [a]Cage = centering age of the latent growth model.
[b]Random effects: varI = variance around the intercept, $varS_1$ and $varS_2$ = variance around slopes, Ris_1 and Ris_2 = correlations between intercept and slope, and Residual indicates residual variance.
$^{*}p < .05$, $^{**}p < .01$.

a two-slope pattern, we can see in Table 3 that the slope estimates differed dramatically, resulting in marked differences in the shape of the trajectories with and without speed correction. The intercepts (indicating mean performance at the centering age) were similar for both uncorrected and speed-corrected versions of the cognitive measures as a result of standardization. Estimates for both Slopes 1 and 2, however, indicated milder rates of decline for the speed-corrected measures. As expected for a measure of crystallized abilities, uncorrected information demonstrated stability up to age 75 followed by significant decline of 1.37% per year, or 13.7% per decade. Speed-corrected Information indicated significant *increases* in performance up to age 75 (.26% per year), followed by significant decline. Both Block Design and General Cognitive Ability showed modest but significant decline before age 65 and steeper decline after age 65. When corrected for speed variance, however, both variables demonstrated *stability* up to age 65 (i.e., nonsignificant Slope 1 estimate), followed by decline. The effect of speed-correction on Thurstone's Picture Memory was more limited. For both original and speed-corrected versions of the measure, participants demonstrated stability up to age 65, followed by modest decline. These results were replicated in the Twin B sample.

Estimates of the random effects (variance around the intercept and slopes) were fairly similar for the uncorrected and speed-corrected measures. Perfect correlations between the intercept and slope that do not achieve statistical significance indicate an extreme or boundary condition. This is not an uncommon result when fitting models to sparse patterns of data (e.g., Hamagami & McArdle, 2001). Differences in the estimate of residual variance (it was higher for the speed-corrected measures) reflect the loss of systematic age-related variance that occurs when the effect of processing speed is regressed out of the cognitive measures.

Quantitative Genetic Analysis

Quantitative genetic latent growth curve analysis provides two types of information about influences on change with age. First, genetic and environmental influences on individual variation around the group mean level of performance (intercept) and rate of change (slope) are estimated. In other words, varI, $varS_1$, and $varS_2$ are decomposed into genetic and environmental components. Second, changes with age in genetic and environmental components of variance can be calculated from the latent growth curve parameters for both the uncorrected and speed-corrected variables.

The quantitative genetic model fit to the latent growth curve model for each cognitive measure included all four components of variance: genetic, shared environmental, correlated environmental, and nonshared environmental effects. Estimates of genetic and environmental influences on intercept and slope parameters for both original and speed-corrected cognitive measures are presented in Table 4. Note that there is little relationship between heritability estimates for the intercepts and the slopes. Factors that influence individual differences in initial level of cognitive performance are not necessarily the same factors that influence changes in performance. The heritability estimate for the intercept indicates heritability at the centering age and is analogous to heritability estimates from cross-sectional analyses. Correcting for processing speed resulted in lower heritability estimates for the intercept for all four cognitive measures because the genetic factors associated with processing speed have been removed. The reduction in heritability ranged from 13% for Information to 38% for Block Design. For all but Thurstone's Picture Memory, heritability estimates for Slope 1 were higher for the speed-corrected measures. With respect to Slope 2, heritability estimates for the speed-corrected measures were lower in two cases (Block Design and Thurstone's Picture Memory) and higher for the remaining two measures (Information and General Cognitive Ability). Shared and correlated environmental effects tended to be relatively small for both original and speed-corrected variables. In fact, these two components of environmental variance could be dropped from the full model without significant loss of fit in all cases.

Using the estimates of genetic and environmental influences on the intercept and slope generated by the quantitative genetic latent

Table 4. Genetic and Environmental Influences on Intercept and Slope Parameters from the Latent Growth Curve Model for Both Original and Speed-Corrected Cognitive Measures.

Parameter	Information		Block Design		Thurstone		Gen. Cog. Ab.	
	Orig.	−Speed	Orig.	−Speed	Orig.	−Speed	Orig.	−Speed
Intercept								
Genetic	.69	.60	.63	.39	.87	.64	.86	.70
Shared Env.	.01	.00	.10	.21	.06	.08	.04	.06
Correlated Env.	.04	.00	.03	.12	.00	.01	.00	.00
Nonshared Env.	.26	.40	.24	.28	.07	.27	.10	.24
Slope 1								
Genetic	.01	.39	.29	.36	.75	.38	.17	.46
Shared Env.	.00	.00	.01	.01	.18	.14	.14	.00
Correlated Env.	.22	.01	.05	.27	.02	.25	.00	.21
Nonshared Env.	.77	.60	.65	.36	.05	.23	.69	.33
Slope 2								
Genetic	.16	.34	.94	.85	.36	.14	.07	.26
Shared Env.	.00	.00	.00	.03	.17	.27	.09	.15
Correlated Env.	.06	.07	.00	.02	.27	.31	.00	.01
Nonshared Env.	.78	.59	.06	.10	.20	.28	.84	.58

growth model, we also can estimate the change with age in genetic and environmental components of total variance. Table 5 presents genetic and environmental variance components over age for original and speed-corrected cognitive measures. Two general patterns in age changes in genetic variance are apparent in the table. Information, both uncorrected and corrected, shows a u-shape pattern for genetic variance with generally small decreases up to age 65 and an upturn 70 years and after. The remaining cognitive measures, both original and speed-corrected, show a general decrease in genetic variance with age. For Block Design the decline in genetic variance is continuous. Thurstone's Picture Memory and General Cognitive Ability both demonstrate an inverted u-shape pattern for genetic variance, suggesting initial increases in genetic variance followed by decreasing genetic variance by age 80.

In contrast, age changes in nonshared environmental variance are generally stable or increasing, with only speed-corrected Block Design demonstrating modest declines with age. Shared environmental effects were indicated for Block Design and Thurstone's Picture Memory; whereas correlated environmental variance was evident for all measures except General Cognitive Ability. For all measures except General Cognitive Ability, correlated environmental variance exhibited a pattern of small estimates before age 65 and larger estimates after age 65. This pattern resulted from the overall smaller slope variances reported for Slope 1 than for Slope 2 (see Table 3). Recall, however, that neither correlated environment nor shared environment contributed significantly to model fit.

When we focus on comparing the variance components for original and speed-corrected measures, again we find two patterns of results. For Information and Thurstone's Picture Memory, greater nonshared environmental variance and less genetic variance were evident for the speed-corrected measures. However, the shape of the trajectory of change in genetic and environmental variance with age was the same for both uncorrected and speed-corrected versions of these cognitive measures. In contrast, removing the speed variance from Block Design and General Cognitive Ability resulted in a marked difference in the shape of the age trajectory. For both variables, estimates of genetic variance began at comparable levels at age 50, but decline at a much faster rate for the speed-corrected measure. This effect is illustrated in Figure 3, which presents genetic and

Table 5. Estimated Components of Variance Across Ages Based on Latent Growth Curve Parameters for Both Original and Speed-Corrected Cognitive Measures.

Measure	Information		Block Design		Thurstone		Gen. Cog. Ab.	
	Orig.	−Speed	Orig.	−Speed	Orig.	−Speed	Orig.	−Speed
Genetic variance								
Age 50	210	157	189	155	66	51	66	70
Age 60	192	128	133	90	145	109	76	77
Age 70	175	135	70	23	133	106	73	58
Age 80	235	227	43	18	101	70	57	22
Shared environmental variance								
Age 50	2	1	24	44	41	52	0	7
Age 60	2	1	19	38	21	27	2	7
Age 70	2	2	18	21	14	18	2	3
Age 80	2	1	16	3	61	68	0	1
Correlated environmental variance								
Age 50	0	52	1	3	1	5	0	8
Age 60	6	43	4	9	0	0	0	1
Age 70	27	35	16	19	12	6	0	0
Age 80	69	12	57	50	47	42	0	1
Nonshared environmental variance								
Age 50	47	92	6	31	7	15	10	29
Age 60	30	67	24	37	12	42	8	26
Age 70	63	96	46	34	22	30	8	24
Age 80	114	197	57	15	85	61	22	50

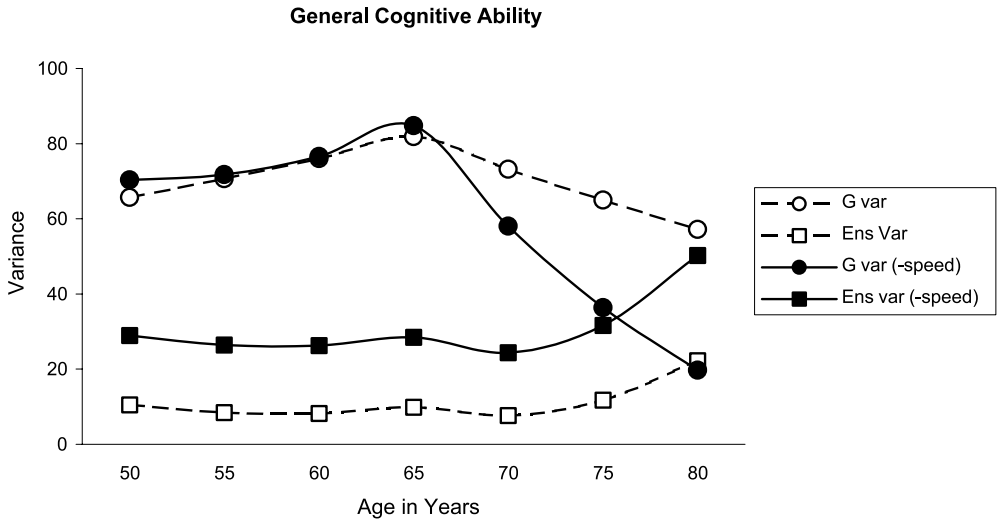

Fig. 3. Changes in genetic and environmental variance, as estimated by the latent growth curve model. Gvar = Genetic variance, Ens = Nonshared environmental variance. Open symbols and dotted lines indicate the estimates for uncorrected General Cognitive Ability; closed symbols and solid lines indicate the estimates for speed-corrected General Cognitive Ability.

environmental components of variance for original and speed-corrected General Cognitive Ability. As estimates of shared and correlated environmental variance were quite small, they have not been included in the figure, in the interest of clarity. Figure 3 demonstrates the increasing divergence in genetic variance for uncorrected and speed-corrected General Cognitive Ability after age 65. Results for Block Design indicate a similar effect in genetic variance.

DISCUSSION

Based on the cognitive aging literature, we predicted that regressing processing speed out of measures of cognitive ability would have two results. First, the trajectory of decline estimated by latent growth curve modeling would be less severe, and second, the extent of genetic and environmental influences on the intercept and slope of the latent growth curve model would be affected. Our first hypothesis was supported: all slope estimates from the speed-corrected cognitive measures reflected less decline with age than slope estimates for the uncorrected measures. For three of the four measures, correcting for speed variance produced a change in the shape of the trajectory of change with age. Whereas Information demonstrated stability up to age 75 followed by decline, speed-corrected Information showed modest but significant increases in performance up to age 75 followed by decline. Age trajectories for Block Design and General Cognitive ability changed from modest decline before age 65 to stability before age 65 when speed variance was removed. A two-slope latent growth curve model fit to Symbol Digit indicated decline in middle adulthood, followed by steeper decline in late adulthood (Finkel, Reynolds et al., 2003). It is not surprising, then, that removing the effect of processing speed results in a reduction in the rate of decline with age in measures of fluid ability, memory, and "*g*". What is not predicted by the processing-speed hypothesis is the effect on Information, a measure of crystallized abilities. Although many investigations of the processing-speed hypothesis fail to include measures of crystallized abilities, when they are included significant speed mediation of age effects in crystallized abilities have been reported (e.g., Sliwinski & Buschke, 1999). Not only do we find speed mediation of the latent growth curve for Information, but when speed variance is removed, performance on this "purified" measure of crystallized ability increased until very late in adulthood, as predicted by the earliest conceptions of the crystallized-fluid model of intelligence (Cattell, 1963; Horn, 1970).

Other investigations report that statistical control for processing speed accounts for most or all of the cross-sectional age effects in cognitive performance, but has a much smaller impact on longitudinal aging effects (e.g., Hultsch et al., 1992; Sliwinski & Buschke, 1999; Zimprich, 2002). Similarly, we find that correcting for speed diminishes, but does not erase longitudinal age effects in cognitive ability. Using the two-slope latent growth curve model, however, we have been able to expand on previous results. In middle-age, longitudinal aging effects were eliminated when the cognitive measure was corrected for the effects of processing speed. After the transition to late adulthood correcting for processing speed had a smaller effect on longitudinal aging effects. Therefore, we may have identified a transition point in the aging process when the mediating effects of processing speed become less powerful. It is also possible that processing speed assessed at the first measurement has limited mediating effects on general cognitive ability measured several years later. A dynamic modeling approach (McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; McArdle & Hamagami, 2003) would be necessary to test whether *change* in processing speed mediates change in other cognitive abilities very late in life.

By applying quantitative genetic methods to the latent growth curve model, both the heritability of change and the change in heritability can be estimated. More precisely, the genetic influences on intercept and slopes can be estimated, as well as changes over age in the genetic and environmental components of variance. As we predicted, correcting cognitive measures for speed variance resulted in lower heritability estimates for the intercept. Heritability estimates for processing speed are substantial (e.g., Finkel & Pedersen, 2000; McClearn et al., 1997; Pedersen et al.,

1992); therefore, we expected that removing speed variance would have the parallel effect of removing some portion of genetic variance as well. Effects of the speed correction on heritability estimates for the slope parameters were mixed. It is important to note that, for the most part, heritability of the slope parameters was lower than heritability of the intercepts. A similar pattern was reported by Reynolds, Finkel, et al. (2002). The one exception to this pattern was Slope 2 for Block Design. Higher heritabilities for rate of decline in Block Design after age 65 may reflect sampling issues (the data become increasingly sparse in late adulthood) or genetic variance for decline related to the onset of dementia or terminal decline. Overall, we can conclude that whereas genetic factors play a significant role in an individual's initial level of performance, rate of change in cognitive performance with age is influenced to a much greater extent by environmental factors. Quantitative genetic methods thus allow us to highlight the difference between level of performance and rate of decline: the magnitude of genetic and environmental influences varies for static and dynamic measures of the cognitive aging process.

Patterns of change with age in genetic and environmental variance replicate results from previous investigations employing different methods of analysis (Finkel et al., 1996; Finkel et al., 1998). We found increasing genetic variance for Information and decreasing genetic variance for the remaining cognitive measures. Environmental variance was stable or increasing for all cognitive measures. Evidence for a decline in late adulthood in the genetic variance of General Cognitive Ability replicates previous findings from other twin studies of aging as well (McClearn et al., 1997; McGue & Christensen, 2001). While genetic variance is decreasing, environmental factors begin to account for more total variance in general cognitive ability in late adulthood. This change may reflect the accumulation of environmental influences that begin to impinge on cognitive performance, especially after the transition to late adulthood. Nonshared environmental variance may include onset of diseases or terminal decline that differ between members of a twin pair. In general, these results serve to emphasize that heritability is not a static number. The relative influence of genetic factors will change as the dynamic combination of genetic and environmental factors changes over the lifespan.

Correcting for processing speed had a marked effect on the trajectories for genetic influences on Block Design and General Cognitive Ability. When corrected for processing speed, genetic variance decreased steadily, diverging from the genetic variance estimate for the uncorrected cognitive measure. Thus, with increasing age, genetic variance associated with processing speed becomes a more prominent component of genetic variance for fluid abilities and for "*g*". Previous cross-sectional comparisons of the cognitive factor demonstrated that processing speed becomes the dominant component of the cognitive factor in late adulthood (Finkel et al., 1995). In addition, quantitative genetic analysis of cross-sectional data indicates that a significant portion of the genetic variance in General Cognitive Ability arises from genetic variance in processing speed (Finkel & Pedersen, 2000). The results of the current analysis serve to replicate and combine these results. Genetic influences on general cognitive ability and fluid abilities act primarily via an indirect path through processing speed, as opposed to direct genetic influences on the ability, per se. In addition, the proportion of genetic variance acting via this indirect path through processing speed increases with increasing age. The processing speed hypothesis proposes the strongest speed mediation effects for fluid abilities and, by extension, general cognitive abilities or "*g*". In this combination of quantitative genetic and latent growth curve methodologies, we provide additional support for the processing speed hypothesis. We are able to highlight the increasingly important roles that processing speed and the genetic influences on processing speed play in longitudinal age changes in fluid abilities and general cognitive abilities.

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