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Genetic Variance in Processing Speed Drives Variation in Aging of Spatial and Memory Abilities

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Abstract

Previous analyses have identified a genetic contribution to the correlation between declines with age in processing speed and higher cognitive abilities. The goal of the current analysis was to apply the biometric dual change score model to consider the possibility of temporal dynamics underlying the genetic covariance between aging trajectories for processing speed and cognitive abilities. Longitudinal twin data from the Swedish Adoption/Twin Study of Aging, including up to 5 measurement occasions covering a 16-year period, were available from 806 participants ranging in age from 50 to 88 years at the 1st measurement wave. Factors were generated to tap 4 cognitive domains: verbal ability, spatial ability, memory, and processing speed. Model-fitting indicated that genetic variance for processing speed was a leading indicator of variation in age changes for spatial and memory ability, providing additional support for processing speed theories of cognitive aging.

Keywords

cognitive aging; longitudinal twin study; behavioral genetics; temporal dynamics; speed–cognition relationship

Mediational theories of age-related cognitive decline focus on identification of a general factor that underlies demonstrated declines in higher cognitive abilities. One candidate is processing speed (Birren, 1964; Salthouse, 1996), and it has gained considerable empirical support. A meta-analysis of cross-sectional studies indicated that up to 79% of age-related variance in many cognitive abilities could be explained by age-related variance in measures of processing speed (Verhaeghen & Salthouse, 1997). There is some question, however, as to whether processing speed or working memory is a stronger candidate for the leading

factor in cognitive aging (Hertzog, Dixon, Hultsch, & MacDonald, 2003; Hultsch, Hertzog, Dixon, & Small, 1998; McArdle, Hamagami, Meredith, & Bradway, 2000; but see Verhaeghen & Salthouse, 1997). In addition, initial results from longitudinal investigations of the speed–cognition relationship did not concur with the results of cross-sectional analyses. Several longitudinal studies reported that a much smaller proportion of the intraindividual age changes in cognitive abilities were 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).

Inconsistent evidence for speed–cognition relationships between cross-sectional versus longitudinal investigations may be due to length of follow-up and analytical considerations. Longer longitudinal periods of assessment allow researchers to model more complex relationships between processing speed and cognitive ability. For example, with sufficient measurement occasions, it is possible to model nonlinear age changes in both processing speed and higher cognitive abilities and examine the relationship between accelerating age trajectories. In their analysis of the longitudinal relationship between speed and cognition, Sliwinski and Buschke (1999) reported that processing speed mediated only a modest portion (6% to 29%) of the longitudinal age effects in cognition. Using data from a study with up to five measurement occasions over a 16-year interval, a nonlinear growth curve analysis of the speed–cognition relationship found that for cognitive traits that were hypothesized to have the strongest relationship with processing speed, accelerating age changes in cognition were related to accelerating age changes in speed (Finkel, Reynolds, McArdle, & Pedersen, 2005). The longitudinal relationship in the quadratic (accelerating) parameters was of the same magnitude as the cross-sectional results summarized by Verhaeghen and Salthouse (1997). Thus, it is not the linear age changes but accelerating age changes in cognitive performance that share variance with processing speed.

Estimating correlations between rates of decline does not address the issue of causation; we can only conclude that aging in processing speed is related to aging of higher cognitive abilities. The development of dual change score models (DCSM) to characterize age changes has allowed researchers to more fully use longitudinal data to specify and test dynamic hypotheses about cognitive aging (McArdle, 2001; McArdle & Hamagami, 2003; McArdle et al., 2000). In other words, the models allow for the identification of leading indicators of cognitive change: the extent to which changes in one variable impact subsequent changes in a related variable. Applications of DCSM to the speed–cognition relationship indicate that processing speed is, in fact, the leading indicator of age changes in higher cognitive abilities (Finkel, Reynolds, McArdle, & Pedersen, 2007a; Ghisletta & de Ribaupierre, 2005; Ghisletta & Lindenberger, 2003; McArdle et al., 2000). These analyses provide the strongest evidence to date in support of a central role of processing speed as a general factor underlying the age changes observed in higher cognitive abilities.

In investigations of variance shared between two traits, behavioral genetic methods can be used to focus the analysis on genetic and environmental contributions to the common variance. Several cross-sectional studies have examined genetic contributions to the relationship between processing speed and various measures of cognitive ability at different points in the adult lifespan (see Posthuma, de Geus, & Boomsma, 2002, for a review). The results indicate that 65% to 100% of the covariance between speed and cognition at any point in the lifespan is explained by a common genetic mechanism. Evidence from longitudinal twin studies indicates that the genetic covariance between speed and cognition is amplified in late adulthood, such that an increasing proportion of genetic variance for cognitive ability can be attributed to genetic influences on processing speed (Finkel & Pedersen, 2004). Furthermore, an analysis incorporating nonlinear aging trajectories for both

processing speed and higher cognitive abilities concluded that it is not the linear age changes but the accelerating age changes in cognitive performance that share genetic variance with processing speed, at least for fluid ability and general cognitive ability (Finkel et al., 2005).

Decomposition of the covariance between speed and cognition into genetic and environmental components can focus our search for causal mechanisms of the speed–cognition relationship, but it cannot identify temporal causality, per se. Even behavioral genetic investigations of the correlation between the rate of change for speed and cognition (e.g., Finkel et al., 2005) cannot determine the leading indicator of age changes. As Posthuma et al. (2003) explained, genetic variance shared between processing speed and higher cognitive abilities can arise in three ways: (a) pleiotropic effects, in which a common set of genes influences both speed and cognition; (b) directional effects, in which genes influencing variation in processing speed in turn influence variation in cognitive abilities (or vice versa); or (c) a dynamic process in which the influence is bidirectional, such that genes for processing speed influence cognitive abilities and genes for cognitive abilities influence processing speed. Clearly, differentiating between these options is difficult. The direction-of-causation method (Heath et al., 1993) uses cross-sectional twin data and relies on differences in heritability between the two correlated traits. If the covariance between the two traits is largely genetic, then the trait with higher heritability is assumed to be the causal agent. On the other hand, if the covariance between the two traits is largely environmental, then the trait with the greater environmental component is assumed to be the causal agent. A recent application of this method to the speed–cognition relationship in a sample of young and middle-aged twins found no support for any causal relationship between genetic variance in speed and cognitive abilities; the researchers concluded that the pleiotropic model provided the best fit to the data (Luciano et al., 2005). The direction-of-causation method is limited by its reliance on cross-sectional data and the requirement that the genetic influence on each trait be sufficiently different in magnitude to support causal inferences. In addition, in the absence of multiple indicators of the traits in question, directional causal models should only be tested if there are at least three sources of variation in the two traits (Heath et al., 1993). The inspection time and IQ variables used by Luciano et al. (2005) were assessed by single measures and were influenced by only two sources of variation: additive genetic effects and nonshared environmental effects.

An extension of the DCSM that incorporates longitudinal twin data (McArdle & Hamagami, 2003) allows for a more direct investigation of the three possible mechanisms for genetic covariance between speed and cognition. By directly modeling the genetic and environmental decomposition of the lead–lag relationships between two variables, the biometric DCSM can determine whether genetic factors impacting processing speed are a leading indicator of variation in aging trajectories for higher cognitive abilities, genetic factors impacting cognitive abilities are a leading indicator of aging changes in processing speed, or a dynamic relationship exists with bidirectional genetic influences. In addition, the biometric DCSM is not subject to the requirements of the direction-of-causation method (e.g., three sources of variance, significant differences in heritability). In the first application of the biometric DCSM model, McArdle and Hamagami (2003) reported a modest but significant bidirectional relationship between genetic influences on block design and vocabulary. They concluded that changes in genetic influences on one variable may be impacted by dynamic changes in genetic influences from another variable.

The goal of the current analysis was to apply the biometric DCSM model to consider the possibility of temporal dynamics underlying the genetic covariance between aging trajectories for processing speed and higher cognitive abilities. The longitudinal Swedish Adoption/Twin Study of Aging (SATSA; Finkel & Pedersen, 2004) includes a cognitive battery that allows for the identification of latent factors to indicate three cognitive domains

in addition to processing speed: verbal abilities, spatial abilities, and memory. Five waves of data collection have been completed for a sample of 806 nondemented individuals with at least one data point and a longitudinal follow-up period of up to 16 years. The cross-sectional age range of the sample at the first wave was 50 to 88 years; thus, the focus of the current analysis is the aging process during the second half of the lifespan. Based on previous results indicating that processing speed is a leading indicator of age changes in spatial and memory abilities (Finkel et al., 2007a), we expect to find that genetic influences on processing speed impact the genetic influences on variation in subsequent change in spatial and memory abilities; hence, such a pattern would suggest temporal causality, though not necessarily direct causation. Genetic influences on processing speed are hypothesized to have a much smaller impact on verbal ability.

Method

Participants

Ascertainment procedures for SATSA have been described previously. In brief, the sample is a subset of twins from the population-based Swedish Twin Registry (Finkel & Pedersen, 2004). The base population comprises all pairs of twins 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 and date and county of birth. Twins were first mailed questionnaires; a subsample of those pairs age 50 or older in which both twins responded was invited to participate in an additional examination of health and cognitive abilities, which occurred in person (Pedersen et al., 1991). In-person testing (IPT1) took place in a location convenient to the twins, such as district nurses' offices, health-care schools, and long-term-care clinics. Testing was completed during a single 4-hr visit. The second (IPT2) and third (IPT3) waves of in-person testing occurred after 3-year intervals. In-person testing did not occur during Wave 4; therefore, the next wave of in-person testing is labeled IPT5 and occurred after a 7-year interval (see Finkel & Pedersen, 2004). The fifth wave of in-person testing (IPT6) took place 3 years after IPT5. In-person testing was conducted in Swedish by registered nurses who were native Swedish-speakers or fluent in Swedish.

The SATSA sample accurately represents the Swedish population age 50 and above (Pedersen et al., 1991). The sample mirrors the ethnic homogeneity of the birth cohorts represented (1900–1948). As expected from population demographics in this age range, 59% of the sample was female. In SATSA, education is rated on a 4-point scale from 1 (*elementary school*) to 4 (*university or higher*). Mean education in the current sample was 1.6 (standard deviation = 0.9). Participants were drawn from all 21 of Sweden's counties. Zygosity was determined via questionnaire for all twins and via blood serum testing for almost all twins who completed in-person testing.

Dementia status was determined by clinical diagnosis based on current diagnostic criteria (Gatz et al., 1997); participants who developed dementia up to the time of IPT5 testing were not included in the current analyses. It is possible that some participants may have developed dementia subsequent to the completion of IPT5. The number of participants at each in-person testing occasion who remained free of dementia as of IPT5 is 594, 558, 538, 516, and 441. Not all SATSA participants were tested at each wave; some twins dropped out or participated intermittently, whereas others entered at later waves once they met the age requirements. In total, 806 nondemented individuals had cognitive data available from at least one testing occasion across a 16-year time span, and 67% of these participants had data at three or more time points.

Table 1 presents the number of complete and incomplete twin pairs available for the cognitive factors in each 3-year interval as a function of age at assessment and the total

number of individuals in each interval. The full age trajectory spanned 45 years, from the first IPT of the youngest participant to the last IPT of the oldest participant. However, the data for each cognitive factor were too sparse after age 89 to support statistical modeling; therefore, only data up to age 88.9 were included in the present analyses. Data from complete twin pairs is fairly limited between ages 83 and 89; however, fitting the structural models without these two age intervals had only a small impact on either parameter estimates or model fit. Because SATSA is a cohort-sequential design, the sample sizes are not constant at each age of assessment. The sample sizes diminish at the higher ages as a result of attrition, and the number of available data points is smaller at the earlier ages because of the nature of the design. Using the 3-year age interval allowed us to maximize the age range available for inclusion in the analyses.

Measures

Four cognitive domains are represented in the SATSA cognitive test battery (see Nesselroade, Pedersen, McClearn, Plomin, & Bergeman, 1988; Pedersen, Plomin, Nesselroade, & McClearn, 1992): verbal, spatial, memory, and processing speed abilities. Verbal abilities are tapped by tests of Information, Synonyms, and Analogies. The Figure Logic, Block Design, and Card Rotations tests assess spatial abilities. Memory tests include Digit Span, Picture Memory, and Names and Faces. Finally, Symbol Digit and Figure Identification measure processing speed. Reliabilities for these tests range from .82 to .96 (Pedersen et al., 1992). Principal components analysis was used to construct latent factors from the individual tests within each domain: verbal, spatial, memory, and speed. For the verbal, spatial, and speed factors, factor loadings ranged from .78 to .92, and internal consistency was .85, .78, and .82, respectively. The memory factor was more diverse, including measures of short-term, long-term, and picture memory. Factor loadings ranged from .64 to .78, and the internal consistency was .60. Previous comparisons of factor structure between cohorts and across testing occasions indicate that the factor structure does not vary systematically across age or time (Finkel, Reynolds, McArdle, & Pedersen, 2005; see also Salthouse & Davis, 2006). An invariant definition of factors at each testing occasion was created by standardizing the cognitive measures relative to the respective means and variances at IPT1, and the loadings from the factor analyses conducted at IPT1 were used to construct the verbal, spatial, and memory factors. The speed measures were combined into a speed factor using unit weighting. Finally, for ease of interpretation, all factor scores were translated to T-scores using factor means and variances from IPT1.

Longitudinal age plots of subsets of the twin data are presented in Figure 1. Longitudinal data for 10 randomly selected twin pairs are presented in each graph as a function of each individual's age at testing. Each twin pair is indicated by the letter used to symbolize data points (A through J). For each twin pair, repeated scores for one twin are connected over time with a solid line, and repeated scores for the co-twin are connected over time with a dashed line. Thus, on the verbal factor, identical (monozygotic; MZ) twin pair J expressed very similar trajectories of change over time, although Twin 1 participated in four waves of measurement and Twin 2 participated in only three. In contrast, the cross-time performances on the verbal factor for fraternal (dizygotic; DZ) twin pair J are quite different. The graphs demonstrate both the complexity of data modeling issues and the general trends that can be observed. Although there are clearly individual and within-pair differences for both twin types on all four cognitive measures, the spatial and speed factors manifest steeper decline trajectories than the verbal and memory factors. In addition, MZ pairs are generally more similar in trajectories of decline than DZ pairs across the four cognitive factors.

Statistical Method

Univariate analysis—A univariate biometric DCSM was used to examine genetic and environmental influences on age changes for each of the cognitive factors independently. Extensive discussions of the DCSM are available (McArdle, 2001; McArdle & Hamagami, 2003; McArdle et al., 2004), as well as comparisons of DCSM with latent growth curve models (Ghisletta & de Ribaupierre, 2005; Lövdén, Ghisletta, & Lindenberger, 2005). As presented in Figure 2, the model is based on latent difference scores to create a growth curve based not on performance at one age but on change from one age to the next. Thus, Y_{50} represents observed performance on measure Y at age 50, with y_{50} indicating the latent true score and $u_{y,50}$ signifying error. Error variance (σ_u) is assumed to be constant at each age. Performance at age 53 is a function of both performance at Y_{50} and of the change in performance that occurred between ages 50 and 53 (Δy_{53}). The focus of the DCSM is to predict changes in the latent trait score (Δy), which are modeled as a function of both constant trends in change across time and a self-feedback component (e.g., added changes in the trait given the previous score). *Constant change* (α) is related to the slope factor (y_s), such that age changes accumulate over time in an additive fashion. *Proportional change* signifies that the difference between performances at any two adjacent ages is directly proportional to the previous score, through the parameter β . Thus, the model for change in Y at time point t can be written as in Equation 1:

$$\Delta y[t] = \alpha y_s + \beta y[t-1] \quad (1)$$

The values of α and β are commonly assumed to be constant across time (although this assumption is testable), and they allow for the generation of a family of curves capturing the trajectories of change with age. In the full DCSM model, α is set to 1 and the parameter β differs from zero to the extent that the longitudinal change is nonlinear. In describing change with age, then, the DCSM estimates four fixed effects: α , β , mean intercept (μ_0), and mean slope (μ_s).

Twin data allow for the decomposition of individual variation (random effects) around the group mean intercept and slope into genetic and environmental components. The variance in any trait can be divided into four separate components: additive genetic effects (V_a), correlated environmental effects or general cultural effects that can be shared by twins regardless of rearing status (V_c), shared rearing environmental effects that serve to make the members of a family more similar (V_s), and nonshared environmental effects, including error (V_e). 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.

$$\text{covMZT} = V_a + V_c + V_s \quad (2)$$

$$\text{covMZA} = V_a + V_c \quad (3)$$

$$\text{covDZT} = \frac{1}{2} V_a + V_c + V_s \quad (4)$$

$$\text{covDZA} = \frac{1}{2} V_a + V_c \quad (5)$$

Whereas MZ twins share 100% of their genes, DZ twins share on average only half of their segregating genes, and thus only half of the additive genetic effects can contribute to DZ twin similarity ($\frac{1}{2}V_a$). Although the general cultural effects of V_c can impact the similarity of all twin pairs, V_s can only contribute to the similarity of twins reared together. By definition, the individual-specific (nonshared) effects of V_e cannot contribute to twin similarity. By fitting structural models to the observed MZA, MZT, DZA, and DZT covariance matrices, we can estimate the proportion of total variance accounted for by the variance in genetic factors, shared environment factors, correlated environment factors, and nonshared environment factors. Heritability is defined as the proportion of total variance attributed to genetic variance.

To decompose both the individual variation around the mean intercept and slope and the correlation between the intercept and slope, a standard Cholesky model (Neale & Cardon, 1992) was implemented (as in McArdle & Hamagami, 2003). As shown in Figure 2, the model includes genetic and environmental effects specific to the intercept (a_0 and e_0) and genetic and environmental effects specific to the slope (a_s and e_s). The paths from a_0 and e_0 to the slope (y_s) generate the Cholesky decomposition of the correlation between the intercept and slope (for simplicity, only additive genetic and nonshared environmental paths are included in the figure).

Bivariate analysis—A bivariate biometric DCSM was used to examine genetic and environmental contributions to the dynamic relationships between age changes in the cognitive factors (McArdle & Hamagami, 2003). The extension of the basic DCSM allows for constant change (α), proportional change (β), and a coupling mechanism (γ), where change in trait X depends on the previous value of Y , and vice versa. The model, presented in Figure 3, includes time series for two traits, X and Y , with the dynamic interrelationship between changes in the two traits modeled by two additional fixed effects. The new parameters allow the two traits to affect each other in a dynamic way by modeling the effect of Y on subsequent changes in X (γ_{yx}) and the effect of X on subsequent changes in Y (γ_{xy}). Thus, the model for the dynamic interaction of changes in X and Y at time point t can be written as in Equations 6 and 7,

$$\Delta y[t] = \alpha_{y,y_s} + \beta_{y,y} [t-1] + \gamma_{xy,x} [t-1], \quad \text{and} \quad (6)$$

$$\Delta x[t] = \alpha_{x,x_s} + \beta_{x,x} [t-1] + \gamma_{yx,y} [t-1], \quad (7)$$

so that change in one trait is a time-based function of both itself and another trait. As with the dual change parameters α and β , the coupling parameters (γ_{xy} and γ_{yx}) are assumed to be constant over the whole time series.

Again, twin data allow us to decompose the individual variation (random effects) around the group mean intercept and slope parameters for each variable. As shown in Figure 3, the model includes genetic and environmental effects specific to the each intercept (a_{x0} and e_{x0} , a_{y0} and e_{y0}) and genetic and environmental effects specific to the slope (a_{xs} and e_{xs} , a_{ys} and e_{ys}). The paths from a_{x0} and e_{x0} to x_s and from a_{y0} and e_{y0} to y_s generate the Cholesky decomposition of the correlation between the intercept and slope for X and Y . Coupling parameters estimate dynamic relationships between variables within individual. The biometric portion of the model estimates relationships between twins, but within variables. Thus, in the biometric bivariate DCSM, the only paths between variables are the coupling parameters. Model testing is used to determine the impact of the coupling parameters on the estimates of genetic and environmental variance.

It is important to note that one of the fundamental assumptions of DCSM is that data are missing at random. Without complete mortality data, it is difficult to establish whether the missing-at-random assumption is met, although previous investigations of SATSA data suggest that participants who continue in the study are significantly different from those who drop out (e.g., personality ratings; Pedersen & Reynolds, 1998). Of most importance in investigations of aging is to demonstrate that the pattern of missing data does not differ for older and younger participants. Cohort comparisons of missing data (Finkel, Reynolds, McArdle, & Pedersen, 2007b) indicate that the patterns of participation are fairly similar, although older participants are somewhat more likely (71%) than younger participants (64%) to participate in at least three time points, simply as a result of a greater number of opportunities for participation. At IPT2, IPT3, and IPT5, any twins who had responded to the initial questionnaire and reached age 50 were invited to participate in SATSA (Finkel & Pedersen, 2004).

Both univariate and bivariate biometric DCSM were fit with the structural equation modeling program Mx (Version 1.66b; Neale, Boker, Xie, & Maes, 2003), using the variable length datafile option that takes advantage of data from both complete and incomplete pairs. The raw maximum likelihood estimation procedure was used throughout. Hypotheses were tested by comparing model fit indices; nested models were compared using the difference chi-square test obtained by taking the difference between the obtained model fits ($-2LL$) and testing its significance with the degrees of freedom equal to the difference in the number of parameters of the two models.

Results

Univariate Analysis

In the first step of the analysis, the univariate biometric DCSM was fit to each of the four cognitive factors separately, to verify the shape of the change trajectory over age and to provide starting values for the bivariate DCSM model. Parameter estimates (and 95% confidence intervals) resulting from fitting the full model are presented in Table 2, along with model fit statistics. Estimates of change parameters must be interpreted with caution; consider that the mean slope estimates presented in Table 2 represent only one component of change and can only be interpreted in the context of the proportional change component, β . The magnitude of β indicates the extent to which current performance is predicted by previous performance. Thus, larger values of β indicate a stronger relationship between current and past performance, which signifies stability in the age trajectory. In contrast, smaller values of β show that previous performance is not contributing much to the prediction of current performance, indicating greater change over age. Positive values for β indicate that change accumulates or accelerates with age; in combination with a negative mean slope estimate (μ_s), accelerating decline with age is indicated. Thus, accelerating decline in performance is indicated for all four cognitive factors.

Previous analyses tested hypotheses about the fixed effects (Finkel et al., 2007a); the focus of the current analysis was on the genetic and environmental decomposition of random effects. Therefore, four reduced models were used to test the significance of genetic and environmental contributions to individual variation in the intercept and slope parameters. In Model 2, genetic influences on the mean intercept were dropped from the model (set $a_0 \rightarrow y_0 = 0$). In Model 3, genetic influences specific to the slope were dropped from the model (set $a_s \rightarrow y_s = 0$). In Model 4, genetic influences on the correlation between the intercept and slope were dropped from the model (set $a_0 \rightarrow y_s = 0$). Finally, in Model 5 all shared rearing environment and correlated environment parameters were dropped from the model. The results of model testing are presented in Table 3. Model testing indicates significant genetic influences on the intercept for each cognitive factor, but no significant genetic

influences specific to the slope. Genetic influences impact the slope parameters only through genetic influences on the intercepts via the intercept–slope correlation. The results presented in Table 2 indicate that the 95% confidence interval for all estimates of shared rearing environment and correlated environment effects encompassed zero, suggesting no significant impact of these components of variance. Model fitting results support this conclusion: Dropping the shared rearing environment and correlated environment effects resulted in nonsignificant changes in model fit for all four factors.

Bivariate Analyses

The focus of the current analysis was investigation of the impact of genetic and environmental influences on the leading role for processing speed in cognitive aging. For that reason, three dynamic relationships were investigated: processing speed as a leading indicator of change in verbal ability, spatial ability, and memory ability. Parameter estimates (and 95% confidence intervals) resulting from fitting the full biometric bivariate DSCM to these pairs of factors are presented in Tables 4 through 6, along with model fit statistics. The addition of the coupling parameter changes the prediction of Δy and Δx : They are now functions of α , β , and γ . Therefore, estimates of α , β , mean intercept, and mean slope parameters in the bivariate model may differ from the univariate model results. The estimates of the fixed effects are similar to the estimates from the phenotypic bivariate DCSM analysis reported by Finkel et al. (2007a).

The bivariate biometric analysis of processing speed and verbal ability suggests modest bidirectional coupling between the two variables (see Table 4). Although the estimates of γ are small (.07 and .03), the confidence intervals indicate that they are significantly different from zero. Model testing results, summarized in Table 7, also supported the conclusion that neither the speed $\rightarrow$ verbal coupling parameter (Model 2) nor the verbal $\rightarrow$ speed coupling parameter (Model 3) could be dropped from the model without significantly reducing model fit. However, setting the two coupling parameters equal to each other (Model 4) did not significantly impact model fit. As in the univariate model, strong genetic influences for the intercept are indicated, but genetic influences specific to the slope did not differ significantly from zero; all genetic influences on the slope act through genetic influences on the intercept. As expected from the univariate model fitting results, shared rearing environment and correlated environment effects could be dropped from the model without reducing model fit (Model 6 in Table 7). Model parameters were used to calculate the expected values of the age-based dynamics, providing an indication of the change in variance components over the age range.¹ Figure 4 presents the expected values for each age interval for both genetic variance (top) and environmental variance (bottom) resulting from two of the models tested: a model assuming full coupling between the two variables (Model 1) and a model assuming no coupling between the two variables (Model 5). Although Model 5 did not fit the data as well as Model 1, comparing the results with and without coupling demonstrates the impact of coupling on the age-based estimates of genetic and environmental influences on the speed and verbal factors. Figure 4 shows little difference between the estimates of the full coupling and no coupling models for either the speed or the verbal factor; both indicate increasing genetic and environmental variance with age. The limited difference between the full coupling and no coupling models for these factors reflects the modest size of the coupling parameters between the speed and verbal factors.

¹Computation of the genetic and environmental components of variance from the parameters of the bivariate biometric DCSM involves the following steps: (a) Compute the expected covariance matrix according to the full bivariate biometric latent difference score model that includes additive genetic parameters in the model; (b) compute the expected covariance matrix based on the submodel in which all genetic parameters are removed from the full biometric model; (c) isolate additive genetic effects (i.e., variance due to the additive genetic effects); and (d) derive heritability, using quantification obtained from steps (a), (b), and (c). Matrix algebra associated with these steps is available from Deborah Finkel.

In contrast, the bivariate biometric DCSM analysis of processing speed with spatial ability suggests not bidirectional coupling but a leading role for processing speed (see Table 5). Although neither coupling parameter listed in Table 5 can be dropped from the model without significantly impacting model fit (Models 2 and 3 in Table 7), equating the two parameters resulted in a significant reduction in model fit (Model 4). Given the modest estimate for the spatial $\rightarrow$ speed coupling parameter ($\gamma = .02$), we can infer a unidirectional relationship between the speed and spatial factors, with speed as the leading indicator of age changes in spatial ability ($\gamma = .65$). Again, parameter estimates indicate significant genetic influences on the intercept for both cognitive factors, but genetic influences on the slopes act through genetic influences on the intercepts. Results for Model 6 (see Table 7) indicate that shared rearing environment and correlated environment did not contribute significantly to the individual variation captured by the bivariate DCSM of the speed and spatial factors. Figure 5 presents the expected values at each age interval for both genetic and environmental variance of the speed and spatial factors as predicted by the full coupling and no coupling models. The dip that occurs at age 53 in the full-coupling curve for genetic variance in the spatial factor results from the fact that γ does not contribute to the estimation of change and the genetic contributions to change until age 53 (see Figure 3). Unlike the results for the verbal factor, coupling with processing speed has a dramatic impact on the age-based estimates of genetic variance for the spatial factor. Increasing genetic variance for the spatial factor in the full model changes to decreasing genetic variance in the no coupling model. When the coupling with speed is removed, a large part of the genetic contribution to individual variation in the spatial factor is also removed. Thus, comparison of the full coupling and no coupling models indicates that an increasing portion of the genetic influences on change in the spatial factor is acting through dynamic coupling with processing speed. In contrast, genetic variance estimates for the speed factor differ little with and without coupling with spatial ability. Moreover, environmental variance is largely unaffected by coupling between the factors for either speed or spatial ability.

Results for the bivariate biometric DCSM analysis of processing speed with memory ability are very similar to the results for spatial ability (see Table 6). The memory $\rightarrow$ speed coupling parameter is estimated at .01 with a 95% confidence interval from .00 to .02. In addition, when the memory $\rightarrow$ speed coupling parameter is dropped from the model, the change in model fit is significant at only the .05 level (Model 3 in Table 7). Equating the memory $\rightarrow$ speed coupling parameter with the speed $\rightarrow$ memory coupling parameter ($\gamma = .34$) results in a significant reduction in model fit (Model 4). Therefore, we can conclude that processing speed is the leading indicator of age changes in memory ability. As expected, dropping shared rearing environment and correlated environment effects from the model did not significantly reduce model fit (Model 6). The expected values at each age interval for both genetic and environmental variance of the memory and speed factors as predicted by the full coupling and no coupling models are presented in Figure 6. As with the spatial factor, coupling with processing speed significantly changes the age-based estimates of genetic variance for the memory factor but has little effect on environmental variance. Removing the genetic variance associated with processing speed diminished the genetic variance remaining for memory; this effect is amplified with increasing age, such that an increasing proportion of genetic variance for memory ability can be attributed to genetic influences on processing speed. As expected from the results of model testing, coupling with memory had little impact on genetic and environmental variance for the speed factor.

Discussion

To test hypotheses about the possibility of temporal dynamics underlying the genetic covariance between aging trajectories for processing speed and higher cognitive abilities, biometric DCSMs were fit to the four cognitive factors in two steps: univariate and

bivariate. In the univariate analyses, models were fit to the verbal, spatial, memory, and speed factors separately. Model comparisons indicated two general conclusions. First, similar to results from latent growth curve analyses of cognitive data (e.g., McArdle, Prescott, Hamagami, & Horn, 1998; McGue & Christensen, 2001; Reynolds, Finkel, Gatz, & Pedersen, 2002; Reynolds et al., 2005), model testing indicated significant genetic influences on the intercept for each cognitive factor, but no significant genetic influences specific to the slope. Genetic influences impacted the slope parameters only through genetic influences on the intercepts via the intercept–slope correlation. Although the parameterization of the slope differs in DCSM and latent growth curve models, the results concur. Therefore, current results support the conclusion that although genetic variance plays a significant role in cognitive performance at any one point in time, changes over time result primarily from environmental factors. Second, as is true in most biometric analyses of cognitive aging (for a review, see McGue & Johnson, 2007), individual variation in late adulthood is explained primarily by additive genetic and nonshared environmental influences; model fitting provided no evidence for correlated or shared environmental effects. Although rearing environmental effects make a significant contribution to cognitive abilities during childhood, their impact begins to decline early in adulthood (Plomin, DeFries, McClearn, & McGuffin, 2001).

Application of bivariate DCSMs to the four cognitive factors allowed us to address the central hypotheses of temporal dynamics in the genetic covariance between speed and cognitive aging. Three dynamic relationships were investigated: processing speed as a leading indicator of age changes in verbal, spatial, and memory ability. Given that processing speed has a stronger relationship with fluid ability than crystallized ability (Salthouse, 1996), we expected to find that genetic variance in processing speed would play a stronger leading role in age changes in spatial and memory ability than in verbal ability. Model fitting results supported our expectations. The dynamic relationship of genetic variance between the speed and verbal factors was bidirectional, but modest. Dropping the coupling between the two variables had little impact on the age changes in genetic and environmental variance for the verbal factor. In contrast, model fitting results indicated a strong unidirectional coupling between speed and spatial factors and between speed and memory factors. When coupling with the speed factor was removed from the model, a large portion of the genetic variance for the cognitive factor (spatial or memory) was also removed. Thus, we can conclude that genetic variance in processing speed drives variation in higher cognitive abilities, particularly for measures of fluid ability. In other words, coupling tells us that genetic factors that impact speed at an earlier time point also impact the cognitive factor (spatial, memory) at a later time point. The results also suggest that an increasing portion of the genetic influences on age changes in the spatial and memory factors acts through dynamic coupling with processing speed.

The conclusion of unidirectional temporal dynamics as the explanation of the genetic covariance between aging trajectories for processing speed and higher cognitive abilities differs from the results of Luciano et al. (2005) for several reasons. Clearly, the difference in analytical methods (cross-sectional vs. longitudinal) is a major source of differing conclusions. Although the direction of causation method for use with cross-sectional twin data has the potential to elucidate issues of causation, in their application of it Luciano et al. were limited by single indicators of each trait and the existence of only two components of variance for each trait. In contrast, the DCSM method uses longitudinal twin data to allow direct estimation of leading indicators of genetic influences on change. Additionally, there was very little overlap in the age ranges of the samples used by Luciano et al. and in the present analyses. Twins included in the cross-sectional analyses ranged in age from 16 to 56, whereas twins in the SATSA sample were measured at ages ranging from 50 to 89. Given that the dynamic relationship between speed and cognition indicates that the proportion of

genetic variance in cognition explained by genetic factors for processing speed increases throughout the age range, it is possible that the dynamic relationship was not of sufficient magnitude to be detected earlier in the lifespan. Although Luciano et al. (2005) concluded that the genetic covariance for speed and cognition resulted from pleiotropic effects, the results of the current analysis were consistent with a hypothesis of directional causation, at least during the second half of the lifespan.

One of the limitations of the current analysis is clearly the sparseness of the data in very late adulthood. Although as many as 42 individuals were included in the 86.0 to 88.9 age range, that encompasses very few twin pairs to provide genetically informative data. In the current analyses, we chose to use all available information, regardless of co-twin status or participation patterns. In other words, every available data point was included in the model, thereby reducing bias that would otherwise occur if only complete data were used. It is important to note that attrition in the SATSA sample has been kept to a minimum of 10% to 15% at each wave. However, attrition and mortality are important factors (e.g., Dominicus, Palmgren, & Pedersen, 2006). In future applications on the model, we plan to incorporate modeling of the effects of attrition and mortality. An additional limitation of the model is the use of constant values over age for the proportional change and coupling parameters. Our goal in the current analyses was to start with the basic DCSM model, which required a considerable computational burden. Future directions include investigating alternative models, including examination of increases or decreases in coupling before and after a certain age and investigating the genetic and environmental contributions to individual variation around the coupling and proportional change parameters. It could be argued that the 3-year age interval used in the current application of the dual change score model created between-person age variance within the age intervals. The age-based model used here (change from age 50 to age 53), as opposed to a time-based model (change between Wave 1 and Wave 2), minimizes the impact of between-person age variance. Instead, individuals from different cohorts are included in the same age interval, raising the possibility of cohort heterogeneity. However, previous analyses have identified little or no cohort effects in the SATSA data (Finkel et al., 2007b). In the future, it would be beneficial to establish the optimal age intervals to use in the DCSM to minimize possible between-person age variation.

The results of the biometric DCSM analysis suggest that researchers attempting to identify genes that impact not just static cognition at one point in time but the dynamic process of cognitive aging should focus their efforts on genetic factors influencing processing speed. Genetic factors impacting processing speed that drive variation in higher cognitive abilities likely include genes coding for fundamental aspects of the neural system, such as connectivity, myelination, quantity and quality of ion channels, and speed of synaptic transmission (Jensen, 1998). For example, processing speed has been genetically related to white matter volume, which includes all myelinated axons in the cerebrum (Posthuma et al., 2003). The positive correlation between processing speed and white matter volume ($r = .25$) was completely explained by genetic factors. Myelination increases the speed of neural transmission; therefore, it is likely that higher levels of myelination lead to faster processing speed. Several candidate genes have been identified that may be involved in myelination processes (Posthuma et al., 2003); the current analysis suggests that these genes may also serve as indicators of higher cognitive abilities in general and cognitive aging in particular.

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References

- Birren, JE. The psychology of aging. Prentice-Hall; Englewood Cliffs, NJ: 1964.
- Dominicus A, Palmgren J, Pedersen NL. Bias in variance components due to nonresponse in twin studies. *Twin Research and Human Genetics*. 2006; 9:185–193. [PubMed: 16611486]
- Finkel D, Pedersen NL. Processing speed and longitudinal trajectories of change for cognitive abilities: The Swedish Adoption/ Twin Study of Aging. *Aging, Neuropsychology, and Cognition*. 2004; 11:325–345.
- Finkel D, Reynolds CA, McArdle JJ, Pedersen NL. The longitudinal relationship between processing speed and cognitive ability: Genetic and environmental influences. *Behavior Genetics*. 2005; 35:535–549. [PubMed: 16184483]
- Finkel D, Reynolds CA, McArdle JJ, Pedersen NL. Age changes in processing speed as a leading indicator of cognitive aging. *Psychology and Aging*. 2007a; 22:558–568. [PubMed: 17874954]
- Finkel D, Reynolds CA, McArdle JJ, Pedersen NL. Cohort differences in trajectories of cognitive aging. *Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*. 2007b; 62:P286–P294.
- Gatz M, Pedersen NL, Berg S, Johansson B, Johansson K, Mortimer JA, et al. Heritability for Alzheimer's disease: The study of dementia in Swedish twins. *Journals of Gerontology, Series A: Biological Sciences and Medical Sciences*. 1997; 52:M117–M125.
- Ghisletta P, de Ribaupierre A. A dynamic investigation of cognitive dedifferentiation with control for retest: Evidence from the Swiss Interdisciplinary Longitudinal Study of the Oldest Old. *Psychology and Aging*. 2005; 20:671–682. [PubMed: 16420141]
- Ghisletta P, Lindenberger U. Age-based structural dynamics between perceptual speed and knowledge in the Berlin Aging Study: Direct evidence for ability dedifferentiation in old age. *Psychology and Aging*. 2003; 18:696–713. [PubMed: 14692858]
- Heath AC, Kessler RC, Neale MC, Hewitt JK, Eaves LJ, Kendler KS. Testing hypotheses about direction of causation using cross-sectional family data. *Behavior Genetics*. 1993; 23:29–50. [PubMed: 8476389]
- Hertzog C, Dixon RA, Hultsch DF, MacDonald WS. Latent change models of adult cognition: Are changes in processing speed and working memory associated with changes in episodic memory? *Psychology and Aging*. 2003; 18:755–769. [PubMed: 14692862]
- Hultsch, DF.; Hertzog, C.; Dixon, RA.; Small, BJ. Memory change in the aged. Cambridge University Press; New York: 1998.
- Hultsch DF, Hertzog C, Small BJ, McDonald-Miszczak L, Dixon RA. Short-term longitudinal change in cognitive performance in later life. *Psychology and Aging*. 1992; 7:571–584. [PubMed: 1466826]
- Jensen, AR. The g factor. Praeger; Westport, CT: 1998.
- Lövdén M, Ghisletta P, Lindenberger U. Social participation attenuates decline in perceptual speed in old and very old age. *Psychology and Aging*. 2005; 20:423–434. [PubMed: 16248702]
- Luciano M, Posthuma D, Wright MJ, de Geus EJC, Smith GA, Geffen GM, et al. Perceptual speed does not cause intelligence, and intelligence does not cause perceptual speed. *Biological Psychology*. 2005; 70:1–8. [PubMed: 16038769]
- MacDonald SWS, Hultsch DF, Strauss E, Dixon RA. Age-related slowing of digit symbol substitution revisited: What do longitudinal age changes reflect? *Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*. 2003; 58:P187–P194.
- McArdle, JJ. A latent difference score approach to longitudinal dynamic structural analyses.. In: Cudeck, R.; duToit, S.; Sorbom, D., editors. *Structural equation modeling: Present and future*. Scientific Software; Lincolnwood, IL: 2001. p. 342–380.
- McArdle JJ, Hamagami F. Structural equation models for evaluating dynamic concepts within longitudinal twin analyses. *Behavior Genetics*. 2003; 33:137–159. [PubMed: 14574148]
- McArdle JJ, Hamagami F, Jones K, Jolesz F, Kikinis R, Spiro A III, Albert MS. Structural modeling of dynamic changes in memory and brain structure using longitudinal data from the normative aging study. *Journals of Gerontology, Series B: Psychological Sciences and Social Sciences*. 2004; 59:P294–P304.

- McArdle JJ, Hamagami F, Meredith W, Bradway KP. Modeling the dynamic hypotheses of Gf-Gc theory using longitudinal life-span data. *Learning and Individual Differences*. 2000; 12:53–79.
- McArdle JJ, Prescott CA, Hamagami F, Horn JL. A contemporary method for developmental–genetic analyses of age changes in intellectual abilities. *Developmental Neuropsychology*. 1998; 14:69–114.
- McGue M, Christensen K. The heritability of cognitive functioning in very old adults: Evidence from Danish twins aged 75 years and older. *Psychology and Aging*. 2001; 16:272–280. [PubMed: 11405315]
- McGue, M.; Johnson, W. Genetics of cognitive aging.. In: Craik, FIM.; Salthouse, TA., editors. *Handbook of aging and cognition*. Erlbaum; Hillsdale, NJ: 2007. p. 55-96.
- Neale, MC.; Boker, SM.; Xie, G.; Maes, HH. *Mx: Statistical modeling*. 6th ed.. Department of Psychiatry, Virginia Commonwealth University; VCU Box 900126, Richmond, VA 23298: 2003.
- Neale, MC.; Cardon, LR. *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic; London: 1992.
- Nesselroade JR, Pedersen NL, McClearn GE, Plomin R, Bergeman CS. Factorial and criterion validities of telephone-assessed cognitive ability measures: Age and gender comparisons in adult twins. *Research on Aging*. 1988; 10:220–234. [PubMed: 3175323]
- Pedersen NL, McClearn GE, Plomin R, Nesselroade JR, Berg S, de Faire U. The Swedish Adoption/Twin Study of Aging: An update. *Acta Geneticae Medicae et Gemellologiae*. 1991; 40:7–20.
- Pedersen NL, Plomin R, Nesselroade JR, McClearn GE. Quantitative genetic analysis of cognitive abilities during the second half of the lifespan. *Psychological Science*. 1992; 3:346–353.
- Pedersen NL, Reynolds CA. Stability and change in adult personality: Genetic and environmental components. *European Journal of Personality*. 1998; 12:365–386.
- Plomin, R.; DeFries, JC.; McClearn, GE.; McGuffin, P. *Behavioral genetics*. Worth; New York: 2001.
- Posthuma D, Baaré WFC, Pol HEH, Kahn RS, Boomsma DI, de Geus EJC. Genetic correlations between brain volumes and the WAIS-III dimensions of verbal comprehension, working memory, perceptual organization, and processing speed. *Twin Research*. 2003; 6:131–139. [PubMed: 12723999]
- Posthuma, D.; de Geus, EJC.; Boomsma, DI. Genetic contributions to anatomical, behavioral, and neurophysiological indices of cognition. *Behavioral genetics in the postgenomic era*. Plomin, R.; DeFries, JC.; Craig, IW.; McGuffin, P., editors. American Psychological Association; Washington, DC: 2002. p. 141-161.
- Reynolds CA, Finkel D, Gatz M, Pedersen NL. Sources of influences on rate of cognitive change over time in Swedish twins: An application of latent growth models. *Experimental Aging Research*. 2002; 28:407–433. [PubMed: 12227921]
- Reynolds CA, Finkel D, McArdle JJ, Gatz M, Berg S, Pedersen NL. Quantitative genetic analysis of latent growth curve models of cognitive abilities in adulthood. *Developmental Psychology*. 2005; 41:3–16. [PubMed: 15656733]
- Salthouse TA. The processing-speed theory of adult age differences in cognition. *Psychological Review*. 1996; 103:403–428. [PubMed: 8759042]
- Salthouse TA, Davis HP. Organization of cognitive abilities and neuropsychological variables across the lifespan. *Developmental Review*. 2006; 26:31–54.
- Sliwinski M, Buschke H. Cross-sectional and longitudinal relationships among age, cognition, and processing speed. *Psychology and Aging*. 1999; 14:18–33. [PubMed: 10224629]
- Taylor JL, Miller TP, Tinklenberg JR. Correlates of memory decline: A 4-year longitudinal study of older adults with memory complaints. *Psychology and Aging*. 1992; 7:185–193. [PubMed: 1610506]
- Verhaeghen P, Salthouse TA. Meta-analyses of age–cognition relations in adulthood: Estimates of linear and nonlinear age effects and structural models. *Psychological Bulletin*. 1997; 122:231–249. [PubMed: 9354147]
- Zimprich D. Cross-sectionally and longitudinally balanced effects of processing speed on intellectual abilities. *Experimental Aging Research*. 2002; 28:231–251. [PubMed: 12079576]
- Zimprich D, Martin M. Can longitudinal changes in processing speed explain longitudinal age change in fluid intelligence? *Psychology and Aging*. 2002; 17:690–695. [PubMed: 12507364]

Verbal Factor MZ

Verbal Factor DZ

Spatial Factor MZ

Spatial Factor DZ

Figure 1.

Longitudinal twin data on the four cognitive factors for random subsets of 10 identical (MZ) and 10 fraternal (DZ) twin pairs. Each twin pair is indicated by the letter used to symbolize data points (A–J). Repeated scores for one twin are connected over time with a solid line, and repeated scores for the co-twin are connected over time with a dashed line.

Figure 2.

A univariate biometric dual change score model to examine genetic and environmental influences on age changes for each of four factors (verbal, spatial, memory, and processing speed) independently (Y). Error variance (σ_u) is assumed to be constant at each age; α represents constant change and is related to the slope factor y_s ; β represents proportional change. The model includes an estimate for intercept (y_0), mean intercept (μ_0), and mean slope (μ_s). The model includes genetic and environmental effects specific to the intercept (a_0 and e_0) and specific to the slope (a_s and e_s). The paths from a_0 and e_0 to the slope (y_s) generate the decomposition of the correlation between the intercept and slope.

Figure 3.

A bivariate biometric dual change score model to examine genetic and environmental influences on the relationship between age changes in two cognitive factors (Y and X). Error variance (σ_u) is assumed to be constant at each age within each factor; α_y and α_x represent constant change related to the slope factors y_s and x_s ; β_y and β_x represent proportional change in Y and X ; cross-trait coupling is indicated by γ_{yx} and γ_{xy} . The model includes estimates for intercepts (y_0 and x_0), mean intercepts (μ_{y0} and μ_{x0}), and mean slopes (μ_{ys} and μ_{xs}). The model includes genetic and environmental effects specific to the each intercept (a_{x0} and e_{x0} , a_{y0} and e_{y0}) and specific to each slope (a_{xs} and e_{xs} , a_{ys} and e_{ys}). The paths from a_{x0} and e_{x0} to x_s and from a_{y0} and e_{y0} to y_s generate the decomposition of the correlation between the intercept and slope for X and Y .

Figure 4. Expected values of the age-based dynamics of genetic variance (top) and nonshared environmental variance (bottom) for the speed (left) and verbal (right) factors, with and without full coupling.

Figure 5.

Expected values of the age-based dynamics of genetic variance (top) and nonshared environmental variance (bottom) for the speed (left) and spatial (right) factors, with and without full coupling.

Figure 6.

Expected values of the age-based dynamics of genetic variance (top) and nonshared environmental variance (bottom) for the speed (left) and memory (right) factors, with and without full coupling.

Table 1

Range in Number of Complete Pairs (and Incomplete Pairs) in Each 3-Year Age Interval as a Function of Age at Assessment

Age interval	Twin type				Total individuals
	MZA	MZT	DZA	DZT	
50–52.9	4 (2)	10 (0)	16 (4)–18 (2)	11 (2)–12 (1)	91–92
53–55.9	9 (3)–10 (2)	17 (2)–18 (1)	22 (8)–23 (6)	22 (4)–24 (2)	158–160
56–58.9	12 (6)–13 (5)	17 (4)	28 (15)–29 (15)	19 (8)–22 (5)	186–189
59–61.9	14 (6)–15 (5)	19 (7)–20 (6)	40 (6)–42 (5)	21 (4)–22 (3)	212–217
62–64.9	12 (7)–13 (6)	30 (4)–31 (3)	36 (23)–40 (20)	26 (15)–28 1(13)	261–264
65–67.9	15 (8)–16 (8)	33 (10)–36 (8)	44 (24)–46 (23)	30 (16)–34 (12)	301–315
68–70.9	15 (5)–16 (4)	27 (9)–29 (8)	33 (29)–37 (26)	33 (15)–35 (14)	274–282
71–73.9	9 (11)–11 (10)	22 (10)–23 (8)	35 (28)–40 (23)	27 (21)–31 (17)	260–267
74–76.9	10 (13)–13 (10)	19 (9)–22 (9)	27 (22)–31 (20)	30 (17)–35 (13)	233–253
77–79.9	8 (8)–9 (7)	11 (12)–13 (11)	9 (18)–14 (20)	15 (22)–19 (21)	147–168
80–82.9	6 (6)–10 (3)	5 (9)–7 (11)	5 (13)–9 (16)	9 (18)–11 (19)	98–123
83–85.9	2 (1)–3 (3)	2 (8)–2 (9)	1 (11)–3 (16)	1 (12)–4 (9)	44–63
86–88.9	1 (3)–1 (5)	0 (5)–1 (8)	0 (4)–2 (7)	1 (8)–2 (10)	25–42

Note. Range in sample size is presented because the number of data points varied for each cognitive factor.

MZA = monozygotic twins reared apart; MZT = monozygotic twins reared together; DZA = dizygotic twins reared apart; DZT = dizygotic twins reared together.

Table 2

Parameter Estimates (and 95% Confidence Intervals) From Fitting the Univariate Biometric Dual Change Score Model to the Four Latent Factors

Parameters and fit	Verbal factor	Spatial factor	Memory factor	Speed factor
Fixed effects				
Mean intercept, μ_0	52.76 (51.89, 53.65)	55.00 (54.00, 56.04)	53.96 (52.86, 55.18)	57.19 (56.19, 58.20)
Mean slope, μ_s	-17.39 (-24.14, -11.71)	-11.43 (-14.02, -9.06)	-17.56 (-23.61, -11.81)	-8.39 (-10.04, -6.82)
Constant change, α	= 1	=1	=1	=1
Proportional change, β	0.33 (0.22, 0.46)	0.20 (0.15, 0.25)	0.32 (0.21, 0.44)	0.13 (0.10, 0.16)
Random effects				
Intercept deviation, σ_0				
Additive genetic	8.54 (7.72, 9.25)	8.89 (7.64, 9.66)	8.60 (6.85, 9.92)	7.18 (5.55, 8.07)
Shared environment	0.48 (-2.54, 2.54)	0.44 (-3.00, 3.00)	1.26 (-3.55, 3.55)	1.89 (-4.04, 4.04)
Correlated environment	<.01 (-3.25, 3.25)	<.01 (-4.44, 4.44)	3.51 (-6.26, 6.26)	<.01 (-4.29, 4.29)
Nonshared environment	3.51 (2.88, 4.23)	2.57 (1.13, 3.56)	3.21 (2.12, 4.38)	2.98 (1.76, 4.06)
Slope deviation, σ_s				
Additive genetic	0.07 (-0.18, 0.18)	<.01 (-0.18, 0.18)	<.01 (-0.09, 0.09)	<.01 (-0.30, 0.30)
Shared environment	<.01 (-0.14, 0.14)	<.01 (-0.19, 0.19)	<.01 (-0.13, 0.13)	0.16 (-0.31, 0.31)
Correlated environment	<.01 (-0.10, 0.10)	<.01 (-0.15, 0.15)	<.01 (-0.09, 0.09)	<.01 (-0.21, 0.21)
Nonshared environment	0.07 (-0.15, 0.15)	0.22 (-0.30, 0.30)	0.04 (-0.13, 0.13)	0.24 (-0.33, 0.33)
Slope on level				
Additive genetic	-2.82 (-3.94, -1.91)	-1.86 (-2.34, -1.39)	-2.83 (-3.90, -1.79)	-0.94 (-1.28, -0.55)
Shared environment	-0.11 (-0.90, 0.90)	-0.02 (-0.74, 0.74)	-0.37 (-1.26, 1.26)	-0.46 (-0.84, 0.84)
Correlated environment	<.01 (-1.16, 1.16)	<.01 (-1.02, 1.02)	-1.15 (-2.32, 2.32)	<.01 (-0.74, 0.74)
Nonshared environment	-1.18 (-1.66, -0.78)	-0.58 (-0.86, -0.05)	-1.04 (-1.58, -0.58)	-0.53 (-0.80, -0.13)
Error deviation, σ_u	8.71 (8.01, 9.48)	15.41 (14.23, 16.71)	26.37 (24.36, 28.53)	15.76 (14.55, 17.09)
Misfit index: -2LL	14,499	14,837	15,886	15,590
Degrees of freedom	2,356	2,280	2,316	2,416

Table 3

Model-Fitting Results for Univariate Biometric Dual Change Score Model

Models	Parameters	Overall misfit ($-2LL$)			
		Verbal	Spatial	Memory	Speed
1. Full model	18	14,499	14,837	15,886	15,590
2. Drop $a_0 \rightarrow y_0$	17	14,563 **	14,895 **	15,921 **	15,628 **
3. Drop $a_0 \rightarrow y_s$	17	14,500	14,837	15,886	15,590
4. Drop $a_0 \rightarrow y_s$	17	14,558 **	14,894 **	15,921 **	15,612 **
5. Drop S and C	12	14,499	14,838	15,889	15,593

Note. S refers to shared rearing environmental effects and C refers to correlated environmental effects.

** Change in model fit is significant at $p < .01$.

Table 4

Parameter Estimates (and 95% Confidence Intervals) From the Bivariate Biometric Dual Change Score Model: Speed and Verbal Factors

Parameters	Speed factor	Verbal factor
Fixed effects		
Mean intercept, μ_0	57.56 (56.47, 58.67)	52.73 (51.59, 53.78)
Mean slope, μ_s	-8.16 (-9.63, -6.78)	-3.71 (-7.01, -0.61)
Constant change, α	= 1	=1
Proportional change, β	0.10 (0.06, 0.13)	<.01 (-0.09, 0.09)
Coupling, γ speed $\rightarrow$ verbal		0.07 (0.04, 0.10)
Coupling, γ verbal $\rightarrow$ speed	0.03 (0.02, 0.04)	
Random effects		
Intercept deviation		
Additive genetic	7.63 (5.53, 8.61)	9.38 (8.37, 10.42)
Shared environment	2.29 (-4.45, 4.45)	0.14 (-2.83, 2.83)
Correlated environment	0.06 (-4.78, 4.78)	0.05 (-3.38, 3.38)
Nonshared environment	2.29 (0.89, 3.64)	3.86 (2.83, 5.00)
Slope deviation		
Additive genetic	0.27 (-0.45, 0.45)	0.51 (0.21, 0.80)
Shared environment	0.22 (-0.37, 0.37)	<.01 (-0.52, 0.52)
Correlated environment	<.01 (-0.25, 0.25)	<.01 (-0.29, 0.29)
Nonshared environment	0.18 (-0.32, 0.32)	0.29 (-0.53, 0.53)
Slope on level		
Additive genetic	-0.96 (-1.29, -0.44)	-0.52 (-1.13, 0.11)
Shared environment	-0.45 (-0.88, 0.88)	-0.29 (-0.60, 0.60)
Correlated environment	-0.01 (-0.80, 0.80)	<.01 (-0.37, 0.37)
Nonshared environment	-0.24 (-0.59, 0.12)	-0.23 (-0.56, 0.19)
Error deviation, σ_u	3.95 (3.80, 4.11)	2.91 (2.79, 3.04)
Misfit index: -2LL	29,903	
Degrees of freedom	4,774	

Table 5

Parameter Estimates (and 95% Confidence Intervals) From the Bivariate Biometric Dual Change Score Model: Speed and Spatial Factors

Parameters	Speed factor	Spatial factor
Fixed effects		
Mean intercept, μ_0	57.00 (56.03, 58.00)	56.00 (54.17, 57.77)
Mean slope, μ_s	-9.12 (-10.61, -7.70)	3.04 (0.59, 5.90)
Constant change, α	= 1	=1
Proportional change, β	0.13 (0.09, 0.16)	-0.73 (-0.88, -0.59)
Coupling, γ speed $\rightarrow$ spatial		0.65 (0.53, 0.78)
Coupling, γ spatial $\rightarrow$ speed	0.02 (0.01, 0.03)	
Random effects		
Intercept deviation		
Additive genetic	7.71 (6.58, 8.52)	7.46 (4.12, 9.55)
Shared environment	1.33 (-3.53, 3.53)	<.01 (-4.38, 4.38)
Correlated environment	<.01 (-3.62, 3.62)	3.19 (-7.15, 7.15)
Nonshared environment	2.94 (1.91, 3.89)	2.25 (-4.44, 4.44)
Slope deviation		
Additive genetic	<.01 (-0.30, 0.30)	<.01 (-2.37, 2.37)
Shared environment	0.12 (-0.30, 0.30)	<.01 (-1.44, 1.44)
Correlated environment	<.01 (-0.21, 0.21)	1.55 (-2.54, 2.54)
Nonshared environment	0.26 (0.19, 0.34)	<.01 (-1.85, 1.85)
Slope on level		
Additive genetic	-1.12 (-1.42, -0.81)	-3.12 (-4.23, -1.80)
Shared environment	-0.41 (-0.78, 0.78)	<.01 (-1.57, 1.57)
Correlated environment	<.01 (-0.64, 0.64)	-0.21 (-2.66, 2.66)
Nonshared environment	-0.61 (-0.84, -0.32)	-1.65 (-2.26, 2.26)
Error deviation, σ_u	3.90 (3.75, 4.05)	3.98 (3.84, 4.14)
Misfit index: -2LL	29,927	
Degrees of freedom	4,698	

Table 6

Parameter Estimates (and 95% Confidence Intervals) From the Bivariate Biometric Dual Change Score Model: Speed and Memory Factors

Parameters	Speed factor	Memory factor
Fixed effects		
Mean intercept, μ_0	56.73 (55.70, 57.77)	54.46 (52.34, 56.44)
Mean slope, μ_s	-9.34 (-11.01, -7.78)	6.20 (2.95, 11.56)
Constant change, α	= 1	=1
Proportional change, β	0.14 (0.10, 0.18)	-0.47 (-0.68, -0.34)
Coupling, γ speed $\rightarrow$ memory		0.34 (0.26, 0.47)
Coupling, γ memory $\rightarrow$ speed	0.01 (0.00, 0.02)	
Random effects		
Intercept deviation		
Additive genetic	7.82 (6.09, 8.64)	7.24 (3.09, 10.84)
Shared environment	1.59 (-3.74, 3.74)	0.73 (-5.37, 5.37)
Correlated environment	0.14 (-4.64, 4.64)	5.18 (-9.08, 9.08)
Nonshared environment	2.08 (0.81, 3.26)	4.85 (2.57, 7.13)
Slope deviation		
Additive genetic	0.18 (-0.35, 0.35)	0.81 (-2.80, 2.80)
Shared environment	0.18 (-0.31, 0.31)	<.01 (-1.73, 1.73)
Correlated environment	<.01 (-0.21, 0.21)	<.01 (-1.71, 1.71)
Nonshared environment	0.17 (-0.29, 0.29)	<.01 (-1.26, 1.26)
Slope on level		
Additive genetic	-1.23 (-1.57, -0.76)	-2.81 (1.17, 4.33)
Shared environment	-0.38 (-0.84, 0.84)	-0.85 (-1.76, 1.76)
Correlated environment	-0.02 (-0.90, 0.90)	-0.18 (-1.66, 1.66)
Nonshared environment	-0.28 (-0.63, 0.08)	-0.98 (-1.76, -0.17)
Error deviation, σ_u	3.97 (3.82, 4.13)	5.08 (4.89, 5.28)
Misfit index: -2LL		31,221
Degrees of freedom		4,734

Table 7

Model-Fitting Results for Bivariate Biometric Dual Change Score Models

Models	Parameters	Overall misfit ($-2LL$)		
		Speed and verbal	Speed and spatial	Speed and memory
1. Full model	34	29,903	29,927	31,221
2. Drop γ speed $\rightarrow$ factor	33	29,946 ^{**}	30,039 ^{**}	31,306 ^{**}
3. Drop γ factor $\rightarrow$ speed	33	29,947 ^{**}	29,938 ^{**}	31,226 [*]
4. Equate coupling	33	29,906	30,022 ^{**}	31,284 ^{**}
5. Drop all coupling	32	30,099 ^{**}	30,440 ^{**}	31,490 ^{**}
6. Drop S and C	22	29,907	29,932	31,224

Note. S refers to shared rearing environmental effects, and C refers to correlated environmental effects.

* Change in model fit is significant at $p < .05$.

** Change in model fit is significant at $p < .01$.