A previous investigation reported that cross-sectional age differences in Digit Symbol Substitution (DSS) test performance reflect declines in perceptual processing speed. Support for the tenability of the processing speed hypothesis requires examining whether longitudinal age-related change in DSS performance is largely mediated by changes in speed. The present study used data from the Victoria Longitudinal Study to examine patterns and predictors of longitudinal change in DSS for 512 older adults ($M_{age} = 68.37$ years, $SD = 7.43$). On the basis of multilevel modeling, baseline DSS performance was poorer for older participants and men, with longitudinal declines more pronounced with increasing age and decreasing speed. In contrast to the present cross-sectional findings, statistical control of change trajectories in perceptual speed using the same data did not substantially attenuate cognitive change. These discrepancies suggest different sources of variance may underlie cross-sectional age differences and longitudinal age changes for DSS.

NUMEROUS recent investigations have assessed mediational theories of age-related memory loss (see Luszcz & Bryan, 1999, for a review). Such processing resource accounts posit that age associations across many cognitive tasks are not independent, but rather reflect the influence of relatively few general factors (Salthouse, 1991, 1996). “Processing speed” has garnered particular attention as a mediator of age differences in cognitive functioning: age-related declines are attributed to a general slowing of perceptual and cognitive processes (see Birren, 1965; Salthouse, 2000). We focus on an age-sensitive behavioral indicator of the processing speed construct, “digit symbol substitution (DSS”).” Salthouse (1992) first established the DSS-processing speed relationship by partialing variance associated with simple speeded tasks requiring same and/or different judgements (e.g., are pairs of letters the same or different) from total variance in DSS performance. Consistent with the general slowing hypothesis, controlling for individual differences in the “letter comparison” and “pattern comparison” tasks attenuated over 95% of the age-related variance in DSS. These results imply considerable overlap between what the DSS test measures and processing speed (although recent findings suggest substitution coding tasks are not pure measures of cognitive speed; Piccinin & Rabbitt, 1999).

In light of age-related associations of DSS with processing speed (Salthouse, 1992) and intelligence (e.g., Wechsler, 1958), it is plausible that DSS measures processes important to the association between age and normal cognitive decline. Indeed, Salthouse noted that a “potentially fruitful research agenda might therefore involve focusing on the causes and consequences of the processes responsible for age-related variations in digit-symbol performance” (Salthouse, 1992, p. 121). Many follow-up investigations have examined DSS, and other speeded tasks, as mediators of age-related variance across various cognitive measures.

Despite mounting cross-sectional evidence for DSS’s capacity to mediate age differences in cognition, analysis of longitudinal change has been neglected. Only a few studies (e.g., Carmelli et al., 2000; Sliwinski & Buschke, 1999; Wielgos & Cunningham, 1999) have examined age-related change in DSS performance. A strong test of the processing speed hypothesis requires that proposed indicators exhibit both between- and within-person declines. It is well known that cross-sectional investigations can only approximate age change within individuals. Moreover, research has shown that cross-sectional and longitudinal designs can yield different patterns of cognitive performance with increasing age (e.g., Hultsch, Hertzog, Dixon, & Small, 1998; Schaie, 1996). Therefore, to evaluate the importance of DSS as an indicator of processing speed specifically, and the tenability of the processing speed hypothesis more generally, it is essential to replicate cross-sectional mediation using longitudinal data. If age differences in DSS performance truly reflect a general slowing of cognitive operations, it follows that statistically controlling for changes in processing speed should largely mediate age-related change in DSS performance. Of the few studies to consider DSS change, none have focused on the underlying sources of within-person age variance. Longitudinal mediation of change for other cognitive measures, including controlling for perceptual (Sliwinski & Buschke, 1999) and verbal processing (Hultsch, Hertzog, Small, McDonald-Miszczak, & Dixon, 1992) speeds, has yielded outcomes inconsistent with the general hypothesis. Specifically, partialing change in these speed measures did not substantially attenuate cognitive change. Testing within-person theories of cognitive aging using within-person data and analyses represents a critical next step in the evaluation of the processing speed hypothesis.

The purpose of the present investigation was to replicate Salthouse’s (1992) findings in a longitudinal framework. We examined (a) whether there is age-related change in DSS, and if so; (b) whether change in processing speed accounts for this age-related change. We used 6-year (3-wave) data from the Victoria Longitudinal Study (VLS). To evaluate the processing
speed hypothesis, we used multilevel modeling to compare how well a speed composite mediated age differences versus age change in DSS performance.

**Methods**

The VLS consists of longitudinal sequences in which multiple cross-sectional samples of adults are re-tested at 3-year intervals with new samples added every 6 years. This study employed data from the first three waves of Sample 2. The design, participants, measures, and procedures of the VLS have been described extensively elsewhere (Dixon et al., in press; Hultsch et al., 1998) and are only summarized here as they pertain to this report.

**Participants**

VLS participants were community-dwelling adults living in a medium-sized metropolitan area (Victoria, British Columbia, Canada) and were recruited for study through advertisements in the public media. At the first measurement wave, 512 adults (344 women and 168 men) ranging in age from 54 to 87 years ($M = 68.37, SD = 7.43$) participated in the study. More than 89% of these individuals had 12 years or more education ($M = 14.85; SD = 3.13$), more than 86% rated their general health as good or better on a 5-point scale ranging from (0) very poor to (4) very good ($M = .76, SD = .72$), and more than 92% reported 3 or fewer physician-diagnosed chronic health conditions ($M = 1.51; SD = 1.35$) from a list of 26 ailments (e.g., vision and hearing loss, stroke, arthritis, diabetes). Standardized linear age regressions indicated significant decreases for education ($ß = −.10, p < .05$) with increases in poorer self-reported health ($ß = .09, p < .05$) and greater number of chronic conditions ($ß = .26, p < .001$). At baseline, univariate analyses of variance (ANOVA) indicated more years of education for men ($M = 15.62; SD = 3.18$) than for women, $M = 14.47, SD = 3.05$, $F(1, 510) = 15.58, p < .01$, $ß^2 = .03$, with significantly more chronic conditions experienced by women ($M = 1.65, SD = 1.39$) than by men, $M = 1.21, SD = 1.23$, $F(1, 510) = 12.55$, $p < .01$, $ß^2 = .02$. There were no gender differences in age or self-reported health.

A total of 400 adults (260 women and 140 men) aged 57 to 90 years ($M = 70.21, SD = 6.82$) returned for the second occasion with 322 adults (203 women and 119 men) aged 61 to 90 years ($M = 73.39, SD = 6.85$) participating on the third occasion. Eighteen individuals did not complete the initial measurement wave and were excluded from further consideration. Of the 190 individuals who dropped out of the study after Wave 1, 29 died, 55 had personal or family health problems, 4 had memory problems, 48 were busy and/or not interested, 21 moved, and 33 could not be located. We examined attrition effects separately for (a) demographic characteristics and (b) cognitive measures using a 2 (gender) $×$ 2 (attrition status) MANCOVA, partialing the influence of chronological age. We compared baseline performance for the 322 participants who returned for all three testing waves with the 190 individuals who did not continue. For the demographic characteristics, we observed significant omnibus effects for “gender,” Wilks’s $λ = .940$, $F(3, 505) = 10.78$, $p < .001$, $ß^2 = .06$. “Attrition status,” Wilks’s $λ = .978$, $F(3, 505) = 3.82$, $p < .05$, $ß^2 = .02$; and “age” as a covariate, Wilks’s $λ = .947$, $F(3, 505) = 9.50$, $p < .001$, $ß^2 = .05$. Individuals who failed to return for subsequent waves of testing had lower levels of education, $F(1, 507) = 6.12$, $p < .05$, $ß^2 = .01$, and more chronic health problems, $F(1, 507) = 3.92$, $p < .05$, $ß^2 = .01$. Similarly, we observed significant omnibus effects for the set of cognitive variables for gender, Wilks’s $λ = .944$, $F(5, 503) = 5.93$, $p < .01$, $ß^2 = .06$. Attribution status, Wilks’s $λ = .974$, $F(5, 503) = 2.63$, $p < .05$, $ß^2 = .03$; and age as a covariate, Wilks’s $λ = .693$, $F(5, 503) = 44.48$, $p < .001$, $ß^2 = .31$. Independent of age, those who failed to return exhibited poorer cognitive performance for DSS, $F(1, 507) = 6.47$, $p < .05$, $ß^2 = .01$, and “word span,” $F(1, 507) = 8.51$, $p < .01$, $ß^2 = .02$, but not for “number comparison,” “identical pictures,” or “computation span.” Observed patterns for the nonsignificant findings were in the expected direction.

**Measures**

The VLS measurement battery consisted of multiple questionnaires, tests, and tasks assessing various domains including cognitive, perceptual, and physiological functioning. Five cognitive measures were pertinent to the present investigation.

**Digit symbol substitution.**—We administered the DSS task from the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1958) to all participants. We presented the participants with a coding key pairing nine numbers (1 through 9) with nine symbols. Printed under the coding key were rows of randomly ordered numbers with empty boxes below. We gave participants 90 seconds to transcribe as many symbols as possible into the empty boxes based on the digit–symbol associations specified in the coding key. The number of correctly completed items represented the outcome measure.

**Perceptual speed.**—We chose several well-established indicators of perceptual speed, similar to those used in previous investigations (Salthouse, 1992), from the Kit of Factor-Referenced Cognitive Tests (Ekstrom, French, Harman, & Dermen, 1976). Both tasks required participants to make simple perceptual comparisons as rapidly as possible within a limited time period. For the Identical Pictures test, participants chose the one line drawing of five that matched a target figure. For the Number Comparison test, participants were asked to decide whether two strings of digits were identical (e.g., 5167 and 5177). We scored speed as the number of correctly completed items in 90 seconds. Following Salthouse (1992), we standardized the two speed measures (wave 1: $r = .54$, wave 2: $r = .54$, wave 3: $r = .56$) across all individuals and waves, summed them within waves, and converted them to T-scores ($M = 50, SD = 10$), yielding composite processing speed measures (one per wave).

**Working memory.**—We also administered two working memory tasks developed by Salthouse and Babcock (1991). For Computation Span, participants solved arithmetic problems while holding one number from each problem in memory for later recall. For Word Span, participants listened to orally presented sentences and wrote answers to simple questions about each sentence while retaining the last word of each sentence for later recall. For both tasks, the number of items
(problems, sentences) increased from one to seven, with three trials at each series length. We scored working memory span as the highest span (1–7) correctly recalled on two out of three trials. We standardized the two measures (Wave 1: $r = .52$; Wave 2: $r = .55$; Wave 3: $r = .59$) across individuals and waves, summed them, and converted them to T-Scores ($M = 50$, $SD = 10$) to form composite working memory measures.

**Statistical Procedure**

We used hierarchical linear modeling (HLM; Bryk & Raudenbush, 1987, 1992) to examine associations between age, candidate mediators, and 6-year change in DSS performance. Relative to other statistical approaches (e.g., repeated-measures ANOVA), multilevel modeling affords several distinct advantages including (a) calculation of parameter estimates despite missing data, participant attrition, and unequal intervals between waves of testing; and (b) simultaneous assessment of between-person differences (Level 2) and within-person change (Level 1). These advantages complement our present objective to contrast the magnitude of age-variance mediated across persons at Wave 1, and within persons over time.

**Multilevel equations.**—To replicate and extend previous cross-sectional findings (Salthouse, 1992), we analyzed six primary predictors (age, health, education, gender, processing speed, and working memory) in a series of five nested HLM equations. First, we fit a fully unconditioned model (Equation 1) with DSS as the dependent measure and no predictors.

\[
\text{Level 1: } \text{DSS}_i = B_{0i} + R_{ij} \quad (1a)
\]

\[
\text{Level 2: } B_{0i} = \gamma_{00} + U_{0i} \quad (1b)
\]

This model provides a baseline against which to assess how much total DSS variance (both between and within person) was accounted for by age.

For Equation 2, we included separate cross-sectional ($x_{ij} = \text{age at baseline centered at the sample average of 68.37 years}$) and longitudinal ($\text{age}_{ij} - \text{age}_{0i} = \text{age change between baseline and a given follow-up wave}$) age predictors. At the within-subjects level (Equation 2a), the HLM equation represents a single regression equation for each individual case where DSS performance for a given individual ($i$) at a given wave ($j$) is a function of that individual’s performance at baseline testing ($B_{0i}$: the intercept) plus her/his average individual rate of DSS change per unit change in age ($B_{1i}$: the slope), plus an error term. To estimate average effects for the entire sample, individual parameters ($B_{0i}$ and $B_{1i}$) representing Level 1 model predictors became outcome measures to be predicted for Level 2 between-person equations. Equation 2b models a given individual’s baseline DSS performance ($B_{0i}$) as a function of (a) average sample DSS performance on the first wave ($\gamma_{00}$), (b) average sample difference in baseline DSS performance per year difference in age ($\gamma_{10}$), plus (c) a random effect ($U_{0i}$) estimating each person’s deviation about the mean intercept holding other predictors constant. Similarly, Equation 2c models a given individual’s rate of DSS change ($B_{1i}$) as a function of (a) average sample rate of DSS change per unit change in age ($\gamma_{10}$), plus (b) a random effect ($U_{1i}$) estimating individual deviations about the mean age slope. Contrasting the cross-sectional fixed age effect from Equation 2b ($\gamma_{10}$) with the longitudinal fixed age effect from Equation 2c ($\gamma_{10}$) yields a direct comparison of between-person age differences to within-person age change.

The third set of equations builds on the second by covarying the influence of three demographic variables. Equation 3b includes individual differences predictors for years of education (centered at the sample average of 14.85 years), self-reported health (coded as 0 = very good to 4 = very poor), and gender (coded as 0 = women and 1 = men). Estimates of education, health, and gender were also computed for Equation 3c (the age slope).

\[
\text{Level 1: } \text{DSS}_i = B_{0i} + B_{1i} \times (\text{AGE}_{ij} = \text{age}_{ij} - \text{age}_{0i}) + R_{ij} \quad (3a)
\]

\[
\text{Level 2: } B_{0i} = \gamma_{00} + \gamma_{01} \times (\text{AGE}_{ij} = x_{ij}) + \gamma_{02} \times (\text{EDUC}) + \gamma_{03} \times (\text{HEALTH}) + \gamma_{04} \times (\text{GENDER}) + U_{0i} \quad (3b)
\]

\[
B_{1i} = \gamma_{10} + U_{1i} \quad (3c)
\]

However, these covariates were not of primary interest nor were they included in the final model because Level 2 predictors cannot explain within-person DSS change (only between-person differences in change). For all models, we retained only significant covariates as predictors of DSS performance.

Equation 4 augments Equation 3 by including separate cross-sectional ($x_{2i} = \text{speed at baseline mean centered at 50 T-score units}$) and longitudinal ($\text{speed}_{ij} - \text{speed}_{0i} = \text{speed change between baseline and follow-up wave}$) predictors of processing speed in addition to the corresponding age effects and covariates. Equation 4a models individual DSS change as a function of baseline DSS performance ($B_{0i}$), average rate of DSS change per unit change in age adjusting for change in processing speed ($B_{1i}$), average rate of DSS change per unit change in processing speed adjusting for age change ($B_{2i}$), plus an error term. Equations 4b through 4d have similar interpretations to those already outlined where $\gamma_{10}$ and $\gamma_{10}$ respectively represent the cross-sectional age and speed effects, with $\gamma_{10}$ and $\gamma_{20}$ reflecting the longitudinal age and speed slopes (see Appendix, Note).

\[
\text{Level 1: } \text{DSS}_i = B_{0i} + B_{1i} \times (\text{AGE}_{ij} = \text{age}_{ij} - \text{age}_{0i}) + B_{2i} \times (\text{SPEED}_{ij} = \text{speed}_{ij} - \text{speed}_{0i}) + R_{ij} \quad (4a)
\]

\[
\text{Level 2: } B_{0i} = \gamma_{00} + \gamma_{01} \times (\text{AGE}_{ij} = x_{ij}) + \gamma_{02} \times (\text{SPEED}_{ij} = x_{2ij}) + \gamma_{03} \times (\text{EDUC}) + \gamma_{04} \times (\text{HEALTH}) + \gamma_{05} \times (\text{GENDER}) + U_{0i} \quad (4b)
\]

\[
B_{1i} = \gamma_{10} + U_{1i} \quad (4c)
\]

\[
B_{2i} = \gamma_{20} \quad (4d)
\]

We modeled a fifth set of equations, identical to Equations 4a through d, incorporating cross-sectional ($x_{2i}$ = working memory...
at baseline mean centered at 50 T-score units) and longitudinal (working memory_{ij} – working memory_{i0} = working memory change between baseline and follow-up wave) predictors of working memory in lieu of processing speed.

**Calculating percentage of age variance attenuated.** Multi-level analyses assessing mediators of age differences and change must necessarily focus on fixed age effects (Sliwinski & Buschke, 1999). Explaining variance associated with the longitudinal fixed age effect (γ_{10j} from Equation 2c) requires examining time-varying (i.e., Level 1) covariates that change within persons. Similarly, explaining variance associated with the cross-sectional fixed age effect (γ_{01j} from Equation 2b) requires examining between-person (i.e., Level 2) individual differences at baseline. To compare the relative influence of mediators, differences between corresponding cross-sectional and longitudinal fixed age effects are compared before and after partialing covariates of interest. For example, comparing fixed age effects at cross-section, both before (γ_{01i}; Equation 2b) and after (γ_{01i}; Equation 4b) partialing individual differences in processing speed, yields the importance of speed as a mediator of age differences. Similarly, comparing longitudinal age effects before (γ_{10i}; Equation 2c) and after (γ_{10i}; Equation 4c) partialing change in processing speed reflects the importance of speed as a mediator of age change. The percentage of age variance attenuated, both at cross-section and over time, is calculated as the ratio of the **indirect age effect** (i.e., total age effect minus direct age effect after partialing the mediator) to the total age effect multiplied by 100 (see Salthouse, 1992; Sliwinski & Buschke, 1999).

**RESULTS**

We reported results in three sections. First, we examined cross-sectional age differences for DSS performance. Second, we partitioned age-related variability in DSS performance into between- and within-person sources. Finally, we fit multilevel models to simultaneously examine age differences and change in DSS, including potential predictors underlying change.

**Accounting for Age Differences in Digit Symbol Performance**

Table 1 summarizes hierarchical multiple regression analyses examining age-DSS associations before and after covarying other predictors. As expected, observed patterns paralleled those of Salthouse (1992). Age differences accounted for 23.2% of the total variance in DSS (compared with 28.7% reported by Salthouse, 1992). We calculated the percentage of age-related variance mediated by the predictors by comparing the ratio of indirect to total age effects. Total age-related variance in DSS was attenuated by (a) 9.48% for education and self-reported health (compared with 4.90%), (b) 33.81% for working memory (compared with 64.47%), and (c) 90.95% for processing speed (compared with 96.7%).

Consistent with Salthouse (1992), our results suggested age differences in DSS largely reflected an age-related slowing of processing speed. However, these cross-sectional findings focus solely on individual differences at a single point in time. An untested assumption is that between-person variance and covariance estimates will approximate within-person variance and covariance estimates (for further discussion, see Hofer & Sliwinski, 2001; Hofer, Sliwinski, & Flaherty, 2002; Sliwinski & Hofer, 1999). If age differences in DSS reflect underlying differences in processing speed, it follows that partialing change in speed should largely attenuate age-related DSS change. This is an important test given that the speed hypothesis posits that between-person variance and covariance estimates (for further discussion, see Hofer & Sliwinski, 2001; Hofer, Sliwinski, & Flaherty, 2002; Sliwinski & Hofer, 1999). If age differences in DSS reflect underlying differences in processing speed, it follows that partialing change in speed should largely attenuate age-related DSS change.

**Partitioning Age-Related Variance in Digit Symbol Performance**

Multilevel modeling permits partitioning of total age-related DSS variance (Var DSS) into constituent between- (τ: Var U_{ij} + Var U_{i0}) and within-person (σ^2: Var R_{ij}) components. We calculated the amount of variance associated with cross-sectional and longitudinal age effects by contrasting variance estimates from Equations 1 and 2. When estimates from Equation 1 are used (τ = 105.115 and σ^2 = 23.897), the intraclass correlation (τ/τ + σ^2) indicated that 81.48% of total DSS variance was due to between-person differences whereas 18.52% (1 − .8148) reflected within-person differences over time. Comparing these estimates to corresponding age-covaried estimates from Equation 2 (τ̂ = 85.146; τ̂ = 0.55751; σ^2̂ = 17.372) yielded the amount of (a) total between-person DSS variance accounted for by Wave 1 age differences (18.47%) and (b) total...
within-person DSS variance accounted for by longitudinal age change (27.30%). Notably, percentage of variance attenuated represents a separate index of model fit for the cross-sectional and longitudinal age trends (Sliwinski & Buschke, 1999). Our longitudinal age change model fit the within-person data comparatively better than the cross-sectional age difference model fit the between-person data. Together, both age effects accounted for 20.10% of total DSS variance.

**Accounting for Age Differences and Change in Digit Symbol Performance**

Following variance partitioning, we examined what proportion of between- and within-person age-related variance estimates were mediated by select Level 1 and Level 2 predictors. Specifically, we used HLM 5.0 to examine the effectiveness of processing speed and working memory as mediators of cross-sectional and longitudinal age effects. Of particular interest was whether processing speed attenuated similar amounts of between- and within-person variance, as predicted by the processing speed hypothesis. Each row of Table 3 summarizes fixed effect coefficients associated with specific mixed models (Equations 2 through 5). All models included a random intercept, a random age slope, and a fixed mediator slope (see Appendix, Note). In all cases, the likelihood ratio test indicated significant improvement in model fit with the addition of random effects for intercept and age slope.

Table 3 Row 1 (Age) reflects the cross-sectional and longitudinal age coefficients derived using Equation 2. As expected, we observed significant age differences at Wave 1 and age-related declines across all 3 waves for DSS performance. At cross-section, for every additional year of age above the sample average, there was a corresponding 0.738 unit decrease for DSS performance (a DSS unit reflects 1 correct raw score response). Over time, each additional year of age change from baseline was associated with an average individual declining 0.539 DSS units. Unstandardized estimates of cross-sectional age differences (spanning 54 to 87 years) were slightly larger than estimates of age change over the longitudinal interval (M = 6.63 years). To compare these values, we obtained standardized estimates by dividing each fixed age effect by its corresponding standard deviation (Sliwinski & Buschke, 1999). The standardized between-subject estimate (−0.738/0.7467 = −0.988) was larger than its within-subject counterpart (−0.539/0.168 = −0.329), indicating that cross-sectional age effects overestimated actual longitudinal DSS change.

In the second row of Table 3 (Age/Demographic), we used Equation 3 mixed models to estimate cross-sectional and longitudinal age effects after partialing self-reported health and gender (education was not significant, so we omitted it from all final models). We observed significant gender differences, holding age and self-reported health constant, for Wave 1 DSS performance with the average woman (52.46) performing slightly higher than the average man (52.46 − 2.81 = 49.65). In addition, those who reported their health as very good (52.46) performed better than those who reported their health as good or lower (52.46 − 1.98 = 50.48). Age differences and change in DSS performance remained significant after we partialed the demographic measures. We derived variance attenuation estimates at cross-section by comparing the respective indirect with total age effects [(−0.738) − (−0.729)]/−0.738; 1.22% of between-subject age-related variance was accounted for by individual differences in self-reported health and gender. As noted, longitudinal age-related variance cannot be accounted for by these Level 2 covariates; only Level 1 mediators can account for DSS change. We found no evidence suggesting rates of age change for DSS varied as a function of gender or health.

We summarized mixed model results from Equation 4 in Table 3 Row 3 (Age/PS). This equation assesses the magnitude of age effects after we partialed the cross-sectional and longitudinal processing speed predictors. Both speed at Wave 1 and change in speed were significantly associated with DSS performance, even subsequent to controlling for corresponding...
age effects. Notably, significant age differences remained after we partialed speed at Wave 1, with adjustments for change in speed also failing to fully attenuate longitudinal age change in DSS performance. The focus of this investigation was to examine whether change in speed accounted for similar proportions of age-related change variance in DSS performance relative to between-subject findings. Independent of health and gender effects, 65.57% of the cross-sectional age-related variance was accounted for by between-person differences in processing speed. In addition, partialed speed from DSS performance at baseline (a) eliminated significant gender differences, attenuating 55.16% of the Level 2 gender-related variance; and (b) attenuated 44.95% of the Level 2 health-related variance. In contrast, with no additional Level 1 covariates, only 20.96% of the longitudinal age effect was accounted for by within-person changes in processing speed. There were no gender or health differences for the speed slopes (i.e., no between-person differences in within-person change). Relative to both the present and previous (Salthouse, 1992) cross-sectional attenuation findings, considerably less age-related change variance for DSS was mediated by changes in processing speed.

We summarized results from Equation 5 in Table 3 Row 4 (Age/WM), where we examined the respective age effects after we partialed cross-sectional and longitudinal predictors of working memory. When we controlled for between- and within-person age effects, DSS performance shared significant associations with both Wave 1 level and change in working memory. Similarly, both cross-sectional and longitudinal fixed age effects remained significant subsequent to our controlling for individual differences and change in working memory. Compared with processing speed, working memory was a markedly poorer mediator of DSS age differences and change. When we controlled for health and gender, 16.74% of the baseline age effect was accounted for by between-person differences in working memory. Our partialing of working memory exerted little effect on gender and health differences. Comparatively, only 0.93% of the longitudinal age effect was accounted for by within-person change in working memory. Consistent with our speed mediation findings, considerably less age-related change variance in DSS was mediated by changes in working memory relative to between-person age variance accounted for by Wave 1 individual differences in working memory.

**Discussion**

The focus of this investigation was to replicate and extend Salthouse’s (1992) analysis of the DSS-processing speed association. As expected, hierarchical regression results replicate previous cross-sectional findings: Processing speed is the most influential mediator attenuating over 90% of age differences in DSS. The divergence in magnitude of age-DSS associations (23.2% vs. 28.7%) is likely a function of the restricted range in our sample (54 to 87 years vs. 18 to 84 years).

Although the HLM results demonstrate both significant age differences and age change, several important dissociations distinguish the multilevel between- versus within-person age effects. First, age change accounts for a larger proportion of total within-person DSS variance relative to between-person variance accounted for by age differences; this pattern reflects better model fit for the longitudinal data. Second, comparison of standardized coefficients indicates the between-subject fixed age effect overestimated the amount of actual within-person DSS decline. In contrast to our findings, Wielgos and Cunningham (1999) reported that 7-year DSS change closely approximated age differences. Observed discrepancies likely reflect the design of the study and comparison technique used. Whereas Wielgos and Cunningham (1999) compared cross-sectional and longitudinal age trajectories by examining plotted comparisons (see Figure 3, p. 116), we contrasted standardized between- and within-person fixed age effects. Notably, other findings have reported that the age change effect for DSS is larger than the age difference effect (Sliwinski & Buschke, 1999). In part, this dissimilarity may reflect the smaller number of occasions present in our data (e.g., we could not assess the quadratic age effect for a given follow-up period).

The third and most important dissociation distinguishing the between- versus within-person age effects pertains to Level 1 and Level 2 mediation of age variance. Revisiting the question of what age differences and change in DSS performance reflect, we assessed the relative importance of processing speed and working memory as mediators of these separate age effects. The cross-sectional age estimate is markedly reduced after we partial Wave 1 differences in speed, although age differences remain significant. Of all baseline mediators of DSS performance, speed clearly attenuates the most age-related variance. However, contrary to postulates of the processing speed hypothesis, statistically controlling for within-person change in speed fails to mediate sizeable proportions of DSS age change variance. Moreover, a consistent dissociation emerges when contrasting variance attenuated for the separate age effects: within-person change is attenuated less than between-person differences after we control for respective variance in perceptual speed or working memory. Of total DSS-age variance mediated, speed is the better predictor, accounting for more age differences (over 3 times more variance attenuated) and change (over 22 times more variance attenuated).

In contrast to present and prior (Salthouse, 1992) cross-sectional results, our longitudinal mediation findings do not support the interpretation that age declines for DSS reflect declines in processing speed. These opposing findings beget an important follow-up question: Why the discrepancy between predictors of age differences and age changes in DSS performance? One possibility is that different sources of variance underlie these separate age effects. Indeed, estimates of between-person variance and covariance may not closely approximate corresponding within-person estimates. At the between-subjects level, for example, variance shared between measures of processing speed and cognition may be spuriously related to age (Sliwinski & Buschke, 1999). In studies examining speed mediation of age differences, age-related variance typically is partitioned into direct (i.e., unique) and indirect (i.e., speed mediated) components, resulting in very little unique age variance remaining to explain. A critical caveat of this approach is that estimates of attenuated age variance are a function of both age-related and non-age-related associations between the dependent and mediator variables (for further discussion, see Hertzog, Hultsch, Dixon, & Small, 2002; Lindenberger & Pöpper, 1998; Sliwinski & Hofer, 1999). Consequently, cross-sectional findings depicting processing speed as a pivotal mediator of age-related variance for DSS, or any cognitive measure, may largely reflect stable associations be-
b tween variance sources orthogonal to age. In contrast, at the within-subjects level, spurious associations between dependent measures, mediators, and age do not influence longitudinal outcomes, which subsequently yield less biased effects for mediators (Sliwinski & Buschke, 1999). This may account for the observed discrepancy between partialing average between-group differences versus within-individual change.

Given that few longitudinal investigations have examined DSS performance, as well as the fact that this study and that of Sliwinski and Buschke (1999) have reported discrepancies between longitudinal and cross-sectional analyses of the same data, further longitudinal tests of the processing speed hypothesis should be conducted. These should consider not only DSS, but other process measures of cognition. Several caveats are associated with the present findings. First, future studies should include more than three measurement waves for assessing Level 1 mediators such as change in processing speed. Additional waves permit (a) more reliable estimates of change, (b) more precise examination of the shape of change (e.g., polynomial Level 1 age effects), and (c) more powerful assessment of whether rates of age-related slowing vary as a function of individual differences (e.g., gender, health). Second, additional within-person mediators of DSS change should be examined. Although speed and working memory do not account for the preponderance of age-related declines in the present study, other theoretically relevant Level 1 mediators should be assessed. Possible time-varying candidates include markers of biological age such as sensory and physiological function (e.g., Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994). Relative to using cognitive measures (e.g., processing speed) as mediators of age-cognition associations, such biological performance indicators are less subject to circularity criticisms (e.g., Anstey, 1999; Anstey & Smith, 1999; MacDonald, Dixon, Cohen, & Hazlitt, in press).

In summary, although the actual or definitive causes of DSS performance declines with increasing age are still unknown, it is apparent that processing speed may not represent the most rudimentary cause. The appeal of the speed hypothesis lies in its parsimony; identifying a general mediating factor would be of considerable scientific value. Nevertheless, the present results and those from other recent investigations (e.g., Hultsch et al., 1998; Sliwinski & Buschke, 1999) suggest it is premature to generalize the processing speed account to explanations of actual cognitive change. Although many investigations have adopted the age variance partitioning approach, cross-sectional estimates of variance attenuated may be spurious. Consequently, cognitive aging hypotheses and theories derived from these findings may not generalize to actual within-person phenomena. Future efforts should seek convergent findings exploring the same issues in both cross-sectional and longitudinal designs. In particular, it appears prudent to explore further the role of speed as a mediator of age-related cognitive decline in a longitudinal framework.

Acknowledgments

The Victoria Longitudinal Study (VLS) is funded by a grant from the National Institute on Aging (AG08235) to Roger Dixon. Stuart MacDonald’s participation was supported by a postgraduate scholarship from the Natural Sciences and Engineering Research Council of Canada. We thank the volunteer participants of the VLS for their time and effort and the VLS research assistants for their invaluable assistance in data collection and preparation.

Address correspondence to Stuart W. S. MacDonald, Stockholm Gerontology Research Center, Olofssonovägen 4, Box 6401, S-113 82 Stockholm, Sweden. E-mail: stuart.macdonald@neurotec.ki.se. Address correspondence regarding the Victoria Longitudinal Study to Roger A. Dixon at rdixon@ualberta.ca.

References


Received April 26, 2001
Accepted December 9, 2002

Appendix

Note

Given only three occasions of measurement, there were no remaining degrees of freedom to estimate $\sigma^2$ for Equation 4d so we omitted the random effect associated with processing speed ($U_{ij}$) from the model. Of note, the fixed effect estimates are identical regardless of whether we estimated associated random coefficients; however, we may have underestimated the associated standard errors thereby increasing the probability of Type I errors (Sliwinski & Buschke, 1999). We computed an equivalent model where we estimated the random effect for speed by fixing the $\sigma^2$ estimate equal to a constant (17.25344) derived from an earlier model where the random coefficient for speed was fixed. Results indicated that, irrespective of fixing the speed slope, change in speed was significantly related to change in DSS independent of age change.