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Modeling Intraindividual Cognitive Change in Aging Adults: Results from the Einstein Aging Studies

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ABSTRACT

This paper reviews research from the longitudinal Einstein Aging Studies (EAS) that has focused on modeling intraindividual cognitive change in aging adults. We describe how to separate models of intraindividual change at the within-person level from models of individual differences in change at the between-person level. We illustrate this distinction by analyzing new data from the EAS to test the speed hypothesis at the intraindividual level of analyses. The present findings replicate those of Sliwinski and Buschke (1999) by showing that within-person changes in speed predict within-person changes in cognition, but that speed does not substantially attenuate estimates of within-person cognitive decline. We conclude that correct measurement and explanatory modeling of intraindividual change should be the primary focus of longitudinal aging research, and that the more common practice of modeling individual differences in change should be a secondary focus.

Longitudinal studies of cognitive aging provide the opportunity to measure and model changes in cognitive function exhibited by aging individuals. Because cross-sectional comparisons of individual differences in age and cognition cannot unambiguously disentangle variability caused by aging versus stable individual differences characteristics (Lindenberger & Potter, 1998), longitudinal studies provide a unique advantage over cross-sectional designs. Baltes and Nesselroade (1979) enumerated the following five rationales for longitudinal research:

1. Direct identification of intraindividual change
2. Direct identification of interindividual differences in intraindividual change
3. Analysis of interrelationships in behavioral change
4. Analysis of causes of intraindividual change
5. Analysis of causes of interindividual differences in intraindividual change.

The first two rationales pertain to the correct measurement of change, while the remaining three pertain to modeling (i.e., explaining) change. These rationales clarify an often neglected complexity of longitudinal research, namely the possibility (or, as we will argue, the necessity) of measuring and modeling change at two levels of analysis. The first level of analysis is the intraindividual (within-person) level, and the second level of analysis if the interindividual (between-person) level. The main thesis of this paper is that correct measurement and explanatory modeling of intraindividual change should be the primary focus of longitudinal research, and that the more

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common practice of modeling individual differences in change should be a secondary focus. To support this proposition, we review some research from the longitudinal Einstein Aging Studies (EAS), and present some new analyses of these data.

The EAS represent a merging of three cohorts of older adults that have been followed longitudinally. The first cohort of individuals were recruited (from 1980 to 1982) as part of the Bronx Aging Study (BAS) (e.g., Hall, Lipton, Sliwinski, & Stewart, 2000; Hall et al., 2001, 2002; Masur et al., 1994; Sliwinski et al., 1996; Sliwinski & Buschke, 1997; Sliwinski, Hofer, Hall, Buschke, & Lipton, in press; Verghese et al., 2000, in press) with the purpose of studying rates and predictors of incident dementia and the neuropathological correlates of cognitive function. A second cohort of individuals was recruited (from 1986 to 1990) as part of the Teaching Nursing Home program project (e.g., Grober, Lipton, Hall, & Crystal, 2000; Sliwinski & Buschke, 1999). The aims of this study were quite similar to that of the BAS. In 1992 these two projects were merged and combined with a third sample that was recruited to continue research on developing methods for early detection and screening of dementia (Buschke, Sliwinski, Kuslansky, & Lipton, 1997; Buschke et al., 1999), testing cognitive aging theories (Buschke, Sliwinski, Kuslansky, & Lipton, 1995; Sliwinski & Buschke, 1997), and neuropathologic correlates. The new analyses presented in this paper use data from the more recent 1992 EAS sample. The purpose of these analyses is to replicate the findings from Sliwinski and Buschke by (1) demonstrating within-person associations between changes in speed and memory, and by (2) demonstrating that within-person changes in speed do not account for within-person memory decline. We will also conduct a formal test for the equivalence of within-person and between-person effects (i.e., to test whether the effects are 'ergodic'; Nesselroade & Molenaar, 1999; Sliwinski & Hofer, 1999).

The following review of research from the EAS will encompass work that has attempted to shift the focus of longitudinal analyses from the examination of individual differences in rates of change to more detailed modeling of intraindividual change and its predictors. In the first section we describe how to formulate predictions at both the within-person level and at the between-person level of analysis. In the second section we integrate recent methodological developments in modeling change with theoretical distinctions between normative and nonnormative aging processes (Baltes & Nesselroade, 1979) that determine cognitive decline. In the third section we explore some of the difficulties of modeling individual differences in rates of change when the model for intraindividual change is incorrect or incomplete. Then, we propose an alternative method for testing theoretical predictions using longitudinal data and describe new analyses that extend the ideas covered in the introduction by illustrating how to test whether aging effects and the effects of explanatory variables are ergodic.

DISTINGUISHING WITHIN-PERSON AND BETWEEN-PERSON ANALYSIS OF CHANGE

Although cognitive aging theories seek to explain why individuals lose (or maintain) cognitive function as they grow older, most theories have been evaluated only by examination of between-person age-related cognitive differences at cross-section. Longitudinal studies allow for more direct tests of aging theories by examination of within-person change as opposed to between-person age differences. However, most longitudinal analyses have focused on between-person differences in change, and have not systematically examined within-person predictors of change. In fact, hypotheses regarding predictors of change can be tested at both the within-person and between-person level of analysis, and the results need not converge (Sliwinski & Hofer, 1999) The clearest method for indicating what it means to model within-person change and between-person change is by presenting a formal statistical model for change in hierarchical or multilevel format (Raudenbush, 2001).

Suppose that each individual's status with respect to a cognitive measure, *Y*, changes with age at a constant rate, and that these rates vary randomly across the population of aging persons.

We denote each person, i ($i = 1, \ldots, M$), measured at T_i time points ($t = 1, \ldots, T_i$). According to this notation, M is the total number of persons studied, and T_i is the total number of observations for person i. We can then represent the simple linear model for intraindividual change as:

$$Y_{ti} = b_{0i} + b_{1i}\,\text{age}_{ti} + r_{ti}, \tag{1}$$

where age_{ti} is the age for person i at time t. This model represents intraindividual change as a linear function of chronological age. Two parameters are estimated, b_{0i}, and b_{1i}, which reflect the intercept and rate of age-based change for person i. Age need not be used as the basis variable for structuring intraindividual change. Instead, actual time in years from an arbitrary time point, such entry to study (e.g., Sliwinski & Buschke, 1999) or time of dropout (e.g., Sliwinski, Hofer, Hall, Buschke, & Lipton, in press) or time of disease diagnosis (e.g., Hall, Lipton, Sliwinski, & Stewart, 2000) can be used to structure intraindividual change. This within-person (Level-1) model implies a family of between-person models (Level-2) that capture random person-to-person variability in the two Level-1 parameter estimates:

$$\begin{aligned} b_{0i} &= \beta_0 + u_{0i}, \\ b_{1i} &= \beta_1 + u_{1i}. \end{aligned} \tag{2}$$

These between-person models represent each person's intercept and slope as a function of the population mean, β_0 as the population average intercept, and β_1 as the population average slope, and randomly varying unique (i.e., person specific) effects for the intercept (u_{0i}) and slope (u_{1i}). This model has been referred to as the hierarchical linear model, the random coefficients model, the multilevel model (MLM), latent curve analysis and the mixed model. Raudenbush (2001) points out that the Level-1 within-person model is analogous to the measurement model, and the Level-2 between-person model is analogous to the structural model in structural equation modeling (SEM) terminology. And like any SEM approach, the validity of any structural model requires that the underlying measurement model is correct.

By highlighting the similarity between the MLM and SEM we do not mean to imply that measurement is the only purpose of the Level-1 or within-person model, and that all explanatory modeling occurs at Level-2, the between-person level. In fact, we believe that most interesting and informative explanatory modeling of cognitive aging must occur at the within-person level. Moreover, the analysis of individual differences in rates of change at Level-2 assume that the between-person variability in rates of change (i.e., the Level-2 residual, u_{1i}) reflects individual differences in the underlying causal process producing change. We will argue that the variance of the random slopes can reflect numerous sources, such as measurement error (at Level-1) and between-group differences in average rates of change.

There are three sources of variance to explain in the MLM framework; the within-person variance (r_{ti}), the between-person variance in level (u_{0i}) and the between-person variance in rate of change (u_{1i}). It is commonly acknowledged that a variable may not predict individual differences in level and rate of change equally well, or that entirely different variables may be required to model level and rate of change. Examples in aging research have mostly involved demonstrating that although individual differences in processing speed account for age-related individual differences in level of performance, individual differences in speed do not account for individual differences in rate of cognitive change (Hultsch, Hertzog, Dixon, & Small, 1998; Hultsch et al., 1992; Zimprich & Martin, 2003). However, it is not widely recognized that variables that predict change at Level-2 (between-person) need not predict change at Level-1 (within-person), and visa versa. For example, Albert et al. (1995) demonstrated that level of educational attainment predicted individual differences in rate of cognitive change. But because level of educational attainment is constant (i.e., does not vary) within-persons (at least in older adults), educational attainment cannot predict within-person change. The same is true for variables such as gender and race.

The situation becomes more complicated when one asks whether changes in one variable predict changes in another. Again, this question can be asked at either the within-person or

between-person level (Sliwinski, Hofer, & Hall, in review). At the between-person level, correlated change implies that individuals changing faster than average on one variable are also changing faster (or slower) than average on another variables. A growing number of studies have demonstrated correlations among rates of cognitive change on different variables in aging individuals (e.g., Hultsch et al., 1998; Wilson et al., 2002; Zimprich & Martin, 2003). This approach is based on modeling smoothed (e.g., linear) deviations of individual trajectories from the population average (the u_{1i} component from Equation (2)) with time-to-time dynamics (the r_{ti} component from Equation (1)) considered as unmodeled error components. At the within-person level, correlated change implies that the relative amount of change (or variability) exhibited by an individual during a given time period on one variable would be similar to the relative change (or variability) on other variables. There are fewer examples of studies examining within-person covariability among cognitive measures (but see MacDonald, Hultsch, Strauss, & Dixon, in press; Sliwinski & Buschke, 1999; Sliwinski et al., in review).

THE IMPORTANCE OF MODELING WITHIN-PERSON COGNITIVE CHANGE

A key challenge facing cognitive aging theorists involves correct formulation of theoretical predictions in the context of longitudinal models of cognitive change. By ''correct formulation'' we mean that the Level-1 model of intraindividual change should include variables that measure putative time-varying causal processes. That is, unless our models of intraindividual change are theoretically informed, modeling individual differences in intraindividual change may not be theoretically informative. Most formulations of Level-1 models of intraindividual change are limited to include only a variable that indicates time from an event (i.e., time from birth, time from baseline) and are consequently theoretically impoverished. However, other time metrics can serve as the basis for structuring intraindividual change. The analyses of Sliwinski et al. (in press) and Hall et al. (2000) illustrate this point.

Even measurement models of change are theory laden. When analysts represent intraindividual change as a function of chronological age, they make the assumption that the underlying causal processes driving change are well-measured by age (e.g., normative aging influences, Baltes & Nesselroade, 1979). However, despite the evidence for common factor aging theories (e.g., Baltes & Lindenberger, 1997) there are likely multiple causes of cognitive loss in aging individuals. One important cause is the development and progression of dementia (Katzman & Kawas, 1994). Unfortunately, structuring change as a function of chronological age may not accurately capture the rate and form of disease related cognitive decline because individuals of the same age need not be at the same stage of their disease. This problem was illustrated in an analyses described by Sliwinski et al. (in press). They analyzed change in memory function in a sample of initially nondemented individuals that were followed for up to 15 years. Approximately 25% of individuals developed clinical dementia during the follow-up period and were classified as having 'preclinical dementia' at points in time prior to their diagnosis. Sliwinski et al. (in press) predicted that individuals with progressive dementia should exhibit more rapid memory decline than their non-dementing age-peers. However, their results failed to provide strong support for this prediction when statistical analyses showed no significant differences between the age-based decline trajectory for the nondementing and preclinical individuals. Figure 1 shows the predicted age-based trajectories for both groups. A diseased-based model was then fit to the dementing individuals that structured change according to the time at which individuals crossed clinical threshold for diagnosis. Thus, the model in Equation (1) did not use chronological age, but instead used years from diagnosis to represent memory change. The predicted trajectory for the disease-based change is shown by the open-circles in Figure 1. Re-aligning dementing individuals with respect to disease progression revealed much more rapid and accelerating decline (and resulted in better model fit). Thus, aligning individuals

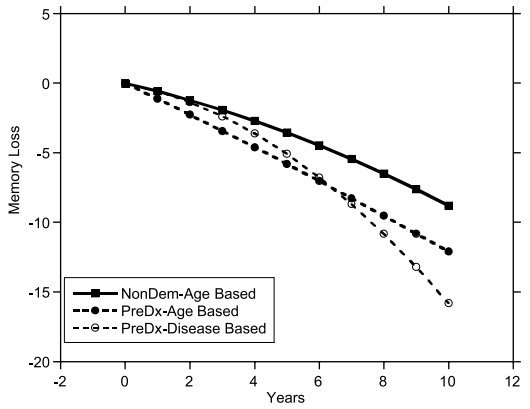

Fig. 1. Age-based memory decline nondementing and preclinical aging adults (from Sliwinski et al., in press).

with respect to their chronological age obscured the decline trajectory caused by the progression of their disease. In analyses of the same data set, Hall et al. (2000, 2001) applied a change-point method and detected a point of abrupt decline approximately 7 years prior to diagnosis that could not be detected by an age-based change model. These results imply that estimates of fixed effects (i.e., population averages) depend upon how the data are structured to represent change at the intraindividual level. We now consider how structuring the data at the intraindividual level influences analyses of individual differences in rates of change.

WHAT DO INDIVIDUAL DIFFERENCES IN RATE OF CHANGE REFLECT?

The primary focus of most longitudinal analyses of cognitive aging is on the examination of individual differences in rates of change. A number of studies have demonstrated that sublinical disease is an important predictor of differential decline (e.g., Haan, Shemanski, Jagust, Manolio, & Kuller, 1999; Rubin et al., 1998). Some studies have described the magnitude of correlations among rates of change on different cognitive measures (e.g., Wilson et al., 2002), and others have evaluated the predictions of cognitive aging theory by modeling the correlations among rates of cognitive change (e.g., Hultsch et al., 1998; Zimprich & Martin, 2003). Underlying all these analyses is the assumption that differences among individuals in rates of change reflect individual differences in underlying causal process(es). Although this assumption is undoubtedly true to some degree, between-person heterogeneity can also reflect other sources of unmeasured variation.

Between-person correlations among rates of cognitive change depend upon the specification of the Level-1 model of intraindividual change. If the model for intraindividual change is incorrect, then the correlations among between-person rates of change may not inform regarding underlying causal processes of change. Hall et al. (2000) and Sliwinski et al. (in press) showed that restructuring the time-basis for the intraindividual model for change (from chronological age to time from diagnosis) produced better fitting change models for intraindividual change in preclinical dementia. Sliwinski et al. (in review) extended this work by examining how changes in the Level-1 model of intraindividual change influences estimates of between-person variance and covariance components for rates of change.

Equation (1) is a very simple model for intraindividual change in which the rate of change is constrained to be constant over time (i.e., linear in time). We can relax this constraint somewhat by allowing the rate of change to vary over time by adding a quadratic term. In fact, there is good reason to believe that the rate of cognitive change is not constant across the life span (cf. McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; Schaie, 1996) or even across shorter time intervals in old age (Arenberg, 1982; Giambra, Arenberg, Zonderman, Kawas, & Costa, 1995; Hall et al., 2000; Sliwinski et al., in press). The failure to find evidence of accelerating change in many longitudinal studies may be a result of too few measurement occasions that are too widely spaced (e.g., Sliwinski & Buschke, 1999). However, even when demonstrations of statistically significant accelerating decline obtain, there is rarely adequate statistical power to estimate or model individual differences in quadratic effects. Therefore, linear change models are often employed because they account for the sample data as well as more complex models, even though we may acknowledge that true change in

the population is nonlinear. Consider the simple model (Eq. (1)) that represents intraindividual change as a linear function of age applied to the fictitious data from three individuals, sampled at different chronological ages (see Fig. 2a). There are individual differences in both level (i.e., intercepts) and slope (indicated by the solid lines). A typical longitudinal analysis would seek to account for individual differences in slopes by measuring a status variable at baseline (e.g., disease status, education attainment) or by estimating and correlating change slopes from other cognitive variables. These analyses are informative to the extent that individual differences in rates of change reflect individual differences in rates of change on some causal dimension that can be measured. However, person-specific (or random) slopes are in fact residuals and represent deviations from the overall average rate of change, and therefore may reflect measurement error. Consider Figure 2b that assumes we plot cognition as a function of 'true' biological aging. This graph shows the trajectories of the individuals to be perfectly parallel (i.e., there are no individual differences in change). The filled points in Figure 2b are the data plotted in Figure 2a, which represents the random sampling and the subsequent misalignment of persons with respect to the progression of an underlying process that causes change. Such misalignment produces residuals in estimated rates of change that reflect an incorrect measurement model, that is, the fallibility of chronological age (or time) as an indicator of the causal processes producing cognitive change.

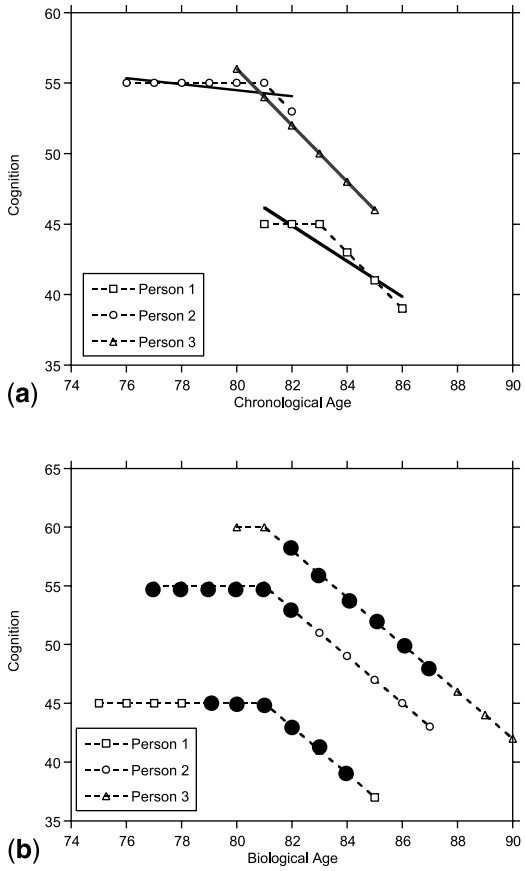

Fig. 2. (a) Hypothetical cognitive change as a function of chronological age. (b) Hypothetical cognitive change as a function of biological age.

What are the consequences of such misalignment? One consequence is that the form of the average change trajectories may not be estimated correctly (e.g., Hall et al., 2000; Sliwinski et al., in press). Even if change is perfectly linear in the population, misalignment will produce a negatively biased (i.e., biased toward zero) slope estimates in samples. (This is analogous to the error-in-predictors problem in simple regression that attenuates estimated slopes.) The consequences of misalignment can influence our interpretations of correlated rates of cognitive change as well. Sliwinski et al. (in review) examined the effects of misalignment on the correlations among slopes using data from the EAS. One finding from this analysis is that sample heterogeneity can inflate correlations among rates of change. Specifically, pooling individuals with and without preclinical dementia resulted in higher change correlations than did analysis of the nondementing group alone. This finding simply represents the effects of mixing two subgroups (nondementing and preclinical dementia individuals) with different average rates of change. Thus, the correlations in the combined sample not only represent the within-group covariation among rates of change, but also the between-group differences in average change.

A second and less intuitive finding was that realigning preclinical individuals with respect to the time from diagnosis produced better model fit

and reduced the estimated covariances among rates of change. Examination of Figure 2 suggests why this reduction in correlations might occur: realigning individuals in the Level-1 measurement model for intraindividual change can reduce (or increase) between-person variability in slopes at Level-2. Figure 3 shows the plot of trajectories for the preclinical individuals from the Sliwinski et al. (in review) data, represented as a function of chronological age (Fig. 3a) and as a function of time to diagnosis (Fig. 3b). The age-based plot suggests that change is (on average) linear, and that there is substantial heterogeneity among individuals in rate of change. However, the disease-based plot suggests that change is not constant, but instead accelerates as the time of diagnosis approaches. Moreover, there is very little visual evidence of heterogeneity in trajectories in the disease-based model, and the variance component for the slope is not significantly larger than zero. Thus, realigning individuals at Level-1 reduced the covariation among rates of change at Level-2 by reducing the between-person variation (i.e., the Level-2 residuals) in slopes. However, analysis of within-person covariation indicated strongly correlated change among the cognitive variables in the demented individuals (correlations ranging between .45 and .51). One important implication of these results is that correlations among rates of cognitive change may not inform regarding the underlying causal dimensionality of change. These correlations can reflect causal heterogeneity at the level of the sample as well as model misspecification at the level of the individual. A second important implication is that change can be correlated within individuals without rates of change being correlated in the population.

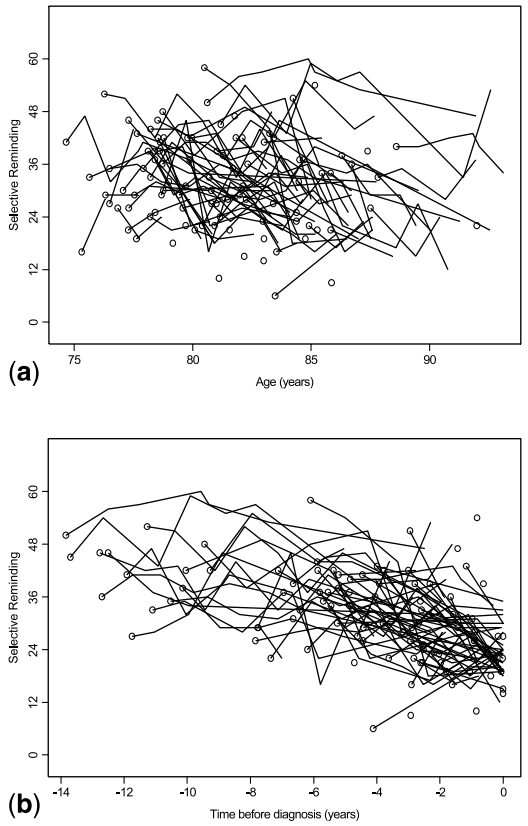

Fig. 3. (a) Age-based trajectories of memory decline for preclinical individual. (b) Disease-based trajectories of memory decline for preclinical individual.

AN ALTERNATIVE APPROACH TO MODELING COGNITIVE CHANGE

The question "Is change on two variables correlated?" is not sufficiently precise to afford a specific statistical evaluation. Change can be correlated in the population, which would result in between-person associations between the rates of change (i.e., the random slopes). This would imply that individuals who are declining more rapidly than average on one variable are also declining more rapidly than average on the other variable. Change can also be correlated in the person, which would result consistent relative change (or variability) exhibited by an individual during a given time period on each of two variables. That is, within-person covariation between two variables would indicate that scores on two variables "travel together" over time. Modeling the between-person and within-person facets of cognitive change provide complementary, but not redundant means for testing theoretical predictions using longitudinal data.

Sliwinski and Buschke (1999) provided a longitudinal test of the speed hypothesis of cognitive aging (Salthouse, 1996). Their test involved modeling within-person changes on cognitive variables

as function of within-person changes on measures of processing speed. Previous studies confirming the speed hypothesis had come from cross-sectional analyses of between-age differences and between-person covariation among cognition, speed and age. These demonstrations are quite limited for two reasons. First, Lindenberger and Potter (1998) provided a formal demonstration of the problems inherent in drawing inferences from statistical mediation analyses of developmental effects. Second, aging theories must account for aging related changes, which analyses of age differences only approximate. Therefore, Sliwinski and Buschke reasoned that the speed hypothesis must predict that statistically accounting for intraindividual change on speed should substantially reduce the estimate of within-person change. Figure 4 graphically depicts this prediction. The open squares fall on the average within-person slope for memory change before controlling on speed. The open squares show the adjusted within-person slope after controlling on within-person changes in speed. Such a result would support the contention that within-person changes in speed can account for within-person changes in memory. Testing this prediction requires a simple extension to the model described by Equations (1) and (2). The Level-1 model for intraindividual change is written as follows:

$$Y_{ti} = b_{0i} + b_{1i}(\text{age}_{ti} - \text{age}_{1i}) + b_{2i}(\text{speed}_{ti} - \text{speed}_{1i}) + r_{ti} \quad (3)$$

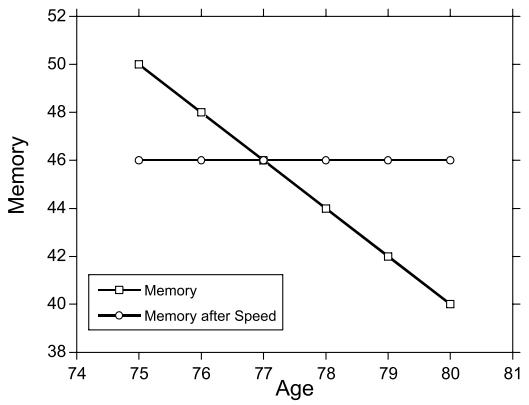

Fig. 4. Predictions of the speed hypothesis for average within-person change estimates.

This model for intraindividual change differs in two regards from the model presented in Equation (1). First, this model includes an additional variable and therefore represents within-person changes as a function of changes in age and changes in speed. The second difference is that the influence of individual differences at baseline in age and speed are removed. This adjustment is required for the Level-2 population averages to reflect pure within-person sources of variation. If the Level-1 model is not so adjusted, then the Level-2 average effects would reflect both within-person and between-person sources of variation (Snijders & Bosker, 1999). The between-person differences removed at Level-1 are put back into the model at Level-2:

$$\begin{aligned} b_{0i} &= \beta_{00} + \beta_{01}\text{age}_{1i} + \beta_{02}\text{speed}_{1i} + u_{0i}, \\ b_{1i} &= \beta_{01} + \beta_{11}\text{age}_{1i} + \beta_{12}\text{speed}_{1i} + u_{1i}, \\ b_{2i} &= \beta_{02} + u_{2i}. \end{aligned} \quad (4)$$

This Level-2 model allows the estimates of the intraindividual intercept (b_{0i}) and the intraindividual age slope (b_{1i}) to vary as a function individual differences in baseline values of age and speed. The β_{02} parameter estimates the average within-person effect of speed on within-person variation in *Y*. If we treat the response variable as memory performance, then the β_{02} parameter reflects the average within-person slope obtained by regressing memory change on speed change, after detrending on age.

We will use the model described by Equations (3) and (4) to replicate the findings of Sliwinski and Buschke (1999) in a new sample of participants in the EAS. Therefore, one purpose will be to assess to what extent intraindividual change in speed 'mediates' intraindividual change in memory. A second purpose will be to illustrate how statistical tests for the equivalence of within-person and between-person effects can be conducted in the context of MLM. We will then discuss the specific implications of these results for aging theory, and their broader implications for analysis of data from a variety of repeated measures designs.

METHOD

Participants

All participants gave informed consent approved by the Committee on Clinical Investigations of the Albert Einstein College of Medicine. Six-hundred-and-fifty community residing older adults born between 1902 and 1930 were recruited for participation. Individuals were recruited by obtaining a list from the Health Care Finance Administration (HCFA) of all adults receiving medicare in the Bronx. All individuals over the age of 65 who were residing within Bronx county were sent introduction letters describing the study and inviting them to participate. Each person received a telephone call two weeks after receiving the introduction letter and was invited to participate. Approximately 50% of all those contacted by telephone agreed to participate. To be eligible, individuals had to be fluent English and make fewer than eight errors on the Blessed mental status test (Blessed, Tomlinson, & Roth, 1968). Individuals were excluded if evidence of disturbance in consciousness, neurological disease, current psychiatric disorder, alcohol or drug dependence, endocrine or hematological disease or malignancy not in remission for more than 2 years, or current use of psychotropic or antidepressant drugs. The baseline characteristics of the 650 individuals meeting inclusion criteria are described in Table 1. Table 2 shows the number of annual measurement occasions for individuals. Of the original participants, 294 (45%) are still actively participating in the study.

Table 1. Participant Characteristics ($N = 650$, *SD* in Parentheses).

Age at baseline	78.1 (5.4)
Sex (% female)	63%
Education (years schooling)	
<6	5%
7–12	45%
12+	50%
Average Follow-up	3.5 years (1.3)

Table 2. Frequency Distribution of Maximum Follow-Up.

Testing occasion	*N* (% of sample)
Wave 1	244 (37)
Wave 2	133 (21)
Wave 3	95 (15)
Wave 4	87 (13)
Wave 5	53 (8)
Wave 6	38 (6)

Procedures

Memory

Free recall was measured using a controlled learning procedure (Buschke, 1984). Individuals were presented with a list of 16 words and had to associate each word with a category cue. After the study phase and a 20 second distracter period during which subjects had to count backwards, free recall was tested. Subjects were prompted with category cues for any word not freely recalled. This procedure was repeated three times and the score on this test was the sum of word freely recalled across the three trials (0–48).

Speed

Processing speed was assessed using the digit symbol substitution test (Wechsler, 1981). Although commonly used to indicate processing speed, performance on this test has a substantial memory component (Piccinin et al., 1999). We discuss how this might bias the following analyses in the discussion section.

Fluency

The controlled word association test was used to assess verbal fluency. Participants were required to name as many words as possible beginning with the letters F, A and S. Participants were given 90 s for each letter. The score was the total number of unique correctly retrieved words summed across the three letters.

Analyses

Data were analyzed using general linear mixed models with random coefficients (Laird & Ware, 1982). Statistical models were implemented using the PROC MIXED procedure in the Statistical Analysis System (SAS). Fixed effects were estimated to analyze average performance and change in performance on the SRT. Random effects and their variances were used to model individual differences in performance and individual differences in change. The variance component for the within-person speed effect was never significantly larger than 0, indicating no evidence for statistically significant individual differences in the within-person relationship. This does not imply that the within-person speed effect is identical for every person in the population. Rather, failure to estimate a variance component larger than 0 likely results from designs (e.g., intervals that are issues between testing waves) and power issues (e.g., too few observations). Therefore, this variance component was constrained to be 0 for all models. Baseline age was centered at 80 to facilitate

interpretation of the intercept. Alpha level was set to .05 for all significance tests.

RESULTS

The first step in modeling the change in memory, fluency and speed involves fitting separate descriptive MLMs to each of these variables. The results are displayed in Table 3. The between-person age effect (age_{1i}) was significant for all three variables, indicating that older individuals performed worse on the memory, speed and fluency measures at baseline than younger individuals. The within-person age effects ($age_{ti} - age_{1i}$) were also statistically significant, indicating that individuals' performance decreased over time. The magnitude of the within-person age effect depended upon age at baseline for all three variables ($age_{1i} \times [age_{ti} - age_{1i}]$), indicating that older individuals were declining on average faster than younger individuals. There was no evidence of accelerating within-person decline on any of the variables.

Analysis of the random effects indicated significant between-person variability in intercepts and within-person slopes. Although not central to any of our predictions, the correlations among the random intercepts and random slopes were computed. Scores at baseline were significantly correlated for all pairs of the three dependent variables: .41 for memory and speed, .54 for speed and fluency, and .32 for fluency and memory. The rates of change were also significantly correlated, .33 for memory and speed, .22 for speed and fluency, and .16 for fluency and memory.

Next, the relative magnitudes of the between-person and within-person age effects were examined for each variable. The within-person age effect was significantly larger than the between-person age effect for the memory measure (difference = 0.43, $SE = .09$). Although within-person age effects were larger than the between-person age effects for the speed and fluency measures, these differences were not statistically significant. The within-person age effect accounted for 21%, 27% and 17% of the total within-person variance in memory, speed and fluency, respectively. The between-person age effect accounted for 8%, 17% and 2% of the total between-person variability in memory, speed and fluency, respectively. Thus, the within-person aging effects were at least as large and at least as important in accounting for variance in the dependent variables as were the between-person age effects.

Table 3. Descriptive Longitudinal Models.

	Memory		Speed		Fluency	
	Par.	*SE*	Par.	*SE*	Par.	*SE*
Fixed effects						
Intercept	30.9	0.25	34.8	0.44	34.9	0.55
age_{1i}	−0.19	0.04	−0.80	0.08	−0.29	0.09
$(age_{ti} - age_{1i})$	−0.62	0.08	−0.88	0.09	−0.48	0.11
$(age_{ti} - age_{1i}) \times age_{1i}$	−0.06	0.01	−0.04	0.02	−0.04	0.02
Between-person random effects						
Var (Intercept)	26.9		103.9		161.5	
Var $(age_{ti} - age_{1i})$	0.44		0.69		0.99	
Corr (Int, Slp)	0.00		−0.02		−0.37	
Within-person random effects						
Residuals	14.85		19.3		32.6	

Note. Age is centered at 80. Par. = parameter estimate, SE = standard error. Var (Intercept) is the variance of random intercepts, Var $(age_{ti} - age_{1i})$ is the variance of random slopes, and Corr (Int, Slp) is the correlation between random slopes and intercepts. The residuals value is the residual within-person variance.

Table 4. Longitudinal Models to Test the Speed Hypothesis.

	Memory		Fluency	
	Par.	*SE*	Par.	*SE*
Fixed effects				
Intercept	23.2	0.73	12.5	1.48
age_{ti}	−0.19	0.04	−0.21	0.09
$Speed_{1i}$	0.22	0.02	0.65	0.04
$(age_{ti} - age_{1i})$	−0.52	0.08	−0.39	0.10
$(age_{ti} - age_{1i}) \times age_{1i}$	−0.06	0.01	−0.03	0.02
$(Speed_{ti} - Speed_{1i})$	0.12	0.03	0.25	0.04
Between-person random effects				
Var (Intercept)	21.3		121.5	
Var $(age_{ti} - age_{1i})$	0.35		0.82	
Corr (Int, Slp)	0.00		−0.31	
Within-person random effects				
Residuals	14.9		32.4	

Note. Age is centered at 80. Par. = parameter estimate, *SE* = standard error. Var (Intercept) is the variance of random intercepts, Var $(age_{ti} - age_{1i})$ is the variance of random slopes, and Corr (Int, Slp) is the correlation between random slopes and intercepts. The residuals value is the residual within-person variance.

The second step was to fit the model described in Equations (3) and (4) to the memory and fluency measures. The results are displayed in Table 4. For the memory measure, individual differences in speed at baseline predicted individual differences in memory, and adding baseline speed to the model reduced the effect of baseline from −0.19/year (*SE* = .04) to −0.01/year (*SE* = .04). Baseline speed predicted individual differences in fluency, and introduction of baseline speed to the model resulted in a reversal of sign of the age effect, from −0.80/year (*SE* = .08) to 0.21/year (*SE* = .09). The within-person speed effects were also statically significant for both variables, indicating that occasion-to-occasion changes (or variability) in speed predicted occasion-to-occasion changes in memory (0.12/year, *SE* = .03) and influency (0.25/year, *SE* = .04), after adjusting for mean changes over time. The within-person aging effects remained significantly greater than 0, and relatively unchanged, for both memory (−0.61/year without speed, −0.52/year after speed) and fluency (−0.49/year without speed, −.39/year after speed).

Analysis of the descriptive models revealed that the between-person and within-person age effects differed significantly for the memory, but not for the fluency or speed measures. When between-person and within-person effects do not differ, they can be combined to obtain a more precise estimate of the overall effect of interest. Such convergence models (Bell, 1953) are common in longitudinal studies (e.g., McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; Sliwinski et al., in press), but were not used here because the current emphasis was to distinguish between-person and within-person effects and not to estimate of age effects in the population. Comparisons of the magnitudes of the between-person and within-person speed effects indicated that the between-person speed effect was significantly larger for both memory (between-within difference = 0.10 [*SE* = .03]) and fluency (between-within difference = 0.40 [*SE* = .06]). The results illustrate the importance of maintaining the distinction between effects that reflect individual difference and effects that reflect intraindividual variation (Nesselroade & Molenaar, 1999; Sliwinski & Hofer, 1999), since these two types of effects may not converge on a common point estimate or measure the same underlying process.

DISCUSSION

The discussion is organized into two sections. First, we examine the implications of the present results for cognitive aging theory. And second, we describe possible applications and limitations of our analytic approach for studying the relations among variables at the within-person level.

Analysis of Within-Person Change and Cognitive Aging Theory

The present results replicate the findings of Sliwinski and Buschke (1999) in several respects. First, individual differences in speed (as measured by the digit symbol substitution test) predicted individual differences in cognitive performance at baseline. Second, the effect of individual differences in age on individual difference in cognition is greatly attenuated after statistically adjusting for speed. This finding simply illustrates the comparability of the present data with a vast number of aging studies that illustrate the importance of speed measures in predicting individual differences in cognition in age heterogeneous samples (see Salthouse, 1996 for a review). The more interesting finding was that within-person changes in speed predicted within-person changes in free recall and fluency, after adjusting for mean decline. This result and that of Sliwinski and Buschke (1999) illustrate that, as predicted by the speed hypothesis, changes in speed correlate with changes in cognition at the intraindividual level of analyses. Previous demonstrations of the association between speed and cognition have been restricted to the analysis of individual differences. Although speed does predict cognitive change within individuals, accounting for changes in speed does not explain a large percentage of the within-person age effects. Accounting for within-person changes in speed reduced the within-person age effect on free recall by 15% and on fluency by 21%. In comparison, the individual differences in speed essentially eliminated the negative between-person age effect on cognition. We do not wish to make too much of the relative attenuation of either within-person or between-person age effects because collinearity among predictors (in this case, age and speed) can complicate mediational analyses (Lindenberger & Potter, 1998).

However, the present findings do have potentially important implications for the evaluation of aging theories. The present results indicate that the relative importance of variables for predicting between-person differences in cognitive performance can differ substantially from their importance in accounting for within-person change or variation in cognition. Although it has long been recognized that between-person age differences and within-person age change can reflect different processes (e.g., period, cohort, as well as aging influences), the same principle applies to other variables and processes. In fact there are three distinct ways in which two variables can be related to each other. First, two variables can be correlated at the between-person level, which would imply consistency in the rank ordering of individuals on both variables at a specific time. In other words, between-person correlations signify the extent to which individual differences on one variable are similar to individual differences on another variable.

A second way in which two variables can be related to each other is in terms of the correlations among individual differences in rates of change. Although the focus of this analysis is on individual differences in estimates of within-person change, it is essentially a between-person level analysis. Many longitudinal analyses of cognitive aging are concerned with identifying correlated change among cognitive measures (e.g., Hultsch et al., 1998; Wilson et al., 2002; Zimprich & Martin, 2003). The motivation behind this focus on the between-person analysis of correlated change is that cognitive aging theories make predictions about how change on different variables should covary in the population as it ages. Although correct, this is not the only way to formulate predictions regarding cognitive change in aging adults. Another, though less frequently adopted approach (MacDonald et al., in press; Sliwinski & Buschke, 1999; Sliwinski et al., in press, in review) to formulating predictions regarding cognitive change is possible. This approach focuses on developing models of intraindividual change that can better predict changes within-persons, rather than differences in the rate of change between-persons. Thus, the within-person approach asks the question ‘‘Does

performance on these variables travel together over time?'' whereas the between-person approach asks the question ''Are individuals who are changing more (or less) rapidly than average on one variable also changing more (or less) rapidly than average on other variables?'' To keep the distinction clear between these two approaches, we suggest referring to the associations at the within-person level as *coupled change*, and referring to associations at the between-person level as *correlated change*.

There are some important considerations for both the analysis of correlated and coupled cognitive change. The analysis of correlated change appears to be sensitive to the specification of the Level-1 model of intraindividual change and to between-group differences in average change (Sliwinski et al., in review). We would argue that unless the intraindividual model for change includes relevant time-varying measures of psychological, physiological and social process, then the analysis of individual differences in change might be of limited value. Most models of intraindividual change are extremely simplistic, including only a single variable that measures time from some event (e.g., birth, death, the first testing occasion, diagnosis, etc.). We assume that changes in that single time variable accurately measure important changes in the underlying causal processes. We further assume that individuals at the same location of this time metric are at comparable stages of the underlying causal time-varying (e.g., aging) processes. Sliwinski et al. (in press) demonstrated this assumption is not met in the case of preclinical dementia (i.e., individuals of the same age are at different severity levels in their disease progression).

Applications and Limitations for Intraindividual Level Analyses

Our argument is that in order to optimize inferences regarding the analysis of individual differences in change, we must develop more sophisticated, theory-based models of intraindividual change. The present analyses illustrate the utility of this approach by translating the predictions of a specific hypothesis (the speed hypothesis) to the intraindividual level of analysis. One can easily imagine how other theory-driven questions could be asked and answered at the intraindividual level of analysis (e.g., do structural and functional changes in the brain predict changes in cognition; do changes in health behaviors predict loss of cognitive function, etc). Posing such questions at the intraindividual level of analysis of coupled change provides a valuable complement to the between-person analysis of correlated change. The analysis of correlated change is essential to support inferences regarding how the *population* changes as it ages. The analysis of coupled change is necessary to support inferences regarding how *individuals* change as they age. Confusing analysis of population characteristics with analysis of individual characteristics can lead to commission of the ecological fallacy (Robinson, 1950). Because many hypotheses in cognitive aging pertain to individual level inferences, modeling intraindividual cognitive change should be a more frequently adopted analytic strategy. This line of reasoning exposes a potential limitation of the mixed model approach to intraindividual analysis. The mixed model can be used to estimate average within-person effects, and hence can provide information regarding *intraindividual* variability. However, we have not used this type of model to provide *individual* level analysis. That is, the present results cannot be clearly demonstrated in any given individual, but only in the sample of individuals in the aggregate. Individual level modeling would require many more observations per individual than current longitudinal studies provide. Future longitudinal studies that provide adequate data for individual level modeling would be extremely valuable.

Although we believe that theoretical predictions should be translated to intraindividual level analyses whenever possible, there are some important considerations for modeling intraindividual level phenomena. The spacing of measurement occasions is a critical determinant of the utility of intraindividual modeling. In general, the more closely spaced the measurement occasions are, the more useful is intraindividual level analysis. The present analyses had annual measurements, but more dense measurement designs (Nesselroade, 1991) would be optimal. Moreover, a commitment to modeling intraindividual processes demands that the spacing of

measurements be determined by theory, and not just by design (e.g., power) considerations. We know that cognitive, physiological and psychological measures vary within individuals over very short temporal intervals (Hultsch, MacDonald, & Dixon, 2002; Hultsch et al., 2000). Whether variables covary moment-to-moment, day-to-day, or only over longer periods (e.g., months or years) will depend upon the underlying mechanisms that couples these variables. For example, covariability among variables over very short time intervals (e.g., day-to-day) might reflect different mechanisms than would covariability observed over yearly intervals, as in the present study.

Another consideration is the importance of adjusting for overall mean trends in order to infer the coupling of measurements within individuals. Two sequences of repeated measures can be unrelated, but appear correlated if the means of the two series are changing as a function of time. This problem is especially important in the context of aging research (e.g., Hofer & Sliwinski, 2001; Salthouse, 1985). Demonstrating associations between measures after adjusting for overall mean trends is a critical step in establishing coupled processes at the intraindividual level.

There remain more potentially serious threats to the validity of intraindividual modeling of cognitive change. Some of these threats are shared by more conventional approaches to the analysis of individual differences in change. For example, differential practice effects on cognitive tests could obscure the true covariation among tests at both between-person and within-person levels of analysis. Another potential threat derives from the small number of observations used to estimate change. The majority of individuals in the present sample had three or fewer observations, and relatively few (27%) had four or more observations. These observations are widely spaced (1 year), and therefore do not permit separation of short-term variability from long-term change. Assume that short-term variability was quite large relative to long-term change, and that in the short-term speed and memory did not covary, but that they did covary in the long term. The present analysis would, under this assumption, underestimate the magnitude of the within-person speed-memory relationship.

The possibility of uncorrected measurement error presents another potentially serious problem for using mixed models to analyze intraindividual change. What type of measurement error might this be? One type is not really measurement error, but is short-term intraindividual variability of the type that might produce low test-retest correlations. Using a measurement-burst design (Nesselroade, 1991) and aggregating across measurement occasions can readily address this type of error. For example, assume a test has an intraclass correlation of 0.60. Taking the average of six measurements would boost the reliability to 0.90. There is another type of measurement error that cannot be corrected by taking multiple measurements, namely the type that results from using fallible indicators of latent constructs. Latent variable methods are typically used to correct for fallible indicators. Although ubiquitous and accepted in psychological research, such latent variable methods carry with them strong assumptions regarding the correspondence between patterns of covariation between and within person (e.g., local homogeneity) that are by no means clearly met (Borsboom, Mellenbergh, & van Heerden, 2003). Regardless of one's philosophical position regarding statistical approaches to correcting measurement error, unmeasured sources of variability will always exist and can potentially bias results to a significant extent.

CONCLUSIONS

The present analyses replicated the findings of Sliwinski and Buschke (1999) in demonstrating that (1) within-person changes in speed predict within-person changes in cognition, but that (2) the within-person speed effect does not (statistically) explain within-person age effects. We have also argued for the importance and utility of developing more complete and theoretically informed models of intraindividual change. Because analysis of individual differences in change can proceed only after specifying a model for intraindividual change, we propose that intraindividual modeling should be the primary focus of longitudinal cognitive aging research. Simply conducting the type of analyses described in this

paper will not solve many of problems we highlighted regarding analysis of individual differences in change (e.g., sensitivity to Level-1 model specification). However, shifting emphasis toward developing more theoretically sophisticated models of intraindividual change is a necessary step in improving the theoretical value of analyses of individual differences in change. The emphasis on intraindividual modeling has been a long running theme in many longitudinal analyses of the Einstein Aging Studies. These studies have demonstrated the utility of this approach for understanding the natural history of preclinical dementia (Hall et al., 2000, 2001; Hall, Ying, Kuo, & Lipton, 2003; Sliwinski et al., in press), the influence of attrition and mortality on cognitive change (Sliwinski et al., in press), the meaning and interpretation of correlated cognitive change (Sliwinski et al., in press) and for testing predictions from cognitive aging theory. However, our attempts at developing more informative models of intraindividual change have been extremely limited. We hope this discussion will urge aging researchers to give more emphasis to the study of aging as a process that unfolds within individuals.

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