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Predicting Impending Death: Inconsistency in Speed is a Selective and Early Marker

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Abstract

Among older adults, deficits in both level and variability of speeded performance are linked to neurological impairment. This study examined whether and when speed (rate), speed (inconsistency), and traditional accuracy-based markers of cognitive performance foreshadow terminal decline and impending death. Victoria Longitudinal Study data spanning 12 years (5 waves) of measurement were assembled for 707 adults aged 59 to 95 years. Whereas 442 survivors completed all waves and relevant measures, 265 decedents participated on at least one occasion and subsequently died. Four main results were observed. First, Cox regressions evaluating the three cognitive predictors of mortality replicated previous results for cognitive accuracy predictors. Second, level (rate) of speeded performance predicted survival independent of demographic indicators, cardiovascular health, and cognitive performance level. Third, inconsistency in speed predicted survival independent of all influences combined. Fourth, follow-up random-effects models revealed increases in inconsistency in speed per year closer to death, with advancing age further moderating the accelerated growth. Hierarchical prediction patterns support the view that inconsistency in speed is an early behavioral marker of neurological dysfunction associated with impending death.

Keywords

cognitive aging; inconsistency; mortality; neurocognitive resources; speed; terminal decline

Terminal decline refers to accelerated cognitive deterioration that occurs in proximity to death among older adults (Riegel & Riegel, 1972; Small & Bäckman, 1999). Theoretically, this impending mortality phenomenon implies that some late-life cognitive deficits may not reflect primary aging (or time-since-birth) effects, but rather time-to-death effects. Clinically, the interpretable magnitude of normative (i.e., disease-free) aging-related cognitive deficits may be ambiguous if an individual is in a terminal decline phase. Overall, in contrast to typical and primary aging, older adults who are entering a phase foreshadowing death may experience detectable cognitive decrements. Accelerated decline may begin as early as 5–8 years prior to death (Berg, 1996; Bosworth & Schaie, 1999; Sliwinski et al., 2006; Wilson, Beckett, Bienias, Evans, & Bennett, 2003). Increases in mortality risk are predicted by indicators of biological

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aging (e.g., grip strength, visual acuity; Anstey, Luszcz, Giles, & Andrews, 2001), disease processes (e.g., cardiovascular disease; Hassing et al., 2002), and compromised cognitive functioning (e.g., episodic memory, verbal ability; Deeg, Hofman, & Van Zonneveld, 1990; Johansson & Zarit, 1997; Korten et al., 1999; Smits, Deeg, Kriegsman, & Schmand, 1999).

Regarding cognitive predictors of mortality, previous research has focused on associations with mean level accuracy-based behavioral performance. We extend this research by examining whether indicators of basic neurocognitive resources, including both level and inconsistency of speeded performance, predict impending death compared to the traditional cognitive accuracy measures. Theoretically, such neurocognitive resources may occupy an intermediate position on a continuum linking the neurological (and thus overall biological) substrate with complex cognitive performance. Arguably, behavioral indicators of neurocognitive resources may be parsed from other cognitive measures, reflecting their potential sensitivity to neurobiological integrity (Goldberg & Weinberger, 2004; MacDonald, Nyberg, & Bäckman, 2006). Accordingly, markers of such resources may be behavioral proxies of neural integrity and the availability of neural resources. In aging, they may empirically index early neurodegenerative changes and other neurological deficits associated with subtle changes in general biological health, on the one hand, and cognitive abilities, on the other (Dixon, in press; Dixon et al., 2007; Hultsch, Strauss, Hunter, & MacDonald, 2008; Kempler, 2004). Moreover, speed-based markers of neurocognitive resources are well-known as integral predictors of individual differences in normal cognitive aging (Hertzog, Dixon, Hultsch, & MacDonald, 2003; Luszcz & Bryan, 1999; Salthouse & Berish, 2005). As such, they may represent especially promising predictors of pathological aging, including changes in biological health, as reflected in terminal decline and death.

Measures of neurocognitive resources include (a) both cognitive and motor response components, (b) the assessment of processing speed with response latencies, and (c) the possibility of deriving indicators of both mean rate (i.e., average latency) and intraindividual variability (e.g., average inconsistency in latency). Compared to accuracy-based cognitive measures, chronometric response latencies permit a more precise index of underlying or prodromal changes in neurocognitive resources or neurobiological processes (Gazzaniga, Ivry, & Mangun, 2002; Goldberg & Weinberger, 2004; MacDonald et al., 2006). To date, the most common marker has been the central-tendency measure of level (rate) of speeded performance. Although mean rate shares a well-documented association with age-related cognitive decline and neurodegenerative disease, it has not been associated with terminal decline (e.g., Anstey, Dear, Christensen, & Jorm, 2005; Sliwinski, Hofer, Hall, Buschke, & Lipton, 2003; Thorvaldsson, Hofer, & Johansson, 2006). The lesser-used indicator is inconsistency in speeded performance. Defined as fluctuating within-person changes in behavior on speed tasks, it has been associated with impaired cognitive performance, dementia, physical status, and sample attrition (Anstey et al., 2001; Hultsch & MacDonald, 2004; MacDonald, Hultsch, & Dixon, 2003). Regarding attrition from longitudinal studies, selectively increased inconsistency for dropouts may reflect influences such as deteriorating biological health, progressive neurodegenerative disease, or impending death. The logic is as follows: If inconsistency in speeded performance represents a useful (if not unique) behavioral indicator of neurological integrity (Hultsch et al., 2008), it should also predict cognitive decline and impairment, perhaps independently of mean rate of performance (Dixon et al., 2007; Hultsch, MacDonald, & Dixon, 2002). Given that neurological integrity has been linked separately to both terminal decline (Berg, 1996) and speed (rate and inconsistency; Hultsch & MacDonald, 2004), associations between inconsistency and impending death warrant specific investigation.

We examine the relative contribution of three sets of markers of early terminal decline: (a) traditional cognitive performance (mean accuracy level), (b) cognitive speed (level or mean rate), and (c) inconsistency of cognitive speed. The latter two markers have not been previously

explored and compared as predictors of death. We used data from the Victoria Longitudinal Study (VLS), an ongoing investigation of biological, cognitive, and health-related aging. Participants completed between 1 and 5 waves of measurement, providing up to 12 years of performance data given the 3–year retest intervals. On each wave, participants performed a broad battery of tasks, including two speeded tasks that produced both level and inconsistency scores. During the 12-year period, 265 participants died.

We address three research questions. First, for each individual's respective final wave of testing, mean age and mortality group differences were examined for all measures. Second, Cox regression analyses were used to test the hypothesis that inconsistency for individuals' final waves predicted survival status either with, or independent of, mean level of general cognitive and speeded performance, cardiovascular disease, and several demographic measures. Third, multilevel random-effects models assessed the longitudinal covariation between change in speeded inconsistency and time to event, and whether this dynamic coupling was further modified by mortality status or age. The novel objective of this multilevel analysis was to identify how trajectories of inconsistency changed per year closer to event and whether they were accelerated for those who would subsequently die. The first two research questions employ a cross-sectional approach to the analysis of longitudinal survival data, with a number of factors (e.g., life-long poor performance) potentially undermining claims that lower performance for decedents reflects cognitive declines in proximity to death (Berg, 1996). The latter multilevel analyses, not previously used in this context, are required to properly assess terminal decline. Not only can they demonstrate an association between predictor and outcome but also identify the shape of change in inconsistency in relation to the death event.

Method

Participants

VLS participants are initially community-dwelling adults between 55 and 85 years of age with no serious health conditions; they are followed at 3-year retest intervals or "waves" (for details see Dixon & de Frias, 2004; Hultsch et al., 1998). We assembled data from VLS Sample 1 (Waves 1 to 5; 12 years) and VLS Sample 2 (Waves 1 to 3; 6 years). Of the 1014 baseline participants, 447 survivors and 265 confirmed decedents were identified by the censoring date (May 31, 2002). Five survivors scored below 24 on the Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) and were excluded from the sample (O'Connor et al., 1989). Decedents completed at least one wave with subsequent date of death confirmed through Vital Statistics records for the province of British Columbia, Canada. Questionable dementia was listed as an antecedent condition (not primary cause) on death certificates for 33 decedents. Although VLS ledgers confirmed that these individuals were not demented at baseline, definite dementia diagnosis and date of incidence were not available in vital status records. Analyses were computed both with and without these 33 cases; because identical patterns of statistical inference were observed, we include them in the present results (see Wilson et al., 2003). The remaining 302 original VLS participants were excluded from this study because of attrition and unconfirmed status (e.g., moved and could not be located, deceased in another province). Therefore, 707 participants (442 survivors and 265 decedents) were eligible for analyses.

Table 1 presents demographic characteristics as a function of mortality status for participants' respective final wave of testing. A cross-tabulation analysis indicated that the ratio of women to men did not vary significantly by mortality status. Compared with survivors, decedents were less educated, but reported significantly better health relative to same-aged peers. Education was partialled from all subsequent analyses. On average, decedents completed 2.0 waves of testing (range = 1–5), with approximately 5.2 years between last wave of testing and subsequent

death. Survivors completed 3.5 waves of measurement (range = 3–5), with an average of 2.79 years (SD = 0.44) from the last wave of testing to the censoring date.

Assessment of cognitive performance (level)

Traditional cognitive assessment yields information about the level of performance averaged across multiple items or trials. Three cognitive constructs (i.e., working memory, episodic memory, and semantic memory) sensitive to cognitive health, aging, and survival were selected from previous confirmatory factor analyses (Hertzog et al., 2003; Hultsch et al., 1998). Working memory was indexed by sentence construction, listening span, and computation span, all requiring storage and simultaneous processing of information. The VLS sentence construction task (see Hultsch et al., 1990) requires participants to read aloud a series of sentences, detect and maintain a highlighted word for later recall in an order that produces a new sentence. The outcome measure used was the highest number of newly formed sentences correctly recalled for 2 out of 3 passage lengths. The second task, computation span (Salthouse & Babcock, 1991) required participants to solve a series of arithmetic problems while holding the final digit from each problem in memory for later recall. The number of problems in a series increased from one to seven, with three trials at each series length. The highest span correctly recalled for two out of three trials was the measure used. For the third task, listening span (Salthouse & Babcock, 1991), participants answered questions about simple sentences that were orally presented while simultaneously trying to remember the final word of each sentence for later recall in order of presentation. Three trials were given for each series length (ranging from two to seven sentences). The outcome measure was the highest span for which target words were correctly recalled on at least two of three trials. Because the computation and listening span tasks were not included in the VLS measurement battery until 1989, Sample 1 participants who died prior to the second wave of testing (n = 72) did not have scores for these measures. All relevant analyses for these two tasks were computed for n = 635 participants.

Episodic memory was indexed by immediate free recall of two word lists and two stories (see Dixon, Wahlin, Maitland, Hultsch, Hertzog, & Bäckman, 2004; Hultsch et al., 1998). Each word list consisted of six words from five taxonomic categories (e.g., birds) typed on a single page in unblocked order. Participants were given 2 min to study each list and 5 min to write their recall. The average number of correctly recalled words across the two lists was used as the outcome measure. Each equivalent story contained approximately 300 words (about 160 propositions) and was presented in a typed booklet for study (4 min) followed by written recall (10 min). Recall protocols were scored for gist recall using criteria described elsewhere (Dixon et al., 2004) with reliability estimates across all possible pairs of scorers exceeding 90%. The outcome measure was the average number of gist propositions recalled across the two stories.

Semantic memory was indexed by two separate 40-item recall tests of world facts (Hultsch et al., 1998) and a 54-item recognition vocabulary measure (Ekstrom, French, Harman, & Dermen, 1976). Recall of world facts was measured by assessing knowledge in multiple domains (e.g., science, history, sports; see Nelson & Narens, 1980). The questions were presented in booklets, and participants wrote their answers under self-paced timing conditions. The outcome measure reflected average correct across the two tests. Vocabulary was measured in a 15-min session by the best response for each multiple-choice question. The score was the number of correct responses out of 54.

Assessment of speeded performance (mean level)

Level of speeded performance was indexed by two verbal speed tasks. The lexical decision task (Baddeley, Logie, Nimmo-Smith, & Brereton, 1985) required participants to judge as quickly as possible whether a string of 5 to 7 letters formed an English word (e.g., island versus nabion). A total of 60 test trials were presented (30 words and 30 nonwords). For the semantic

verification task (Palmer, MacLeod, Hunt, & Davidson, 1985), participants judged as rapidly as possible the plausibility of sentences (50 trials) presented on a computer screen (e.g., "The tree fell to the ground with a loud crash" versus "The pig gave birth to a litter of kittens this morning"). For both tasks, participants responded by pressing 1 of 2 keys on a custom-built response console. Response latencies were accurate to +/− 1 msec, permitting a more precise index of internal processes compared with accuracy measures (Gazzaniga et al., 2002). Within each task, the level of performance score was computed by averaging latencies across trials. All trials, including error trials, were analyzed for the lexical decision and semantic verification tasks. Previous findings from our laboratory (Burton et al., 2006) have clearly indicated that the inclusion of a small percentage of error trials (typically less than 2–3%) had no impact on the results.

Assessment of speeded performance (inconsistency)

The two verbal speed tasks were also used to compute performance inconsistency information. In contrast to a mean speed-of-performance score, inconsistency represents the within-person and across-trial variability of latencies. Details for computing inconsistency are described in the data preparation section.

Assessment of cardiovascular health

Cardiovascular disease (CVD) was indexed by both a self-report and an objective measure. Self-reported CVD conditions (heart disease, stroke, arteriosclerosis, and high blood pressure) were collected in the VLS intake health instrument administered at all waves. Actual number of currently prescribed medications for the CVD-related conditions of high blood pressure, heart conditions, chest pain, and blood thinning (e.g., Prinivil, Digoxin, Coumadin) were taken from the VLS medication protocol, in which participants use a supplied secure container to bring all prescribed and over-the-counter medications to the laboratory. All pertinent medication information is recorded (e.g., drug identification number) for subsequent classification and analyses. For each CVD predictor, the total score ranged from 0 (no CVD conditions or relevant medications) to 4 (all four of the CVD conditions or medications). The overall CVD index was created by summing across relevant items, with higher values reflecting greater severity.

Data preparation (level and inconsistency)

To facilitate comparisons across tasks in the same metric, indicators of cognitive level were linearly transformed as T-scores ($M = 50$), standardizing across individuals and waves to avoid detrending the data. T-scores were also computed for the response latency markers of speed (level), but were reverse coded so that lower values reflected slower reaction time. Speeded inconsistency was operationally defined as an intraindividual standard deviation (ISD) computed across latency trials of the lexical decision and semantic verification tasks (Hultsch & MacDonald, 2004). Higher ISDs reflect relatively inconsistent performance across trials, whereas lower ISDs indicate relative consistency. As extremely fast or slow responses could be due to errors (e.g., accidental key press) and unduly influence computed values of ISDs, we followed established procedures (e.g., Hultsch et al., 2002; Hultsch et al., 2008) and trimmed extreme raw latency outliers if they fell outside an a priori specified lower or upper bound. The lower bounds for legitimate responses were 400 msec for lexical decision and 1000 msec for semantic verification. The upper bounds were set at 10,000 msec for lexical decision and 20,000 msec for semantic verification. All higher/lower values were dropped, as were subsequent values exceeding 3SD above or below the mortality status by age means. Notably, as has been observed in all previous research using these procedures, the number of trials dropped across the entire Persons × Trials data matrix for each speed measure was negligible (i.e., less than 2.0%). Percentage of missing trials did not vary systematically across groups. To avoid

statistical problems associated with missing data, we imputed values for excluded outlier trials by using a regression procedure in which missing value estimates were based on the relationships among responses across trials. Missing values were imputed using data from all available individuals and trials. Because both dropping outliers and imputing missing values reduces variability, the subsequent analyses represent a conservative approach for examining the predictive influence of inconsistency.

Significant group (e.g., age, status) and trial (e.g., practice) differences in mean response speed are typically observed. These systematic level effects represent potential confounds for the analysis of intraindividual variability. For example, evidence of greater intraindividual variability in older adults as indicated by an ISD computed on raw scores may simply reflect the fact that older adults are on average slower than younger adults, and higher mean RTs are typically related to higher variances. To dissociate systematic between-person differences and within-person changes from lawful but transient changes in performance, raw latency trials for each speed measure were restructured into a person-period data matrix and separately regressed on mortality status, age group, cross-sectional trial and longitudinal occasion effects, and all their higher-order interactions (Hultsch et al., 2002). Resulting residual scores are statistically independent of confounds including group differences in response speed, performance accuracy, and systematic variation across trials or occasions due to practice or learning to learn. The residuals were linearly transformed as T-scores (to permit comparison of the tasks in the same metric) and individual ISDs were then computed for each participant. Figure 1 shows residual T-scores for latencies as a function of mortality status and trials. Patterns demonstrate (a) marked individual differences in variability after partialling systematic between- and within-subject effects from the raw scores, and (b) amplified patterns of inconsistency for decedents.

Statistical analysis

Three statistical procedures, corresponding to the three research questions, were used. Mean performance outcomes and predictors reflect each individual's respective final wave of testing, consistent with prevailing operational definitions and previous procedures and results (e.g., Berg, 1996; Riegel & Riegel, 1972). To evaluate the first research question, multivariate analysis of covariance (MANCOVA), controlling for years of education, was used to assess mortality (survivor and decedent) and age group (young-old and old-old) differences for all measures of cognitive level, speed level, and speed inconsistency. To circumvent listwise case deletion, a conservative imputation approach favoring cognitive stability over change was adopted. Missing values for the cognitive level measures were replaced with an individual's own performance score from the immediately preceding wave. Although more complex imputation techniques exist, such approaches should be used with caution when resulting data estimates will be used as predictors in subsequent analyses. Partial missing data were present for 22 survivors and 15 decedents (5.2% of the total sample): no individual failed to respond to fewer than 5 of 9 cognitive measures for any given measurement wave. For the few instances that a given individual had not completed the first wave, the mean baseline score for equivalent age and mortality status groups was substituted using series mean estimation in SPSS 11 (SPSS for Windows, 2001). For all analyses, patterns of significance with and without missing data were identical.

Given the known association between attrition and mortality (Anstey et al., 2001), the second and third research questions employed analytic methods that examine performance patterns for all available data (survivors and decedents) to minimize underestimation of terminal decline effects. Because general sample attrition could influence our findings (i.e., recall that some VLS participants' survival status could not be verified), the observed associations are likely conservative underestimates of the association between predictors and survival. For the second

research question, hierarchical Cox proportional hazards regression (Cox, 1972) was used to examine whether cognitive level, speed level, and speed inconsistency predicted increased likelihood of mortality over the longitudinal interval. Given that decedents and survivors participated in the study for varying periods of time, modeling the hazard rate (i.e., the probability of dying for a given time interval) was more appropriate than modeling mortality status as the dependent outcome. Time to event was computed as the number of years and months elapsed between each individual's respective final wave of testing and (a) date of death for decedents (M = 5.18, SD = 3.74), or (b) the last date for which all surviving participants were known to be alive (May 31, 2002: M = 2.79, SD = 0.44). All survivors were included in the Cox analyses as right-censored cases (i.e., cases for which survival time was unknown and the death event had not yet occurred). Defining the incident as death, odds ratios indicate the percentage increase in death rate per unit decrease (or increase) in raw cognitive predictor scores holding constant the effects of all other variables in the equation. Cox regression results will reveal which measures best predict likelihood of death over a follow-up interval. Cox regressions were also computed using predictors at baseline performance, yielding identical patterns of significance to those reported in the results section.

For the third research question, multilevel or random-effects regression models (Singer & Willett, 2003) were specified to examine whether individual trajectories of inconsistency increased as a function of proximity to event, and whether such changes were further modified by chronological age or mortality status. These multilevel models do not require samples balanced on time or the equivalent number of measurement occasions per participant, thus representing a distinct advantage relative to other listwise analytic approaches. Hierarchical linear modeling (HLM) version 5.05 software (Raudenbush, Bryk, Cheong, & Congdon, 2000) was used to examine the data, with robust standard errors estimated using restricted maximum likelihood (REML). For each measure of inconsistency, the Level 1 model (see equation 1) examined individual rates of change as a function of time to event (years to death for decedents vs. years remaining in the study prior to the censoring point for survivors) parameterized as the number of years and months elapsed since baseline performance. Inconsistency for a given individual (i) at a given time (t) was modeled as a function of that individual's performance centered at 7.24 years (the grand mean of time to event: the intercept β_{0i}), plus a slope parameter (β_{1i}) reflecting her/his average linear rate of change with increasing proximity to event (date of event$_{it}$ - date of baseline testing$_{ij}$), plus a random within-subject error term (ε_{it}) reflecting residual variance remaining to be explained.

$$\text{Level 1: Inconsistency}_{it}=\beta_{0i}+\beta_{1i}(\text{Time to Event}_{it})+\varepsilon_{it} \quad (1)$$

Associated Level 2 HLM equations examined between-group differences for change in inconsistency. Average population effects were estimated by modeling the Level 1 prediction parameters (β_{0i} and β_{1i}) as outcome measures for the Level 2 between-person equations (see equations 2 and 3). Of primary interest, equation 3 modeled each individual's linear rate of change in inconsistency (β_{1i}) as a function of the population average change in inconsistency for young-old survivors per year closer to event (γ_{10}), the average difference in change per year closer to event between age groups (γ_{11}), mortality groups (γ_{12}), and their interaction (γ_{13}), plus a random effect (u_{1i}) estimating whether variability remains about inconsistency slopes holding these predictors constant.

$$\text{Level 2: } \beta_{0i}=\gamma_{00}+\gamma_{01}(\text{Age Group}_i)+\gamma_{02}(\text{Mortality Status}_i) +\gamma_{03}(\text{Age Group x Mortality Status}_i)+u_{0i} \quad (2)$$

$$\beta_{1i}=\gamma_{10}+\gamma_{11}(\text{Age Group}_i)+\gamma_{12}(\text{Mortality Status}_i) + \gamma_{13}(\text{Age Group x Mortality Status}_i)+u_{1i} \quad (3)$$

The test of the random-effect coefficient for equation 3 (u_{1i}) evaluates whether changes in inconsistency as a function of time to event are equivalent across individuals. Education was entered as a covariate for equations 2 and 3.

Results

Although we emphasize reporting of statistically significant results, we selectively note non-significant effects when they pertain to theoretically- or archivally-related hypotheses.

Mortality effects at final testing wave

Initial MANCOVAs for cross-sectional data assessed whether cognitive level, speed level, and speed inconsistency for each individual's respective final wave of testing varied as a function of mortality status or age group (see Table 2). To maintain consistency with previous age categorizations in the literature (e.g., Bäckman, Small, Wahlin, & Larsson, 2000;MacDonald, Dixon, Cohen, & Hazlitt, 2004), subjects were classified according to age at final testing wave as either young-old (n = 519) ranging in age from 59 to 79 years (M = 71.86, SD = 4.91), or old-old (n = 188) ranging in age from 80 to 95 years (M = 83.66, SD = 3.38). Significant omnibus effects were observed for Mortality status ($p < .001$), Age group ($p < .001$), as well as the Mortality status × Age group interaction ($p < .01$). Univariate F tests were computed for each of the 7 cognitive measures, 2 speed level measures, and 2 speed inconsistency measures. Significant mortality status effects were observed for all 11 measures, with decedents exhibiting lower levels of cognitive performance ($p < .01$) and greater inconsistency across trials ($p < .001$). Reliable age group effects were observed for all but the vocabulary task, with the old-old performing lower and more variably than the young-old group ($p < .001$). Reliable mortality status × age group interactions were observed for 2 of the 7 cognitive level measures ($p < .05$, fact recall and vocabulary) as well as both of the neurocognitive level ($p < .001$) and inconsistency ($p < .001$) measures. In all cases, the old-old decedents were differentially impaired compared to the other groups. As expected, identical patterns of significance were observed when age was entered as a continuous measure using linear regression.

Cohen's d values are presented in Figure 2, which graphs the magnitude of mortality effects for each measure, separately within age group and for the entire sample. For three of the four indices of cognitive and speed level that exhibited significant Mortality × Age interactions, mortality effect sizes increased from small to medium (lexical decision and fact recall) and from small to large (semantic verification) with increasing age; the magnitude of mortality differences for vocabulary approached a medium effect for the old-old. A medium to large age-related increase was also observed for story recall. In contrast, irrespective of age, small mortality effects were observed for the working memory measures, with a medium effect observed for word recall. Of note, for both markers of inconsistency, mortality effect sizes were uniformly small for the young-old and large for the old-old.

Speed inconsistency as a predictor of survival

For the second research question, Cox regression models addressed whether speed inconsistency predicted mortality status beyond contributions of cognitive level and neurocognitive level, as well as selected demographic indicators and CVD status. Table 3 presents Cox regression results predicting mortality status as a function of select covariates entered in five hierarchical blocks. Predictors for each subsequent block are introduced into the model and tested simultaneously with predictors from all previous blocks, yielding partial regression coefficients. Age, education, and self-reported health were entered in Block 1, followed by the presence of CVD conditions and number of CVD medications in Block 2. Mean cognitive performance predictors were examined for Block 3, with separate models computed for each of the seven indicators to reduce multicollinearity (c.f., Small & Bäckman,

1997). Each speed level predictor was entered separately in Block 4, followed by Block 5 entry of the corresponding inconsistency indicator (i.e., Block 4 lexical level followed by Block 5 lexical ISD; Block 4 semantic level followed by Block 5 semantic ISD). The entry order of regression blocks in Table 3 was determined a priori to facilitate a stringent test of whether inconsistency remained a differentially sensitive predictor of mortality even subsequent to partialing for demographic factors, CVD, and mean cognitive accuracy and speed performance. Entering speeded performance level (Block 4) prior to inconsistency (Block 5) reflects the central importance afforded to level in extant research (Hultsch & MacDonald, 2004). Although not reported in Table 3, an identical hierarchical analysis was computed with one modification: the ISD indicators were entered in Block 4 followed by corresponding speeded level indicators in Block 5. For all models, individual regression coefficients reflect the association between each covariate and the likelihood of dying over the longitudinal interval, with corresponding odds ratios (OR) indicating the percentage difference in mortality risk per unit difference in covariate score.

Findings summarized in Table 3 demonstrate that age, number of CVD medications, speed level, and speed inconsistency all share significant associations with mortality status. For the first block of demographic predictors, the odds ratio for age was 1.026, indicating an increase in age-related mortality risk of 2.6% per year or 26% per decade. For Block 2, requiring additional medications to treat cardiovascular disease was associated with a 25.7% increased mortality risk), controlling for all other covariates. This pattern suggests that each additional CVD medication used is a proxy for disease severity; medication use per se does not increase mortality risk. The Block 2 results also suggest that the prescribed medications variable was a more reliable index of CVD and impending death than the self-reported number of CVD conditions. Results for these first two blocks are consistent with previous findings linking age, CVD integrity, cognitive performance, and mortality (Hassing et al., 2002). In contrast to the MANCOVA findings where decedents showed clear impairment in mean level cognitive performance, none of the 7 mean level cognitive indicators evaluated in Block 3 of the Cox models predicted increased risk of mortality across the follow-up interval. Previous investigations have also reported reliable mean cognitive differences between survivors and decedents, only to reveal weak mean level prediction of subsequent mortality risk using survival analysis (Small & Bäckman, 1997). Among potential explanations, mean level mortality group differences in cognitive performance may be confounded by demographic and disease-related factors. Using Cox regression to model the hazard rate for death, controlling for potential confounds including age and the number of CVD medications, likely attenuated the predictive link between cognitive performance level and mortality risk. In contrast, both Block 4 speed level indicators predicted mortality over and above select measures in the first 3 blocks. Specifically, mean lexical decision speed significantly predicted mortality independent of all measures in the first 3 blocks. Depending on the covariates, slower lexical speed (per unit change in T-scores) was associated with a 1.3 to 1.8% increased risk of mortality. Similar patterns were observed for semantic verification speed, which reliably predicted mortality independent of 3 of the 7 cognitive level indicators and approached significance for the remaining 4 ($p < .10$). Slower semantic speed was associated with a 1.1 to 1.9% increased risk of mortality.

Even after controlling for all measures in Blocks 1 to 4, both Block 5 markers of speed inconsistency reliably predicted increased likelihood of death over the follow-up interval. When entered in Block 5, inconsistency in semantic verification significantly predicted mortality ($p < .05$) independent of virtually all predictors in the previous blocks, including the Block 4 speed level indicator of semantic speed. The only exception was for the vocabulary prediction model where the probability for semantic inconsistency in Block 5 reached but did not surpass the criterion ($p = .05$). Each additional T-score increase in semantic ISD (Block 5) was associated with an increased risk of mortality ranging from 6.6% for the vocabulary model

to 12.7% for the computation span model. Corresponding models indicated that inconsistency in lexical decision uniquely predicted mortality status independent of all previous predictors for the computation and listening span models ($p = .055$ for vocabulary). The hazard ratios for lexical ISD (Block 5) indicated a 3.4% (sentence construction and word recall models) to 9.2% (computation and listening span models) increased risk of mortality per T-score increase in variability. As speed level in Block 4 reliably predicted mortality status independent of covariates in the first 3 blocks, the Block 5 partial regression coefficients indicate that inconsistency fully attenuated this predictive association and contributed unique information to the prediction of mortality relative to mean cognitive accuracy and speeded performance.

To compare the relative prediction of speeded level and inconsistency, an additional hierarchical analysis was computed reversing the order of predictor entry in Blocks 4 and 5; entry order in Blocks 1 to 3 remained identical. When entered in Block 4, inconsistency in semantic verification significantly predicted mortality ($p < .01$) independent of all predictors in Blocks 1 to 3, including all seven Block 3 mean level cognitive predictors. Similarly, Block 4 entry of lexical ISD uniquely predicted mortality ($p < .05$) independent of all demographic, CVD, and cognitive level predictors. When subsequently entered in Block 5, neither measure of speed level uniquely predicted mortality independent of the predictors in Blocks 1 to 4. Thus, regardless of whether speeded level was entered prior to (Block 4) or following (Block 5) inconsistency, neither level indicator remained a significant predictor of mortality status independent of inconsistency. However, controlling for speed level did partially attenuate the association between mortality and speed inconsistency relative to the effects observed when only inconsistency was entered in Block 4. In part, the non-significant associations observed for lexical inconsistency independent of lexical level could reflect the part-whole relation between ISD scores and mean level performance. Whereas ISDs reflect residual variance after controlling for mean group differences in response speed and systematic effects over time, mean performance level indicators for the same variable include all relevant sources of variability.

Change in speed inconsistency as a function of time to death

The Cox regression results suggested that the neurocognitive resource indicators are reliable predictors of impending mortality, with speed inconsistency performing as an especially promising predictor. Whereas Cox regression can only speak to the contemporaneous presence or absence of associations between covariates and survival status, multilevel modeling facilitates examination of the time-varying covariation between predictors and outcomes. Our third research question explicitly tests the longitudinal time-varying covariation between speed inconsistency and time to event. Specifically, the multilevel models evaluated whether the magnitude of change in inconsistency was accelerated for decedents vs. survivors per year closer to event. This third analysis is both novel and important as no previous investigations have examined change in inconsistency as a function of mortality status.

Table 4 summarizes multilevel findings associated with equations 1 to 3. Collectively, the equations assessed whether speed inconsistency changed differentially as a function of time to event, mortality status, or age group. Coefficients in the intercept column (γ_{00}) reflect mean ISDs (T-scores) for young-old survivors at the sample midpoint for time to event ($M = 7.24$ years). The first slope (γ_{10}) assessed the average rate of change in inconsistency for young-old survivors per additional year of time remaining in the study. For semantic verification, young-old survivors exhibited increasing inconsistency with increasing proximity to the censoring date. The second slope (γ_{11}) examined age group differences in rates of change for inconsistency. Consistent with expectations, inconsistency increased at a faster rate for old-old relative to young-old adults per year closer to event. The average difference in rates of change per year closer to event between the age groups for semantic inconsistency was 0.06

ISD units per year, with an average difference of 0.22 units for lexical decision. Similar patterns were observed for age as a continuous predictor. The third slope coefficient (γ_{12}) assessed differences in rates of ISD change between survivors and those facing mortality. With increasing proximity to death, inconsistency in lexical decision performance for decedents showed significant linear increases (0.29 ISDs per additional year closer to death). The inconsistency slope for semantic verification was not significant but was in the expected direction. Larger magnitude effects for lexical vs semantic inconsistency may reflect the increased novelty of stimulus words requiring lexical judgment, whereas words presented in the semantic task were comparatively more familiar and provided semantic support through syntactic structure. A similar compensatory explanation has been offered to account for age differences in performance inconsistency between sensorimotor vs. verbal response latency measures, where tasks that benefit less from strategies or existing knowledge (e.g., non-verbal response latency tasks) show greater performance inconsistency (Hultsch et al., 2002). For each corresponding fixed effect, random-effect coefficients were significant ($p < .01$) indicating that, even after controlling for the specified predictors, significant between-group differences remained to be explained.

Qualifying the main effects, a significant Age group × Mortality status interaction was observed for lexical decision (see Figure 3). Simple regression slopes were calculated within each age group to examine associations between change in inconsistency, time to death, and mortality status. For the young-old, significant differences in ISD change were observed between mortality groups, with decedents increasing an additional 0.16 ISDs per year closer to death ($t = -2.77$, $p < .01$). Similarly, relative to same-age survivors, old-old decedents exhibited accelerated increases in inconsistency (0.40 ISD units) per year closer to death ($t = -4.33$, $p < .001$). These findings suggest that increases in speed inconsistency are amplified as a function of both age and impending mortality.

Discussion

Convergent findings from three central research questions supplement current knowledge by demonstrating links between level and inconsistency in speeded performance, survival status, and proximity to death. We discuss results pertaining to each research question in turn.

The objective of the first research question was to replicate previous mortality patterns for cognitive outcomes and to extend such results to the novel outcome measures of level (rate) and inconsistency in speeded performance. As expected, decedents exhibited uniformly lower levels of cognitive performance, slower speeded performance, and greater inconsistency than survivors. Increasing age modified the mortality effects for all four speed-related outcomes, but only 2 of the 7 cognitive-level measures. The consistently reliable and large magnitude differences (see Figure 2) observed for the markers of speeded level and inconsistency (as compared to traditional cognitive level) foreshadow their importance as predictors of mortality status (see below). Larger magnitude mortality effects for the old-old reflect the differential sensitivity of inconsistency measures to chronological age, with the old-old being closer to death (on average). Three novel and significant aspects of the results should be highlighted. First, this is the initial demonstration of mortality-related effects for markers of neurocognitive level and inconsistency. Similar markers have recently been linked to normal age-related cognitive decline, mild cognitive impairment, and neurodegenerative disease (Dixon et al., 2007;Hultsch & MacDonald, 2004). Second, the regression approach used to estimate ISD scores ensured that inconsistency estimates were uncontaminated by mortality or age differences in performance speed or by systematic variation across trials. That robust mortality differences remained for the inconsistency measures underscores the durable association and the possibility that speeded inconsistency is a plausible behavioral proxy of neural integrity. Third, as mortality effects are typically reported within 5 years of death (Berg, 1996;Wilson

et al., 2003) and the duration between last wave of testing and death averaged 5.2 years in the present study, the observed reliable mean differences between survivors and decedents likely underestimates the true effect. The duration between measurement occasion and event is directly modeled in the Cox regression and multilevel analyses, consequently yielding accurate time-dependent associations.

The second research question evaluated whether the two markers of neurocognitive resources predicted mortality risk either with, or independent of, mean level of cognitive performance, cardiovascular disease, and several demographic measures. Theoretically, changes in neurocognitive resources are important behavioral reflections of neurodegenerative changes (Dixon et al., 2007; MacDonald et al., 2006), and potentially differentially sensitive predictors of mortality. Results from the Cox regressions demonstrated that age, number of CVD medications and both speeded level and inconsistency predicted mortality risk as many as 15 years in advance (the maximum span in this study between final testing wave and subsequent death). As expected, regarding the central importance of neurocognitive resource predictors, Block 4 entry of either speed level or speed inconsistency reliably predicted mortality over and above the demographic, CVD, and cognitive level predictors from Blocks 1 to 3. These partial regression coefficients ascribe differential predictive status to speed-related performance relative to cognitive level performance, with both speeded level and inconsistency predicting mortality risk for select models.

The importance of this finding is notable given that the traditional (mean-level) cognitive accuracy predictors (e.g., working memory, episodic memory) are known to be sensitive to both normative and pathological aging processes. Moreover, we computed additional analyses to contrast the relative predictive importance of speeded level vs. inconsistency. Whereas speed inconsistency predicted mortality independent of speed level (rate), the opposite was not observed. That inconsistency was a generally better predictor than mean rate confirms and extends previous findings (Strauss, MacDonald, Hunter, Moll, & Hultsch, 2002). The differential importance of inconsistency in predicting abnormal cognitive functioning and impending death is in line with recent theoretical proposals that intraindividual performance fluctuations may provide a useful behavioral index of neurological integrity (Hultsch & MacDonald, 2004; Hultsch et al., 2008). Specifically, in the aging literature, performance inconsistency is known to increase as a function of age (Hultsch et al., 2002), traumatic brain injury (Stuss, Pogue, Buckle, & Bondar, 1994), neurodegenerative disease (Hultsch et al., 2000; Murtha, Cismaru, Waechter, & Chertkow, 2002), and severity of neurological disturbance (de Frias, Dixon, Fisher, & Camicioli, 2007; Dixon et al., 2007). Thus, inconsistency appears to be primarily a central nervous system phenomenon (Hultsch & MacDonald, 2004) reflective of the integrity and availability of neurocognitive resources.

Despite documenting mean survival differences for both cognitive level and speed-related performance for the first research question, only the speed indicators reliably predicted mortality risk independent of all other covariates. This predictive dissociation underscores the utility of differentiating between traditional cognitive and speeded markers for theoretical and future assessment purposes (Dixon et al., 2007; Goldberg & Weinberger, 2004). For example, for some neuropsychological or health conditions, a single performance or average may be insufficient for optimally classifying an early condition or for validly characterizing a given individual's true score or competence level (even with the use of normed cut-off scores in clinical settings; Strauss et al., 2002). To underscore this point, within-person fluctuations in cognitive performance from one occasion to the next correspond to as much as several decades of between-person age differences (Nesselroade & Salthouse, 2004). Moreover, the differential importance of inconsistency as a predictor of death likely reflects the central importance of neurocognitive resources for multiple cognitive and clinical outcomes. Given the selective sensitivity of speed inconsistency for predicting outcomes such as impairment, dementia, and

death, it may be clinically useful to develop task-related inconsistency norms that could facilitate early identification and intervention. Markers with clinical advantages in terms of cost and ease of administration must be developed.

The third research objective focused specifically on trajectories of longitudinal change in inconsistency as a function of time to death. Compared to more conventional tools that demonstrate mean mortality group differences (e.g., MANCOVA) or the link between predictors and survival status (e.g., Cox regression), the parameterization of time to event using multilevel modeling permits a more precise assessment of the relative influence of impending mortality on change in inconsistency per year closer to event (death vs last known date alive). This approach allows for one of the most stringent tests of the terminal decline hypothesis to date.

Inconsistency in speed reliably increased per year closer to death, with slightly different patterns observed as a function of mortality status versus chronological age. Independent of mortality status, inconsistency increased for both age groups per year closer to event. Independent of age, decedents exhibited increasing inconsistency (for lexical decision) per additional year closer to death. For the latter, a significant mortality status by age group interaction revealed larger mortality group differences for the old-old group, with correspondingly more shallow slopes for survivors and young-old adults. These findings not only demonstrate the association between speed inconsistency and terminal decline, but also model the magnitude of change in inconsistency per year closer to death. As shown in Figure 3, those at greatest risk (i.e., impending death) show the largest longitudinal increases in inconsistency. These longitudinal findings support and extend the above-noted cross-sectional observation regarding the differential importance of inconsistency in speeded performance as an early predictor of mortality risk. The distinct age and mortality effects imply both normative and pathological influences on changes in inconsistency, with the accelerated age increases for decedents likely reflecting both compromised neurobiological functioning and limited availability of neurocognitive resources. Notably, these results underscore the perils of treating elderly samples as neurocognitively homogeneous; namely, averaging across subgroups (including those in a terminal decline phase) may generate misleading estimates of cognitive aging trajectories (Bosworth & Schaie, 1999).

Despite increasingly systematic knowledge about speed inconsistency, a key unresolved question concerns the nature of the underlying mechanisms that link within-person variability, neurological compromise, and mortality. Among potential neurobiological explanations for within-person variability, theorists have identified random errors or neural “noise” in the transmission of CNS signals (Hendrickson, 1982; Welford, 1980), a decrease in the frequency of neuronal oscillations reflecting the excitatory potential of neurons (Jensen, 1992), or the dysregulation of neurotransmitter systems (Li, Lindenberger, & Sikström, 2001; MacDonald et al., 2006). Although such mechanisms do not directly result in death, the increased inconsistency in sensitive speed tasks accompanying them may provide early behavioral indicators of neurobiological compromise, which may reflect broader or inchoate biological (disease-related) processes that more directly presage death. For example, in research associated with the frontal-lobe deficit hypothesis of aging (e.g., Raz et al., 2005; West, 1996), more intraindividual variability has been observed as a function of focal frontal lobe lesion location (e.g., frontal-temporal dementia) than for relatively non-frontal lesions or for Alzheimer’s dementia (e.g., Stuss et al., 1994; Stuss, Murphy, Binns, & Alexander, 2003). The present study established a link between increased speed inconsistency, terminal cognitive decline, and subsequent mortality. Future research may investigate further biological-disease changes associated with these neurobiological foundations of inconsistency (MacDonald et al., 2006).

Although the results are unique, converging, and robust, several limitations should be noted. First, because we used only semantic speed tasks, it is unclear whether the differential importance of speeded measures as predictors of mortality are task-specific or generalize across multiple indicators (e.g., perceptual speed, sensorimotor response latency, verbal speed). Sampling a broader continuum of speed measures would further inform the differential relevance of specific tasks for the prediction of mortality. Second, as is typical for longitudinal studies, the sample was positively selected (particularly the elderly men) and this could potentially account for the non-significant association between sex and mortality. However, the observed mortality effects may be even stronger for a less selected sample. Third, some typical design concerns include the proportion of participants completing 3 or more waves and a sometimes relatively long interval between last wave and death. In addition, decedents completed fewer waves of measurement than survivors; enough to facilitate multilevel analysis of linear change with random effects but not enough to assess curvilinear decline. Despite these limitations, the consistent intraindividual trends likely underestimate the magnitude of terminal decline in the elderly population. Fourth, one facet of our cardiovascular disease assessment did not exclude the possibility of patient-reporting bias, but the number of CVD medications was a more objective and statistically reliable predictor of mortality than self-reported presence of CVD. Nevertheless, future terminal decline studies may benefit from more precise assessments of presence and severity of CVD, the inclusion of other health-related or genetic predictors, and an upgrading of initial neurocognitive resource assessments even in initially healthy older adults.

In sum, the present study is the first to demonstrate not only a clear link between speeded inconsistency and impending death, but that speed inconsistency may be a more sensitive marker of mortality risk than the more common predictors of mean level cognitive performance and background variables. In addition, reliable prediction is maintained across a broad interval between last wave and death. The results underscore the importance of attending to fluctuations, cycles, and oscillations of behavior, as well as average levels of performance, when testing theories of cognitive aging (Hultsch et al., 2008), identifying predictors of preclinical neurological compromise (Dixon et al., 2007), and now even estimating future basic health or mortality status (i.e., survival or death). Ultimately, ascertaining whether speeded level and inconsistency represent behavioral proxies for neurological integrity requires objective assessment of brain structure and function. For now, given documented and potential relations among health/disease, mortality, biological aging, and cognition (e.g., Anstey et al., 2001; Hassing et al., 2002; MacDonald et al., 2004; Wahlin, MacDonald, de Frias, Nilsson, & Dixon, 2006), research on neurocognitive performance continues to have a promising future.

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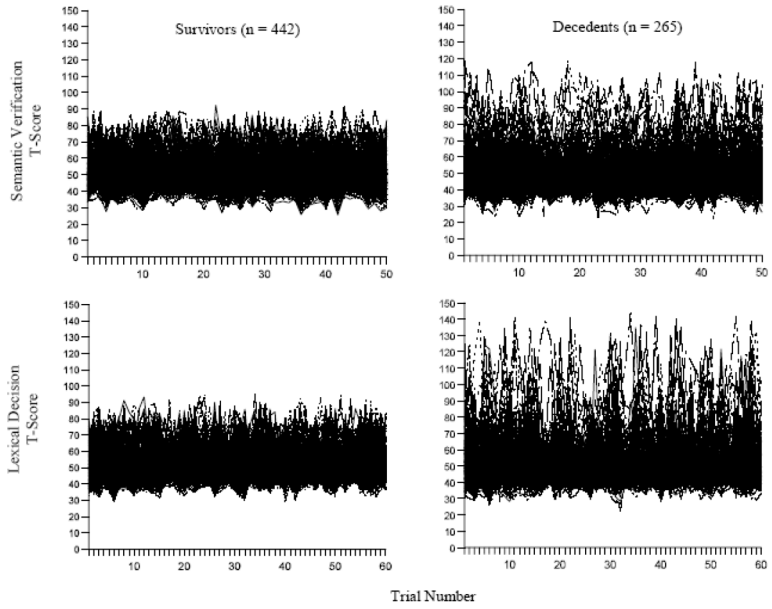

Figure 1.
Intraindividual variability profiles for semantic verification and lexical decision: Residualized T-scores for survivors and decedents across trials.

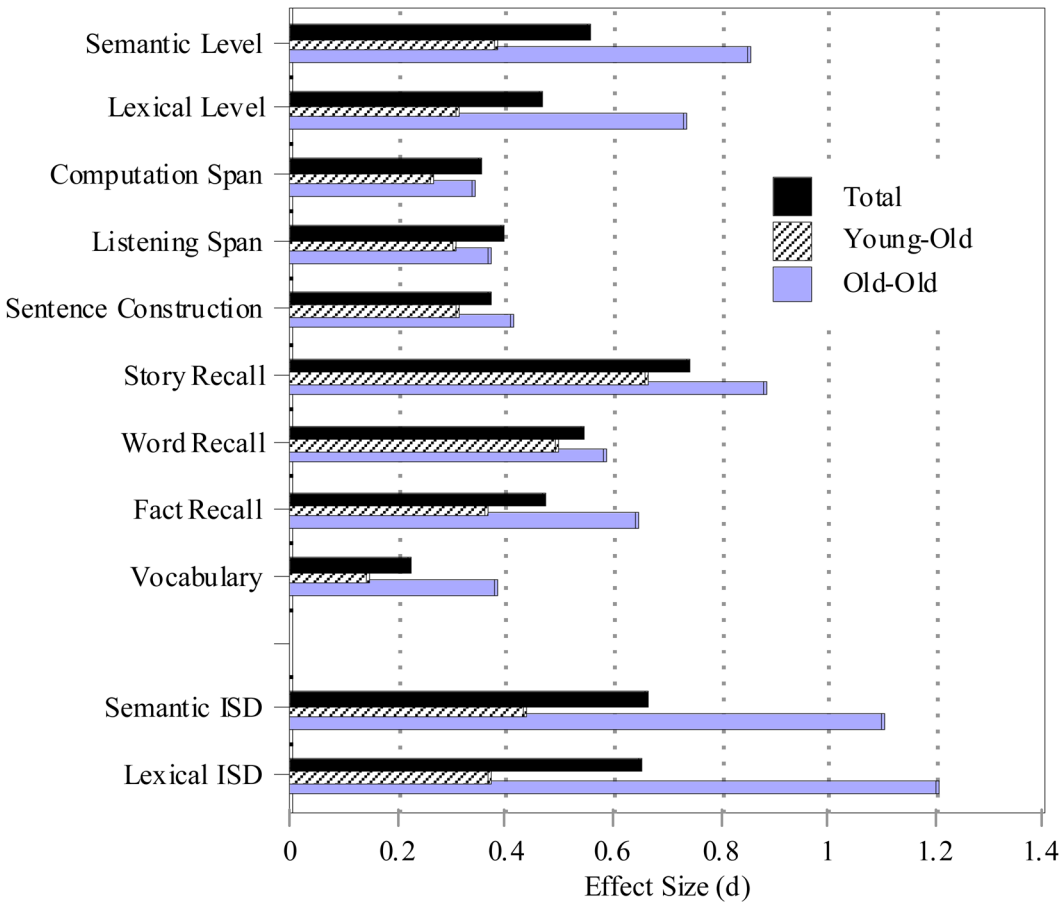

Figure 2.
Cohen's d for mortality status differences as a function of age group at the final wave of testing. ISD = intraindividual standard deviation.

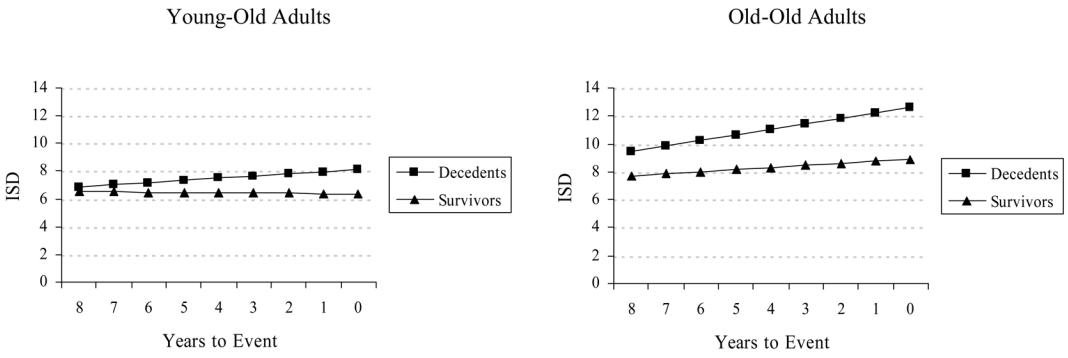

Figure 3.
Change in speed inconsistency for lexical decision as a function of time to event, mortality status, and age.

Table 1

Demographic characteristics by mortality status for respective final testing wave.

Variable	Survivors (n = 442)	Decedents (n = 265)	*p* value
Gender (% Female)	61.80	56.20	n.s.
Age	74.64 (6.78)	75.61 (7.12)	n.s.
Years of education	14.92 (3.06)	14.03 (3.49)	< 0.01
Relative health	1.39 (0.85)	0.88 (0.79)	< 0.01
Years to death	---	5.18 (3.74)	

Note. Standard deviations appear in parentheses. Subjects rated their health relative to same-aged peers on a 5-point scale ranging from (0) very good to (4) very poor.

Table 2

Mortality and age group mean T-scores for cognitive and speeded measures at last testing wave.

	Survivors			Decedents		
Level	**Young-Old (n = 337)**	**Old-Old (n = 105)**	**Total (n = 442)**	**Young-Old (n = 182)**	**Old-Old (n = 83)**	**Total (n = 265)**
Semantic Speed						
Semantic verification	53.00 (7.69)	48.95 (7.73)	52.04 (7.88)	50.14 (7.46)	38.85 (15.99)	46.60 (12.05)
Lexical decision	52.68 (7.49)	48.75 (7.24)	51.75 (7.61)	50.50 (6.68)	39.58 (18.00)	47.08 (12.53)
Working Memory						
Computation span[a]	52.39 (9.45)	46.86 (8.27)	51.08 (9.47)	49.86 (9.86)	43.59 (11.06)	47.52 (10.73)
Listening span[a]	52.50 (9.00)	46.99 (10.15)	51.19 (9.57)	49.69 (9.79)	43.21 (10.34)	47.27 (10.45)
Sentence construction	52.21 (8.06)	48.75 (10.71)	51.39 (8.87)	49.41 (9.84)	43.90 (13.24)	47.68 (11.29)
Episodic Memory						
Story recall	53.86 (8.80)	48.65 (9.26)	52.62 (9.17)	47.91 (9.30)	40.63 (9.05)	45.63 (9.81)
Word recall	53.58 (8.54)	46.95 (9.49)	52.00 (9.21)	49.25 (9.05)	40.99 (10.92)	46.66 (10.39)
Semantic Memory						
Fact recall	52.82 (9.16)	48.19 (9.40)	51.72 (9.42)	49.49 (9.54)	41.97 (10.07)	47.13 (10.30)
Vocabulary	50.79 (8.24)	51.03 (9.13)	50.85 (8.45)	49.46 (11.24)	46.68 (13.50)	48.59 (12.04)
Inconsistency						
Semantic Speed						
Semantic ISD	5.77 (1.57)	6.88 (1.98)	6.03 (1.74)	6.46 (1.60)	10.04 (3.76)	7.59 (2.98)
Lexical ISD	5.56 (1.79)	6.98 (2.23)	5.90 (2.00)	6.26 (2.03)	11.75 (5.72)	7.98 (4.41)

Note. Standard deviations appear in parentheses; ISD = intraindividual standard deviation.

[a]These measures were not administered for Sample 1Wave 1.

Table 3

Cox regressions predicting mortality status as a function of demographic and cognitive covariates.

Covariates	*β*	Odds Ratio	95% CI	*p*
Block 1				
Age	0.026	1.026	1.008 – 1.045	0.005
Education	0.024	1.024	0.989 – 1.061	0.173
Relative health	0.013	1.013	0.864 – 1.188	0.870
Block 2				
CVD conditions	−0.033	0.967	0.808 – 1.158	0.717
CVD medications	0.229	1.257	1.065 – 1.483	0.007
Block 3				
Computation span [a]	−0.004	0.996	0.981 – 1.011	0.582
Block 4: Level Semantic	−0.020	0.981	0.967 – 0.994	0.006
Block 4: Level Lexical	−0.018	0.982	0.971 – 0.994	0.002
Block 5: ISD Semantic	0.119	1.127	1.044 – 1.216	0.002
Block 5: ISD Lexical	0.088	1.092	1.035 – 1.153	0.001
Listening span [a]	−0.007	0.993	0.978 – 1.009	0.412
Block 4: Level Semantic	−0.019	0.982	0.968 – 0.995	0.009
Block 4: Level Lexical	−0.017	0.983	0.971 – 0.994	0.004
Block 5: ISD Semantic	0.118	1.126	1.043 – 1.214	0.002
Block 5: ISD Lexical	0.088	1.092	1.035 – 1.152	0.001
Sentence construction	−0.003	0.997	0.985 – 1.008	0.592
Block 4: Level Semantic	−0.011	0.989	0.977 – 1.001	0.079
Block 4: Level Lexical	−0.013	0.987	0.977 – 0.998	0.024
Block 5: ISD Semantic	0.068	1.070	1.001 – 1.145	0.048
Block 5: ISD Lexical	0.034	1.034	0.987 – 1.034	0.156
Story recall	−0.001	0.999	0.985 – 1.014	0.919
Block 4: Level Semantic	−0.013	0.987	0.974 – 1.000	0.052
Block 4: Level Lexical	−0.013	0.987	0.976 – 0.998	0.018
Block 5: ISD Semantic	0.070	1.072	1.002 – 1.147	0.042
Block 5: ISD Lexical	0.035	1.035	0.988 – 1.035	0.145
Word recall	−0.002	0.998	0.986 – 1.011	0.813
Block 4: Level Semantic	−0.012	0.988	0.976 – 1.001	0.065
Block 4: Level Lexical	−0.013	0.987	0.976 – 0.998	0.021
Block 5: ISD Semantic	0.069	1.071	1.001 – 1.146	0.046
Block 5: ISD Lexical	0.034	1.034	0.987 – 1.083	0.158
Fact recall	−0.003	0.997	0.983 – 1.010	0.626
Block 4: Level Semantic	−0.012	0.988	0.974 – 1.001	0.072
Block 4: Level Lexical	−0.013	0.987	0.976 – 0.998	0.024
Block 5: ISD Semantic	0.069	1.071	1.001 – 1.146	0.046
Block 5: ISD Lexical	0.034	1.035	0.988 – 1.084	0.149
Vocabulary	0.008	1.008	0.996 – 1.020	0.185
Block 4: Level Semantic	−0.019	0.982	0.969 – 0.994	0.005
Block 4: Level Lexical	−0.016	0.984	0.973 – 0.994	0.003
Block 5: ISD Semantic	0.066	1.068	1.000 – 1.141	0.050
Block 5: ISD Lexical	0.047	1.048	0.999 – 1.099	0.055

Note. Statistical models were computed separately for each cognitive and speed measure. Results represent parameters for a given regression block controlling for the influence of all predictors on previous blocks. Values in column 1 reflect unstandardized regression coefficients, whereas values in column 2 are odds ratios denoting percentage change in morality risk per unit increase of predictor. Mortality status was coded as either 0 (survivor) or 1 (decedent). CI = confidence interval; CVD = cardiovascular disease. Subjects rated their health relative to same-aged peers on a 5-point scale ranging from (0) very good to (4) very poor. CVD conditions represent self-reported presence of 4 cardiovascular diseases (heart disease, stroke, arteriosclerosis, and high blood pressure) for any wave of testing. CVD medications reflect self-reported use of medications prescribed to treat cardiovascular symptoms and disease (heart, chest, high blood pressure, and anticoagulants). ISD = intraindividual standard deviation.

[a] N = 707 for all measures except computation span and listening span (n = 635).

Table 4

Change in speed inconsistency as a function of time to death, age group, and mortality status.

Variable	Intercept γ_{00}	Slope $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t
Semantic verification	6.8	−0.09	0.016	−5.53**
		−0.06	0.030	−2.13*
		−0.04	0.036	−1.02
Lexical decision[a]	6.46	0.03	0.021	1.56
		−0.22	0.040	−5.54**
		−0.29	0.049	−5.80**

[a] *Note*. A significant Age group × Mortality status interaction was observed for this variable; associated mortality coefficients are reported separately for each age group in the text. $\gamma 00$ = inconsistency for Young-Old Survivors at the sample midpoint for time remaining in the study ($\underline{M}$ = 7.24 years); $\gamma 10$ = average rate of change in inconsistency per additional year of time remaining in the study for Young-Old Survivors; $\gamma 11$ = average difference in rate of inconsistency change between Young-Old and Old-Old adults partialing the influence of mortality status; $\gamma 12$ = average difference in rate of inconsistency change between Survivors and Decedents controlling for age group differences; SE = standard error; t = t-ratio.

* $\underline{p} < .05$

** $\underline{p} < .01$