

Longitudinal Profiles of Terminal Decline:
Associations between Cognitive Decline, Age, Time to Death, and Cause of Death

by

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ABSTRACT

Normative age differences and declines in cognition may be overestimated due to influences reflecting impending mortality. The terminal decline hypothesis posits that accelerated cognitive decline for older adults is a function of proximity to death.

Although previous research has demonstrated mortality-cognition associations, key questions remain unresolved. This study examined five neglected aspects of terminal decline research: (a) are mortality deficits uniform across age? (b) does impending mortality differentially influence cognitive domains? (c) does cause of death influence magnitude of mortality deficits? (d) do individuals closer to death show accelerated cognitive declines? and (e) do mortality deficits share associations with indicators of neurological disturbance such as performance inconsistency?

The sample consisted of 707 adults from the Victoria Longitudinal Study (VLS) who completed between 1 to 5 waves of measurement over a 12-year period. Participants were classified as either Young-Old (59 to 79 years, $M = 71.86$) or Old-Old (80 to 95 years, $M = 83.66$). A total of 442 Survivors completed all waves and relevant measures compared with 265 Decedents who participated on at least one occasion and subsequently died. An extensive battery of tests was administered including measures of verbal speed, working memory, episodic memory, semantic memory, and crystallized verbal ability.

Results were informative for each of the five research questions. First, mortality-related cognitive deficits were magnified with increasing age. Old-Old decedents exhibited steeper decline compared with similarly aged and younger survivors. Further, multilevel analyses demonstrated that Decedents declined at significantly faster rates per

year increase in age. For the second research question, terminal decline was found to differentially influence select cognitive measures. Relative to Survivors, Old-Old Decedents displayed large variation across measures exhibiting poorer performance for verbal speed and episodic memory with considerably better performance for vocabulary. Results for the third research goal demonstrated that specific cause of death differentially influenced cognitive performance. Greater cross-sectional differences and declines were found for those who died of cardiovascular disease (CVD). A fourth contribution to the terminal decline literature found that the shape of cognitive decline for Decedents was accelerated in closer proximity to death. Evidence for the final research question revealed that impending death, presence of CVD, and older age were all associated with increased performance inconsistency. Considered together, these results provide both converging evidence and novel contributions to the terminal decline literature.

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Dedication

Dedicated with love to my parents, Jane and George MacDonald.

For I am what they have given; my accomplishments are theirs.

Chapter I

INTRODUCTION

Research evidence suggests a robust association between cognitive functioning and proximity to death in older adults. This observed cognition-mortality association has critical implications for identifying sources of cognitive decline. Most notably, patterns of decline attributed to age may actually reflect impending death. If true, reports of age-related differences in cognitive performance may be overestimated due to influences reflecting “terminal decline” (Kleemeier, 1962). The terminal decline hypothesis states that cognitive deficits do not reflect age per se but are instead a function of time to death. The argument is that most people maintain stable or slightly declining functions into old age with more marked decline indicating impending mortality. Moreover, important distinctions have been proposed to differentiate the magnitude of cognitive decline preceding death. Terminal decline represents a steady linear decline whereas terminal drop reflects an accelerated curvilinear decline (Palmore & Cleveland, 1976).

Although well-documented for the past four decades, there has been a recent resurgence of interest in the association between cognitive performance and time to death (e.g., Anstey, Luszcz, Giles, & Andrews, 2001; Berg, 1996; Bosworth & Schaie, 1999; Small & Bäckman, 1997, 1999). This renewed interest reflects several precipitating influences. First, from a theoretical perspective, a better understanding of cognition-mortality relations will also inform understanding of age-cognition relations. Chronological age is not a meaningful independent variable that explains variance in cognitive performance; rather, it is a simple proxy for other underlying biological and environmental influences that operate along the age continuum (MacDonald, Dixon,

Cohen, & Hazlitt, in press; Salthouse, 1999). Thus, examining impending mortality and factors underlying it (e.g., chronic disease) may help us more directly pinpoint sources of cognitive decline. Second, many unanswered questions remain from previous examinations of cognitive performance and proximity to death (for a review, see Small & Bäckman, 1999). Unresolved issues in the time to death literature include: (a) whether mortality-related deficits are similar across the latter part of the adult life span (e.g., those aged 55 to 90); (b) whether the impact of impending mortality on performance is differential across various cognitive domains (e.g., more pronounced declines for crystallized vs. fluid abilities); (c) whether specific causes of death (e.g., cardiovascular vs. non-cardiovascular disease) influence the magnitude of observed cognitive deficits; (d) whether individuals closer to death show accelerated cognitive declines consistent with terminal drop; and (e) whether time to death is associated with increased performance inconsistency, a potential indicator of central nervous system (CNS) atrophy (Hultsch & MacDonald, in press).

This study uses multi-wave data from the Victoria Longitudinal Study (VLS) to examine these neglected aspects of the cognitive performance-time to death association. Consistent with the purpose of a dissertation, novel contributions to the time to death literature will be made in several ways: (a) by re-examining recent findings that have yet to be replicated; (b) by extending recent findings using new analyses of longitudinal data; and (c) by investigating novel research questions not previously considered. I begin by reviewing select findings documenting cognition-mortality relations. After reviewing relevant literature, core hypotheses are outlined and potential unique contributions to the extant literature are discussed.

Chapter II

REVIEW OF THE LITERATURE

Documenting Relations between Cognitive Functioning and Mortality

In a recent review chapter, Berg (1996) summarized over three decades of research on aging and terminal decline. The following sections provide a brief overview of relevant literature on cognition-mortality relations. Although survival shares known associations with many constructs including personality (e.g., Field & Schaie, 1985; Maier & Smith, 1999) and health (e.g., Korten et al., 1999), the present review focuses exclusively on cognitive functioning.

Seminal Findings

The first investigation of cognition-mortality relations was conducted by Kleemeier (1962). Seventy men who lived in an institution for the elderly were administered the Wechsler-Bellevue intelligence test battery. During a 12-year period, the battery was administered on at least 2 occasions. For both survivors and decedents, the mean age at the last testing occasion was 79 years (approximately 50% of the original sample had died). To equate for differential intervals between test administrations, a mean annual rate of change for each individual was calculated. Kleemeier observed considerable individual differences in intelligence over time. Significant declines in performance subtest change scores were observed for decedents compared with survivors. Moreover, although only a few men ($n = 13$) completed the battery more than two times, performance curves for decedents who died shortly after the final testing occasion were accelerated relative to survivors. These observed differences between terminal decline and drop profiles can be considered conservative given the number of survivors also

approaching death.

Kleemeier proposed a novel interpretation to account for these findings. Rather than implicating age as the mechanism accounting for cognitive decline, he suggested that observed individual differences in intelligence were largely mediated by impending death. From these seminal findings, he proposed the terminal decline hypothesis to account for cognitive performance-mortality associations. He argued that late-life changes in cognitive performance are not directly a function of age but instead reflect distance to death. The implications of this investigation were and still remain wide-reaching for the field of cognitive aging. Age group differences in cognitive performance may be overestimated with poorer performance observed for older age groups because likelihood of death increases with age (Riegel & Riegel, 1972). Following from this assertion, age group differences in cognitive performance should attenuate if participants in close proximity to death are excluded from cross-sectional analyses.

Kleemeier's investigation spawned numerous studies examining the association between cognitive abilities and mortality. Representative findings are reviewed in turn.

Robust Relations Between Cognitive Performance and Impending Death

Early research focused on documenting the nature of the association between cognition and time to death. By the 1970's, comprehensive reviews of this literature were available (e.g., Botwinick, 1973; Palmore & Jeffers, 1971; Siegler, 1975) outlining numerous replications of Kleemeier's (1962) original findings. For example, Botwinick, West, and Storandt (1978) found decedents performed more poorly than survivors for perceptual and psychomotor tasks. Not surprisingly, significant differences were also found between decedents and survivors on measures of personality and health. However,

despite many similar demonstrations of the robust relation between mortality and cognitive functioning (for a review, see Berg, 1996), a number of questions remained unanswered.

Does Mortality Uniformly Influence Cognitive Performance?

Unanswered queries of particular relevance to the terminal decline hypothesis include whether impending mortality uniformly influences cognitive performance across age, cognitive domain, and cause of death. The following sections examine critical aspects of each of these relationships.

Age- and mortality-related cognitive deficits. Deriving from early research in this area, a key question focused on whether impending death influenced cognitive decline similarly across the adult age continuum spanning young-old (55 to 64 years), mid-old (65 to 74 years), and old-old (75 years and older) age cohorts. Riegel and Riegel (1972) suggested that the association between mortality and cognitive performance may be of greater magnitude among young-old adults compared with those older than 65 years. Their hypothesis was based on defining characteristics of mortality for each age group. Specifically, Riegel and Riegel suggested that because death is more random for old-old adults than for young-old, there should be less-marked performance differences between survivors and decedents (i.e., both old-old groups are closer to their life expectancy and have experienced considerable cognitive decline).

The general claim is that old-old participants experience a weakening of many biological systems as well as decline in physiological vigor. Thus, irrespective of cause (e.g., heart failure, stroke, compromised immune system), the actual death event occurs largely at random (Cunningham & Brookbank, 1988). In contrast, among the young-old,

death strikes individuals who are clearly differentiable from same-aged survivors with regard to psychological qualities (including memory and cognition). Specific diseases typically underlie death in young-old and mid-old adults. As a consequence, impending death for young-old participants (far removed from their natural life expectancy) would be expected to attenuate cognitive performance relative to normative same-aged survivors. Riegel and Riegel's (1972) findings supported this hypothesis. As expected, cross-sectional analysis of scores from the first measurement occasion revealed significant differences in verbal ability between survivors and decedents. Moreover, compared with old-old decedents, young-old decedents had lower verbal test scores suggesting that their performance is more markedly influenced by impending death.

Recent findings, however, are inconsistent with the claim that mortality-related cognitive deficits are strongest among the young-old. For example, impending mortality has been found to influence cognitive performance for individuals older than 70 years of age (Anstey et al., 2001; Bosworth, Schaie, & Willis, 1999; Small & Bäckman, 1997) and even for persons considered very-old (over 90 years of age; Maier & Smith, 1999). The limited number of measures used by Riegel and Riegel represents one possible reason for these discrepant findings. They used five verbal achievement measures (synonyms, antonyms, selections, classification, and analogies) that indexed crystallized verbal abilities. In contrast, aforementioned studies demonstrating cognition-mortality relations for those 70 years and older used more diverse cognitive performance measures examining both fluid and crystallized abilities. Clearly, addressing whether cognition-mortality associations hold uniformly across the adult age range requires a broader assessment of cognitive measures.

Are mortality-related deficits observed across cognitive tasks? Subsequent investigations have examined invariance of cognition-mortality relations across various cognitive performance domains. Of particular interest is whether impending death exerts a general or specific influence on cognitive performance. White and Cunningham (1988) hypothesized that mortality-related deficits should be greatest for intellectual abilities typically unaffected by age (e.g., vocabulary) relative to those that demonstrate normal age decrements (e.g., processing speed). Cognitive performance for 97 participants who died within 7 years of testing was compared to a matched-group (age, gender, education) of survivors. They examined whether mortality exerted a greater influence on crystallized vocabulary performance compared with fluid processing speed. Cross-sectional findings indicated that impending death influenced vocabulary performance alone and only for those who died prior to age 70. Moreover, the mortality-cognition association was restricted to those who died within 2 years of testing (a narrower interval than the original 5 years proposed by Riegel and Riegel, 1972). In contrast, no significant differences were observed between survivors and decedents on perceptual speed; changes in speed appear to be a function of normal aging as opposed to terminal drop.

Other recent findings also support the claim that terminal drop exerts domain-specific influences. Small and Bäckman (1997) found that impending death shared stronger associations with cognitively-supported tests of recognition memory that exhibit modest age-related decrements compared with free-recall memory tasks that exhibit larger age-related decrements. Performance on other memory tests resistant to normative aging declines (e.g., Digit Span as an indicator of primary memory) have also

demonstrated mortality-related deficits (Johansson & Berg, 1989). These findings contradict a strong version of the terminal decline hypothesis which suggests that impending mortality exerts a pervasive influence on cognitive abilities. Rather, such patterns imply that: (a) impending death influences select cognitive abilities typically resistant to the aging process (e.g., crystallized vs. fluid, recognition vs. recall); and (b) that age differences for the young-old and mid-old on such age-resistant abilities are indicative of influences other than normal aging (e.g., terminal decline).

However, some findings contradict the cognitive specificity of terminal drop. For example, Anstey and colleagues (2001) examined associations between mortality and cognitive performance across a continuum of measures including verbal ability (e.g., similarities), memory (e.g., symbol recall), and processing speed (e.g., digit symbol substitution). Results indicated that poor performance for each of these cognitive measures was associated with increased mortality risk. Other investigations have also demonstrated similar effects across different cognitive domains indexed by tests of verbal fluency, primary memory, episodic memory, visuospatial ability, and general cognitive ability (e.g., Small, Fratiglioni, von Strauss, & Bäckman, in press). Both cross-sectional and longitudinal results indicated that decedents performed more poorly for all cognitive measures. Maier and Smith (1999) also demonstrated mortality-related deficits for age-sensitive tasks including reaction time and perceptual speed. These findings are inconsistent with the White and Cunningham (1988) hypothesis suggesting that only specific cognitive abilities are related to time to death. Rather, observed mortality-cognition associations across both process and product measures imply the general nature of this phenomenon.

In light of these discrepant findings, additional research across a broad continuum of cognitive measures is required to determine whether mortality exerts a general influence on cognitive functioning.

Does cause of death influence mortality-related deficits? A third unanswered question concerns the association between actual cause of death and cognitive performance. Until quite recently, examining cause of death as a mediator of the mortality-cognition relationship has received little empirical attention. This is surprising because there are obvious theoretical and empirical links between health and cognitive functioning (e.g., Anstey & Christensen, 2000; van Boxtel et al., 1998) as well as chronic illness/disease and mortality itself (e.g., Korten et al., 1999). Some studies report an attenuated relationship between mortality and select cognitive measures after controlling for self-reported health measures (e.g., Smits, Deeg, Kriegsman, & Schmand, 1999) although others have failed to demonstrate such a mediating influence (e.g., Maier & Smith, 1999). In a recent investigation, Anstey and colleagues (2001) demonstrated that the influence of impending mortality on cognitive performance could be explained by measures of self-rated health and disease. Data were collected for over 60 medical conditions reflecting 3 primary disease variables of interest: neurological conditions, cardiovascular conditions, and number of medications taken. Neurological and cardiovascular conditions were of particular interest as they have been shown to predict both mortality and cognitive function (e.g., Haan, Shemanski, Jagust, Manolio, & Kuller, 1999). After partialling measures of self-reported disease and health, previously significant cognitive measures no longer predicted mortality as an outcome. This finding is consistent with the view that disease processes underlie, in part, mortality-cognition

associations. However, even after controlling for self-reported disease variables, a verbal measure (similarities), fluid process measure (processing speed), and a measure of general cognitive ability (MMSE) remained significant predictors of mortality. Thus, in addition to reflecting disease, Anstey and colleagues suggested that poor cognitive performance may also reflect biological aging processes that occur independent of disease; the significant prediction of mortality by these diverse cognitive measures implies shared variance independent of disease perhaps reflecting a biological common cause.

Berg (1996) noted that most investigations in this area do not discuss underlying mechanisms, nor do many even report simple summary statistics including the health or disease status of participants. However, several possible sources of mortality-related deficits have been offered. One possibility is that specific disease processes underlie observed deficits. However, an equally plausible source posits a general biological breakdown reflecting basic aging processes (termed biological vitality). To date, a single investigation has considered the possible link between cause of death and mortality-related cognitive deficits (Small et al., in press). This investigation represents the first attempt to move beyond descriptive assessments of the mortality-cognition association to focus on possible sources underlying the terminal decline phenomenon. To examine cause of death as a possible source of mortality-related cognitive decline, they categorized individuals for whom they had previously obtained performance data into two cause-of-death groups: those who died from cardio- or cerebrovascular diseases (CVD; e.g., heart attack, stroke) versus those who died of other causes (non-CVD; e.g., cancer). Cognitive performance was assessed by a test of global cognitive functioning,

episodic memory, primary memory, visuospatial ability, and verbal fluency. Consistent with expectations, survivors performed better than decedents for all cognitive measures, both cross-sectionally and longitudinally. However, cause of death did not differentially influence baseline cognitive performance for decedents who died before the second testing occasion (i.e., less than 3-years after testing) compared with those who died at least 3-years later, nor did cause of death influence longitudinal cognitive declines (i.e., no cause of death by time interaction for decedents alone). These preliminary results suggest that the association between impending death and cognitive functioning is not disease specific but rather reflects a general influence consistent with Berg's (1996) hypothesized general biological breakdown. However, it should be emphasized that the cause of death categories used (CVD vs. non-CVD) were potentially too broad to properly index specific disease effects. Design limitations notwithstanding (e.g., few decedents, large variation in causes of death), future investigations must further examine the issue of specific vs. general influences on cognitive decline.

Other Unresolved Issues in Terminal Decline Research

In addition to the identified age group, cognitive domain, and cause of death discrepancies, other critical aspects of the terminal decline phenomenon require further study. The first concerns proper attention to the longitudinal analysis of intraindividual terminal decline profiles, and the second focuses on potential relations between terminal decline and other conceptually-related correlates.

Is proximity to death associated with accelerated cognitive decline? Berg (1996) outlined two general approaches for examining the terminal decline hypothesis: (a) a cross-sectional analysis of longitudinal information using techniques such as Cox

regression or survival analysis; and (b) a true longitudinal analysis of longitudinal data using techniques such as hierarchical linear modeling (HLM). Most investigations use the former approach. Typically, data from a single measurement point is categorized on the basis of distance from death (e.g., those who are more than 3 years from death vs. those who are less than 3 years) or survival status (e.g., survivor vs. decedent).

Unfortunately, cross-sectional analysis of longitudinal data is the weaker of the two designs and can only speak to the presence or absence of associations between cognitive performance and group category (e.g., survival status or distance from death).

Ultimately, analysis of longitudinal data is required to track cognitive performance over time and to examine its covariation with time to death (e.g., number of years). Of critical importance, longitudinal designs permit assessment of both linear and curvilinear declines related to death. Among other benefits, this permits a statistical test of terminal-decline versus terminal-drop accounts of mortality-related deficits. For example, with increasing proximity to death, does decline reflect a slow and steady linear function or does it more characteristically reflect accelerating curvilinear decline (Palmore & Cleveland, 1976). Few studies differentiate conceptually or empirically between these two possibilities (Berg, 1996).

Many investigations have considered associations between level of performance and mortality. In contrast, considerably fewer studies have examined longitudinal change in cognitive performance preceding death (e.g., Deeg, Hofman, & Van Zonneveld, 1990; Jarvik & Blum, 1971; Swan, Carmelli, & LaRue, 1995). An early longitudinal study by Jarvik and Falek (1963) examined associations between mortality status and longitudinal patterns of cognitive performance. Reports indicated a positive association between

survival and longitudinal cognitive stability for a 12-year follow-up of twin pairs. Vocabulary, similarities and digit symbol performance were examined from the Wechsler-Bellevue Intelligence test battery; individuals who declined significantly on two or three measures were more likely to die 5 years after the last measurement occasion. Notably, cognitive decline was not examined as a continuous variable for this analysis; annual rate of decline was used to define stability and critical loss.

More recent studies have employed advanced statistical techniques to examine mortality effects on cognitive functioning over time. For example, Bosworth and Schaie (1999) examined survival effects for cognitive performance data from the Seattle Longitudinal Study. The sample consisted of 605 decedents and 613 survivors comparable in age and education. For their respective final occasions of measurement, decedents were hypothesized to exhibit diminished levels of cognitive performance relative to survivors. Analysis of covariance (ANCOVA), controlling for education, was used to examine age and survival group effects at cross-section. Consistent with previous results, survivors significantly outperformed decedents for a variety of cognitive domains reflecting crystallized abilities, visualization abilities, perceptual speed, and psychomotor speed. Significant interactions were also observed between survival status and age indicating that the oldest decedents (75 years and older) exhibited lower levels of crystallized ability and psychomotor speed relative to same-aged survivors. Interestingly, little difference in cognitive performance was observed between survivors and decedents younger than 75 years old.

Complementing these cross-sectional findings, Bosworth and Schaie (1999) examined longitudinal data spanning 14 years and 3 waves of measurement. Decedents

were hypothesized to exhibit greater cognitive declines relative to survivors across the preceding 7- and 14-year intervals. In the first analysis, repeated-measures analysis of covariance (RMANCOVA) was used to examine age group and survival status differences in cognitive change across the last 2 measurement occasions (a 7-year interval). To control for pre-existing differences in ability, education and initial baseline performance were entered as covariates. Decedents exhibited greater declines relative to survivors for tests of verbal meaning and psychomotor speed. In addition, a significant age group by survival status interaction was observed for crystallized numerical ability: decedents 75 years and older showed the most decline across the 7-year interval. Parallel analyses were performed to examine rate of change across the last 3 measurement occasions (14 years) yielding the same results.

Bosworth and Schaie (1999) interpreted patterns observed in both the cross-sectional and longitudinal results to suggest that survival effects are ability specific. Of particular interest, the longitudinal test of the terminal decline hypothesis was only partially supported. For example, examination of fluid abilities produced no marked differences between survivors and decedents; both groups demonstrated age-related declines. In contrast, for tests of crystallized ability (recognition vocabulary), significant differences in decline were magnified for those 75 years and older. Relative to same-aged survivors, older decedents consistently demonstrated lower levels and greater decline in cognitive performance. For younger age groups, decedents performed only slightly below survivors. As verbal ability is known to be less sensitive to age than other cognitive abilities, Bosworth and Schaie suggested that observed declines on this and related verbal tasks may signal impending death (consistent with the White and

Cunningham hypothesis). Notably, the differential influence of mortality as a function of age is consistent with previous claims suggesting that mortality-cognitive functioning associations are more pronounced in older adults (e.g., Cooney, T.M., Schaie, K.W., & Willis, S.L., 1988). In part, this magnified association for the old-old may reflect protracted chronic disease processes. Observed dissociations between cross-sectional and longitudinal findings underscore the importance of further longitudinal analysis. Indeed, collapsing across information at the between-subjects level may conceal true patterns of intraindividual change (e.g., Hultsch, Hertzog, Dixon, & Small, 1998; Sliwinski & Hofer, 2001).

Specific survival analysis techniques have also been used to examine the influence of mortality on cognitive performance across time. For example, Small and Bäckman (1997) used Cox regression to examine whether cognitive performance predicted mortality status (survivor vs decedent) across a longitudinal follow-up period. This approach represents an improvement over examination of performance level alone; time to death from the first occasion of measurement was included in the regression model. Specifically, the model tested whether individuals with certain cognitive performance scores on the first measurement occasion were at increased risk of death over the follow-up period. Potential confounds including demographic and health-related variables were included as covariates. Two crystallized measures of cognition, word recognition and category fluency, independently predicted mortality over the follow-up interval. Based on their findings, Small and Bäckman concluded that not all cognitive tasks were affected by proximity to death; they suggested that clear deficits in typically age-resistant tasks may reflect conditions other than normal aging (e.g., impending death,

dementia). However, in contrast to these ability-specific findings, other comparable Cox regression investigations have found that poor performance across a continuum of cognitive measures predicts increased mortality risk over both 4- and 6-year follow-up periods (e.g., Anstey et al., 2001).

Of the few longitudinal studies that have examined mortality in relation to longitudinal decline, even fewer have properly addressed the issue of terminal drop (i.e., curvilinear as opposed to linear decline) related to distance to death (Berg, 1996). To address this issue, longitudinal studies must focus on the shape of decline. Using data from the Duke Longitudinal Study, Palmore and Cleveland (1976) reported significant linear terminal decline but no curvilinear terminal drop for intelligence test scores. Results from the Gothenburg Longitudinal Study support a terminal drop interpretation but are limited to three measurement occasions (Berg, 1987). Over an 11-year interval, declines in vocabulary and inductive reasoning performance were greater for decedents relative to survivors. Interestingly, the terminal drop pattern was particularly pronounced for older decedents on the vocabulary measure. In comparison, survivors showed little decline on vocabulary but did exhibit decline on the fluid measure.

A more recent study by Anstey and colleagues (Anstey et al., 2001) used an innovative approach for examining evidence in favour of terminal drop. They used Cox regression to examine both level and change in cognitive performance as predictors of mortality. A variety of cognitive predictors were used including tests of verbal ability, processing speed, and memory recall as well as a cognitive composite formed by summing all raw test scores. As noted by Berg (1996), the Cox regression approach represents a cross-sectional investigation of longitudinal mortality data. Typically,

mortality status is ascertained over a specified longitudinal interval and then cross-sectional estimates of cognitive performance (e.g., at baseline) are used to predict risk of mortality. Anstey and colleagues used change scores reflecting 2-year decline in cognitive performance as predictors of subsequent mortality 4 years later. Participants were classified into change score distribution quintiles according to the magnitude of decline exhibited over the 2-year period. The top quintile group (i.e., those exhibiting the least 2-year decline) was chosen as the reference group with the lowest quintile group exhibiting the most decline. For each variable, incident rate ratios (IRRs) were then calculated by comparing the bottom quintile exhibiting the most decline with the top-performing quintile to specifically test whether decline relative to the reference group predicted increased mortality risk. Significant decline was defined as the distribution quintile exhibiting the largest negative change scores. Even after controlling for age, education, and gender, significant declines for the similarities and cognitive composite measures were associated with an increased IRR for mortality. Relative to the reference group, the lowest quintile group experienced a 40% increase in death rate predicted by significant declines in similarities and a 66% increase in death rate predicted by significant cognitive-composite declines. These observed associations between cognitive decline and increased mortality risk are consistent with a terminal drop pattern of accelerated decline before death.

Is proximity to death associated with increased performance inconsistency? A second critical issue for investigation concerns potential relations between terminal decline and other correlates. Investigation of the terminal decline phenomenon has been largely descriptive. However, preliminary evidence suggests that, at least conceptually,

the terminal decline phenomenon may be related to multiple factors including disease, health, and central nervous system function. For example, Riegel and Riegel (1972) proposed the “biological terminal drop-mortality model” which suggests that terminal decline might be a function of physiological or CNS deterioration. More recently, Anstey and colleagues (Anstey et al., 2001) also suggested that the association between cognitive decline and terminal drop preceding death may reflect disease or general biological aging. A parallel literature has examined similar associations between an hypothesized behavioural marker of neural atrophy (i.e., within-person performance variability) and cognitive decline (e.g., Hultsch, MacDonald, & Dixon, 2002). Intraindividual variability, or inconsistency (e.g., Hultsch & MacDonald, in press), refers to within-person change that is relatively rapid and transient (e.g., emotions, moods, fluctuations in performance) over multiple trials or occasions. This section outlines important links between these parallel literatures.

Although not previously examined, there are important reasons for considering terminal decline in relation to performance inconsistency. First, similar to terminal decline (c.f., Riegel & Riegel, 1972), neural deterioration may underlie documented increases in inconsistency. Indeed, inconsistency has been hypothesized to reflect several underlying influences including neural “noise” in the transmission of CNS signals (Hendrickson, 1982), impaired catecholaminergic function (e.g., Li & Lindenberger, 1999), or common-cause atrophy of CNS function (Lindenberger & Baltes, 1994). It is conceivable that terminal decline and inconsistency share the same or similar underlying mechanisms. Second, inconsistency has been shown to increase with age (Hultsch et al., 2002) and as a function of neurological impairment including traumatic brain injury

(Stuss, Pogue, Buckle, & Bondar, 1994) and Alzheimer's disease (Hultsch, MacDonald, Hunter, Levy-Bencheton, & Strauss, 2000). Terminal decline patterns may also increase with age given the greater likelihood of mortality: another potentially important link between the two phenomena. Third, and perhaps most compelling, inconsistency shares strong associations with cognitive decline. Age-related increases in performance inconsistency are associated with poorer mean cognitive performance (Hultsch et al., 2000, 2002; Li, Aggen, Nesselrode, & Baltes, 2001; Rabbitt, 2000) and lower levels of general intelligence (Jensen, 1982; Rabbitt, Osman, Moore, & Stollery, 2001). For example, Rabbitt and colleagues (2001) found that greater within- and across-session inconsistency for older adults' reaction time on a letter identification task was associated with poorer performance on the Culture Fair Intelligence Test. Similarly, Hultsch et al. (2002) found greater reaction time inconsistency was correlated with poorer mean performance for various cognitive measures including perceptual speed, working memory, episodic memory, and crystallized abilities. In addition to these cross-sectional findings, MacDonald and colleagues (MacDonald, Hultsch, & Dixon, in press) found that longitudinal increases in inconsistency over a 6-year period shared significant associations with cognitive decline.

Given independent associations between time to death (i.e., terminal decline) and cognitive performance on the one hand, and inconsistency and cognitive performance on the other, the next logical step is to examine associations between terminal decline and inconsistency. Observed associations between factors reflecting biological aging and mortality (e.g., Anstey et al., 2001) imply that future research should empirically test associations between mortality and behavioural index measures of CNS deterioration. If

performance inconsistency truly represents a marker of CNS disturbance, systematic associations would be expected between inconsistency, time to death, and mortality status. To date, no investigations have examined these relationships.

The Present Investigation: From What We Know to What We Need To Know

The preceding literature review has described robust associations between cognitive performance and mortality, addressed some unanswered questions related to the uniformity of this association across conditions (e.g., age groups, cognitive domain, cause of death), and outlined several shortcomings of terminal decline investigations. In a recent review, Small and Bäckman (1999) summarized critical unanswered issues associated with the terminal decline phenomenon and proposed future avenues of research. In particular, they argued that terminal decline research should include a broader adult age spectrum spanning young-old to very-old (e.g., 55 to 90 years or older), a wider range of cognitive ability measures, and a closer consideration of actual cause of death as an influence of mortality-related cognitive deficits. Consideration of these and related factors may serve two purposes: (a) provide a better understanding of terminal decline and its characteristic nature; and (b) assist with identification of sources underlying mortality-related cognitive deficits. Addressing lacunae in the literature, including inadequate longitudinal investigation of the phenomenon as well an obvious failure to examine terminal decline in the context of other potentially related literatures (e.g., markers of neurological disturbance such as performance inconsistency), will also facilitate these two purposes. These previously unresolved and unaddressed issues represent the foundation for the present study.

Chapter III

OVERVIEW, OBJECTIVES, AND HYPOTHESES

Research Rationale

Critical facets of the terminal decline phenomenon are not well understood. Five primary research questions were identified to examine these issues: (a) to ascertain whether terminal decline is evident across a broad age continuum; (b) to investigate whether mortality-related deficits vary as a function of cognitive domain; (c) to examine whether cause of death differentially influences the magnitude of cognitive decline; (d) to explore whether the shape of terminal decline is mediated by age, time to death, or cause of death; and (e) to consider whether decedents exhibit increased intraindividual variability in cognitive performance relative to survivors. VLS data afford many advantages for investigating these core research questions including a diverse range of cognitive measures, multiple times of measurement, and adequate numbers of deceased participants. The following sections outline each proposed research question as well as associated hypotheses and expected empirical outcomes. Each question identified addresses critical gaps in the terminal decline literature. Where possible, I discuss how answers to these research questions will uniquely contribute to better understanding associations between cognitive performance and time to death.

Question #1: Are mortality-related cognitive deficits uniform across age? Riegel and Riegel (1972) suggested that the impact of impending mortality on cognitive performance is attenuated for the old-old (those 75 years of age and older). They argued that death strikes more randomly at higher ages with fewer differences between survivors and decedents. In comparison, young-old (55 to 64 years) and mid-old (65 to 74 years)

adults should show more pronounced patterns of terminal decline because younger decedents differ in a variety of capacities relative to same-aged and older survivors. For example, individuals who die in their 50's or 60's often suffer specific acute illness or disease setting them apart in many ways (e.g., cognitively) relative to their surviving cohort. With increasing age, death becomes more normative, sources of death tend to be more random, and individual differences in cognitive performance between survivors and decedents may be reduced. At odds with this hypothesis, recent investigations have consistently demonstrated mortality-related deficits for those 75 years and older (e.g., Bosworth & Schaie, 1999; Johansson & Zarit, 1997; Maier & Smith, 1999; Small & Bäckman, 1997). Notably, a strong test of Riegel and Riegel's hypothesis using a broad continuum of older adults is lacking (Small et al., in press).

Thus, a defining question asks whether terminal decline is evident at all points across the adult life span or whether the magnitude of mortality-related cognitive deficits varies as a function of age. A particular strength of the VLS data set is its inclusion of multiple age cohorts spanning a broad segment of the elderly adult life span. The present investigation permits examination of mortality-related deficits for both young-old (59-79 years) and old-old (80-95 years) adults. Unique contributions will be made to the terminal decline literature by assessing whether mortality-related cognitive deficits are attenuated for only select age groups.

Hypothesis 1: It is hypothesized that impending death will negatively impact cognitive performance regardless of age. Specifically, terminal decline effects are expected for both young-old and old-old decedents. This prediction follows from the belief that factors underlying mortality-related deficits are not age specific but rather

reflect other sources (e.g., type of cognitive measure, cause of death).

Question #2: Are mortality-related cognitive deficits uniform across cognitive domain? A second research objective concerns whether performance in various cognitive domains is similarly or differentially influenced by time to death. Obvious age-related performance distinctions can be drawn between cognitive processes and products (Hultsch et al., 1998). Fluid process measures begin to exhibit decline as early as middle adulthood (e.g., Schaie, 1996). Given such early onset of age-normative decline, both decedents and survivors above 55 years of age are expected to exhibit cognitive deficits. In contrast, crystallized products of cognition (e.g., vocabulary) do not typically exhibit age-related declines until the 7th decade of life (e.g., Schaie, 1996). As a consequence of observed differences between age-normative trends for fluid and crystallized abilities, some researchers have suggested that terminal decline effects will be more pronounced for age-resistant cognitive performance measures (e.g., White & Cunningham, 1988). Evidence has been found to support the claim that typically well-preserved abilities exhibit the largest mortality-related effects (e.g., Bosworth & Schaie, 1999; Small & Bäckman, 1997; White & Cunningham, 1988); such deficits are suggested to reflect pathology and impending death as opposed to normative age-related changes. Unfortunately, these issues remain unresolved. Many studies have examined only select measures on a restricted continuum of processes to products of cognition (e.g., fluid to crystallized intelligence), although some investigations have recently used expanded measurement batteries (e.g., Bosworth & Schaie, 1999; Bosworth et al., 1999; Small et al., in press). Limited measurement batteries have restricted examination of cognitive-deficit specificity.

Thus, a second defining question concerns whether performance across various cognitive domains (reflecting both fluid and crystallized skills) is differentially influenced by mortality. Of particular interest is whether impending death influences abilities both well-preserved in old age (e.g., semantic memory, vocabulary) as well as those that show normal age decrements (e.g., working memory, processing speed). VLS data afford several strengths for examining this issue. Multiple domains of cognitive performance are examined, with each of these domains exhibiting diversity in the magnitude of normative age-related decrements. This broad continuum of tasks permits examination of whether impending death influences cognition globally (i.e., across all cognitive measures in VLS battery) or locally (i.e., across select VLS measures). In addition to examining differences in level, an equally important question concerns whether mortality-related cognitive change increases for select tasks relative to others. A unique contribution will be made to the literature by testing the specificity hypothesis across a wide range of cognitive measures within the same sample.

Hypothesis #2: It is hypothesized that mortality-related influences: (a) will be observed across cognitive domains for both process and product measures; (b) will be larger for older adults for typically age-resistant cognitive tasks (i.e., age by cognitive domain interaction), and (c) will be apparent at both cross-section and over time.

Question #3: Does cause of death influence mortality-related cognitive deficits?

A third objective focuses on whether cause of death differentially influences patterns of terminal decline in cognitive performance. Until quite recently, cause of death had not been examined as a possible mediating factor. This is surprising given well-documented associations between cognitive performance and chronic disease (e.g., Hertzog, Schaie, &

Gribbin, 1978; van Boxtel et al., 1998) as well as demonstrated links among health, cognition, and life expectancy in the elderly (e.g., Korten et al., 1999). These patterns imply that health status, and perhaps one's ultimate cause of mortality (e.g., cardiovascular disease), may facilitate understanding of mortality-cognition associations in old age. Among causes of death, cardiovascular disease is of particular interest as it has been shown to predict both mortality and cognitive decline (e.g., Haan, Shemanski, Jagust, Manolio, & Kuller, 1999; Hassing et al., 2002; Hertzog et al., 1978).

To date, only one investigation has directly examined associations between cause of death and terminal decline. Small and colleagues (in press) found that magnitude of cognitive deficits did not differ for those who died from cardiovascular diseases (CVD; e.g., heart attack, stroke) relative to other non-cardiovascular causes (non-CVD; e.g., infectious diseases, cancer). This pattern favours a general compared with disease-specific account of terminal decline. However, in a related study, Hassing and colleagues (2002) found that only three of six initial cognitive measures remained significant predictors of survival after adjusting for stroke and cardiovascular indicators. These findings imply that cerebro- and cardiovascular indicators underlie terminal decline.

To address this unresolved issue, the third defining question of this study considers whether: (a) cause of death shares systematic associations with the magnitude of mortality-related cognitive deficits; or (b) whether various causes of death share similar relations. Berg (1996) noted that if mortality-related deficits are associated with a large number of causes of death, this implicates a global influence such as biological vitality as opposed to a select underlying pathology. It is also conceivable that cause of death may differentially impact cognitive performance for the Young-Old compared with

the Old-Old. Precipitating causes may more forcefully influence cognitive functioning in younger groups of elderly adults (if, for example, the source of mortality-related deficits is more common among the Old-Old). Thus, it is critical to examine whether cognitive performance varies as a function of both age and cause of death. This proposed analysis represents the first attempt to replicate the findings of Small and colleagues (Small et al., in press). It will uniquely contribute to the literature by: (a) examining associations across a broader age range (the participants Small and colleagues examined were 75 to 95 years of age); and (b) considering whether cognitive decline is accelerated for specific causes.

Hypothesis #3: It is hypothesized that mortality-related cognitive deficits will be magnified for those who died of CVD-related causes.

Question #4: Do age, mortality status, or proximity to death influence shape of terminal decline? As potentially the most significant contribution, longitudinal associations between cognitive performance and time to death will be examined. Proximity to death may yield several longitudinal performance trends including “terminal decline” (exhibiting steady linear decreases) or “terminal drop” (reflecting accelerated curvilinear decreases) just prior to death. Although Palmore and Cleveland (1976) stressed the distinction between terminal decline and terminal drop, in practice the terms are used interchangeably (Berg, 1996).

The definitive question of interest concerns whether individuals closer to death show accelerated cognitive decline consistent with the definition of terminal drop. Previous studies have performed cross-sectional analyses of longitudinal data (e.g., survival analysis) or limited longitudinal analyses using repeated measures analysis of

variance; the present study proposes examination of across-time covariation between cognitive decline and actual time to death for decedents. Notably, this type of multilevel analysis of longitudinal mortality data has not been examined previously. In addition to examining associations between cognitive decline and years to death, analyses will also test whether decline is accelerated for specific causes of death or as a function of age group.

The VLS design will facilitate unique contributions to the terminal decline literature on at least three levels. First, longitudinal analysis of longitudinal data provides a stronger test of the terminal decline/drop hypothesis. The VLS data set includes as many as five waves of data for decedents and permits testing of curvilinear change. At minimum, three measurement occasions are required to examine the shape of change. For example, although Anstey and colleagues (Anstey et al., 2001) used cognitive change scores in a novel Cox regression approach for examining survival effects, not all change scores significantly predicted mortality. In part, these findings could reflect the restricted measurement frame. Estimates of change were based on only two measurement occasions and lacked sensitivity for demonstrating significant mortality associations (e.g., limited 2-year retest interval, regression to the mean, age-related increases in performance variability). Using multi-wave VLS data and relevant techniques (e.g., HLM), these analyses can detect curvilinear decline related to mortality informing us about the shape of change prior to death. Previous findings suggest examining the trajectory of decline for mortality-related deficits is important. For example, Small and colleagues (Small et al., in press) found that the influence of impending mortality on cognitive performance was only apparent within a relatively short time frame prior to death. Decedents who

died within 3 years of baseline testing (prior to the second wave of measurement) showed cognitive impairments whereas those who died after the second wave were indistinguishable from survivors. The precipitous decline often observed for the last wave of longitudinal studies is of particular interest; these performance decrements may reflect terminal drop.

Second, using multilevel statistical techniques, the VLS design permits examination of all performance data regardless of number of measurement waves completed. A disadvantage of listwise deletion techniques (e.g., repeated-measures analysis of variance) is the loss of important information for those who attrited from the study and subsequently died after a single occasion of measurement. For example, in Bosworth and Schaie's (1999) repeated measures investigation, 52% of decedents completed a single wave of measurement and then died preventing longitudinal examination of their performance. As a consequence, observed rates of change for decedents who completed two or more measurement occasions likely underestimated actual rate of change associated with terminal decline. Whenever possible, it is important to include all individuals for analysis of terminal decline.

Similarly, change score analyses also result in the exclusion of participants who complete a single occasion of measurement. For example, Anstey and colleagues (Anstey et al., 2001) examined attrition as a risk factor for mortality. They reported that patterns of missing data at baseline were associated with increased mortality risk. This finding has several implications: (a) associations between cognitive performance and mortality were likely underestimated; and (b) missing data and attrition potentially reflect underlying influences including disease and dementia. It is desirable, therefore, to

examine performance patterns for all available data (survivors and decedents) so that observed terminal decline effects are not underestimated. Although correct interpretation of mean differences requires that the number of participants at each wave be limited to those who share all data points (otherwise estimates of longitudinal decline may be overestimated; Siegler, 1975), analytic techniques such as HLM permit examination of individual rather than average growth curves.

Third, the VLS design implements relatively short 3-year retest intervals that are more sensitive for examining the shape of terminal decline. If decline is accelerated, shorter retest intervals facilitate a more accurate test of how many years prior to death curvilinear decline originates. Indeed, some authors suggest that influences of terminal drop are restricted to the last 5 years of life (e.g., Riegel & Riegel, 1972). Consequently, longer change intervals may not be sensitive to the proposed terminal drop phenomenon. Moreover, consider the hypothetical example of adults who die of sudden acute illnesses. These individuals may have been tested on one occasion, developed a terminal disease at some point after testing, and subsequently died thereby preventing follow-up testing. In such a scenario, even if multi-wave data were available, longer retest intervals (e.g., 5 years vs. 3 years) would be less sensitive to terminal decline patterns in cognitive performance data.

Hypothesis #4: Relative to previous investigations, the multiple advantages of the VLS design permit a more sensitive examination of linear (terminal decline) versus curvilinear (terminal drop) trends. Cognitive change will be examined in relation to number of years to death (a more sensitive predictor compared with wave of measurement). It is hypothesized that individual profiles of terminal decline will

accelerate with proximity to death (consistent with the terminal drop hypothesis) and that both age group and cause of death will moderate these profiles.

Question #5: Is survival status and proximity to death associated with inconsistency in cognitive performance? As a fifth and final objective, I will explore whether performance inconsistency for cognitive measures varies as a function of mortality status, age, cause of death, and time to death. Surprisingly, few studies have considered underlying sources of mortality-related cognitive deficits. To facilitate understanding of terminal decline, it is important to bridge time to death investigations with other literatures. Cognitive performance inconsistency represents a potentially promising phenomenon to examine in the context of mortality-related cognitive deficits. Previous findings have demonstrated that inconsistency increases as a function of age (Hultsch et al., in press), time (MacDonald et al., in press), and neurological disturbance (Hultsch et al., 2000) and is associated with poorer cognitive performance across various cognitive tasks. One possibility is that both terminal decline and performance inconsistency reflect a global vs. local underlying source (e.g., both are driven by a common endogenous mechanism such as CNS integrity). For example, Anstey and colleagues (2001) suggest that the association between cognitive decline and terminal drop preceding death may reflect biological aging; similar endogenous mechanisms have been postulated to underlie performance inconsistency (e.g., Hultsch et al., in press; Li et al., 2001). Given similar associations between time to death and cognitive performance on the one hand, and increased inconsistency and cognitive performance on the other (i.e., both are associated with poorer cognitive performance for older adults and with greater decline across time), it seems feasible that performance inconsistency and time to death

may share a direct association (see Figure 1).

The defining focus concerns the nature of the association between inconsistency and mortality. If both time to death-cognition and inconsistency-cognition pathways reflect general neurological disturbance with increasing age and proximity to death, a systematic association between time to death and inconsistency would be expected. Demonstrating such an association would be consistent with the biological vitality claim put forth by Berg (1996). Queries of particular interest concern whether inconsistency is a predictor of: (a) survival status in a Cox regression analysis; or (b) number of years to death in an HLM analysis (i.e., examination of time-varying covariation between change in inconsistency and proximity to death in years). These questions have not been considered previously.

Hypothesis #5: To examine the biological vitality account of cognitive decline, it is hypothesized that: (a) inconsistency in cognitive performance (as measured across trials of the lexical decision and semantic verification tasks) will increase for decedents compared with survivors at their last wave of testing; (b) age and mortality will interact with older decedents exhibiting the greatest performance variability; (c) inconsistency will predict survival status; (d) greater inconsistency will be associated with mortality status compared with cause of death (based on an assumption of global vs. local influences); and (e) longitudinal estimates of performance inconsistency should increase with increasing proximity to death (i.e., fewer years to live). If observed, relations among cognitive deficits, impending death, and inconsistency are compatible with a general CNS disturbance account of cognitive decline (e.g., breakdown in global neural integrity).

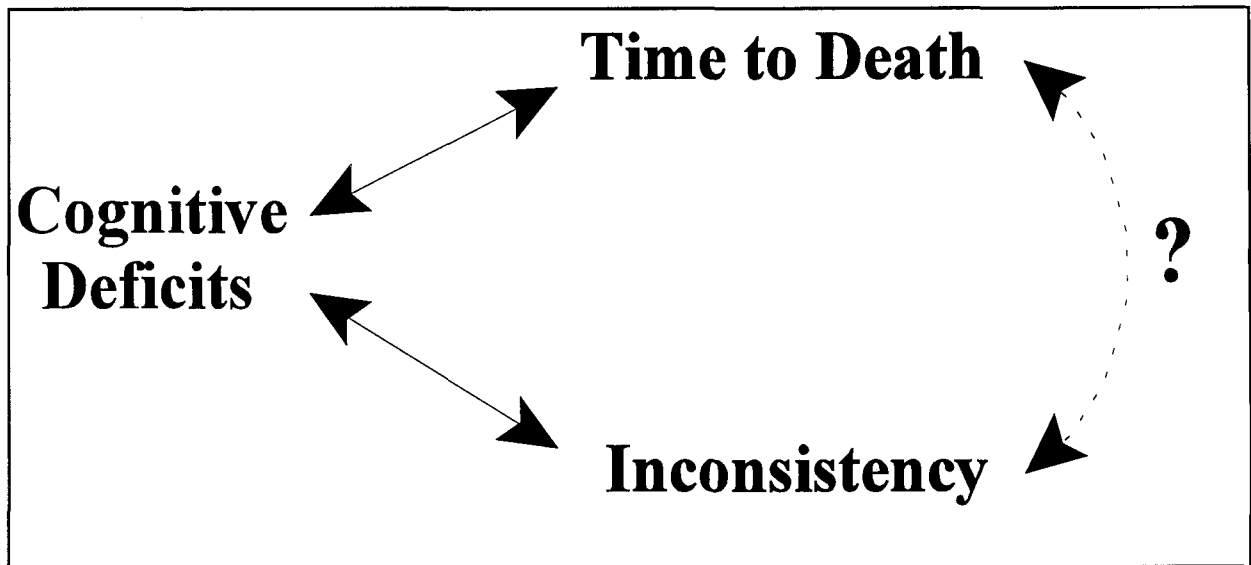


Figure 1

Links between Cognitive Deficits, Time to Death, and Performance Inconsistency

Chapter IV

METHOD

The design of the VLS consists of longitudinal sequences in which multiple cross-sectional samples of adults, initially aged 55 to 89 years, are retested at intervals of 3 years with new samples added every 6 years. Longitudinal data from Sample 1 (Waves 1 to 5) and Sample 2 (Waves 1 to 3) were examined for this study (see Figure 2). Relevant aspects of general design, participants, measures, and procedures are summarized in the following sections. A complete methodological account of the VLS has been described extensively elsewhere (see Hultsch et al., 1998; Dixon, Wahlin, Maitland, Hultsch, Hertzog, & Bäckman, in press).

Participants

Participants in the VLS were community dwelling adults living in a medium-size metropolitan area (Victoria, British Columbia) recruited through advertisements in the public media and appeals to community groups. At the first wave of measurement, 1014 participants were tested (484 from Sample 1 and 530 from Sample 2). Table 1 displays attrition status as a function of wave of testing (3-year retest intervals). Participants dropped out of the study for various reasons including: (a) health and memory problems; (b) too busy or no longer interested; (c) moved from the greater Victoria area; (d) could no longer be located; or (e) death.

The primary focus of this investigation was to contrast cognitive performance of confirmed survivors with confirmed decedents. Based on this rationale, a total of 712 of the original 1014 participants were of interest. A total of 447 confirmed survivors were selected who completed all waves and relevant measures in their respective sample (120

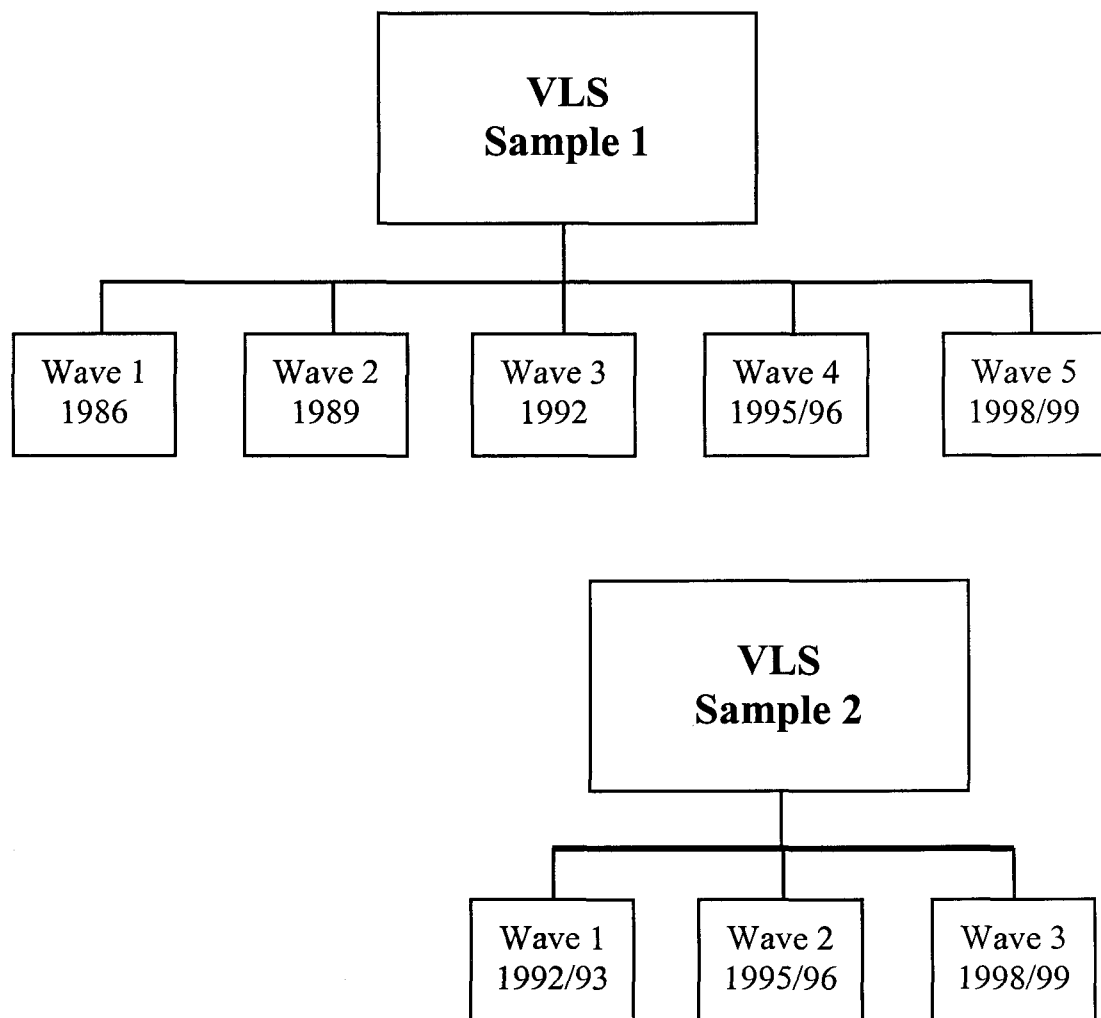


Figure 2

Research Design of the Victoria Longitudinal Study

Table 1
Attrition Status for Each Measurement Wave

Attrition Status	Wave of Testing					
	Wave 1	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6
Sample 1 (year tested)	1986	1989	1992	1995	1998	2002
Returned	484 (100)	333 (68.8)	249 (74.8)	177 (71.1)	120 (67.8)	
Dropped Out						
Health problems		10 (2.1)	15 (4.5)	9 (3.6)	12 (6.8)	
Family health problems		6 (1.2)	1 (0.3)	3 (1.2)	7 (3.9)	
Memory problems		2 (0.4)	0 (0.0)	0 (0.0)	1 (0.6)	
Busy/not interested		48 (9.9)	15 (4.5)	15 (6.0)	7 (3.9)	
Moved		9 (1.9)	8 (2.4)	2 (0.8)	3 (1.7)	
Could not be located		4 (0.8)	7 (2.1)	0 (0.0)	0 (0.0)	
Deceased		72 (14.9)	38 (11.4)	43 (17.3)	18 (10.2)	9 (5.1 ^a)
Sample 2 (year tested)	1992	1996	1999	2002		
Returned	530 (100)	410 (77.3)	327 (79.7)			
Dropped Out						
Health problems		12 (2.3)	18 (4.4)			
Family health problems		2 (0.4)	4 (1.0)			
Memory problems		0 (0.0)	3 (0.7)			
Busy/not interested		34 (6.4)	13 (3.2)			
Moved		12 (2.3)	3 (0.7)			
Could not be located		11 (2.1)	6 (1.5)			
Deceased		49 (9.2)	30 (7.3)	6 (1.5 ^a)		

Values in parentheses reflect percentage of participants associated with a particular reason for attrition. Column totals per sample may not sum to 100% due to rounding error. ^a As of June 2002, these participants were confirmed deceased prior to follow-up testing.

participants completed all 5 waves from Sample 1 and 327 participants completed all 3 waves from Sample 2). Of these 447 survivors, 5 were excluded from all subsequent analyses because they scored below a cut-off score of 24 on the mini mental status examination (MMSE) argued to represent cognitive abnormality (O'Connor et al., 1989). To identify decedents, an initial list of 567 participants (361 from Sample 1 and 206 from Sample 2) was submitted to Vital Statistics based on the following criteria: (a) our laboratory records indicated they were deceased but cause of death information was not available; (b) they discontinued participation for various reasons (e.g., memory or health problems) and may have since died; (c) they were tested for the most recent wave but exhibited signs of deteriorating performance; or (d) they were 85 years of age or older. Based on a search of Vital Statistics records for the province of British Columbia, 265 decedents were identified including date and cause of death information. The remaining 302 participants from the list of 567 were excluded from further analysis because present-day mortality status could not be confirmed (e.g., may be alive but no longer participating in the study, died out of province and do not appear in the Vital Statistics registry, etc.).

The final sample included 707 older adults, aged 59 to 95 years at final testing, who completed between 1 to 5 waves of measurement. Of these 707 participants, there were 442 survivors (273 women, 169 men) who completed all waves and relevant measures compared with 265 decedents (149 women, 116 men) who participated on at least one occasion. Throughout this study, mortality status was defined as the comparison between those who participated on a given measurement occasion but subsequently died (termed decedents) vs. those who participated on all occasions of measurement and remain living (termed survivors). Both mortality status groups were

obviously alive at time of testing; the important distinction is that some individuals would later die during the course of the study. Several phrases (e.g., those facing impending death, terminal decline phase) are used interchangeably with the term decedent.

Two categorical age groups were created representing different birth cohorts. Based on age at last wave of testing, participants were classified as either young-old (YO: $n = 519$) ranging in age from 59 to 79 years ($M = 71.86$, $SD = 4.91$) or old-old (OO: $n = 188$) ranging in age from 80 to 95 years ($M = 83.66$, $SD = 3.38$). For the initial wave of testing, this corresponds to an age classification of 54 to 67 years (YO) contrasted with those 68 years and older (OO). These age classifications are consistent with previous categorizations in the literature (cf., Dixon et al., in press; MacDonald et al., 2002).

To facilitate inferential and multilevel analyses, it was necessary to maximize sample size of deceased participants by collapsing across both VLS samples. As the substantive focus concerns examining associations between cognitive performance and time to death, sample differences will not be reported or further discussed. In support of this decision, a 2 (sample) x 2 (mortality status) multivariate analysis of variance (MANOVA) for baseline demographic measures (age, education, absolute health, and relative health) yielded little evidence of significant sample differences. The only significant univariate main effect associated with sample was for education, $F(1, 703) = 24.86$, $p < .01$, $\eta^2 = .03$, reflecting significantly more years for Sample 2 ($M = 15.05$, $SD = 3.15$) relative to Sample 1 ($M = 13.47$, $SD = 3.20$). This sample difference may reflect a cohort shift in educational attainment. The sample by mortality status interaction was also significant for education, $F(1, 703) = 3.89$, $p < .05$, $\eta^2 = .01$. Survivors and decedents from Sample 1 exhibited similar levels of education ($M = 13.54$ vs. 13.37

years), whereas decedents from Sample 2 reported significantly fewer years relative to survivors ($M = 14.35$ vs. 15.23 years). A one-way analysis of variance examined whether average number of years to death after last date of testing varied as a function of sample differences. The main effect of sample was significant, $F(1, 263) = 28.45$, $p < .01$, $\eta^2 = .10$, indicating longer intervals between wave of last testing and death for Sample 1 ($M = 5.99$, $SD = 4.02$) relative to Sample 2 ($M = 3.49$, $SD = 2.31$). This increased latency to death reflects the 6-year interval between initial waves for Sample 1 (1986) and Sample 2 (1992). The additional 2.5 years is largely influenced by participants who dropped out of the study after Sample 1 Wave 1 and then died many years later.

Table 2 presents demographic characteristics for both baseline and respective final wave of testing as a function of mortality status. For baseline characteristics, significant mortality group differences were observed for chronological age, $F(1, 705) = 152.05$, $p < .01$, $\eta^2 = .18$; years of education, $F(1, 705) = 13.47$, $p < .01$, $\eta^2 = .02$; absolute health, $F(1, 705) = 25.36$, $p < .01$, $\eta^2 = .04$; and relative health, $F(1, 705) = 10.29$, $p < .01$, $\eta^2 = .01$. On average, decedents were older, less educated, and reported poorer health (on a 5-point scale ranging from (0) very good to (4) very poor) than survivors. Although the proportion of women in the sample decreased for the decedent group, a cross-tabulation analysis indicated that mortality status did not significantly differ as a function of gender ($p = .15$). The mean interval between each decedent's first wave of testing and subsequent death was approximately 8.1 years.

For each individual's respective final wave of testing, significant mortality group differences were observed for years of education, $F(1, 705) = 12.68$, $p < .01$, $\eta^2 = .02$; absolute health, $F(1, 705) = 109.73$, $p < .01$, $\eta^2 = .14$; and relative health, $F(1, 705) =$

Table 2
Demographic Characteristics for Survivors and Decedents
at Baseline and Respective Final Wave of Testing (N = 707)

Variable		Survivors	Decedents
N		442	265
Gender (% Female)		61.80	56.20
Baseline Wave			
Age	<u>M</u>	66.56	72.67
	<u>SD</u>	6.34	6.44
Years of education	<u>M</u>	14.73	13.8
	<u>SD</u>	3.10	3.45
Absolute health	<u>M</u>	0.70	0.98
	<u>SD</u>	0.68	0.80
Relative health	<u>M</u>	0.53	0.70
	<u>SD</u>	0.66	0.74
Years to death	<u>M</u>	---	8.11
	<u>SD</u>	---	4.03
Respective Final Wave			
Age	<u>M</u>	74.64	75.61
	<u>SD</u>	6.78	7.12
Years of education	<u>M</u>	14.92	14.03
	<u>SD</u>	3.06	3.49
Absolute health	<u>M</u>	1.85	1.19
	<u>SD</u>	0.78	0.86
Relative health	<u>M</u>	1.39	0.88
	<u>SD</u>	0.85	0.79
Years to death	<u>M</u>	---	5.19
	<u>SD</u>	---	3.74

On a 5-point scale (0 = very good to 4 = very poor), absolute health reflects health relative to perfect; relative health reflects health relative to same-aged peers.

64.21, $p < .01$, $\eta^2 = .08$. Decedents did not significantly differ in age but were less educated than survivors. Notably, decedents' self-reported health was significantly better than survivors relative to perfect (absolute health) and relative to same-aged peers (relative health). This pattern of significant self-reported health differences implies that decedents died of relatively acute (e.g., heart attack, stroke) as opposed to protracted chronic illnesses and underscores the need to examine potential associations between cause of death and cognitive performance. Finally, the mean interval between each decedent's last wave of testing and subsequent death was approximately 5.17 years.

Measures

The VLS measurement battery consists of multiple questionnaires, tests, and tasks assessing various aspects of cognitive (e.g., memory) and non-cognitive (e.g., physiological) functioning. Cognitive performance was assessed using a continuum of measures spanning processes to products of cognition (verbal speed, working memory, episodic memory, and semantic memory). Select indicators of cognitive constructs were chosen based on previous confirmatory factor analyses (Hultsch et al., 1998). Relevant variables indexing both cognitive performance and vital status are summarized below.

Verbal speed. Speed of verbal processing was indexed using two indicators. 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). Participants responded by pressing 1 of 2 keys on the response console. A total of 60 test trials were presented (30 words and 30 nonwords). Per-trial response latencies (msec) as well as average reaction time (RT) across the 60 trials were used as the outcome measures.

The semantic verification task was adapted from procedures used by Palmer, MacLeod, Hunt, and Davidson (1985). Participants were asked to make semantic decisions by judging as rapidly as possible the plausibility of sentences 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). Participants responded by pressing 1 of 2 keys on the response console. A total of 50 test sentences were presented with the individual trial latencies (msec) as well as average RT across the 50 trials serving as the outcome measures.

Working memory. The working memory construct was measured using 3 indicators. The sentence construction task was developed in the VLS laboratory (Hultsch et al., 1990). Participants were asked to read aloud a series of sentences presented on flash cards, with one word in each sentence printed in capital letters and underlined in red. Participants were asked to maintain this target word in memory for later recall while continuing to read subsequent sentences. After reading all sentences for a particular series (ranging from 3 to 6 sentences in length), participants were asked to recall the target words in order. A new sentence was formed by correct sequential recall of target words. Passages of 3, 4, 5, and 6 sentences were used yielding new sentences of 3, 4, 5, and 6 words in length. There were 3 different target sentences associated with each passage length. The outcome measure used was the highest number of newly formed sentences correctly recalled for 2 out of 3 passage lengths.

The computation span task was used as an indicator of working memory requiring storage and simultaneous processing of information (Salthouse & Babcock, 1991). Participants were asked 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.

Similar to computation span, the listening span task (Salthouse & Babcock, 1991) required participants to answer questions about simple sentences that were orally presented while simultaneously trying to remember the final word of each sentence for later recall. At the end of a series of sentences, participants were asked to recall the last words of each sentence in their order of presentation. Three trials were given for each series length (ranging from two to seven sentences). The outcome measure reflected the highest span for which target words were correctly recalled on at least two of three trials. The computation and listening span tasks were not added to the VLS battery until 1989; participants from Sample 1 who died prior to the second wave of testing ($n = 72$) do not have scores for these measures. Analyses for these two tasks were computed for 635 participants.

Episodic memory. The episodic memory construct was indexed by two tasks: word recall and text recall. Episodic word recall was assessed via immediate free recall of two lists of 30 English words (Hultsch, Hertzog, & Dixon, 1990). Each list consisted of six words from five taxonomic categories (e.g., birds, flowers) 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.

Immediate gist recall of two narrative stories about an event in the life (or lives) of an older adult (or couple) was used as a second indicator of episodic memory (Dixon,

Hultsch, & Hertzog, 1989). Each story was structurally equivalent and contained approximately 300 words (about 160 propositions). Stories were presented in a typed booklet for study (4 min) followed by written recall (10 min). Participants were asked to recall as much of the story as possible in their own words. Recall protocols were scored for gist recall using criteria described in Dixon et al. (1989) with reliability estimates across all possible pairs of scorers exceeding 90%. The outcome measure used was the average number of gist propositions recalled across the two stories.

Semantic memory. Semantic memory performance was indexed by two measures. The fact recall test was based on norms developed by Nelson and Narens (1980). World knowledge was measured by two sets of 40 questions that tested individuals' recall of facts about multiple domains including science, history, literature, sports, geography, and entertainment. The questions were presented in booklets, and participants wrote their answers under self-paced timing conditions. The outcome measure reflected average correct out of 40 questions.

The second measure of semantic memory was a 54-item recognition vocabulary measure (Ekstrom, French, Harman, & Dermen, 1976). Participants were asked to provide the best response for each multiple-choice question and were given 15 minutes to complete the test. Number of correct responses out of 54 was used as the target measure.

Cause of death. Vital status of participants was obtained from the Department of Vital Statistics in the province of British Columbia. To ensure proper identification, individuals were matched according to: (a) full name; (b) date of birth; (c) gender; and (d) last known date alive. For those identified as deceased, information obtained included: (a) date of death (day, month, and year); (b) cause of death; and (c) antecedent cause of

death (i.e., contributing conditions). Primary cause of death was operationally defined as the dominant disease or condition temporally preceding death as determined by the attending physician.

Cause of death was classified according to the International Classification of Disease, Injuries, and Causes of Death, Ninth Revision (ICD-9) criteria (World Health Organization, 1975). Table 3 summarizes the five primary and single supplementary ICD-9 axes on which classifications are based. Disease subclassifications are listed below each corresponding primary axis. For the available VLS data, numerous causes of death were identified according to ICD-9 criteria. All causes of death were associated with two primary axes (constitutional or general diseases, local diseases arranged by site) and the one supplementary axis (additional classifications that fall outside description of the primary axes). With regard to classification, the ICD manual states that if a condition can be classified to both a special group chapter and a body system chapter, the code from the special group chapter is employed. For example, Alzheimer's disease refers to both a nervous system disease (body system) and a general mental and behavioural disorder (special group) and is thus coded under the primary axis of constitutional or general diseases (mental or behavioural disorder subclassification; see Table 3).

Using ICD-9 classification categories, Table 4 summarizes observed causes of death for the 265 VLS decedents. Cause of death was categorized into two groups: (a) those who died of cardio- or cerebrovascular disease (CVD); and (b) those who died from non-CVD conditions. Classification resulted in comparable numbers of participants in each group (118 CVD vs. 147 non-CVD). The CVD vs. non-CVD classification was selected for four reasons. First, CVD has been shown to predict impaired cognitive

Table 3
Classification Axes for the International Classification of
Disease, Injuries, and Causes of Death

Primary Axis	Chapter Type
Epidemic diseases Infectious diseases	Special group
Constitutional or general diseases Neoplasms/Cancer Diseases of the Blood and Immune System Endocrine, Nutritional, and Metabolic Diseases Mental and Behavioural Disorders	Special group
Local diseases arranged by site Diseases of the Nervous System Diseases of the Eye Diseases of the Ear Diseases of the Circulatory System Diseases of the Respiratory System Diseases of the Digestive System Diseases of the Skin Diseases of the Musculoskeletal system and Connective Tissue Diseases of the Genitourinary System	Body system
Developmental diseases Pregnancy, Childbirth, and the Puerperium Certain Conditions Originating in the Perinatal Period Congenital Malformations and Chromosomal Abnormalities	Special group
Injuries Injury, Poisoning, and Certain Other Consequences of External Causes	Special group
Additional chapters which fall outside description of the primary axes Symptoms, Signs, and Abnormal Clinical and Lab Findings External Causes of Morbidity and Mortality Factors Influencing Health Status and Contact with Health Services	

Table 4
Causes of Death by Wave of Testing

Cause of Death	Deceased after Wave 1	Deceased after Wave 2	Deceased after Wave 3	Deceased after Wave 4	Deceased after Wave 5
Total CVD (n = 118)					
Cardiac	31	23	17	7	1
Cerebrovascular	15	6	3	1	2
Dementia	8	0	2	2	0
Total non-CVD (n = 147)					
Neoplasms	41	28	9	4	3
Endocrine	3	1	1	1	0
Respiratory	11	2	8	3	2
Nervous system	4	2	2	0	0
Gastrointestinal	1	3	3	0	0
Blood and immune system	0	1	0	0	0
Musculoskeletal	2	0	0	0	0
Genitourinary system	2	1	1	0	0
External causes	3	1	3	0	1
Total	121 (45.6)	68 (25.7)	49 (18.5)	18 (6.8)	9 (3.4)

Values in parentheses reflect percentage of total decedents (n = 265) who died following each wave.

performance (e.g., Hertzog et al., 1978; Fahlander et al., 2000) and represents a potential source of mortality-related cognitive deficits. Second, CVD represents the leading cause of death among older adults in both Canada (Heart and Stroke Foundation of Canada, 1995) and the United States (United States Bureau of the Census, 1996). Third, individual causes of death do not provide sufficiently large cell sizes to analyze separately. Fourth, the CVD/non-CVD classification is consistent with the only previous investigation to examine cause of death as a predictor of cognitive performance (Small et al., in press). Adopting this same categorization permits replication and extension of these previous findings.

Multiple individual causes of death are summarized in Table 4. For the CVD group, circulatory system causes included both cardiac (e.g., heart attack, congestive heart failure, coronary artery disease, arrhythmia, ischemic heart disease, hypertension, ventricular aneurysm, peripheral vascular disease, cardiovascular disease, myocardial degeneration, valve disease) and cerebrovascular (e.g., stroke, intracerebral hemorrhage, cerebrovascular disease, cerebral aneurysm) conditions. Dementia (e.g., vascular dementia, multi-infarct dementia, Alzheimer's disease, organic brain syndrome) was classified with the other CVD causes due to its many underlying vascular associations.

For the non-CVD group, neoplasms were associated with many organs and body systems including breast, prostate, lung, liver, pancreas, colon, uterine, lymph nodes, and blood. Diabetes mellitus was the sole disease associated with endocrine, nutritional, and metabolic causes. Diseases of the respiratory system included respiratory failure, pneumonia, pulmonary fibrosis, chronic obstructive pulmonary disease, and emphysema. Multiple nervous system diseases were observed including motor neuron disease,

Parkinson's disease, and central nervous system (CNS) demyelination. Digestive system causes were associated with gastrointestinal complications (e.g., duodenal ulcer hemorrhage). Several causes associated with the blood and immune system (e.g., autoimmune disease) as well as the musculoskeletal system and connective tissue (e.g., Sjogren's syndrome and osteoarthritis) were identified. Genitourinary system diseases included urinary tract infections and kidney failure. Finally, complications due to falls (e.g., heart attack, respiratory failure, diffuse brain injury), suicide (e.g., asphyxiation, drug overdose), and car accident were classified as external causes of mortality according to ICD-9.

Self-report chronic conditions and medications. A self-report measure was administered inquiring about presence and severity of chronic conditions. Participants were asked about a variety of conditions including vision and hearing loss, arteriosclerosis, high blood pressure, heart disease, stroke, arthritis, diabetes, cancer, osteoporosis, and thyroid. Participants indicated presence or absence of each condition, and if present, rated severity on a 3 point scale: 1 = not serious, 2 = moderately serious, and 3 = very serious. In addition, participants were asked to indicate whether they were presently taking any prescribed medications including those for high blood pressure (e.g., Prinivil), heart or chest pain (e.g., Digoxin), and blood thinning (e.g., anticoagulants such as Coumadin).

A summary index of cardiovascular health was created by summing across the presence/absence data for select self-reported chronicity (heart disease, stroke, arteriosclerosis, and high blood pressure) and medication (heart, chest, high blood pressure, and anticoagulants) questions with higher values reflecting poorer

cardiovascular health. From these data, it was possible to create two additional CVD categories (survivors with and without CVD) corresponding to those already created for decedents using ICD-9 criteria. For the 442 survivors, 180 reported taking no CVD medications or CVD-related health issues across all waves whereas the remaining 262 reported a CVD condition or medication for at least one measurement occasion. The one wave criterion was adopted because some individuals did not report the presence of CVD-related diseases or medications until their last measurement wave (e.g., they either developed or were diagnosed with the condition prior to their last wave of testing and reported it then for the first time). The final categorical variable (referred to as the cardiovascular disease variable) yielded 4 distinct categories comprised of 180 survivors without CVD, 262 survivors with CVD, 147 decedents without CVD, and 118 decedents with CVD. For subsequent analyses, the healthy survivors without CVD (the control group) were compared to survivors with CVD as well as both decedent groups.

Date of last testing and distance to death. For select analyses employing analysis of variance (ANOVA) techniques, a categorical variable reflecting proximity to death was created. Two categories were created: (a) those who died less than 3 years after final wave of testing; and (b) those who died 3 or more years after final wave of testing. For multilevel analyses, more precise time intervals were calculated employing day, month, and year of each testing wave. For decedents, distance to death was calculated in several ways: (a) number of years between the last session of the first wave and actual date of death; and (b) number of years between the last session of the last wave and actual date of death. Corresponding intervals were calculated for survivors using the last date that all surviving participants were known to be alive (May 31, 2002). HLM analyses examined

the rate of cognitive decline for intervals terminating on May 31, 2002. For Cox regression analyses, these cases were included but considered censored (i.e., cases for which the death event had not yet occurred).

Dementia. Probable dementia was identified in several ways. Mini mental status examination (MMSE: Folstein, Folstein, & McHugh, 1975) scores served as markers of dementia for survivors. The MMSE was added to the VLS battery in 1998; scores were only available for those who completed 5 waves for Sample 1 or 3 waves for Sample 2 (i.e., the survivors) and thus could not be used to screen decedents. The MMSE is a brief and objective measure of cognitive functioning comprised of two sections: (a) the first requires vocal responses and covers orientation, memory, and attention; and (b) the second tests ability to name, follow verbal and written commands, write a sentence spontaneously, and copy a complex design consisting of overlapping pentagons. Administration of the MMSE requires five to 10 minutes; it is an untimed task. Maximum scores are 21 for the first section and 9 for section two yielding a total score out of 30. MMSE scores have been shown to reliably differentiate individuals with cognitive disturbance from those who are not impaired (e.g., McDowell, Kristjansson, Hill & Hebert, 1997).

As MMSE scores were only available for survivors, vital statistics information was used to identify dementia in decedents. Physician reports summarized at BC Vital Statistics included both primary and antecedent causes of death. Using this information, a categorical variable was created indicating presence (1) or absence (0) of multiple dementias including: vascular or multi-infarct dementia, organic brain syndrome, Pick's disease, and Alzheimer's disease. A total of 33 decedents (21 from the CVD group and

12 from the non-CVD group) were diagnosed with some form of probable dementia (12 with unspecified dementia, 14 with vascular dementia, 6 with Alzheimer's disease, and 1 with Pick's disease). It is important to control for these cases to reduce the likelihood that any observed cause of death (i.e., CVD vs. non-CVD) differences or declines in cognition were solely a function of dementia (e.g., Bäckman, Jonsson, Wahlin, Small, & Fratiglioni, 2002). All subsequent analyses were examined both with and without controlling for decedents with physician-diagnosed dementia. As expected given the distribution of dementia cases across both the CVD and non-CVD groups, no differences in overall patterns of significance were observed. Consequently, the decision was made to report only those analyses examining all 265 decedents (including the 33 with dementia) for several reasons: (a) records do not indicate when probable dementia diagnosis was made and thus participants may not have been demented at time(s) of testing; and (b) a number of dementia cases were vascular in nature with obvious links to cardiovascular causes of death.

Procedures

All measures were administered during four testing sessions over a period of approximately 4-6 weeks. Order of task administration was invariant for all participants. For all reaction time tasks, stimuli were presented on a computer monitor interfaced with a 166 Mhz IBM compatible computer that controlled stimulus presentation and timing. Participants' responses to stimuli were recorded at an accuracy of ± 1 ms using a custom designed response console. For select analyses, cognitive outcome measures were converted to a T-score metric ($M = 50$, $SD = 10$) to permit cross-domain comparisons.

Statistical Analyses

Analysis of variance (ANOVA) techniques were used for both cross-sectional and longitudinal analyses of data. Group differences were assessed for four primary between-subjects variables: age group (YO vs. OO), mortality status (Survivor vs. Decedent), cause of death (CVD vs. non-CVD), and proximity to death after last wave of testing (3-years or less vs. greater than 3 years). Between-group differences for the last wave of testing were examined using multivariate analysis of covariance (MANCOVA) controlling for education. Repeated-measures analysis of covariance (RMANCOVA), controlling for education and baseline cognitive performance, was used to examine group differences in cognitive change across waves (within-subject variable). Profile analysis was used to assess whether cognitive performance profiles (across the continuum of measures) varied as a function of age group and mortality status. Profile analysis is a special application of MANOVA where multiple dependent tests are all measured on the same scale (in the present case, all cognitive performance measures are in a T-score metric). A doubly multivariate profile analysis was computed to assess whether mortality deficits varied as a function of cognitive domain and time.

Hierarchical linear modeling (Bryk & Raudenbush, 1987; 1992) was used to examine cognitive change as a function of age, mortality status, cause of death, and time to death. Relative to other statistical approaches (e.g., repeated-measures ANOVA), HLM affords several distinct advantages: (a) calculation of parameter estimates despite missing data, participant attrition, and unequal intervals between waves of testing; and (b) simultaneous assessment of between-person differences (Level 2) and within-person change (Level 1). These advantages complement our present objectives in several ways. First, HLM permits examination of cognitive change for individuals with varying waves

of participation: as a consequence, all available VLS measurement waves will be examined (including data from all decedents). Second, HLM permits assessment of whether individual trajectories of cognitive decline vary as a function of group differences. Finally, HLM represents a more powerful technique for differentiating terminal decline vs. terminal drop as a function of time to death.

A Cox proportional hazards analysis (Cox, 1972) was used to predict survival status (Survivors vs. Decedents) across a follow-up period of measurement as a function of cognitive performance. In contrast to ANOVA approaches, an advantage of this technique is that time to death from baseline assessment is incorporated into the prediction model. Models examined using Cox regression assume that the population mortality rate depends upon a continuous time variable (i.e., the interval between Wave 1 baseline testing and subsequent date of death). In addition, multiple predictor blocks can be specified for predicting survival status (e.g., enter covariates in block 1 followed by substantive predictors in block 2). Defining the incident as death, Odds or Incident Rate Ratios (IRR) 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 cognitive measures best predict likelihood of death over a longitudinal follow-up interval.

Data Imputation

The data analytic methods employed (e.g., repeated measures analysis of variance, multilevel modeling) influenced decisions regarding the handling of missing data. Dropping entire cases is never ideal, particularly when using techniques such as multilevel modeling that can maximize information gleaned from partial cases. Data

imputation followed a two-step approach. For individuals who had fully completed at least 1 wave, missing values were replaced with that participant's scores from the immediately preceding wave. Although more complex imputation techniques exist, this method represents a conservative imputation approach that favours cognitive stability as opposed to change. For the few instances when data from previous waves were not available (i.e, for individuals who had not fully completed the first wave), the mean score from Wave 1 for similar age and mortality status groups was used. Missing values were calculated using the missing values module (series mean estimation method) included in the Statistical Package for Social Sciences (SPSS) 11 software (SPSS, 2001). Overall, 22 survivors and 15 decedents were missing some data (approximately 5.2% of the total sample). For any given measurement wave, no individuals were missing more than 4 of 9 cognitive measures.

Several important points must be made with regard to data imputation. First, although 15 of the 265 decedents were missing some data, patterns indicated all were missing data for 3 or fewer tasks. Rather than omit these individuals from further analysis, missing data was imputed according to the approach already outlined. This conservative replacement permits the retention of these important cases. Second, 7 participants missed an entire intermediate testing wave (i.e., not wave 1) and returned 3 years later for reassessment. Consistent with the conservative approach used for partial missing data, scores from the previous wave were substituted for these missing values. Third, 24 of the 44 participants (including those who missed an entire wave) were missing RT trial data for the lexical decision and semantic verification tasks. Missing lexical and semantic trial data were replaced with trial data from the immediately

preceding wave. For the 2 individuals who were missing semantic and lexical data for the first wave, average Wave 1 trial latencies and mean scores for their particular age and mortality status groups were substituted for the missing values. Fourth, the listening span and computation span working memory tasks were not administered for S1W1 in 1986. As these tasks were not administered for this wave, missing values were not imputed. To circumvent listwise case deletion, analyses focusing on these measures were run separately (i.e., separate repeated measures MANOVAs from the other cognitive measures) with corresponding cell sizes reported. For all analyses, results with and without missing data were identical.

Chapter V

RESULTS

This chapter reports statistical analyses and outcomes in five main sections. First, patterns of cognitive performance (at cross-section and over time) were analyzed as a function of mortality status and age to assess whether impending death negatively impacted cognitive performance for both the Young-Old and the Old-Old. Second, differences in cognitive profiles (i.e., performance trends across diverse measures of cognition) were examined as a function of mortality status. Third, patterns of terminal cognitive decline for decedents were analyzed as a function of underlying cause of death. The final two sections summarize novel analyses that make unique contributions to the terminal decline literature. In section four, a multilevel modeling approach was used to examine intraindividual cognitive decline as a function of age and number of years to death at each testing wave (within-person), as well as between-person differences for mortality status and cause of death. For the final section, a potential correlate of terminal decline was examined. Specifically, performance inconsistency on two verbal response latency measures was examined in relation to mortality status and age group differences.

Cognitive Performance as a Function of Mortality Status, Age Differences, and Age Change

Table 5 displays the means and standard deviations for each cognitive measure (raw scores) separately by wave of testing, age group, and mortality status. These descriptive statistics should only be used to characterize the sample within a given testing occasion. Inferences regarding longitudinal trends should not be drawn due to missing data (i.e., dropouts reducing listwise n) and because the repeated measurements are correlated within persons.

Both cross-sectional and repeated-measures longitudinal analyses of variance (ANOVA), as well as multilevel models, were employed to compare mortality-cognitive performance associations for the Young-Old and Old-Old participants. To facilitate comparisons across cognitive domains at cross-section, cognitive outcome measures were linearly transformed to a T-score metric by standardizing raw score values, multiplying these values by 10, and adding a constant of 50 (yielding a distribution with $\underline{M} = 50$ and $\underline{SD} = 10$). Longitudinal data were standardized across individuals and waves of testing to avoid detrending the data (i.e., removing longitudinal declines). F-tests of change for raw and T-score measures yielded identical values; only the metric differed. Reaction time latency measures (lexical decision and semantic verification) were reverse coded so that lower T-scores reflected poorer cognitive performance.

Education was partialled for all subsequent analyses based on the significant mortality group differences (decedents were less educated). Neither age nor health were included as additional covariates; both were analyzed as independent variables of substantive interest in subsequent analyses. Gender was not included as an independent

Table 5
Mean Raw Score Cognitive Performance by Wave of Testing, Age Group, and Mortality Status

Variable	Survivors						Decedents					
	Young-Old			Old-Old			Young-Old			Old-Old		
	TLWave (n = 337)	SLWave (n = 337)	LWave (n = 337)	TLWave (n = 105)	SLWave (n = 105)	LWave (n = 105)	TLWave (n = 36)	SLWave (n = 85)	LWave (n = 182)	TLWave (n = 40)	SLWave (n = 59)	LWave (n = 83)
Verbal Speed												
Semantic	3147.49 (813.55)	3149.47 (826.43)	3202.25 (991.22)	3468.57 (881.59)	3586.50 (862.17)	3724.16 (996.23)	3212.53 (639.23)	3234.21 (772.37)	3571.38 (961.72)	3557.48 (972.79)	4089.81 (1075.2)	5026.72 (2060.46)
Lexical	1038.42 (296.05)	986.03 (264.13)	1015.45 (337.98)	1112.42 (268.85)	1139.72 (319.08)	1193.20 (326.83)	1022.50 (207.40)	1040.33 (269.85)	1113.85 (301.34)	1130.28 (271.73)	1294.44 (393.76)	1606.87 (812.57)
Working Memory												
Computation	3.33 (1.16)	3.37 (1.18)	3.28 (1.26)	2.85 (1.15)	2.87 (1.26)	2.54 (1.10)	3.10 (0.88)	2.93 (1.14)	2.94 (1.31)	2.87 (0.92)	2.55 (1.24)	2.11 (1.47)
Listening	3.30 (1.02)	3.25 (0.89)	3.13 (0.98)	2.81 (0.98)	2.85 (1.06)	2.53 (1.10)	2.80 (1.03)	2.89 (1.07)	2.83 (1.06)	2.74 (1.05)	2.53 (1.22)	2.12 (1.12)
Sentence	5.20 (1.02)	5.42 (0.94)	5.08 (1.12)	4.75 (1.32)	5.10 (1.15)	4.60 (1.49)	5.17 (1.06)	4.86 (1.21)	4.69 (1.37)	4.68 (1.33)	4.00 (1.73)	3.92 (1.84)
Episodic Memory												
Story Recall	39.07 (9.21)	37.56 (8.82)	38.44 (10.14)	34.96 (10.71)	33.16 (9.65)	32.44 (10.67)	33.67 (8.93)	34.36 (9.15)	31.58 (10.72)	30.01 (10.52)	28.17 (10.23)	23.19 (10.44)
Word Recall	18.97 (3.82)	19.11 (3.97)	18.54 (4.17)	17.10 (3.99)	17.25 (5.09)	15.30 (4.64)	18.03 (3.47)	17.96 (3.73)	16.42 (4.42)	16.24 (4.55)	14.55 (4.76)	12.39 (5.33)

Table 5 con't

Variable	Survivors						Decedents					
	Young-Old			Old-Old			Young-Old			Old-Old		
	TLWave (n = 337)	SLWave (n = 337)	LWave (n = 337)	TLWave (n = 105)	SLWave (n = 105)	LWave (n = 105)	TLWave (n = 36)	SLWave (n = 85)	Lwave (n = 182)	TLWave (n = 40)	SLWave (n = 59)	Lwave (n = 83)
Semantic Memory												
Fact Recall	21.90 (5.96)	21.98 (5.98)	21.02 (6.43)	19.94 (6.21)	18.84 (6.44)	17.77 (6.60)	21.64 (6.26)	21.36 (6.27)	18.68 (6.70)	19.06 (6.69)	16.14 (6.76)	13.40 (7.07)
Vocabulary	44.70 (5.94)	45.02 (5.41)	44.09 (5.69)	45.50 (5.85)	45.45 (6.30)	44.26 (6.30)	45.86 (4.61)	45.55 (5.16)	43.18 (7.76)	46.40 (6.18)	44.78 (7.84)	41.25 (9.31)

Semantic = semantic verification; lexical = lexical decision; computation = computation span; listening = listening span; sentence = sentence construction;. TLWave refers to a given individual's third last wave of testing, SLWave refers to her/his second last wave, with Lwave referring to the last (i.e., most recent) wave of testing. For survivors, the total n is constant for each wave as they completed all possible waves of testing. In contrast, fewer than 265 decedents completed the TL and SL waves. A total of 72 decedents died after S1W1 testing; as the listening and computation span tasks were not administered for this wave, the n for these tasks was further reduced to 10 (TL), 55 (SL), and 121 (L) for Young-Old decedents and 23 (TL), 51 (SL), and 72 (L) for Old-Old decedents. Standard deviations are reported in parentheses. These descriptive statistics should only be used to characterize the sample within a given testing occasion. Inferences regarding longitudinal trends should not be drawn due to missing data (i.e., dropouts reducing listwise n) and because the repeated measurements are correlated within persons.

variable or covariate for several reasons: (a) cross-tabulation analysis indicated that mortality status did not significantly differ as a function of gender ($p = .15$); (b) its inclusion resulted in small cell sizes; (c) few previously observed mortality by gender interactions (e.g., Bosworth & Schaie, 1999); and (d) gender effects are difficult to interpret given differing life expectancies for women and men.

The Influence of Age Differences and Impending Death on Terminal Decline

Preliminary cross-sectional analyses were computed to replicate and extend recent terminal decline findings (c.f., Bosworth & Schaie, 1999; Small & Bäckman, 1997). Of specific interest was whether mortality-related deficits for each individual's respective final wave of testing varied as a function of age group. Performance for the final measurement wave (as opposed to baseline performance) was analyzed because it was the most recent testing wave and was expected to be the most sensitive to mortality-related influences due to its proximity to the death event for Decedents. To assess group differences for the last testing wave, a 2 (age group: Young-Old and Old-Old) x 2 (mortality status: Survivor and Decedent) multivariate analysis of covariance (MANCOVA) controlling for education was computed for lexical decision, semantic verification, sentence construction, story recall, word recall, and vocabulary. Because the listening span and computation span tasks were not administered for S1W1, a separate MANCOVA was computed for these two variables to avoid listwise case deletion.

Homogeneity of variance-covariance. Given the unequal sample cell sizes, an initial evaluation of homogeneity of variance-covariance was completed. At the univariate level, the assumption states that the variance for a given cognitive measure must be statistically equivalent across all levels of the independent measure(s). The

multivariate analog requires equivalence of variance-covariance matrices within each cell of the design with the assumption that: (a) these estimates are sampled from the same population variance-covariance matrix; and (b) they can be reasonably pooled to create a single estimate of error (Tabachnick & Fidell, 2001). Heterogeneity of within-cell error matrices can result in a misleading pooled estimate of error variance and subsequently lead to inflated Type I errors (particularly when the greatest dispersion is associated with the smallest cell size). The alpha level is considered conservative if cells with larger sample sizes produce the greater variances and covariances.

Prior to computing the two MANCOVAs, Box's M test was employed to formally test the homogeneity of variance-covariance assumption (i.e., equivalence of covariance matrices for the dependent variables across all groups). The test was significant for both the verbal speed, semantic and episodic memory measures MANCOVA (Box's $M = 479.29$, $p < .001$) and the working memory measures MANCOVA (Box's $M = 19.84$, $p < .05$) indicating a violation of the equality assumption. However, Box's M is a notoriously sensitive test when used with large sample sizes, routine alpha levels, and multivariate applications of ANOVA (as in the present case).

Further examination of the present data suggest that the homogeneity of variance-covariance assumption was satisfactory. Observed cell sizes for the seven cognitive measure (Young-Old Survivors = 337, Young-Old Decedents = 182, Old-Old Survivors = 105, Old-Old Decedents = 83) and two cognitive measure (Young-Old Survivors = 337, Young-Old Decedents = 121, Old-Old Survivors = 105, Old-Old Decedents = 72) MANCOVAs were not sufficiently discrepant as to cause considerable distortion of alpha levels. Second, although Old-Old Decedents represented the smallest cell size and also

exhibited the largest variance estimates (resulting in a liberal significance test), $F_{\max}$ ratios of the largest to smallest cell variances (e.g., 100 vs. 20 = 5) did not violate statistical conventions. For relatively equal sample sizes (i.e., those within a ratio of 4 to 1 or less for the largest to smallest cell size), an $F_{\max}$ value as large as 10 is acceptable. The largest cell size ratio for the present data was for Young-Old Survivors relative to Old-Old Decedents (4.06 and 4.68 respectively for the separate MANCOVAs). $F_{\max}$ ratio comparisons for each cognitive measure for these 2 groups did not exceed 10, with most variance ratios not exceeding 3. It is not until cell-size discrepancies increase markedly (e.g., 9 to 1 as compared with 4 to 1) that an $F_{\max}$ ratio as small as 3 is associated with inflated Type I error, and only then if the smallest group has the largest variance (Milligan, Wong, & Thompson, 1987; Tabachnick & Fidell, 2001). Trends observed for these data suggest the significance of the Box's M test can be safely ignored (i.e., significance tests are expected to be robust to these small violations). The same justification applies to all subsequent ANOVA-based analyses.

Multivariate analysis of covariance for final testing wave. The 2 (Age) x 2 (Mortality) for the seven measure MANCOVA revealed a significant omnibus effect for Age Group, Wilks' $\lambda = .792$, $F(7, 696) = 26.12$, $p < .001$, $\eta^2 = .21$, Mortality Status, Wilks' $\lambda = .862$, $F(7, 696) = 15.92$, $p < .001$, $\eta^2 = .14$, the Age Group x Mortality Status interaction, Wilks' $\lambda = .965$, $F(7, 696) = 3.65$, $p < .01$, $\eta^2 = .04$, and the Education covariate, Wilks' $\lambda = .865$, $F(7, 696) = 15.47$, $p < .001$, $\eta^2 = .14$. Similarly, the MANCOVA for the two working memory measures yielded significant omnibus effects for Age Group, Wilks' $\lambda = .925$, $F(2, 629) = 25.54$, $p < .001$, $\eta^2 = .0821$, Mortality Status, Wilks' $\lambda = .977$, $F(2, 629) = 7.38$, $p < .01$, $\eta^2 = .02$, and the Education covariate, Wilks' λ

= .965, $F(2, 629) = 11.31$, $p < .01$, $\eta^2 = .04$.

Table 6 shows the means and standard deviations (T-scores) for each individual's respective last wave of testing as a function of mortality status and age group. Univariate age and mortality differences in mean cognitive performance were observed subsequent to controlling for years of education. Univariate age group differences were observed for semantic verification, $F(1, 702) = 93.16$, $p < .001$, $\eta^2 = .12$; lexical decision, $F(1, 702) = 85.31$, $p < .001$, $\eta^2 = .11$; computation span, $F(1, 630) = 39.94$, $p < .001$, $\eta^2 = .06$; listening span, $F(1, 630) = 40.82$, $p < .001$, $\eta^2 = .06$; sentence construction, $F(1, 702) = 26.25$, $p < .001$, $\eta^2 = .04$; story recall, $F(1, 702) = 60.09$, $p < .001$, $\eta^2 = .08$; word recall, $F(1, 702) = 84.92$, $p < .001$, $\eta^2 = .11$; and fact recall, $F(1, 702) = 52.05$, $p < .001$, $\eta^2 = .07$. The Young-Old performed significantly better than the Old-Old on all of the cognitive measures with the exception of vocabulary.

Mortality status differences were observed for all cognitive measures: semantic verification, $F(1, 702) = 64.68$, $p < .001$, $\eta^2 = .08$; lexical decision, $F(1, 702) = 48.49$, $p < .001$, $\eta^2 = .07$; computation span, $F(1, 630) = 10.02$, $p < .01$, $\eta^2 = .02$; listening span, $F(1, 630) = 13.04$, $p < .001$, $\eta^2 = .02$; sentence construction, $F(1, 702) = 17.78$, $p < .001$, $\eta^2 = .03$; story recall, $F(1, 702) = 73.34$, $p < .001$, $\eta^2 = .10$; word recall, $F(1, 702) = 37.96$, $p < .001$, $\eta^2 = .05$; fact recall, $F(1, 702) = 28.87$, $p < .001$, $\eta^2 = .04$; and vocabulary, $F(1, 702) = 6.74$, $p < .05$, $\eta^2 = .01$. As expected, Survivors significantly outperformed Decedents for respective final waves of cognitive testing.

Finally, significant Age Group x Mortality Status interactions were observed for semantic verification, $F(1, 702) = 22.99$, $p < .001$, $\eta^2 = .03$; lexical decision, $F(1, 702) = 20.16$, $p < .001$, $\eta^2 = .03$; fact recall, $F(1, 702) = 4.76$, $p < .05$, $\eta^2 = .01$; and vocabulary,

Table 6

Cognitive Performance at Last Testing Wave by Mortality Status and Age Group

Variables	Survivors			Decedents		
	Young-Old (n = 337)	Old-Old (n = 105)	Total (n = 442)	Young-Old (n = 182)	Old-Old (n = 83)	Total (n = 265)
Verbal Speed						
Semantic verif.	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 const.	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)

Standard deviations appear in parentheses. Semantic verif = semantic verification; sentence const. = sentence construction. ^a As these measures were not administered for S1W1, only 121 Young-Old Decedents and 72 Old-Old Decedents had available data.

$F(1, 702) = 5.49, p < .05, \eta^2 = .01$. Old-Old decedents showed significantly poorer performance for the verbal speed and semantic memory measures compared with all other groups. Age group by mortality status interactions were not significant for working memory or episodic memory tasks.

Magnitude of mortality differences. Cohen's d , the difference between means divided by the average standard deviation of both groups, was calculated to estimate the effect size of mortality group differences. The resulting value is an index devoid of arbitrary scaling metric that expresses the distance between means in units of variability. The critical focus concerned which cognitive domains exhibited the most pronounced differences between Survivors and Decedents. According to Cohen's convention, d values of .3, .5, and .8 reflect small, medium, and large effects respectively. Figure 3 graphs the magnitude of mortality effects for each cognitive measure both overall (i.e., Survivors vs. Decedents) and separately for each age group. For three of the four cognitive measures that exhibited significant mortality by age interactions, mortality effect sizes increased from small to medium (lexical decision and fact recall) and from small to large (semantic verification) respectively with increased age; the magnitude of mortality differences for vocabulary approached a medium effect for the Old-Old. In contrast, mortality effect size differences as a function of age group were not observed for the remaining cognitive measures. Small effects were observed for the working memory measures irrespective of age. However, medium to large differences were observed for the episodic memory tasks (word and story recall). Overall, findings indicated that pronounced mortality differences for the final testing wave prior to death were manifested for verbal process measures as well as episodic and semantic abilities.

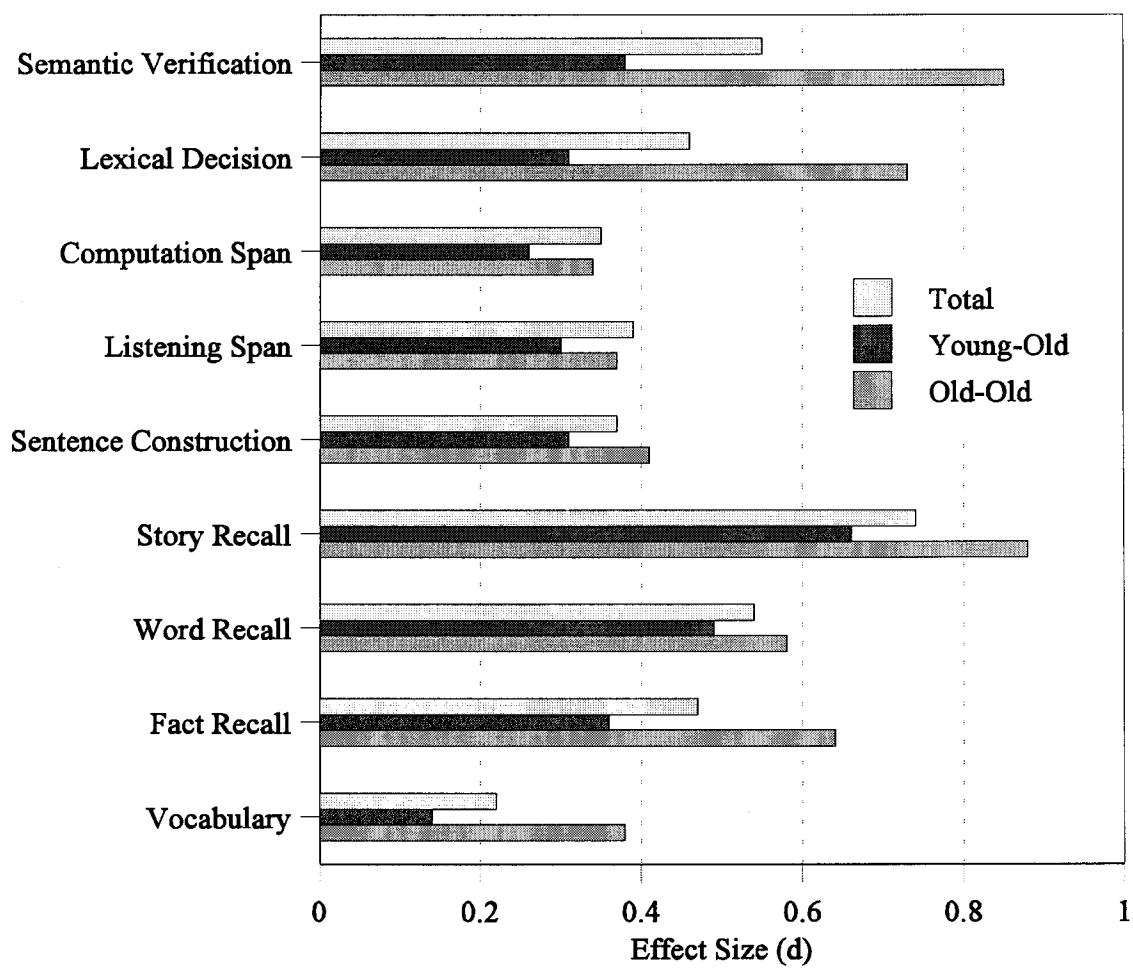


Figure 3

Cohen's d for Mortality Status Differences

as a Function of Age Group for the Final Wave of Testing

The Influence of Age-Related Change and Impending Death on Terminal Decline

First, a mixed model repeated-measures MANCOVA was computed to examine cognitive change across the most recent 3- and 6-year intervals completed. Relative to the cross-sectional analyses, number of decedents with available data were reduced (listwise deletion due to attrition) for the 3-year change analyses, and further reduced for the 6-year change analyses. Because Decedents performed significantly worse than Survivors at baseline for all cognitive measures ($p < .01$), a separate series of mixed-model ANCOVAs were computed examining cognitive change covarying both baseline cognitive performance and education. The mortality x wave and mortality x age group x wave interactions are of particular interest (i.e., do decedents show accelerated decline across the last occasions of measurement even after controlling for baseline performance and education levels).

Repeated-measures multivariate analysis of covariance for 3-year change. Three-year cognitive change occurring between the second-last and last respective waves of testing was examined using a 2 (age group) x 2 (mortality status) x 2 (wave) repeated-measures MANCOVA partialing education (listwise $n = 586$). Table 7 outlines cognitive performance trends over the 3 year period as a function of mortality status and age group. Significant omnibus effects were associated with Age, Wilks' $\lambda = .800$, $F(7, 575) = 20.59$, $p < .001$, $\eta^2 = .20$; Mortality Status, Wilks' $\lambda = .875$, $F(7, 575) = 11.68$, $p < .001$, $\eta^2 = .13$; the Age x Mortality Interaction, Wilks' $\lambda = .972$, $F(7, 575) = 2.38$, $p < .05$, $\eta^2 = .03$; Wave, Wilks' $\lambda = .955$, $F(7, 575) = 3.91$, $p < .001$, $\eta^2 = .05$; the Age Group x Wave interaction, Wilks' $\lambda = .928$, $F(7, 575) = 6.36$, $p < .001$, $\eta^2 = .07$; the Mortality x Wave interaction, Wilks' $\lambda = .932$, $F(7, 575) = 5.95$, $p < .001$, $\eta^2 = .07$; the Mortality x Age

Table 7
Multiwave Cognitive Performance (T-Scores) by Mortality Status and Age Group

Variable	Survivors				Decedents			
	Young-Old (n = 337)		Old-Old (n = 105)		Young-Old (n = 85)		Old-Old (n = 59)	
	SLWave	LWave	SLWave	LWave	SLWave	LWave	SLWave	LWave
Verbal Speed								
Semantic	52.47 (7.79)	51.97 (9.34)	48.35 (8.13)	47.05 (9.39)	51.67 (7.28)	49.86 (8.98)	43.61 (10.13)	36.98 (17.10)
Lexical	52.67 (7.36)	51.85 (9.42)	48.39 (8.89)	46.90 (9.11)	51.16 (7.52)	49.37 (8.59)	44.08 (10.97)	37.73 (17.81)
Working Memory								
Computation	52.28 (9.20)	51.54 (9.84)	48.31 (9.83)	45.78 (8.61)	48.79 (8.89)	49.07 (10.85)	45.83 (9.69)	43.37 (12.18)
Listening	52.59 (8.50)	51.49 (9.35)	48.78 (10.18)	45.77 (10.54)	49.19 (10.21)	47.28 (10.72)	45.73 (11.70)	42.35 (10.44)
Sentence	53.34 (7.23)	50.73 (8.64)	50.94 (8.87)	47.02 (11.49)	49.05 (9.30)	48.68 (10.19)	42.43 (13.35)	41.25 (15.06)
Episodic Memory								
Story Recall	52.20 (8.47)	53.05 (9.73)	47.98 (9.27)	47.28 (10.24)	49.13 (8.79)	47.77 (9.57)	43.18 (9.82)	39.75 (10.28)
Word Recall	53.12 (8.53)	51.90 (8.96)	49.13 (10.93)	44.95 (9.95)	50.67 (8.00)	48.88 (8.74)	43.34 (10.21)	39.16 (39.16)
Semantic Memory								
Fact recall	52.70 (8.86)	51.29 (9.53)	48.06 (9.54)	46.46 (9.78)	51.79 (9.28)	49.89 (10.15)	44.06 (10.01)	40.52 (10.72)
Vocabulary	50.61 (9.03)	49.05 (9.50)	51.32 (10.52)	49.33 (10.52)	51.49 (8.62)	51.77 (8.12)	50.20 (13.10)	45.87 (14.69)

Semantic = semantic verification; lexical = lexical decision; computation = computation span; listening = listening span; sentence = sentence construction. SLWave refers to a given individual's second last wave of testing with Lwave referring to the last (i.e., most recent) wave of testing. Standard deviations are reported in parentheses. Listwise n = 586 for the 3-year repeated-measures analyses except for listening and computation span where listwise n = 548 (337 YO survivors, 105 OO survivors, 55 YO decedents and 51 OO decedents).

Group x Wave interaction, Wilks' $\lambda = .960$, $F(7, 575) = 3.45$, $p < .01$, $\eta^2 = .04$; and the Education covariate, Wilks' $\lambda = .828$, $F(7, 575) = 17.28$, $p < .001$, $\eta^2 = .17$. The corresponding repeated-measures MANCOVA for the two working memory measures not administered at Wave 1 (listwise $n = 548$) yielded significant omnibus effects for: Age, Wilks' $\lambda = .957$, $F(2, 542) = 12.15$, $p < .001$, $\eta^2 = .04$; Mortality Status, Wilks' $\lambda = .976$, $F(2, 542) = 6.64$, $p < .01$, $\eta^2 = .02$; Wave, Wilks' $\lambda = .983$, $F(2, 542) = 4.55$, $p < .05$, $\eta^2 = .02$; the Age Group x Wave interaction, Wilks' $\lambda = .987$, $F(2, 542) = 3.46$, $p < .05$, $\eta^2 = .01$; and the Education covariate, Wilks' $\lambda = .960$, $F(2, 542) = 11.35$, $p < .001$, $\eta^2 = .04$.

Univariate tests of between-subjects effects for age group and mortality status mirrored the main effects reported for the cross-sectional analyses of final wave of measurement. Young-Old adults performed significantly better than the Old-Old for all cognitive measures except vocabulary ($p < .01$). Similarly, collapsing across both final waves of cognitive testing, Survivors significantly outperformed Decedents for all measures except vocabulary ($p < .01$).

Significant Age Group x Mortality Status interactions were observed for semantic verification, $F(1, 581) = 12.45$, $p < .001$, $\eta^2 = .02$; lexical decision, $F(1, 581) = 8.49$, $p < .01$, $\eta^2 = .01$; sentence construction, $F(1, 581) = 6.40$, $p < .05$, $\eta^2 = .01$; fact recall, $F(1, 581) = 5.32$, $p < .05$, $\eta^2 = .01$; and vocabulary, $F(1, 581) = 6.07$, $p < .05$, $\eta^2 = .01$. Consistent with the cross-sectional findings, Old-Old Decedents declined more than Survivors for the verbal speed and semantic memory measures with sentence construction emerging longitudinally. Observed trends for the remaining working and episodic memory tasks were in the expected direction.

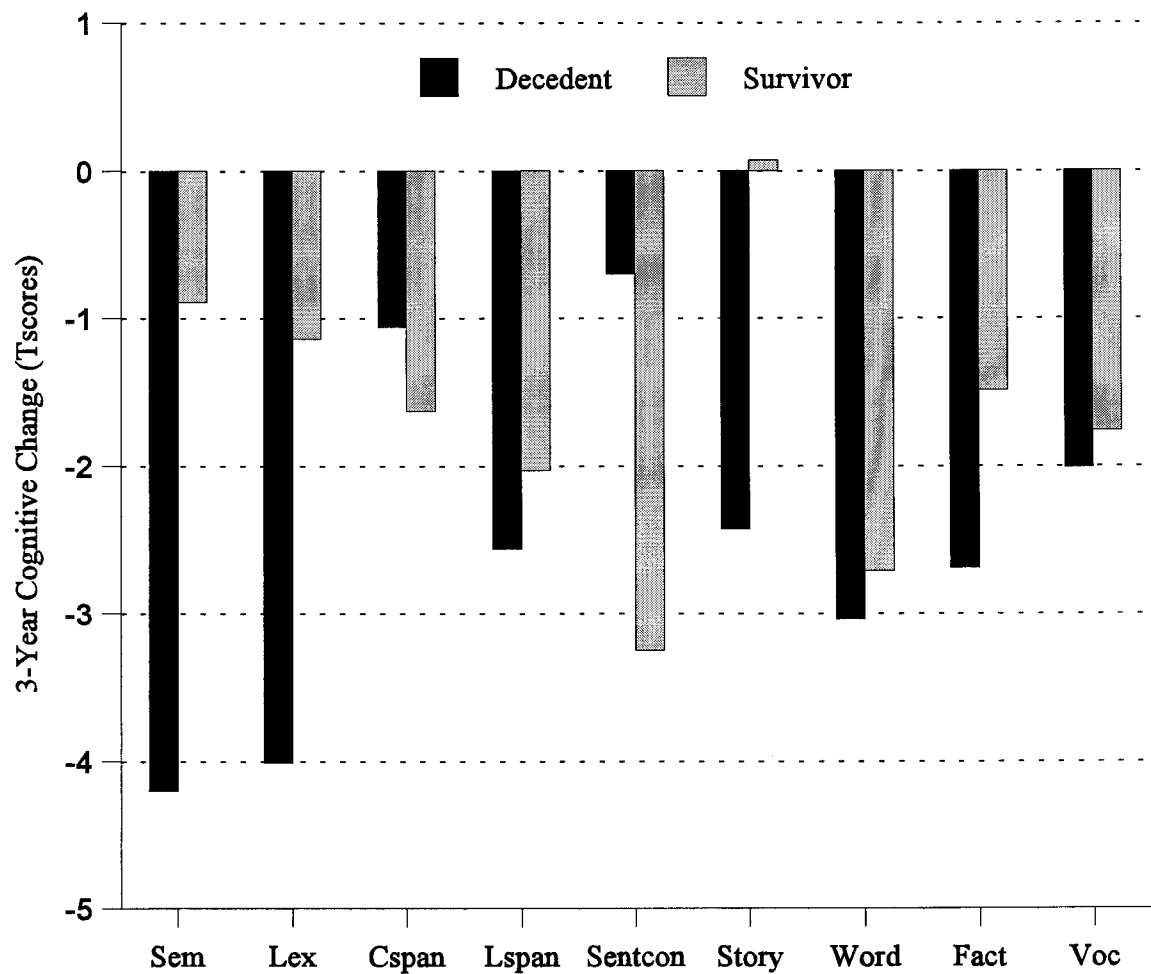
Univariate tests of the within-subjects Wave effect demonstrated significant 3-

year cognitive decline for semantic verification, $F(1, 581) = 5.61, p < .05, \eta^2 = .01$; lexical decision, $F(1, 581) = 6.47, p < .05, \eta^2 = .01$; listening span, $F(1, 543) = 8.32, p < .01, \eta^2 = .02$; sentence construction, $F(1, 581) = 5.56, p < .05, \eta^2 = .01$; fact recall, $F(1, 581) = 9.10, p < .01, \eta^2 = .02$; and vocabulary, $F(1, 581) = 5.53, p < .05, \eta^2 = .01$.

Significant decline was not observed for word recall, story recall, or computation span although trends were in the expected direction.

Significant Age x Wave interactions were observed for semantic verification, $F(1, 581) = 14.51, p < .001, \eta^2 = .02$; lexical decision, $F(1, 581) = 8.20, p < .01, \eta^2 = .01$; computation span, $F(1, 543) = 5.48, p < .05, \eta^2 = .01$; story recall, $F(1, 581) = 7.80, p < .01, \eta^2 = .01$; word recall, $F(1, 581) = 13.06, p < .001, \eta^2 = .02$; and vocabulary, $F(1, 581) = 19.11, p < .001, \eta^2 = .03$. Consistent with previous analyses of these and related cognitive measures (e.g., Hultsch et al., 1998), performance for the Old-Old group was both lower in relative terms and declined more markedly over the 3-year period relative to the Young-Old group.

Further qualifying patterns of 3-year decline, significant Mortality Status x Wave interactions were observed for semantic verification, $F(1, 581) = 20.90, p < .001, \eta^2 = .04$; lexical decision, $F(1, 581) = 10.69, p < .01, \eta^2 = .02$; sentence construction, $F(1, 581) = 7.31, p < .01, \eta^2 = .01$; story recall, $F(1, 581) = 13.80, p < .001, \eta^2 = .02$; and fact recall, $F(1, 581) = 4.68, p < .05, \eta^2 = .01$. Figure 4 shows that, prior to their death, decedents declined more precipitously for semantic verification, lexical decision, story recall, and fact recall. For sentence construction, Survivors declined over time whereas performance for Decedents was stable (albeit at a lower absolute level of performance). Significant 3-way interactions (Mortality Status x Age Group x Wave) also emerged for semantic



Sem = semantic verification, Lex = lexical decision, Cspan = computation span, Lspan = listening span, Sentcon = sentence construction, Story = story recall, Word = word recall, Fact = fact recall, Voc = vocabulary.

Figure 4

Mortality Group Differences for 3-Year Cognitive Change (T-Scores)

verification, $F(1, 581) = 7.78, p < .01, \eta^2 = .01$; lexical decision, $F(1, 581) = 5.01, p < .05, \eta^2 = .01$; and vocabulary, $F(1, 581) = 13.73, p < .001, \eta^2 = .02$. Patterns revealed that Old-Old decedents showed the most 3-year decline for these verbal measures (see Table 7 and Figure 5).

Controlling for baseline performance. The observed Mortality Status interactions may, in part, be related to preexisting differences in cognitive ability for the initial wave of testing. A series of repeated-measures ANCOVAs (one for each of the 9 cognitive measures) were computed to examine whether decedents exhibited accelerated cognitive decline across the last occasions of measurement even after controlling for baseline differences in cognition and education. Only results from the interactions of primary interest (Mortality Status x Wave and Mortality Status x Age Group x Wave) are reported. Corresponding baseline differences in cognitive performance were covaried for each individual measure. Even after controlling for baseline cognitive differences, significant results for the 2-way and 3-way interactions mirrored those previously reported. Mortality Status x Wave interactions remained significant for semantic verification, $F(1, 580) = 18.84, p < .001, \eta^2 = .03$; lexical decision, $F(1, 580) = 10.92, p < .01, \eta^2 = .02$; sentence construction, $F(1, 580) = 6.62, p < .05, \eta^2 = .01$; story recall, $F(1, 580) = 13.52, p < .001, \eta^2 = .02$; and fact recall, $F(1, 580) = 5.11, p < .05, \eta^2 = .01$. Similarly, Mortality Status x Age Group x Wave interactions remained significant for semantic verification, $F(1, 580) = 7.16, p < .01, \eta^2 = .01$; lexical decision, $F(1, 580) = 5.18, p < .05, \eta^2 = .01$; and vocabulary, $F(1, 580) = 13.96, p < .001, \eta^2 = .02$. Except for sentence construction, decedents declined more than Survivors with accelerated decline for Old-Old Decedents.

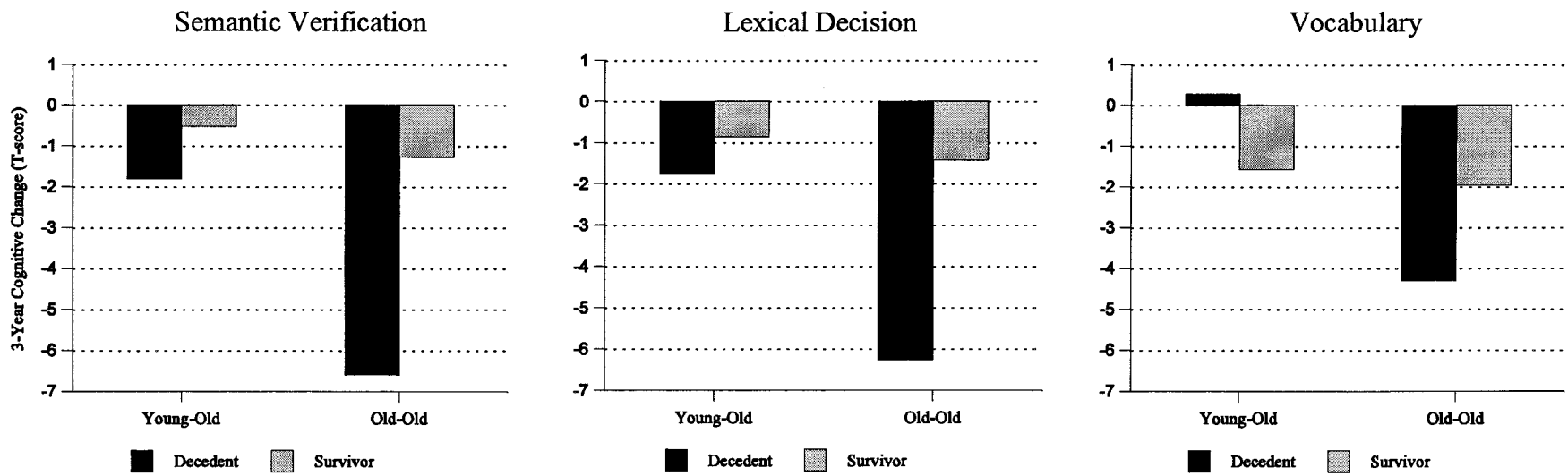


Figure 5

Mortality and Age Group Differences for 3-Year Cognitive Decline (T-scores)

Repeated-measures multivariate analysis of covariance for 6-year change. Six-year cognitive change, spanning the third from last, second from last, and last measurement waves respectively for each participant, was examined using a 2 (age group) x 2 (mortality status) x 3 (wave) repeated-measures MANCOVA partialling education. A total of 189 decedents did not complete all 3 waves of testing yielding a listwise $n = 518$. Table 8 outlines 6-year (3 wave) cognitive performance trends as a function of mortality status and age group.

Significant omnibus effects were associated with Age, Wilks' $\lambda = .860$, $F(7, 507) = 11.75$, $p < .001$, $\eta^2 = .14$; Mortality Status, Wilks' $\lambda = .901$, $F(7, 507) = 7.93$, $p < .001$, $\eta^2 = .10$; Wave, Wilks' $\lambda = .922$, $F(14, 500) = 3.04$, $p < .001$, $\eta^2 = .08$; the Age Group x Wave interaction, Wilks' $\lambda = .893$, $F(14, 500) = 4.26$, $p < .001$, $\eta^2 = .11$; the Mortality x Wave interaction, Wilks' $\lambda = .871$, $F(14, 500) = 5.31$, $p < .001$, $\eta^2 = .13$; the Mortality x Age Group x Wave interaction, Wilks' $\lambda = .924$, $F(14, 500) = 2.93$, $p < .001$, $\eta^2 = .08$; and the Education covariate, Wilks' $\lambda = .831$, $F(7, 507) = 14.70$, $p < .001$, $\eta^2 = .17$. The corresponding repeated-measures MANCOVA for the two working memory measures (listwise $n = 475$) yielded significant omnibus effects for: Age, Wilks' $\lambda = .986$, $F(2, 469) = 3.26$, $p < .05$, $\eta^2 = .01$; Wave, Wilks' $\lambda = .975$, $F(4, 467) = 2.99$, $p < .05$, $\eta^2 = .03$; the Age Group x Wave interaction, Wilks' $\lambda = .980$, $F(4, 467) = 2.42$, $p < .05$, $\eta^2 = .02$; and the Education covariate, Wilks' $\lambda = .951$, $F(2, 469) = 12.02$, $p < .001$, $\eta^2 = .05$. Of note, Mauchly's test of sphericity was significant for the within-subjects wave effect associated with every individual cognitive measure (indicating a violation of the circularity assumption). However, univariate tests were significant for each cognitive measure whether using epsilon adjusted (e.g., Greenhouse-Geisser) or unadjusted degrees of

Table 8
Three Wave (6-Year) Cognitive Performance (T-Scores) by Mortality Status and Age Group

Variable	Survivors						Decedents					
	Young-Old (n = 337)			Old-Old (n = 105)			Young-Old (n = 36)			Old-Old (n = 40)		
	TLWave	SLWave	LWave	TLWave	SLWave	LWave	TLWave	SLWave	LWave	TLWave	SLWave	LWave
Verbal Speed												
Semantic	51.90 (8.45)	51.88 (8.58)	51.33 (10.29)	48.57 (9.15)	47.34 (8.95)	45.91 (10.34)	51.23 (6.64)	51.93 (5.92)	47.87 (8.40)	47.64 (10.10)	43.33 (10.62)	36.49 (18.82)
Lexical	50.84 (9.02)	52.44 (8.05)	51.54 (10.30)	48.59 (8.19)	47.76 (9.72)	46.13 (9.96)	51.33 (6.32)	50.89 (6.98)	47.42 (8.64)	48.04 (8.28)	43.67 (12.15)	37.76 (20.18)
Working Memory												
Computation	51.38 (9.45)	51.77 (9.60)	50.99 (10.27)	47.47 (9.39)	47.63 (10.26)	44.98 (8.99)	49.53 (7.15)	45.45 (10.33)	50.35 (13.22)	47.65 (7.51)	45.52 (9.45)	43.39 (12.70)
Listening	52.20 (10.02)	51.67 (8.70)	50.54 (9.57)	47.39 (9.62)	47.76 (10.42)	44.68 (10.80)	47.30 (10.12)	48.28 (8.58)	46.32 (10.38)	46.70 (10.33)	47.13 (11.81)	41.59 (10.22)
Sentence	51.29 (8.40)	53.03 (7.75)	50.24 (9.26)	47.55 (10.92)	50.46 (9.51)	46.25 (12.32)	50.98 (8.72)	48.91 (8.69)	47.76 (9.90)	46.91 (10.97)	41.96 (14.78)	39.89 (15.98)
Episodic Memory												
Story Recall	52.81 (8.97)	51.34 (8.59)	52.20 (9.87)	48.81 (10.43)	47.06 (9.40)	46.36 (10.39)	47.55 (8.69)	47.01 (8.87)	45.94 (9.91)	43.99 (10.24)	42.03 (10.37)	39.39 (11.27)
Word Recall	52.24 (8.57)	52.55 (8.91)	51.27 (9.36)	48.06 (8.96)	48.38 (11.42)	44.01 (10.40)	50.13 (7.77)	49.60 (8.96)	46.95 (9.66)	46.11 (10.20)	43.64 (11.01)	37.22 (12.88)

Table 8 con't

Variable	Survivors						Decedents					
	Young-Old (n = 337)			Old-Old (n = 105)			Young-Old (n = 36)			Old-Old (n = 40)		
	TLWave	SLWave	LWave	TLWave	SLWave	LWave	TLWave	SLWave	Lwave	TLWave	SLWave	Lwave
Semantic Memory												
Fact Recall	51.98 (9.17)	52.10 (9.21)	50.63 (9.90)	48.97 (9.55)	47.28 (9.91)	45.62 (10.16)	51.58 (9.63)	49.04 (9.77)	46.35 (10.03)	47.62 (10.29)	45.37 (10.88)	41.31 (12.18)
Vocabulary	49.79 (10.14)	50.34 (9.24)	48.75 (9.72)	51.15 (10.01)	51.07 (10.77)	49.03 (10.77)	51.77 (7.87)	50.21 (9.57)	51.39 (7.71)	52.69 (10.56)	53.21 (9.66)	47.22 (14.49)

Semantic = semantic verification; lexical = lexical decision; computation = computation span; listening = listening span; sentence = sentence construction. TLWave refers to a given individual's third last wave of testing, SLWave refers to her/his second last wave, with Lwave referring to the last (i.e., most recent) wave of testing. Standard deviations are reported in parentheses. Listwise n = 518 for the 6-year repeated-measures analyses except for listening and computation span where listwise n = 475 (337 YO survivors, 105 OO survivors, 10 YO decedents and 23 OO decedents).

freedom. For all subsequent analyses, when the adjusted and unadjusted degrees of freedom both yield significant results, only the test associated with the sphericity-assumed degrees of freedom is reported.

Significant univariate tests of age group and mortality status differences were consistent with those reported for the previous analyses. Young-Old adults performed significantly better than the Old-Old ($p < .05$) for all measures but vocabulary. Similarly, collapsing across all waves of cognitive testing, Survivors significantly outperformed Decedents for all measures except vocabulary and computation span ($p < .01$ for all but fact recall which was $p < .05$). However, in contrast to the cross-sectional and 3-year repeated measures analyses, no significant Age Group x Mortality Status interactions were observed for the 6-year repeated-measures MANCOVAs.

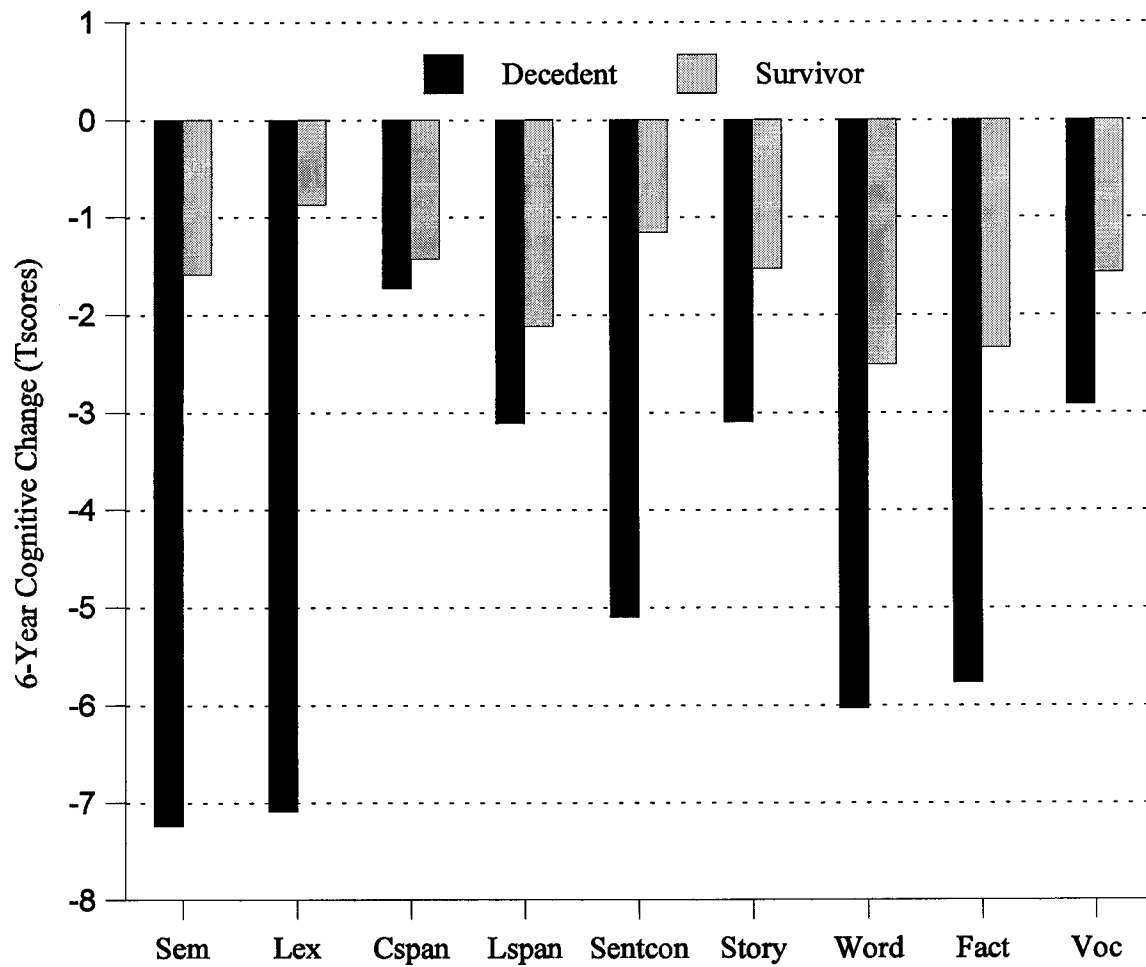
Univariate tests of the within-subjects Wave effect demonstrated significant 3-year cognitive decline for semantic verification, $F(2, 1026) = 7.06, p < .01, \eta^2 = .01$; lexical decision, $F(2, 1026) = 3.29, p < .05, \eta^2 = .01$; listening span, $F(2, 940) = 5.48, p < .01, \eta^2 = .01$; sentence construction, $F(2, 1026) = 3.19, p < .05, \eta^2 = .01$; word recall, $F(2, 1026) = 3.33, p < .05, \eta^2 = .01$; fact recall, $F(2, 1026) = 7.28, p < .01, \eta^2 = .01$; and vocabulary, $F(2, 1026) = 3.19, p < .05, \eta^2 = .01$ (the alpha level associated with 6-year cognitive decline for story recall was $p = .05$). Within-subjects contrasts indicated the shape of decline was linear for all significant wave effects ($p < .05$). A significant quadratic effect was observed for listening span ($p < .05$) indicating accelerated decline between the second and third measurement waves.

Significant Age x Wave interactions were observed for semantic verification, $F(2, 1026) = 13.78, p < .001, \eta^2 = .03$; lexical decision, $F(2, 1026) = 8.61, p < .001, \eta^2 = .02$;

computation span, $F(2, 940) = 3.90, p < .05, \eta^2 = .01$; story recall, $F(2, 1026) = 3.70, p < .05, \eta^2 = .01$; word recall, $F(2, 1026) = 11.26, p < .001, \eta^2 = .02$; and vocabulary, $F(2, 1026) = 15.63, p < .001, \eta^2 = .03$. Relative to the Young-Old, Old- Old participants performed lower in relative terms and declined more markedly over the 6-year period.

Significant Mortality Status x Wave interactions were observed for semantic verification, $F(2, 1026) = 19.93, p < .001, \eta^2 = .04$; lexical decision, $F(2, 1026) = 14.22, p < .001, \eta^2 = .03$; sentence construction, $F(2, 1026) = 10.98, p < .001, \eta^2 = .02$; word recall, $F(2, 1026) = 6.53, p < .01, \eta^2 = .01$; and fact recall, $F(2, 1026) = 11.52, p < .001, \eta^2 = .02$. Longitudinal decline profiles significantly differed between Survivors and Decedents. For each of these interactions, within-subjects contrasts indicated the shape of decline was linear ($p < .01$). Accelerated quadratic declines for decedents were observed for semantic verification ($p < .05$) following the second last wave of testing, and for computation span and sentence construction ($p < .05$) between the third and second last testing waves. In comparison to the 3-year analyses, story recall was no longer significant but word recall (another indicator of episodic memory) demonstrated significant declines as a function of mortality. All other patterns were identical to those reported previously. Figure 6 demonstrates the greater relative decline of Decedents compared with Survivors.

In addition, significant Mortality Status x Age Group x Wave interactions emerged for semantic verification, $F(2, 1026) = 4.92, p < .01, \eta^2 = .01$ and vocabulary, $F(2, 1026) = 11.22, p < .001, \eta^2 = .02$. The 3-way interaction for lexical decision was not significant, although the trends were in the expected direction. For both significant 3-way interactions, within-subjects contrasts indicated linear decline ($p < .01$). Accelerated



Sem = semantic verification, Lex = lexical decision, Cspan = computation span, Lspan = listening span, Sentcon = sentence construction, Story = story recall, Word = word recall, Fact = fact recall, and Voc = vocabulary.

Figure 6

Mortality Group Differences for 6-Year Cognitive Change (T-scores)

cognitive decline for Old-Old decedents was observed for vocabulary between the second last and last waves of testing ($p < .01$). Figure 7 demonstrates that Old-Old decedents showed the most 6-year decline, as well as lower relative performance means (see Table 8), for these measures.

Controlling for baseline performance. Wave 1 cognitive performance was partialled in order to examine whether the Mortality Status x Wave and Mortality Status x Age Group x Wave interactions were attenuated or eliminated. As before, a series of nine repeated-measures ANCOVAs (one for each cognitive measure) were computed to examine whether decedents demonstrate accelerated cognitive decline even after holding constant corresponding baseline differences in cognition and education. Partialling preexisting baseline differences exerted little influence on patterns of significance for the 2-way and 3-way mortality status interactions. Mortality Status x Wave interactions remained significant for semantic verification, $F(2, 1024) = 20.88, p < .001, \eta^2 = .04$; lexical decision, $F(2, 1024) = 15.30, p < .001, \eta^2 = .03$; sentence construction, $F(2, 1024) = 12.37, p < .001, \eta^2 = .02$; word recall, $F(2, 1024) = 8.86, p < .001, \eta^2 = .02$; and fact recall, $F(2, 1024) = 11.90, p < .001, \eta^2 = .02$. In fact, a significant Mortality Status x Wave interaction emerged for story recall, $F(2, 1024) = 4.22, p < .05, \eta^2 = .01$, after controlling for baseline performance differences. The Mortality Status x Age Group x Wave interactions also remained significant for semantic verification, $F(2, 1024) = 5.23, p < .01, \eta^2 = .01$ and vocabulary, $F(2, 1024) = 11.62, p < .001, \eta^2 = .02$. Consistent with previous findings, Decedents declined more than Survivors with Old-Old decedents declining more precipitously.

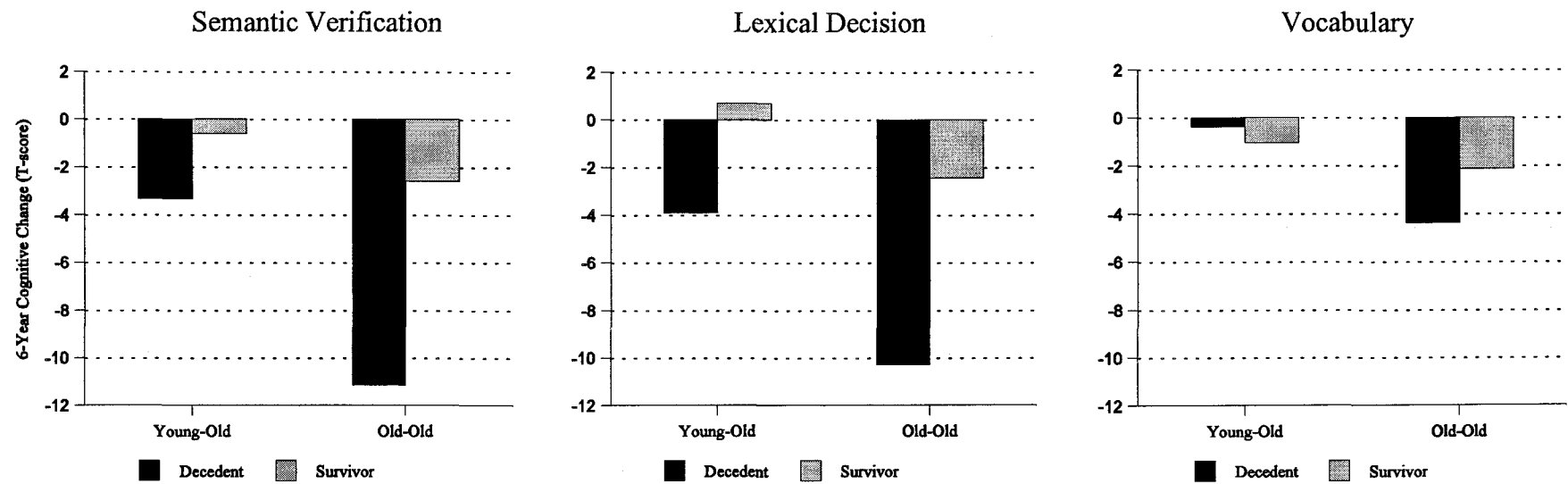


Figure 7

Mortality and Age Group Differences for 6-Year Cognitive Decline (T-scores)

Multilevel Models of Cognitive Change

Previous sections employed repeated measures ANCOVA to examine mortality differences at cross-section and over time. These analyses were important for replicating and facilitating comparisons with published results. The present section employed multilevel analyses to extend these between-group findings. Specifically, intraindividual change for all waves of cognitive measurement was examined using multilevel modeling and the hierarchical linear modeling (HLM) software package (Raudenbush, Bryk, & Congdon, 2001). Among other advantages, HLM circumvents the listwise deletion concerns associated with repeated-measures ANCOVA by examining performance change for all individuals (despite varying waves of participation). For these analyses, cognitive change across all available waves of measurement was examined as a function of increasing age and mortality status. In a later section, a more detailed multilevel analysis examines the shape of cognitive change, specifically differentiating terminal decline vs. terminal drop patterns, as a function of time to death.

HLM equations. HLM examines individual change at two levels: across multiple occasions (Level 1) and for multiple occasions nested within individuals (Level 2). For each cognitive measure, longitudinal change was examined as a function of: (a) chronological age at each wave of measurement (HLM Level 1 variable); and (b) mortality status (HLM Level 2 variable). The following multilevel equations examined whether individual trajectories of cognitive decline varied as a function of increasing age or mortality status.

At the Level 1, or the within-subjects level, HLM equations are tantamount to a single regression equation for each individual case.

$$\text{Level 1: Cognitive Performance}_{it} = B_{0i} + B_{1i} * (\text{Age at Testing}_{it}) + R_{it} \quad (1)$$

For each cognitive measure, the specified Level 1 model (see equation 1) examined individual rates of change as a function of increasing age across measurement occasions (e.g., 73 at Wave 1, 76 at Wave 2, 80 at Wave 3). Specifically, cognitive performance for a given individual (i) at a given time (t) was modeled as a function of that individual's performance for her/his age at the initial Wave of measurement (B_{0i} : the intercept), plus her/his average individual rate of change across waves with increasing age (B_{1i} : the slope), plus a random within-subject error term (R) representing prediction error holding intercept and slope constant. For all analyses, Level 1 chronological age was grand-mean centered (initial $\underline{M}$ = 71.88 years); findings reflect cognitive performance at the sample midpoint on age.

To estimate average effects for the entire sample, individual growth parameters (B_{0i} and B_{1i}) representing predictors in the Level 1 model become dependent measures for the Level 2 between-person equations.

$$\text{Level 2: } B_{0i} = \gamma_{00} + \gamma_{01} * (\text{Mortality Status}_i) + U_{0i} \quad (2)$$

$$B_{1i} = \gamma_{10} + \gamma_{11} * (\text{Mortality Status}_i) + U_{1i} \quad (3)$$

These Level 2 equations assessed whether mortality status differences were associated with different trajectories of individual change with increasing age (across waves).

Equation 2 reflects a given individual's predicted cognitive performance (separately for each measure) for an average aged participant (B_{0i}) as a function of cognitive

performance for Survivors of average age (γ_{00}), the average difference in performance between mortality groups for those of average age (γ_{01}), plus a random effect (U_{0i}) estimating variability about that sample mean holding all predictors constant (i.e., are there additional between-group differences in cognitive performance controlling for predictors entered in the model). Equation 3 examines the predicted linear rate of change for each individual (B_{1i}) as a function of the average change (increment or decrement) in cognitive performance per year increase in age (above the average) for Survivors (γ_{10}), the average difference in change per year increase in age between Survivors and Decedents (γ_{11}), plus a random effect (U_{1i}) estimating whether variability remains about cognitive performance slopes holding these predictors constant (i.e., do between-group differences in within-person cognitive change remain to be explained).

Table 9 summarizes multilevel findings associated with equations 1 to 3. Tabled HLM coefficients are presented in raw score metric. Values were not standardized as t-scores due to unequal listwise n and differing patterns of complete data across individuals, tasks, and measurement occasions. Semantic verification and lexical decision coefficients reflect milliseconds, sentence construction coefficients reflect values on a 0 to 6 scale, listening and computation span coefficients reflect values on a 0 to 7 scale, story recall coefficients reflect percent correct, and word recall, fact recall, and vocabulary coefficients reflect raw scores recalled out of 30, 40, and 54 respectively.

For each of the cognitive measures, coefficients in the intercept column reflect performance for survivors at the average sample age. Although significant ($p < .01$), individual tests of these intercepts as significantly different from 0 are not of theoretical interest. Of greater import was whether intraindividual rates of cognitive change

Table 9

Rate of Cognitive Change as a Function of Increasing Age and Mortality Status

Variable	Intercept γ_{00}	Slope γ_{10}/γ_{11}	SE	t
Semantic verification	3214.14	15.02 53.78	3.44 7.05	4.37** 7.63**
Lexical decision	1021.31	3.41 19.75	1.49 2.90	2.29* 6.82**
Sentence Construction	5.18	-0.02 -0.03	0.005 0.01	-4.24** -2.93**
Listening span	3.11	-0.03 -0.03	0.004 0.01	-6.53** -2.60**
Computation span	3.22	-0.03 -0.03	0.005 0.01	-4.91** -2.23*
Story recall	37.42	-0.09 -0.32	0.03 0.07	-2.93** -4.75**
Word recall	18.54	-0.09 -0.14	0.01 0.03	-6.66** -4.70**
Fact recall	21.16	-0.14 -0.21	0.02 0.03	-8.92** -6.16**
Vocabulary	44.87	-0.06 -0.05	0.01 0.03	-4.34** -1.59

γ_{00} = cognitive performance for Survivors at the midpoint on age ($\bar{M} = 71.88$); γ_{10} =

average rate of linear change per additional year of age for Survivors; γ_{11} = average

difference in rate of linear change between Survivors and Decedents; SE = standard error;

t = t-ratio.

* $p < .05$

** $p < .01$

systematically varied as a function of impending mortality. There are two slopes associated with each cognitive measure. The first slope (γ_{10}) tests the average rate of cognitive change for Survivors with each year of increasing age (similar to a main effect of Time in repeated measures ANCOVA). The second slope (γ_{11}) reflects the average difference in cognitive change (per year increase in age) between Survivors and Decedents. The second slope coefficient is similar to a Mortality Status x Time interaction in repeated measures ANCOVA; it tests how much more Decedents decline relative to Survivors per additional year of age.

Survivors performed more poorly on all cognitive measures with increasing age (see Table 9, slope γ_{10}). Response latencies significantly increased for semantic verification and lexical decision with significant performance declines observed for sentence construction, listening span, computation span, story recall, word recall, fact recall, and vocabulary. Relative to Survivors, performance for those facing impending death deteriorated at significantly faster rates per year increase in age for all measures except vocabulary. Using semantic verification as an example, a Survivor of average age in the sample had a response latency of 3214.14 msec and declined 15.02 msec for each additional year increase in age. Over the course of a decade (i.e., between 71.88 and 81.88 years of age), the average individual's response latency would increase by 150.20 msec. Of particular interest, the second slope coefficient for semantic verification indicated that the average difference between rates of change for Survivors and Decedents was significant. An average-aged individual who would die exhibited significantly slower response latencies per year of increasing age ($15.02 + 53.78 = 68.80$ msec per year or 688.00 msec per decade) relative to an average-aged Survivor (15.02 msec per year or

150.2 msec per decade). For each model, corresponding random effect coefficients for both intercept and slope were examined and found to be significant ($p < .01$). This indicates that, even after controlling for age and mortality differences, significant between-group differences in intercepts (average performance) and slopes (individual rates of change with increasing age) remain to be explained.

Finally, a Level 1 quadratic age term was also assessed for each cognitive measure. To reduce multicollinearity, linear age was centered about its grand mean and the quadratic predictor was computed by squaring the centered linear age variable. Significant quadratic effects were observed for semantic verification ($\gamma_{20} = 0.87, t = 2.88$ and $\gamma_{21} = 2.52, t = 3.73$) and lexical decision ($\gamma_{20} = 0.27, t = 1.97$ and $\gamma_{21} = 1.10, t = 3.75$). For semantic verification performance, response latencies for Survivors increased by an additional .87 msec (over and above the linear rate of 15.02) for each additional year above the average sample age with slower latencies accelerated a further 2.52 msec per year (i.e., 3.39 msec total) for Decedents relative to Survivors. The same interpretation applies to lexical decision. With increasing age, response latencies increased at a faster rate for Decedents relative to Survivors. For the remaining 7 cognitive measures, individual quadratic effects were not significant. Moreover, differences between deviance statistics (distributed as chi-square values) for models with and without the quadratic effect were not significant indicating that the additional complexity of the linear plus quadratic model was not warranted. Accordingly, findings from the linear age models are reported in Table 9 for these measures.

Summary: The Influence of Age and Impending Death on Cognitive Performance.

The defining question for this first section considered whether mortality-related

cognitive deficits were uniform across the adult life span. Overall, findings from this section refute the Riegel and Riegel (1972) suggestion that mortality-related cognitive deficits are attenuated for those over 75 years of age. Rather, both cross-sectional and longitudinal findings indicated that individual differences in cognitive performance between Survivors and Decedents increased with age. Cross-sectional findings for each participant's respective last wave of testing indicated that: (a) Young-Old adults (59-79 years) significantly outperformed Old-Old adults (80-95 years) for all but the vocabulary measure; (b) Survivors significantly outperformed Decedents for all cognitive measures; and (c) mortality status differences were further modified by age group for semantic verification, lexical decision, fact recall, and vocabulary (Old-Old Decedents exhibited the poorest performance). Although not all age by mortality status interactions were significant, Cohen's *d* indicated the magnitude of mortality status differences was usually larger for the Old-Old age group. Similarly, longitudinal repeated-measures ANCOVA across 3- and 6-year change intervals indicated that, prior to their death, Decedents declined more precipitously than Survivors for multiple cognitive measures. Moreover, significant 3-way interactions (Mortality Status x Age Group x Wave) for semantic verification, lexical decision, and vocabulary indicated that Old-Old Decedents exhibited steeper decline compared with similarly aged and younger Survivors. Finally, examination of intraindividual cognitive decline using HLM indicated: (a) significant decline with increasing age for all cognitive measures; and (b) significant mortality differences in rates of cognitive decline with those facing impending death declining at significantly faster rates per year increase in age. Relative to the repeated measures findings, HLM results demonstrated more consistent mortality differences (including age

by mortality interactions) across the range of cognitive measures.

Having demonstrated that impending death negatively impacts cognitive performance across age groups, a related question concerns whether mortality-related cognitive deficits are uniform across cognitive domain. Of particular interest is whether impending death influences both well-preserved cognitive abilities as well as those that typically exhibit age decrements. Although analyses from the preceding section speak to this issue (e.g., comparison of T-scores in Tables 7 and 8 suggest decline is greater for some measures than others), such differences were not examined using inferential test statistics. Direct (as opposed to informal) mean comparisons between cognitive measures require use of alternative statistical techniques. In the next section, profile analysis was employed to examine the domain specificity of mortality-related deficits across a range of cognitive measures.

Cognitive Performance Profiles as a Function of Mortality Status

The following profile analyses were designed to examine whether terminal decline represents a global phenomenon influencing multiple cognitive domains or a local phenomenon affecting select cognitive abilities (c.f., Berg, 1996, Small & Bäckman, 1999). Previous findings suggest that measures typically resistant to the aging process (e.g., crystallized abilities) exhibit the largest mortality-related effects. To examine this assertion, both cross-sectional differences and longitudinal changes in cognitive profiles (i.e., performance trends across a process-to-products continuum of cognitive measures) were examined as a function of mortality status and age group. The continuum ranged from basic process measures (e.g., verbal speed and working memory), through memory measures (e.g., episodic and semantic memory), to cognitive products indexed by crystallized verbal ability (Hultsch et al., 1998). To facilitate cross-domain comparisons, cognitive outcome measures were linearly transformed to T-scores ($M = 50$ and $SD = 10$). Reaction time latency measures were reverse coded so that lower T-scores reflected poorer cognitive performance.

Profile analysis is a special application of MANOVA where multiple dependent tests are all measured on the same scale (a T-score metric in the present case). This technique was used to assess whether cognitive performance profiles exhibited the same mean patterns as a function of age group and mortality status. For present purposes, two profile analysis effects were of primary interest. The test of flatness collapses across groups and assesses the similarity of responses across all dependent cognitive measures (specifically comparing adjacent segments of the continuum), evaluating the same

hypothesis as the within-subjects main effect in a repeated-measures ANOVA. Of greater import, the test of parallelism examines the group by cognitive task profile interaction; it tests whether survivors and decedents have the same performance profiles across the continuum of cognitive measures. The parallelism test is the same as the test of the mixed-model interaction in repeated-measures ANOVA. Although not of interest in the present context, profile analysis also includes a test of level. This test is tantamount to a group main effect in repeated-measures ANOVA.

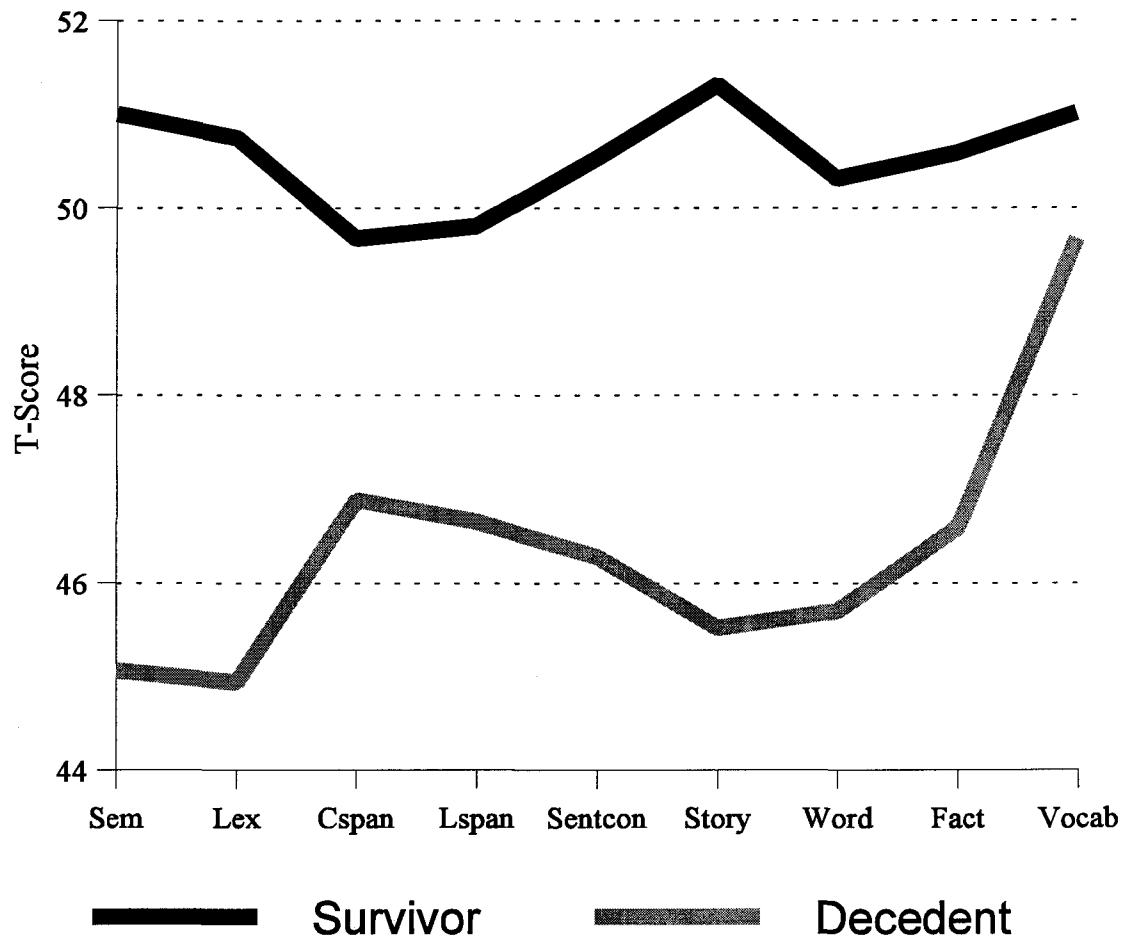
Follow-up post-hoc analyses were computed to decompose significant parallelism interactions. Multiple mean comparisons were made across groups and repeated cognitive measures. As recommended by SaVille (1990), the unrestricted least significant difference (LSD) procedure was employed to examine mean differences. Relative to other procedures that adjust alpha for multiple comparisons (e.g., Tukey's Honestly Significant Difference, Scheffé's adjustment) and consequently increase Type II errors, the LSD procedure returns more consistent results (i.e., significance of mean differences depends only on observed magnitude of differences, standard error of the differences, error degrees of freedom, and alpha level); it is the most appropriate for examining pairwise multiple comparisons. All post-hoc comparisons were computed using single-degree-of-freedom F tests and appropriate error terms from the general linear model (GLM) module in SPSS 11. For all LSD comparisons, a minimum alpha level of .05 was adopted; observed mean differences satisfying the more stringent $\alpha = .01$ criterion reflect more conservative tests of significance (SaVille, 1990).

Comparative Analysis of Mortality Deficits Across Diverse Cognitive Domains

Cross-sectional differences for final measurement wave. To test whether

mortality deficits varied as a function of cognitive domain, a 2 (age group) x 2 (mortality status) x 9 (cognitive task) profile ANOVA was computed for the last wave of testing (covarying for education). The nine cognitive tasks (semantic verification, lexical decision, computation span, listening span, sentence construction, story recall, word recall, fact recall, and vocabulary) were treated as a within-subjects variable. This analysis complements the previously reported Age Group x Mortality Status MANCOVA results with the added focus of contrasting cognitive constructs. The inclusion of the listening span and computation span measures resulted in listwise $n = 635$. Consistent with the objective (i.e., examining terminal decline in relation to global vs. local cognitive influences), only main effects and interactions associated with mortality group differences are discussed.

The within-subjects effect across cognitive profiles was significant, $F(8, 5040) = 5.59, p < .001, \eta^2 = .01$, indicating deviation from flatness. Collapsing across groups, mean performance was inconsistent across cognitive measures. Qualifying this effect, a significant Mortality Status x Cognitive Profiles interaction was observed, $F(8, 5040) = 5.07, p < .001, \eta^2 = .01$, implying deviation from parallelism. Figure 8 shows divergent performance profiles for survivors and decedents across the cognitive measures continuum. To further probe this significant two-way parallelism effect, post-hoc simple effects (using the LSD procedure) examined mean mortality differences individually for each of the nine measures comprising the cognitive profile (e.g., the first simple effect examined mean differences between survivors and decedents for semantic verification). Significant mortality group differences ($p < .001$) were observed for all measures of the continuum except vocabulary. Decedents performed more poorly than survivors for



Sem = semantic verification, Lex = lexical decision, Cspan = computation span, Lspan = listening span, Sentcon = sentence construction, Story = story recall, Word = word recall, Fact = fact recall, and Vocab = vocabulary.

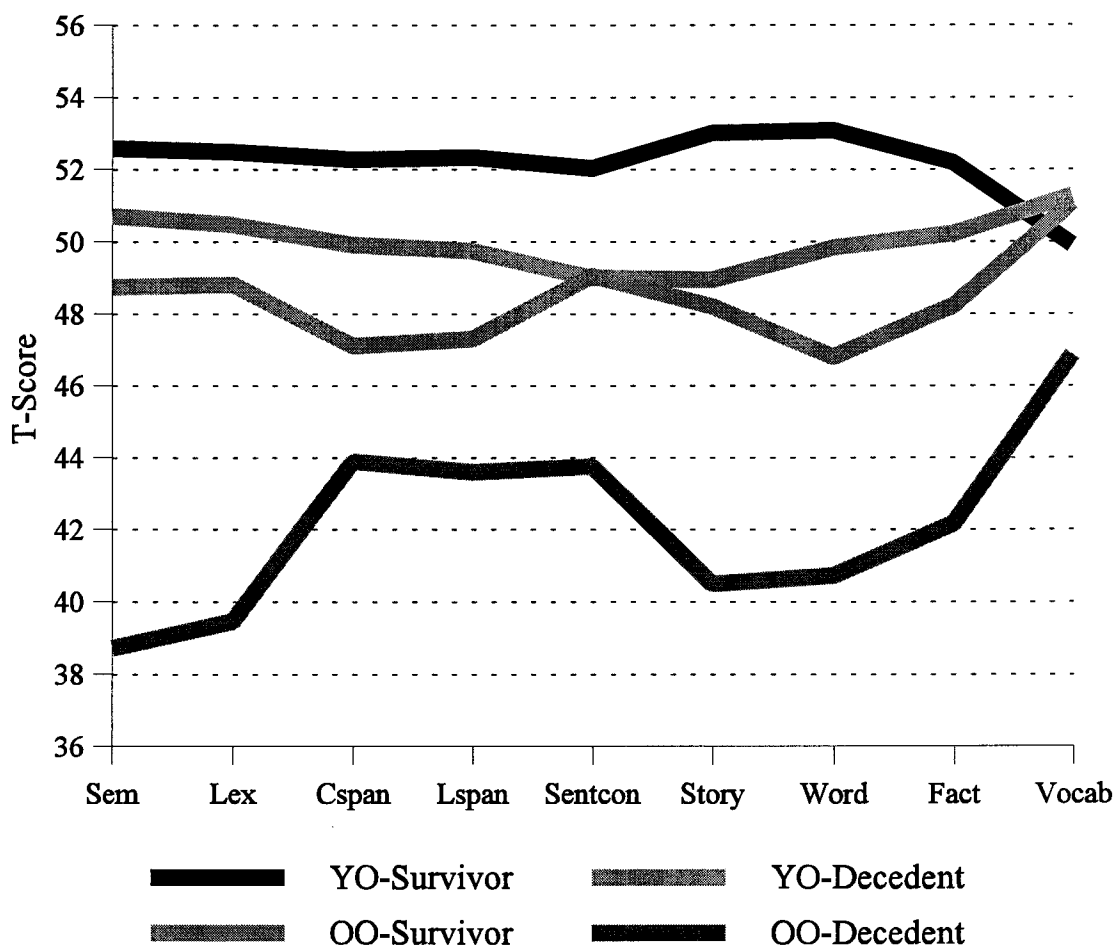
Figure 8

Last Wave Performance Profiles Across Cognitive Domains for Survivors and Decedents

process and memory measures with mortality differences narrowing toward the product end of the continuum.

A second simple effects analysis was computed to examine differences across cognitive measures within each level of mortality group. The omnibus effect for survivors was not significant, $F(8, 623) = .73$, $p = .67$, indicating no mean performance differences across the cognitive continuum (see Figure 8). In contrast, a significant omnibus difference across cognitive measures was observed for decedents, $F(8, 623) = 6.01$, $p < .001$, $\eta^2 = .07$. Multiple comparisons for decedents indicated significant differences between: the two verbal speed measures and computation and listening span ($p < .05$); the three working memory measures and story recall ($p < .05$) as well as computation span and word recall ($p < .05$); and vocabulary and the other eight cognitive measures ($p < .01$). A difference contrast coefficient was applied to the within-subjects cognitive profile to compare vocabulary with the mean performance of the other eight cognitive measures; the contrast was significant, $F(1,192) = 11.97$, $p < .005$, $\eta^2 = .06$. Decedents exhibited the largest mean differences between vocabulary and all other cognitive measures. More modest differences were observed between indicators of verbal speed, working memory, and episodic memory.

The Mortality Status x Age Group x Cognitive Profiles interaction was also significant, $F(8, 5040) = 3.53$, $p < .001$, $\eta^2 = .01$. Figure 9 plots the three-way interaction outlining the departure from parallelism as a function of age group and mortality status. Visual inspection indicates the profile for the Old-Old Decedents is markedly different from the other three profiles. Post-hoc comparisons were computed to pinpoint performance differences within levels of group and task. Relative to the other three



Sem = semantic verification, Lex = lexical decision, Cspan = computation span, Lspan = listening span, Sentcon = sentence construction, Story = story recall, Word = word recall, Fact = fact recall, Vocab = vocabulary, YO = Young-Old, and OO = Old-Old.

Figure 9
Last Wave Performance Profiles Across Cognitive Domains for
Survivors and Decedents as a Function of Age Group

groups, Old-Old decedents performed significantly below mean levels for all cognitive tasks ($p < .001$) except for computation and listening span ($p < .05$ relative to Old-Old Survivors), and vocabulary ($p < .05$ relative to Young-Old Survivors). Similarly, Young-Old survivors significantly outperformed Young-Old decedents for lexical decision, computation span, and fact recall ($p < .05$) as well as listening span, sentence construction, story recall, and word recall ($p < .01$). Old-Old survivors differed from Young-Old decedents for computation span and word recall ($p < .05$). Finally, Young-Old survivors performed significantly better than Old-Old survivors for all cognitive measures except vocabulary ($p < .01$).

Post-hoc analyses were also computed to examine differences across cognitive profiles within levels of age and mortality group. Omnibus effects were significant for Young-Old survivors, $F(8, 624) = 4.30$, $p < .001$, Old-Old survivors, $F(8, 624) = 2.42$, $p < .05$, and Old-Old decedents, $F(8, 624) = 6.83$, $p < .001$, reflecting mean performance differences across the cognitive continuum for these groups. A simple effects analysis of the within-subjects cognitive profile was examined within level of age and mortality status. Young-Old survivors performed significantly worse on vocabulary relative to all other measures ($p < .01$) but exhibited no other mean differences between cognitive tasks. Old-Old survivors demonstrated significant differences between vocabulary and semantic verification and lexical decision ($p < .05$) as well as listening span, computation span, word recall, story recall, and fact recall ($p < .01$). Old-Old decedents exhibited significant mean differences between: the two verbal speed and three working memory measures ($p < .01$) as well as fact recall ($p < .05$); the three working memory measures and the two episodic memory measures ($p < .01$); and vocabulary and the two verbal

speed and two episodic memory measures ($p < .01$) as well as the three working memory measures ($p < .05$). Difference contrasts comparing vocabulary performance with the mean of the other eight cognitive measures were significant for Young-Old survivors, $F(1,336) = 36.60$, $p < .001$, $\eta^2 = .10$; Old-Old survivors, $F(1,104) = 12.36$, $p < .005$, $\eta^2 = .11$; and Old-Old decedents, $F(1,71) = 11.10$, $p < .005$, $\eta^2 = .14$. Overall, the simple effects revealed an interesting dissociation for mortality differences as a function of age. Young-Old survivors exhibited the poorest performance for crystallized vocabulary relative to indicators of verbal speed, working memory, episodic memory, and semantic memory constructs, whereas Old-Old decedents exhibited their best performance on vocabulary relative to these other cognitive domains.

Longitudinal change: Doubly multivariate profile analysis. A doubly multivariate profile analysis (Tabachnick & Fidell, 1996) was computed to test whether cognitive decline profiles varied as a function of mortality status, age group, and wave of testing. In addition to the between-subjects factors (age group and mortality status), two within-subject factors were incorporated into this analysis: wave (cognitive change across the 3-year interval) and cognitive profile (performance trends across the continuum of nine common-metric cognitive measures). This analysis complements the repeated-measures analyses reported earlier with the added focus of contrasting profiles of cognitive change over time. Of primary interest was whether Old-Old Decedents exhibited more precipitous decline for the last testing wave relative to the second last, and if so, whether rate of decline varied across cognitive task profiles. Main effects and interactions relevant to this question (i.e., those associated with mortality group differences) are reported.

To test whether mortality deficits varied as a function of cognitive domain, a 2 (age group) x 2 (mortality status) x 9 (cognitive task) x 2 (wave) profile analysis of variance was computed for the last two waves of testing (statistically covarying for education with listwise $n = 548$). The within-subjects effects across Cognitive Profiles, $F(8, 4344) = 5.88, p < .001, \eta^2 = .01$, and Waves, $F(1, 543) = 17.01, p < .001, \eta^2 = .03$, were significant indicating deviation from flatness. Mean performance was inconsistent across cognitive measures (collapsing over groups and waves). Similarly, significant decline was observed between the second last and last waves of testing (collapsing across groups and cognitive measures). Qualifying these main effects, multiple omnibus interactions were significant including: Mortality Status x Cognitive Profiles, $F(8, 4344) = 3.61, p < .001, \eta^2 = .01$; Mortality Status x Age Group x Cognitive Profiles, $F(8, 4344) = 2.19, p < .05, \eta^2 = .01$; Mortality Status x Wave, $F(1, 543) = 5.13, p < .05, \eta^2 = .01$; Mortality Status x Age Group x Wave, $F(1, 543) = 4.01, p < .05, \eta^2 = .01$; and Mortality Status x Cognitive Profile x Wave, $F(8, 4344) = 6.45, p < .001, \eta^2 = .01$. These significant interactions implied deviation from parallelism (i.e., group differences across cognitive profiles and waves of testing).

Post-hoc analyses were used to decompose relevant interactions. Two of the interactions (Mortality Status x Cognitive Profiles and Mortality Status x Age Group x Cognitive Profiles) replicated observed patterns for the preceding profile analysis (see Figures 8 and 9 respectively). Figure 10 shows 3-year change in cognitive performance (collapsed across all measures) for Survivors and Decedents. Relative to Survivors, Decedents declined at an accelerated rate over the 3-year period (although mortality group differences were significant at each wave, $p < .01$). This two-way association was

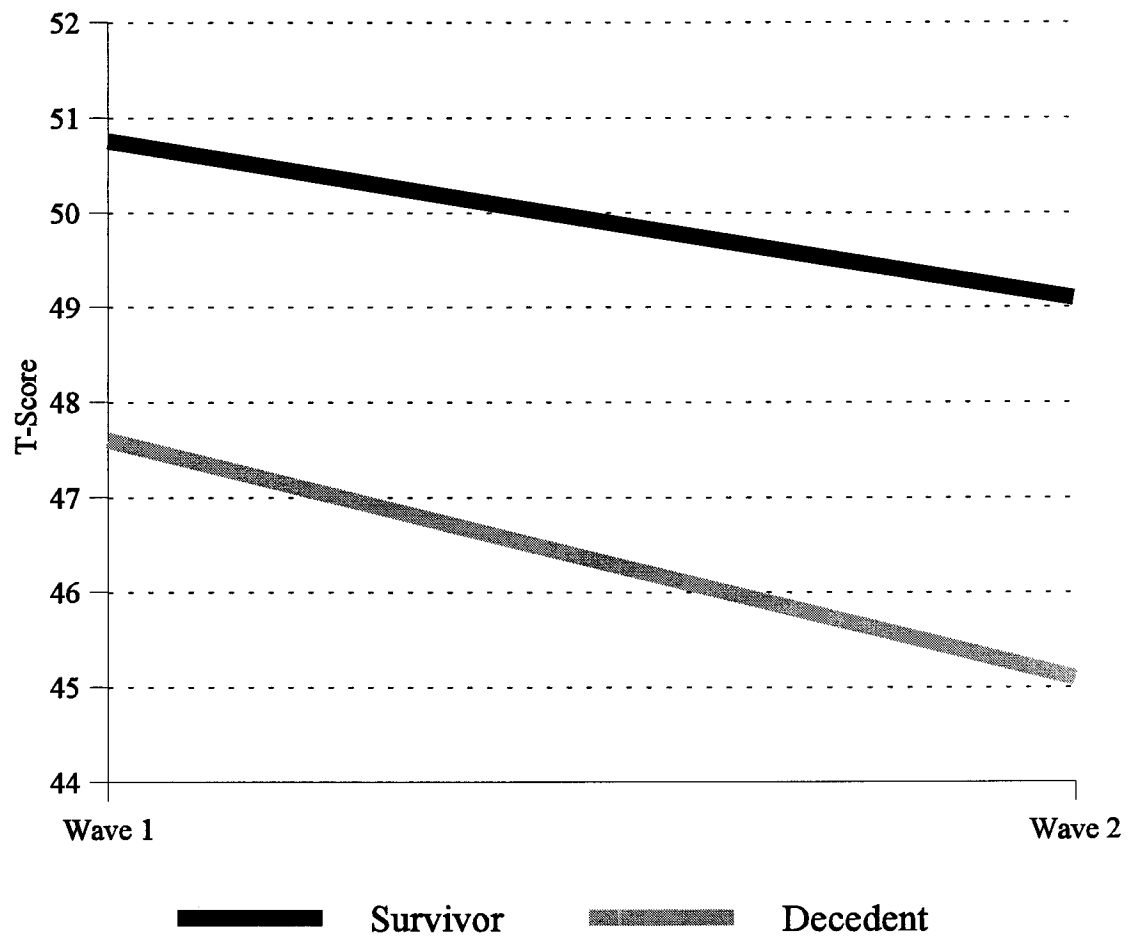


Figure 10

3-Year Change in Aggregate Cognitive Performance for Survivors and Decedents

further modified by age group with Old-Old Decedents exhibiting the most aggregate cognitive decline across the 3-year period (see Figure 11). Post-hoc simple effects for the Mortality Status x Age Group x Wave parallelism interaction were examined using the LSD approach to pinpoint performance differences as a function of wave and group. Group differences (Young-Old Survivor, Young-Old Decedent, Old-Old Survivor, Old-Old Decedent) for cognitive performance collapsed across the profiles was examined separately for each measurement wave. For Wave 1 performance, pairwise comparisons indicated that Young-Old Survivors performed significantly better than all other groups ($p < .01$) with Old-Old Decedents performing significantly worse than all groups ($p < .01$). Old-Old Survivors did not significantly differ from Young-Old Decedents. Identical group differences were observed for Wave 2. In addition, 3-year change was examined separately for each of the four groups; significant linear decline was observed for Young-Old Survivors ($p < .01$), Old-Old Survivors ($p < .01$), Young-Old Decedents ($p < .05$), and Old-Old Decedents ($p < .01$). The effect size associated with the simple effect of Wave was largest for Old-Old Decedents ($\eta^2 = .11$) relative to Young-Old Survivors ($\eta^2 = .06$), Old-Old Survivors ($\eta^2 = .08$), and Young-Old Decedents ($\eta^2 = .01$).

Finally, several simple effects analyses were computed to decompose the change in cognitive profiles for Survivors and Decedents by wave of testing. Relative to Survivors, Decedents appeared to decline more markedly for select cognitive measures (including verbal speed, episodic memory, and semantic memory). The first post-hoc analysis (simple effect for Wave) examined change in cognitive performance within each level of mortality status and cognitive profile (see Figure 12). Survivors demonstrated significant 3-year decline for verbal speed (semantic verification, $p < .05$, $\eta^2 = .01$; lexical

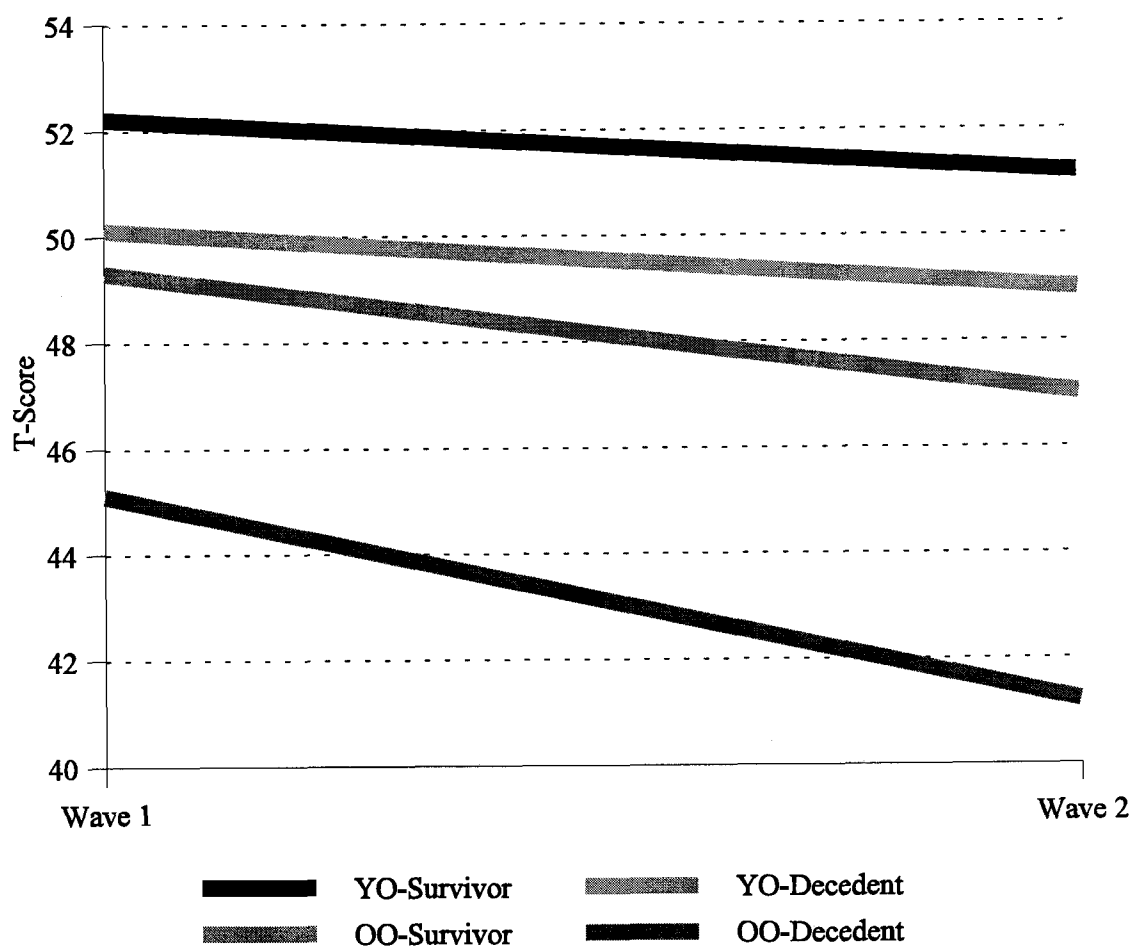
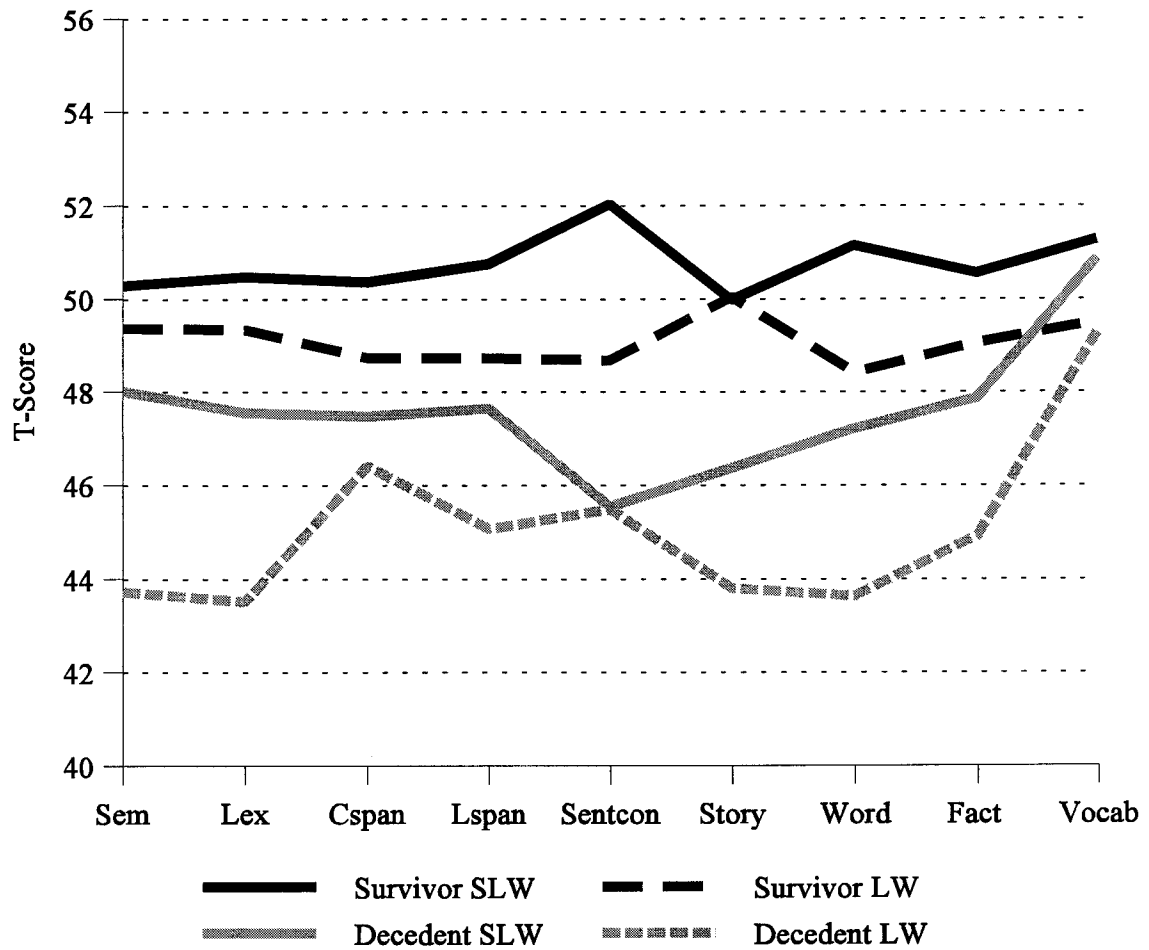


Figure 11

3-Year Change in Aggregate Cognitive Performance as a Function of
Mortality Status and Age Group



SLW = second last wave of testing, LW = last wave of testing, Sem = semantic verification, Lex = lexical decision, Cspan = computation span, Lspan = listening span, Sentcon = sentence construction, Story = story recall, Word = word recall, Fact = fact recall, Vocab = vocabulary, YO = Young-Old, and OO = Old-Old.

Figure 12
Mortality Differences in Cognitive Profiles
for the Second Last and Last Waves of Testing

significant Mortality Status x Cognitive Profile x Wave interaction. Figure 12 displays decision, $p < .05$, $\eta^2 = .01$), working memory (computation span, $p < .01$, $\eta^2 = .02$; listening span, $p < .001$, $\eta^2 = .03$; sentence construction, $p < .001$, $\eta^2 = .07$), episodic memory (word recall, $p < .001$, $\eta^2 = .07$), semantic memory (fact recall, $p < .001$, $\eta^2 = .04$), and verbal ability (vocabulary, $p < .001$, $\eta^2 = .06$). In comparison, Decedents demonstrated significant 3-year decline for verbal speed (semantic verification, $p < .001$, $\eta^2 = .07$; lexical decision, $p < .001$, $\eta^2 = .04$), working memory (listening span, $p < .01$, $\eta^2 = .02$), episodic memory (story recall, $p < .001$, $\eta^2 = .03$; word recall, $p < .001$, $\eta^2 = .04$), semantic memory (fact recall, $p < .001$, $\eta^2 = .05$), and verbal ability (vocabulary, $p < .01$, $\eta^2 = .02$). Contrasting these simple effects yielded several interesting trends: (a) Survivors significantly declined for all working memory measures whereas Decedents declined for only a single indicator; and (b) effect sizes associated with 3-year decline for Decedents were largest for verbal speed measures whereas working memory measures exhibited the largest change effects for Survivors (effect sizes for other domains were roughly comparable across mortality group).

As expected, simple effects for mortality status within each level of wave and cognitive profile showed significant differences between Survivors and Decedents for all cognitive measures (except vocabulary) for both testing waves ($p < .05$). As reflected in Figure 12, the effect size for mortality group differences increased between the second last and last wave for semantic verification (η^2 from .01 to .04), lexical decision (η^2 from .02 to .04), story recall (η^2 from .03 to .06), word recall (η^2 from .03 to .04), and fact recall (η^2 from .01 to .03) with remaining effect sizes demonstrating stability across waves except for sentence construction which decreased (η^2 from .08 to .01).

Finally, simple effects for cognitive profiles examined differences between cognitive measures (i.e., is performance parallel across cognitive domains or does it deviate from flatness) within level of mortality group and wave of testing. Significant omnibus differences across the 9-measure profiles were observed for Decedents at both the second last and last waves ($p < .001$, $\eta^2 = .06$ and $.08$ respectively) but only the second last wave of testing for Survivors ($p < .05$, $\eta^2 = .04$; see Figure 12). For Survivors, significant differences were observed between the following cognitive measures for the second last measurement wave: sentence construction performance was significantly higher than semantic verification ($p < .01$), lexical decision ($p < .05$), computation span ($p < .01$), listening span ($p < .05$), story recall ($p < .05$), and fact recall ($p < .05$); story recall performance was significantly lower than word recall ($p < .05$) and vocabulary ($p < .05$). Notably, Figure 12 shows that the profiles for Survivors at both waves of testing were relatively flat. In contrast, deviation from flatness was far more pronounced across cognitive profiles for Decedents. For the second last wave of testing, Decedents scored higher on the vocabulary test relative to each of the remaining eight cognitive measures ($p < .01$) with mean sentence construction falling significantly below performance for semantic verification ($p < .05$), listening span ($p < .05$), and fact recall ($p < .05$). For their respective final wave of testing, Decedents' vocabulary scores remained significantly higher than performance for the other eight measures ($p < .01$) with mean computation span scores significantly higher than semantic verification ($p < .05$), lexical decision ($p < .05$), story recall ($p < .05$), and word recall ($p < .01$). Interestingly, both Young-Old and Old-Old Decedents performed significantly better on the crystallized verbal ability measure relative to performance for any other cognitive domain.

Longitudinal 6-year change: doubly multivariate profile analysis. The inclusion of the computation and listening span tasks in the cognitive profile resulted in listwise $n = 475$ for the 6-year change analyses. Decedent group cell sizes (Young-Old Decedents = 10; Old-Old Decedents = 23) were too small to compute a second doubly multivariate profile analysis across the last three waves of testing. In a later section of the results, corresponding HLM analyses using all available data will examine multiwave decline.

Summary: Are Mortality-Related Deficits Uniform Across Cognitive Domain?

The focal question considered in this second section was whether mortality-related cognitive deficits were similar across various cognitive domains. The analyses showed that terminal decline exerted more influence on some specific cognitive measures than others. Evidence from two profile analyses found that terminal decline varied as a function of age, mortality status, cognitive domain, and measurement wave. Cross-sectional analyses revealed mean performance differences for Survivors and Decedents across the continuum of cognitive measures. Whereas profiles for Survivors were relatively flat, significant differences for Decedents were observed across the cognitive profile (e.g., Decedents' mean vocabulary performance was significantly better relative to their performance on all other cognitive indicators). Observed trends were further modified by age group with Old-Old Decedents showing the most marked variation between measures on the continuum (poorer performance for verbal speed and episodic memory but better performance for vocabulary). Profiles for the other three groups were relatively flat. Findings from the longitudinal profile analysis established that: (a) Old-Old Decedents declined the most over the 3-year retest interval; (b) Decedents declined at a faster rate for select cognitive measures including verbal speed and episodic memory;

and (c) Decedents performed best on the vocabulary test relative to all other measures.

Following these findings, a logical research progression was to examine mortality-related deficits in relation to possible underlying influences. The third research goal examined whether cause of death and cardiovascular disease influenced patterns and magnitude of terminal decline.

Cardiovascular vs Non-Cardiovascular Influences of Age Differences and Change in Cognition

For this section, analysis of covariance and Cox regression were used to examine whether cause of death or specific disease was differentially associated with cognitive functioning. In theory, cause of death may represent a more sensitive predictor of cognitive performance than mortality status. For example, previous analyses have demonstrated associations between cognitive performance and various chronic illnesses and disease (e.g., Hertzog et al., 1978; Korten et al., 1999; van Boxtel et al., 1998). However, based on current findings, it is unclear whether specific causes of death differentially influence cognitive performance or whether multiple causes exert similar influences. To examine this issue, cause of death was dichotomously coded to contrast cognitive performance for those who died from cardiovascular disease (e.g., heart attack, stroke) relative to other causes of death (e.g., cancer, infectious diseases). Finer-grained analyses of smaller cause of death subcategories were not possible due to restricted sample size. In addition, proximity to death was categorized to replicate an earlier study (Small et al., in press): (a) those who died less than 3 years after final wave of testing ($n = 102$); and (b) those who died 3 or more years after final wave of testing ($n = 163$). As for previous analyses, cognitive outcome measures were linearly transformed to T-scores ($M = 50$ and $SD = 10$).

Cause of Death as a Predictor of Cognitive Deficits

Both cross-sectional differences and longitudinal changes in cognition were examined as a function of age group, cause of death, and proximity to death. In addition to the cause of death main effect, several interactions were of interest. First, the age

group by cause of death interaction represents a test of Berg's (1996) hypothesis that death is more random for older adults. Support for this interpretation would reflect minimal differences in cognitive performance for Old-Old adults as a function of cause of death (with magnified differences for the Young-Old). The cause of death by proximity to death interaction assesses whether individuals closer to death exhibit significantly poorer cognitive performance as a function of differential causes. Evidence for this interaction would imply that magnitude of terminal decline is modified not only by proximity to death, but also by specific underlying cause.

Prior to computing the analyses, the cause of death and proximity to death variables were examined in relation to demographic variables. A chi-square test of the ratio of CVD to non-CVD causes of death (see Table 4) indicated the two categories were equivalent ($\chi^2(1) = 3.17, p = .08$); decedents were as equally likely to die of non-cardiovascular ($n = 147$) as cardiovascular ($n = 118$) diseases. However, findings from a MANOVA indicated that those who died of cardiovascular disease were significantly older at last wave of testing, $F(1, 263) = 22.40, p < .001, \eta^2 = .08$, compared with those who died of other causes ($M = 77.83$ $SD = 6.72$ vs. $M = 73.82$ $SD = 6.96$ respectively). No significant differences were observed for education, self-reported relative health, or self-reported absolute health. All subsequent analyses incorporate age as a categorical predictor (rather than a covariate) to examine whether observed cause of death effects varied as a function of age group. A separate MANOVA indicated that proximity to death was not systematically related to age or years of education for the last wave of testing. However, those who would die within 3 years disclosed significantly poorer self-reported absolute, $F(1, 263) = 7.81, p < .01, \eta^2 = .03$, and relative health, $F(1, 263) =$

7.38, $p < .01$, $\eta^2 = .03$. Bivariate partial correlations, controlling for age, indicated no significant associations between years to death from last wave of testing and last wave cognitive performance for each of the 9 measures. Any significant findings associated with proximity to death are not a function of age differences.

Cause of death and last wave of measurement. To replicate and extend previous analyses (Small et al., in press), a 2 (age group) x 2 (cause of death) x 2 (proximity to death) MANCOVA (listwise $n = 265$), covarying education, was computed to examine whether these factors influenced cognitive performance for decedents. Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .748$, $F(7, 250) = 12.05$, $p < .001$, $\eta^2 = .25$; Cause of Death, Wilks' $\lambda = .935$, $F(7, 250) = 2.50$, $p < .05$, $\eta^2 = .07$; Proximity to Death, Wilks' $\lambda = .935$, $F(7, 250) = 2.47$, $p < .05$, $\eta^2 = .07$; the Cause of Death x Proximity to Death interaction, Wilks' $\lambda = .904$, $F(7, 250) = 3.80$, $p < .01$, $\eta^2 = .10$, and the Education covariate, Wilks' $\lambda = .880$, $F(7, 250) = 4.88$, $p < .001$, $\eta^2 = .12$. No interactions involving age were significant. A separate MANCOVA (listwise $n = 193$) for the two working memory measures yielded a significant omnibus effect for Age Group alone, Wilks' $\lambda = .913$, $F(2, 183) = 8.73$, $p < .001$, $\eta^2 = .09$.

Univariate effects for age were in the expected direction with Young-Old adults performing significantly better than the Old-Old for all cognitive measures except vocabulary ($p < .01$). Table 10 displays cognitive performance means and standard deviations for decedents' last wave of testing by cause of death and proximity to death. Cause of death differentially influenced performance for multiple cognitive measures including: semantic verification, $F(1, 256) = 4.65$, $p < .05$, $\eta^2 = .02$; word recall, $F(1, 256) = 11.85$, $p < .01$, $\eta^2 = .04$; story recall, $F(1, 256) = 7.40$, $p < .01$, $\eta^2 = .03$; sentence

Table 10
Decedents' Mean Cognitive Performance (T-scores) at Last Testing Wave
by Cause and Proximity to Death

Variables	Cardiovascular			Non-Cardiovascular		
	Within 3 Years (n = 62)	3 Years or More (n = 85)	Total (n = 147)	Within 3 Years (n = 40)	3-Years or More (n = 78)	Total (n = 118)
Verbal Speed						
Semantic verif.	42.45 (16.55)	44.77 (11.63)	43.99 (13.48)	49.68 (11.37)	47.98 (9.52)	48.70 (10.34)
Lexical decision	46.17 (11.91)	44.76 (12.74)	45.23 (12.43)	47.41 (17.12)	49.41 (7.42)	48.57 (12.45)
Working Memory						
Computation span ^a	46.84 (11.69)	45.62 (11.01)	46.13 (11.25)	49.34 (10.33)	47.91 (10.15)	48.64 (10.22)
Listening span ^a	45.15 (9.35)	44.36 (11.10)	44.69 (10.35)	50.29 (10.29)	48.35 (9.92)	49.35 (10.11)
Sentence const.	45.99 (11.30)	46.04 (11.51)	46.02 (11.39)	51.36 (9.57)	47.30 (11.79)	49.01 (11.06)
Episodic Memory						
Story recall	43.92 (9.49)	42.70 (8.42)	43.12 (8.77)	50.19 (10.72)	45.78 (9.36)	47.64 (10.16)
Word recall	42.60 (10.23)	44.38 (11.76)	43.78 (11.25)	51.78 (7.19)	46.92 (9.71)	48.97 (9.03)
Semantic Memory						
Fact recall	46.60 (11.47)	44.61 (9.83)	45.28 (10.41)	51.08 (9.50)	46.82 (10.03)	48.61 (10.00)
Verbal Ability						
Vocabulary	49.97 (13.00)	46.75 (12.50)	47.84 (12.71)	52.85 (8.04)	46.51 (12.84)	49.19 (11.48)

Standard deviations appear in parentheses. Within 3 years = died within 3 years of last testing wave; 3 years or more = died 3 or more years following last testing wave; Semantic verif = semantic verification; Sentence const. = sentence construction. ^a As these measures were not administered for S1W1, only 193 Decedents had available data (n = 86 who died of Cardiovascular disease and n = 107 who died of other causes).

construction, $F(1, 256) = 3.91, p < .05, \eta^2 = .02$; and listening span, $F(1, 184) = 3.96, p < .05, \eta^2 = .02$. Those who died of cardiovascular disease performed significantly worse than those who died of other non-cardiovascular causes (see total columns in Table 10). Cause of death effects were particularly pronounced for verbal speed and working memory as well as episodic memory but not for semantic memory or verbal ability. Even for non-significant comparisons, marginal means for all cause of death effects were in the expected direction.

Significant proximity to death effects were associated with: story recall, $F(1, 256) = 6.31, p < .05, \eta^2 = .02$; fact recall, $F(1, 256) = 5.84, p < .05, \eta^2 = .02$; and vocabulary, $F(1, 256) = 8.23, p < .01, \eta^2 = .03$. In contrast to previous findings (Small et al., in press), these trends paradoxically indicated that individuals within 3 years of death performed better on average than those who would die 3 or more years following the last wave of testing. Finally, the Cause of Death x Proximity to Death interaction was significant for word recall, $F(1, 256) = 7.75, p < .01, \eta^2 = .03$. Those who died of cardiovascular disease on the last testing wave performed worse within 3 years of death, whereas those who died of other causes exhibited the opposite trend. Table 10 displays the Cause of Death main effect but does not clearly demonstrate a Proximity to Death effect nor a Cause of Death x Proximity to Death interaction.

Cause of death and 3-year cognitive change. A corresponding longitudinal analysis was computed to examine whether cause of death influenced magnitude of cognitive declines for decedents. A 2 (Age Group) x 2 (Cause of Death) x 2 (Proximity to Death) x 2 (Wave) repeated measures MANCOVA (controlling for education) examined mortality-related cognitive change. Significant omnibus effects were observed

for: Age Group, Wilks' $\lambda = .735$, $F(7, 129) = 6.63$, $p < .001$, $\eta^2 = .27$; the Cause of Death x Proximity to Death interaction, Wilks' $\lambda = .891$, $F(7, 129) = 2.26$, $p < .05$, $\eta^2 = .11$; the Age Group x Wave interaction, Wilks' $\lambda = .833$, $F(7, 129) = 3.70$, $p < .01$, $\eta^2 = .17$; and the Education covariate, Wilks' $\lambda = .787$, $F(7, 129) = 5.00$, $p < .001$, $\eta^2 = .21$. No other main effects or interactions were significant at the omnibus level. Although a corresponding repeated measures MANCOVA was computed for listening and computation span, the results are not reported or interpreted due to exceedingly small cell sizes.

The univariate effect for age group was significant for all cognitive measures save for vocabulary ($p < .01$) indicating superior performance for the Young-Old. Word recall was the only significant univariate test, $F(1, 135) = 4.84$, $p < .05$, $\eta^2 = .04$, associated with the omnibus cause of death by proximity to death interaction. Finally, significant univariate effects associated with the Age Group x Wave interaction were observed for: lexical decision, $F(1, 135) = 5.67$, $p < .05$, $\eta^2 = .04$; semantic verification, $F(1, 135) = 6.25$, $p < .05$, $\eta^2 = .04$; and vocabulary, $F(1, 135) = 15.04$, $p < .001$, $\eta^2 = .10$. For all three measures, the Old-Old demonstrated more accelerated decline between the second last and last waves of testing. Notably, the trends for the Cause of Death x Wave interaction and the Age Group x Cause of Death x Wave interaction were all in the expected direction, albeit not significant. Due to limited sample size, it was not possible to examine 6-year change in a repeated-measures framework.

Examining differences in survival. A Kaplan-Meier analysis was computed to compare the survival functions between those who died of CVD vs non-CVD conditions. The Kaplan-Meier procedure estimates the probability of survival (i.e., likelihood that

death has not occurred) over the interval between the last wave of testing and the death event. Survival functions reflect the proportion of those dying as a function of number of months since last wave of testing. Results indicated that survival functions for each cause of death did not significantly differ, $\text{Log Rank}(1) = 2.87, p = .09$. The number of years to death did not significantly differ between those who died of CVD ($M = 5.65, SD = 3.85$) vs. other non-CVD causes ($M = 4.81, SD = 3.62$). Corresponding Life Tables revealed that 50% of the non-CVD decedents had succumbed by 3.7 years following the final testing wave compared with 5.07 years for CVD-related causes. Trends indicated that CVD decedents survived slightly longer than those who died of other causes, with CVD decedents exhibiting poorer last wave cognitive performance as demonstrated by the MANCOVA findings. Thus, any observed cause of death effects are not confounded by time to death.

Cardiovascular Disease as a Predictor of Cognitive Deficits for Survivors and Decedents

In light of small cell sizes for the preceding decedent analyses, the lack of comparison group for decedents, and null findings associated with the proximity to death variable, subsequent analyses in this section focused on contrasting cardiovascular vs non-cardiovascular disease for both survivors and decedents. These analyses examine cognitive performance as a function of mortality status and disease. To supplement the cause of death categories (CVD vs non-CVD) already created for decedents using ICD-9 criteria, a summary index of cardiovascular health was created for survivors by summing across self-reported questions reflecting chronic cardiovascular conditions (e.g., heart disease, stroke, arteriosclerosis, and high blood pressure) and associated medications (e.g., heart, chest, high blood pressure, and anticoagulants). A composite variable was

created reflecting both presence/absence of cardiovascular disease and mortality status (survivors with CVD, survivors without CVD, decedents with CVD, decedents without CVD). For subsequent analyses, the healthy survivors without CVD represent a control group for comparison with the other three groups. These four groups permit assessment of the combined effects of both impending death and cardiovascular disease on cognitive functioning. Analyses of this type have not been reported previously. As survivors are included in these analyses, the categorical proximity to death variable is no longer relevant; the main focus concerns presence/absence of cardiovascular disease in relation to cognitive performance.

Mortality status, cardiovascular disease, and last wave of measurement. To examine differences at the last wave of measurement, a 2 (Age Group) x 4 (Cardiovascular Disease: survivor non-CVD, survivor CVD, decedent non-CVD, decedent CVD) MANCOVA (listwise $n = 707$), covarying education, was computed. Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .816$, $F(7, 692) = 22.33$, $p < .001$, $\eta^2 = .18$; Cardiovascular Disease, Wilks' $\lambda = .828$, $F(21, 1987.6) = 6.43$, $p < .001$, $\eta^2 = .06$; the Age Group x Cardiovascular Disease interaction, Wilks' $\lambda = .954$, $F(21, 1987.6) = 1.57$, $p < .05$, $\eta^2 = .02$, and the Education covariate, Wilks' $\lambda = .866$, $F(7, 692) = 15.25$, $p < .001$, $\eta^2 = .13$. A separate MANCOVA (listwise $n = 635$) for the two working memory measures yielded a significant omnibus effect for: Age Group, Wilks' $\lambda = .941$, $F(2, 625) = 19.74$, $p < .001$, $\eta^2 = .06$; Cardiovascular Disease, Wilks' $\lambda = .962$, $F(6, 1250) = 4.02$, $p < .01$, $\eta^2 = .02$; and the Education covariate, Wilks' $\lambda = .966$, $F(2, 625) = 11.12$, $p < .001$, $\eta^2 = .03$.

Significant univariate age effects indicated Young-Old adults performed

significantly better than the Old-Old for all cognitive measures except vocabulary ($p < .01$). For each cognitive measure, Table 11 displays means and standard deviations (T-scores) for last wave of testing by mortality status, presence/absence of cardiovascular disease, and age group. Significant group differences for the Cardiovascular disease variable were observed for: semantic verification, $F(3, 698) = 23.09$, $p < .001$, $\eta^2 = .09$; lexical decision, $F(3, 698) = 16.27$, $p < .001$, $\eta^2 = .07$; sentence construction, $F(3, 698) = 7.00$, $p < .001$, $\eta^2 = .03$; listening span, $F(3, 626) = 6.55$, $p < .001$, $\eta^2 = .03$; computation span, $F(3, 626) = 4.77$, $p < .01$, $\eta^2 = .02$; story recall, $F(3, 698) = 29.42$, $p < .001$, $\eta^2 = .11$; word recall, $F(3, 698) = 15.96$, $p < .001$, $\eta^2 = .06$; and fact recall, $F(3, 698) = 10.59$, $p < .001$, $\eta^2 = .04$. The univariate effect for vocabulary was not significant.

Post-hoc LSD contrasts indicated that decedents with CVD performed significantly below levels of the other three groups for semantic verification, sentence construction, listening span, story recall, and word recall ($p < .01$). Decedents with CVD performed similarly to decedents without CVD but significantly below performance of both survivor groups for lexical decision, computation span, and fact recall ($p < .01$). Non-CVD survivors performed significantly better than CVD survivors for story recall alone and better than non-CVD decedents for all measures except sentence construction and computation span ($p < .01$). These trends demonstrate not only the influence of impending death, but indicate that individuals with the added cardiovascular disease risk factor showed disproportionately poorer cognitive performance for measures of verbal speed, working memory, and episodic memory.

Qualifying this cardiovascular effect, significant Age Group x Cardiovascular Disease interactions were observed for semantic verification, $F(3, 698) = 6.72$, $p < .001$,

Table 11
Cognitive Performance at Last Testing Wave by Mortality Status,
Presence/Absence of Cardiovascular Disease, and Age Group (n = 707)

Variable	Survivors				Decedents			
	Non-CVD		CVD		Non-CVD		CVD	
	Young-Old (n = 151)	Old-Old (n = 29)	Young-Old (n = 186)	Old-Old (n = 76)	Young-Old (n = 112)	Old-Old (n = 35)	Young-Old (n = 70)	Old-Old (n = 48)
Verbal Speed								
Semantic	53.54 (8.34)	48.83 (8.98)	52.56 (7.11)	49.00 (7.26)	51.23 (7.20)	40.60 (14.19)	48.39 (7.60)	37.56 (17.21)
Lexical	52.80 (9.09)	48.55 (9.01)	52.59 (5.91)	48.82 (6.18)	51.05 (6.85)	40.63 (20.68)	49.64 (6.34)	38.81 (15.96)
Working Memory								
Computation ^a	52.49 (9.60)	49.00 (8.30)	52.32 (9.35)	46.05 (8.17)	51.00 (9.58)	43.12 (9.65)	48.01 (10.13)	43.97 (12.18)
Listening ^a	52.82 (8.54)	47.16 (9.41)	52.24 (9.37)	46.93 (10.48)	51.79 (9.33)	43.64 (9.69)	46.28 (9.66)	42.86 (10.93)
Sentence	52.32 (8.02)	46.70 (12.01)	52.12 (8.11)	49.53 (10.15)	49.60 (10.23)	47.12 (13.37)	49.09 (9.24)	41.54 (12.77)
Episodic Memory								
Story Recall	54.93 (7.97)	51.13 (8.39)	52.99 (9.35)	47.70 (9.45)	49.11 (9.91)	42.95 (9.64)	45.98 (7.95)	38.93 (8.29)
Word Recall	54.49 (7.94)	46.62 (9.38)	52.84 (8.95)	47.08 (9.59)	50.65 (8.41)	43.59 (8.96)	47.00 (9.63)	39.09 (11.88)
Semantic Memory								
Fact Recall	52.41 (8.89)	46.78 (8.43)	53.16 (9.38)	48.72 (9.74)	50.07 (9.81)	43.96 (9.28)	48.56 (9.07)	40.51 (10.47)
Verbal Ability								
Vocabulary	51.22 (7.24)	49.91 (9.00)	50.44 (8.98)	51.46 (9.20)	49.56 (11.36)	48.01 (11.94)	49.31 (11.14)	45.70 (14.57)

CVD = cardiovascular disease; sentence = sentence construction; listening = listening span; computation = computation span; lexical = lexical decision; semantic = semantic verification. Standard deviations are reported in parentheses. ^a As these measures were not administered for S1W1, only 635 individuals had available data (equivalent n for survivors with $\underline{n}$ = 75, 32, 46, and 40 respectively for the decedent groups).

$\eta^2 = .03$, and lexical decision, $F(3, 698) = 6.05$, $p < .001$, $\eta^2 = .03$. Post-hoc simple effects were examined within each level of age group. Young-Old Decedents with CVD performed similarly to non-CVD decedents but significantly below levels for both survivor groups ($p < .05$). Non-CVD decedents did not differ significantly from any of the other 3 groups. Survivors without CVD did not differ from CVD survivors. In contrast, comparisons for the Old-Old indicated that decedents with CVD performed similarly to non-CVD decedents with both groups performing significantly below levels for each survivor group ($p < .05$). Non-CVD survivors performed similarly to survivors with CVD. For both measures, the significant interaction reflects further deterioration of the non-CVD decedents relative to both survivor groups. Decedents with the added CVD risk factor were already performing significantly below both survivor groups for the Young-Old. Table 11 shows that marginal means for all other cognitive measures were in the expected direction but did not surpass conventional significance levels.

Mortality status, cardiovascular disease, and 3-year cognitive change. To extend the cross-sectional analyses, a 2 (Age Group) x 4 (Cardiovascular Disease) x 2 (Wave) repeated measures MANCOVA (listwise $n = 586$) was computed covarying for education. Significant omnibus effects for this mixed-model MANCOVA were observed for: Age Group, Wilks' $\lambda = .819$, $F(7, 571) = 18.03$, $p < .001$, $\eta^2 = .18$; Cardiovascular Disease, Wilks' $\lambda = .828$, $F(21, 1640.2) = 5.31$, $p < .001$, $\eta^2 = .06$; Wave, Wilks' $\lambda = .955$, $F(7, 571) = 3.80$, $p < .001$, $\eta^2 = .05$; the Age Group x Wave interaction, Wilks' $\lambda = .936$, $F(7, 571) = 5.62$, $p < .001$, $\eta^2 = .06$; the Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .903$, $F(21, 1640.2) = 2.82$, $p < .001$, $\eta^2 = .03$; the Age Group x Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .927$, $F(21, 1640.2) = 2.10$, $p < .01$, $\eta^2 = .03$; and

the Education covariate, Wilks' $\lambda = .827$, $F(7, 521) = 17.06$, $p < .001$, $\eta^2 = .17$. A separate MANCOVA (listwise $n = 548$) for the two working memory measures yielded a significant omnibus effect for: Age Group, Wilks' $\lambda = .970$, $F(2, 538) = 8.27$, $p < .001$, $\eta^2 = .03$; Cardiovascular Disease, Wilks' $\lambda = .955$, $F(6, 1076) = 4.14$, $p < .01$, $\eta^2 = .02$; Wave, Wilks' $\lambda = .982$, $F(2, 538) = 4.96$, $p < .01$, $\eta^2 = .02$; and the Education covariate, Wilks' $\lambda = .955$, $F(2, 538) = 12.55$, $p < .001$, $\eta^2 = .05$. The omnibus tests for the higher order interactions were not significant.

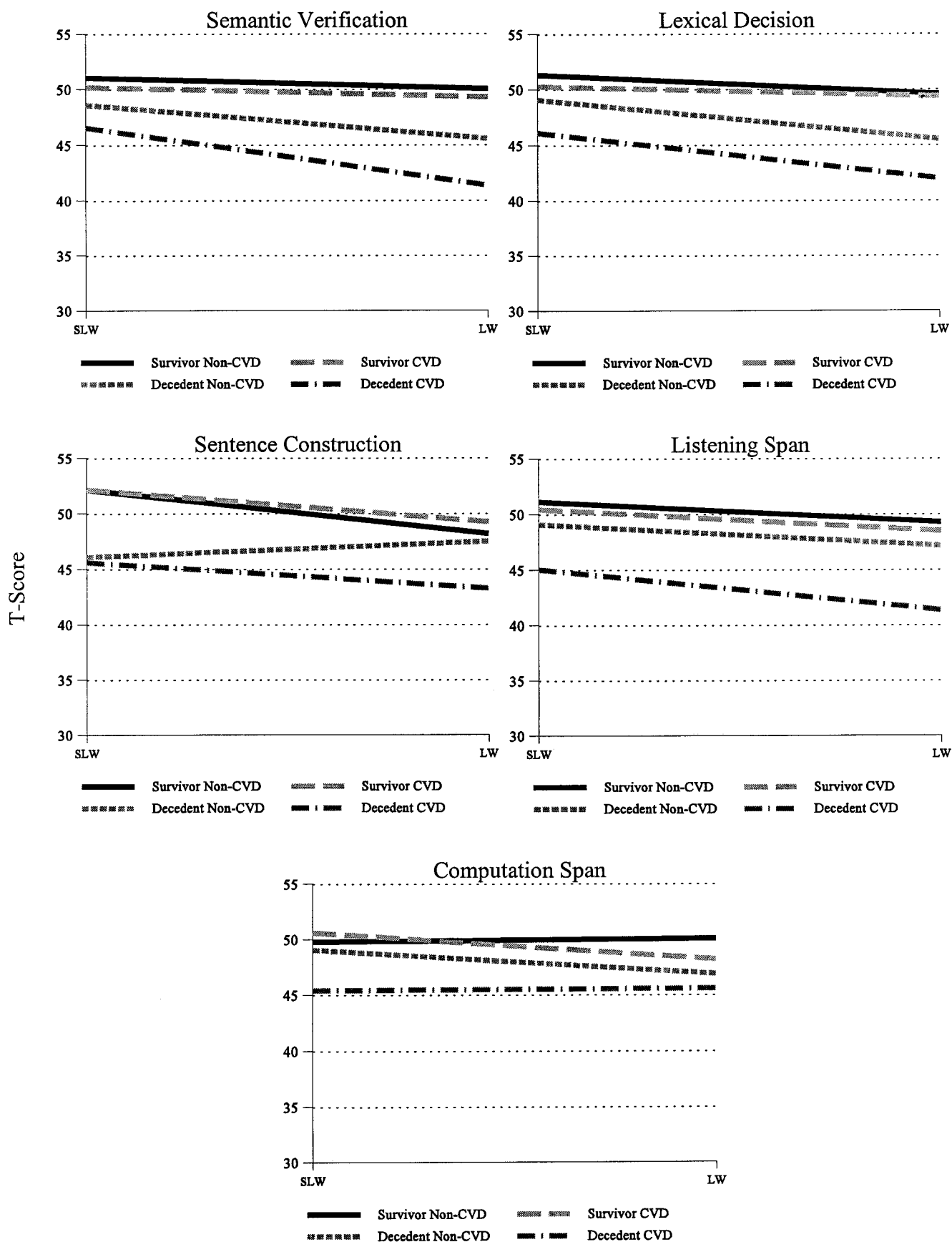
Because univariate effects for the between-subjects variables (Age Group and Cardiovascular Disease) were consistent with those reported for the previous section, only within-subject effects are reported. Univariate Wave effects indicated significant 3-year cognitive decline for the following measures: semantic verification, $F(1, 577) = 5.22$, $p < .05$, $\eta^2 = .01$; lexical decision, $F(1, 577) = 5.92$, $p < .05$, $\eta^2 = .01$; sentence construction, $F(1, 577) = 5.12$, $p < .05$, $\eta^2 = .01$; listening span, $F(1, 539) = 9.25$, $p < .01$, $\eta^2 = .02$; fact recall, $F(1, 577) = 9.52$, $p < .01$, $\eta^2 = .02$; and vocabulary, $F(1, 577) = 4.93$, $p < .05$, $\eta^2 = .01$. These main effects were further informed by Age Group x Wave interactions for: semantic verification, $F(1, 577) = 13.48$, $p < .001$, $\eta^2 = .02$; lexical decision, $F(1, 577) = 9.06$, $p < .01$, $\eta^2 = .02$; story recall, $F(1, 577) = 6.27$, $p < .05$, $\eta^2 = .01$; word recall, $F(1, 577) = 7.49$, $p < .01$, $\eta^2 = .01$; and vocabulary, $F(1, 577) = 19.15$, $p < .001$, $\eta^2 = .03$. Old-Old adults declined more precipitously between the first and second waves.

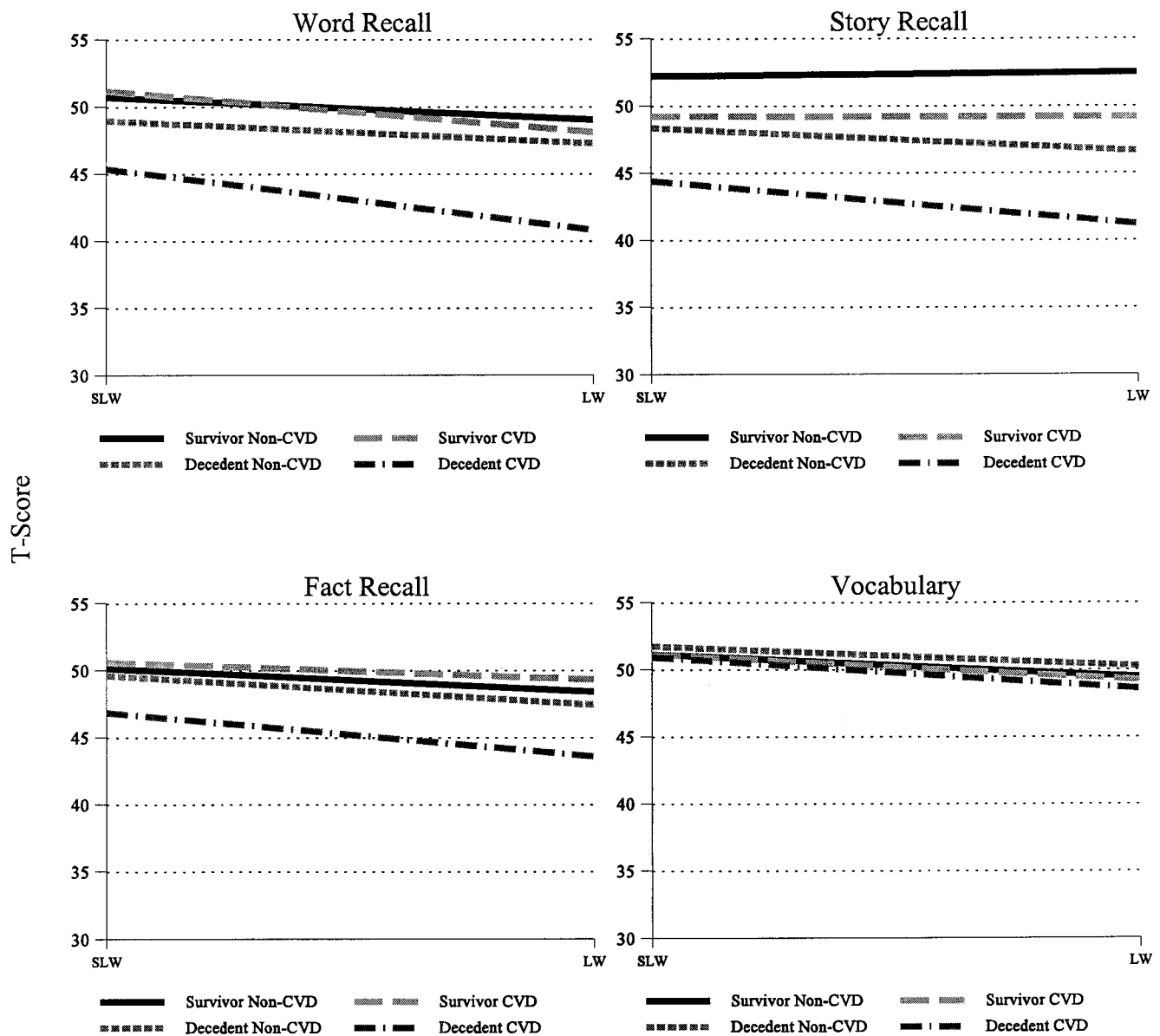
Univariate tests of the Cardiovascular Disease x Wave interaction indicated significant differences for: semantic verification, $F(3, 577) = 7.11$, $p < .001$, $\eta^2 = .04$; lexical decision, $F(3, 577) = 3.25$, $p < .05$, $\eta^2 = .02$; sentence construction, $F(3, 577) =$

5.22, $p < .01$, $\eta^2 = .03$; computation span, $F(3, 539) = 3.01$, $p < .05$, $\eta^2 = .02$; and story recall, $F(3, 577) = 4.98$, $p < .01$, $\eta^2 = .03$ (with word and fact recall demonstrating marginal trends, $p = .06$ and $.07$ respectively). Figure 13 shows cardiovascular group differences for 3-year change in cognitive performance. Significant interactions were decomposed using post-hoc simple effects analyses.

Both verbal speed measures yielded significant interactions. For semantic verification, the simple effects for cardiovascular group indicated significant 3-year declines for the two decedent groups alone; irrespective of disease, survivors did not exhibit significant declines. Simple effects were also computed separately for each wave. For the second last wave of semantic verification testing, decedents with CVD performed significantly below both survivor groups but similarly to non-CVD decedents. By the last wave of testing, CVD decedents performed significantly below all 3 groups. Non-CVD decedents did not differ significantly from CVD survivors for the second last wave but did for the last testing wave. Performance for survivors was equivalent for both testing waves. Significant 3-year declines for lexical decision were observed for all groups except survivors with CVD. Within both testing waves, decedents with CVD performed significantly below all other groups. Non-CVD decedents did not differ from CVD survivors for the second last wave but did for the last wave of testing. Irrespective of disease, survivor groups did not differ significantly for either wave of testing.

Significant interactions were also decomposed for two working memory measures (see Figure 13). Sentence construction performance exhibited significant 3-year declines for all groups except non-CVD decedents. For the second last testing wave, decedents with CVD performed similarly to non-CVD Decedents but significantly below both





CVD = cardiovascular disease; SLW = second last wave; LW = last wave.

Figure 13

Cardiovascular Group Differences for 3-Year Cognitive Change

survivor groups. By the last testing wave 3 years later, CVD decedents performed significantly below all 3 groups. Non-CVD decedents performed significantly below both survivor groups for the second last testing wave but did not differ from either for the last wave. Consistent with the verbal speed findings, survivor groups did not significantly differ for either testing wave. Significant 3-year declines for computation span were observed for survivors with CVD and decedents without CVD. CVD decedents performed similarly to non-CVD decedents but significantly below both survivor groups for the second last testing wave. Three years later, CVD decedents performed significantly below only the non-CVD survivor group. Non-CVD decedents did not significantly differ from any group for either wave of testing with both survivor groups performing equivalently across waves as well.

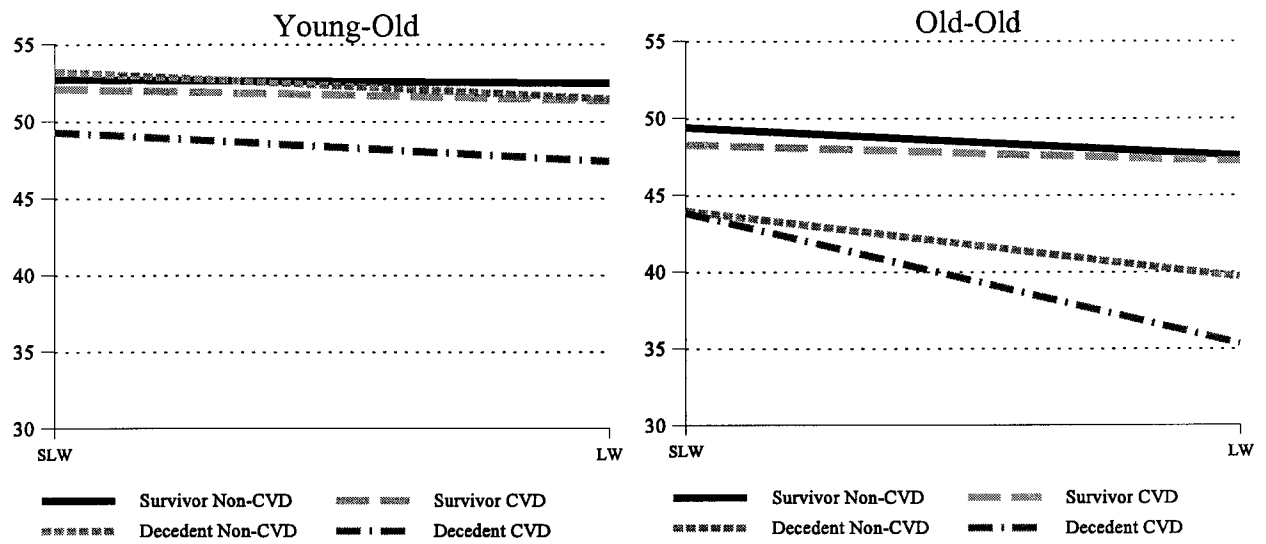
Finally, simple effects were calculated for the episodic memory interaction (see Figure 13). Both decedent groups significantly declined on story recall. For both testing waves: (a) decedents with CVD performed significantly below the other three groups; (b) non-CVD decedents scored below non-CVD but not CVD survivors; and (c) non-CVD survivors performed significantly better than CVD survivors. For 4 of the 5 cognitive measures (semantic verification, lexical decision, sentence construction, and story recall), simple effects indicated that both impending mortality and presence of cardiovascular disease interacted marking accelerated cognitive decline over a 3-year period. In particular, presence of cardiovascular disease differentially influenced cognitive decline for decedents with survivors performing similarly irrespective of disease. For all measures, trends revealed the lowest performance levels for decedents with CVD.

Qualifying these two-way interactions, significant univariate Age Group x

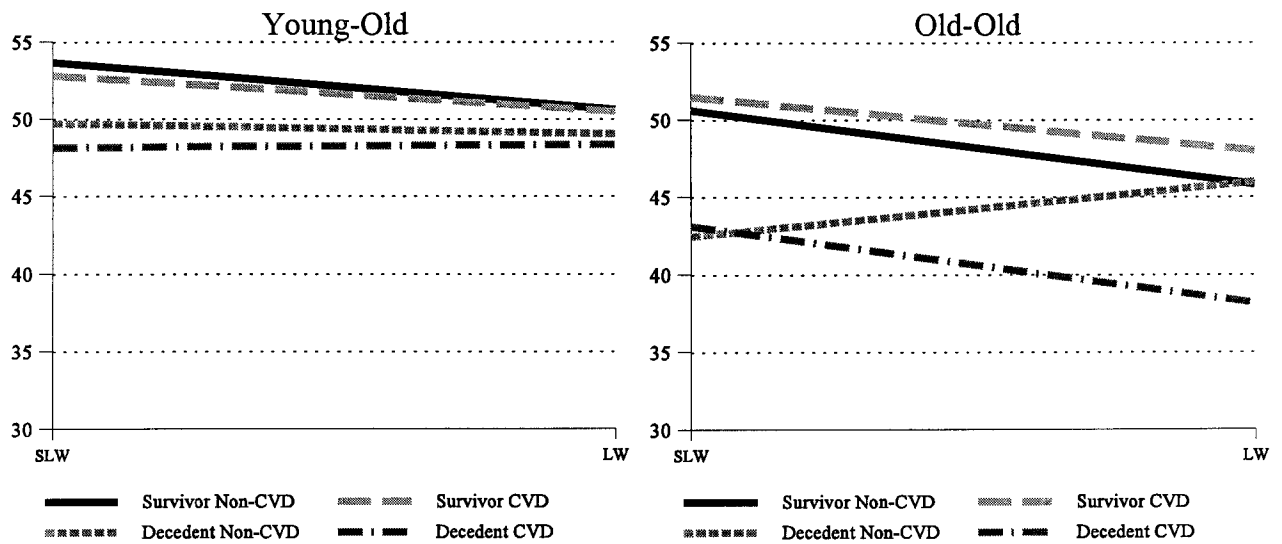
Cardiovascular Group x Wave interactions were observed for: semantic verification, $F(3, 577) = 3.31, p < .05, \eta^2 = .02$; sentence construction, $F(3, 577) = 3.04, p < .05, \eta^2 = .02$; and vocabulary, $F(3, 577) = 5.85, p < .01, \eta^2 = .03$ (with lexical decision demonstrating marginal trends, $p = .06$). For these three measures, Figure 14 shows age, mortality, and cardiovascular group differences for 3-year cognitive change. Post-hoc simple effect analyses were used to decompose these significant interactions. Of particular interest was whether patterns of change varied as a function of age, mortality, and cardiovascular disease. Pairwise comparisons for semantic verification indicated significant 3-year declines for both Old-Old decedent groups. For sentence construction, significant 3-year decline was observed for: (a) both Young-Old survivor groups with and without CVD; and (b) both Old-Old survivor groups as well as decedents with CVD. Mean differences for vocabulary revealed significant cognitive decline for: (a) Young-Old survivors with and without CVD; and (b) all 4 Old-Old survivor and decedent groups with and without CVD. Notably, these simple effects indicate that cognitive decline was most pronounced for Old-Old decedents with cardiovascular disease. Moreover, albeit not significant, trends for all other measures were similar; Old-Old decedents with CVD showed the greatest amount of 3-year cognitive decline.

Mortality status, cardiovascular disease, and 6-year cognitive change. To examine the 3 wave data, a 2 (Age Group) x 4 (Cardiovascular Disease) x 2 (Wave) repeated measures MANCOVA (listwise $n = 518$) was computed covarying for education. Significant omnibus effects for this mixed-model MANCOVA were observed for: Age Group, Wilks' $\lambda = .875, F(7, 503) = 10.22, p < .001, \eta^2 = .13$; Cardiovascular Disease, Wilks' $\lambda = .847, F(21, 1444.9) = 4.11, p < .001, \eta^2 = .09$; Wave, Wilks' $\lambda = .956,$

Semantic Verification

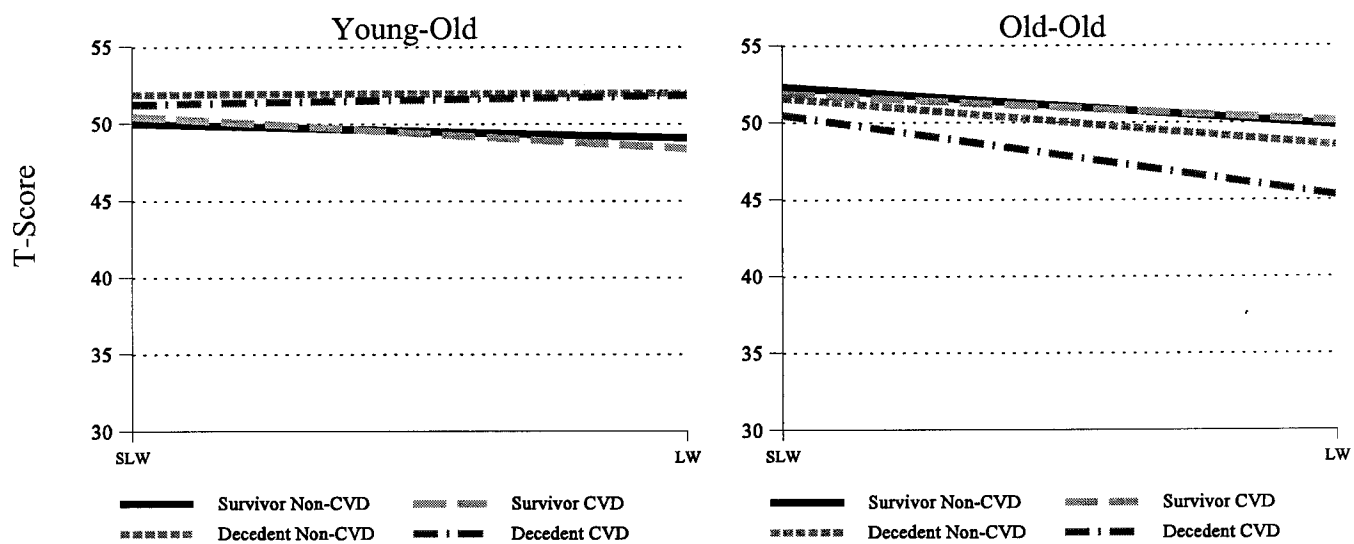


Sentence Construction



T-Score

Vocabulary



CVD = cardiovascular disease; SLW = second last wave; LW = last wave.

Figure 14

Cardiovascular and Age Group Differences for 3-Year Cognitive Change

$F(14, 2024) = 3.30, p < .001, \eta^2 = .02$; the Age Group x Wave interaction, Wilks' $\lambda = .942, F(14, 2024) = 4.37, p < .001, \eta^2 = .03$; the Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .883, F(42, 4750.2) = 3.03, p < .001, \eta^2 = .02$; the Age Group x Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .924, F(42, 4750.2) = 1.93, p < .01, \eta^2 = .01$; and the Education covariate, Wilks' $\lambda = .827, F(7, 503) = 15.01, p < .001, \eta^2 = .17$. Results from a separate MANCOVA (listwise $n = 475$) for the two working memory measures are not reported due to small cell sizes and unstable estimates.

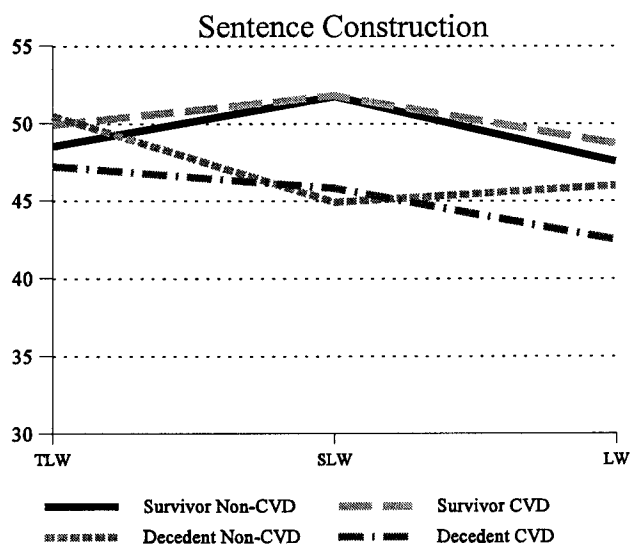
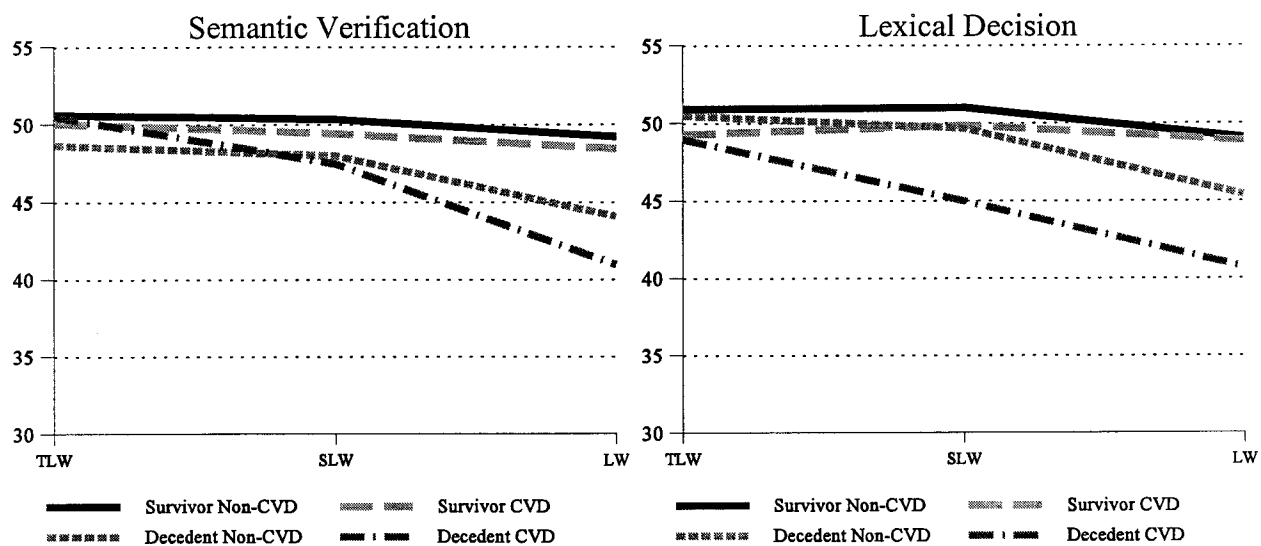
Univariate between-subjects effects for Age Group and Cardiovascular Disease were identical to those reported for the previous cross-sectional and 3-year longitudinal analyses. In addition to the significant within-subject Wave effects reported previously (semantic verification, lexical decision, sentence construction, fact recall, and vocabulary), significant 6-year decline was observed for word recall, $F(2, 1018) = 4.09, p < .05, \eta^2 = .01$. Except for story recall which was no longer significant, the within-subjects Age-Group x Wave interactions also replicated previous findings. Greater linear decline was exhibited by Old-Old adults across the 3 waves for semantic verification, lexical decision, word recall, and vocabulary ($p < .01$) with accelerated quadratic decline on vocabulary ($p < .01$) for the Old-Old.

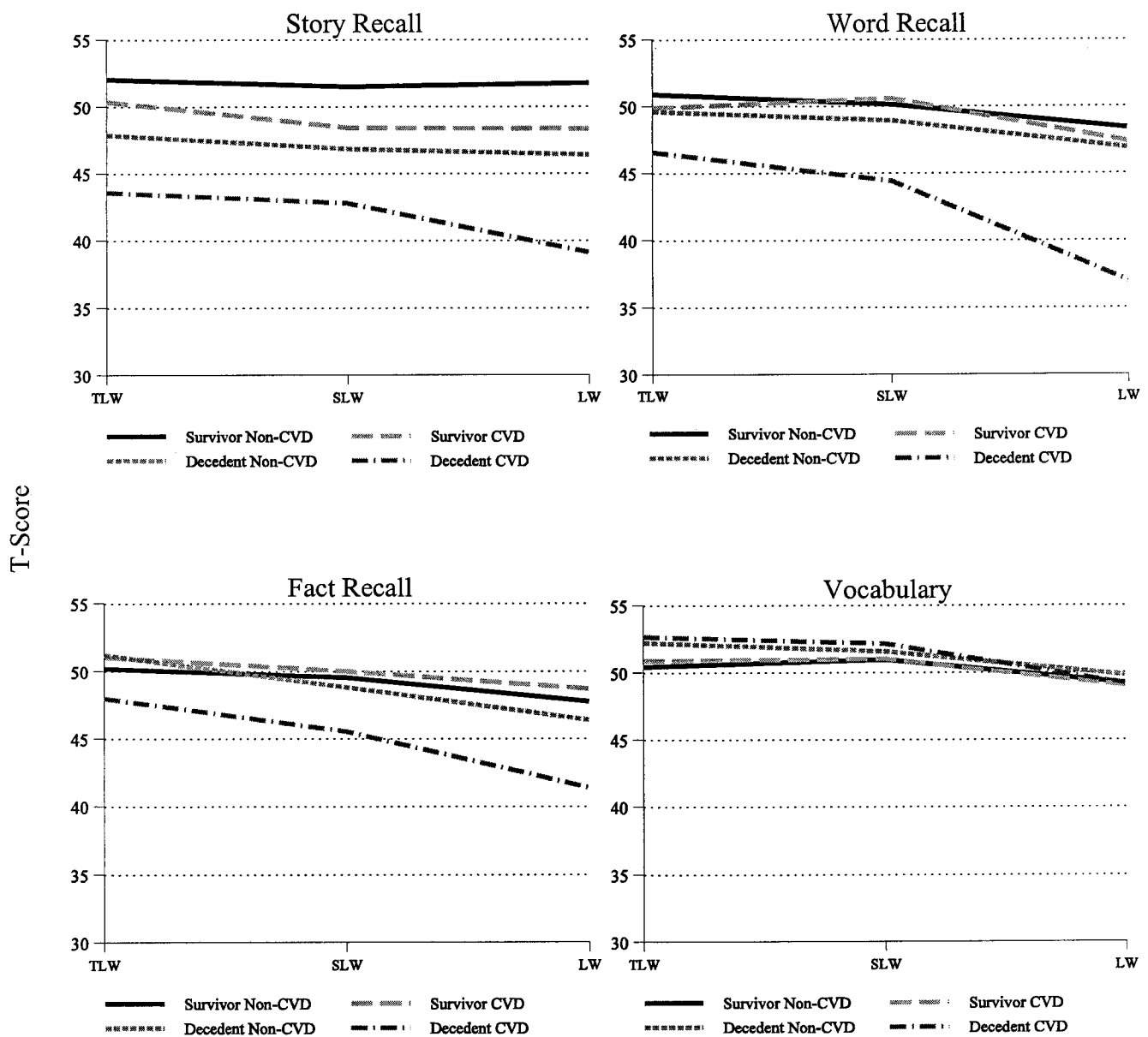
The Cardiovascular Disease interactions are of particular importance for examining how cognitive performance is influenced by mortality status and disease. Univariate Cardiovascular Disease x Wave interactions were significant for the following cognitive measures: semantic verification, $F(6, 1018) = 6.94, p < .001, \eta^2 = .04$; lexical decision, $F(6, 1018) = 4.57, p < .001, \eta^2 = .03$; sentence construction, $F(6, 1018) = 4.67, p < .001, \eta^2 = .03$; story recall, $F(6, 1018) = 2.31, p < .05, \eta^2 = .01$; word recall, $F(6, 1018)$

$= 5.19, p < .001, \eta^2 = .03$; and fact recall, $F(6, 1018) = 3.83, p < .01, \eta^2 = .02$. Patterns of cognitive decline varied as a function of mortality status and presence/absence of cardiovascular disease for all cognitive domains except verbal ability. Figure 15 demonstrates these cardiovascular group differences for 6-year change in cognitive performance.

Post-hoc simple effects were employed to decompose the significant interactions. For each cognitive measure, significant differences between waves of testing were examined within each of the four cardiovascular groups. Patterns of 6-year change for both semantic verification and lexical decision indicated that decedents with CVD demonstrated accelerated decline after both the third and second last testing waves. In comparison, decline for non-CVD decedents emerged after the second last wave with each survivor group demonstrating relatively stable 6-year performance. More complex patterns emerged for sentence construction where decedents with CVD showed decline between the second last and last waves, non-CVD decedents declined between the third and second last waves, with both survivor groups exhibiting significant improvement between the third and second last waves followed by significant decline between the second last and last waves. Simple effects for the episodic memory measures revealed that CVD decedents significantly declined after the second last wave of testing for both story and word recall. In comparison, declines for adjacent testing waves were not significant for non-CVD decedents. Both survivor groups exhibited significant decline CVD survivors significantly declined between the third and second last waves of testing. Significant fact recall performance declines were observed between all adjacent waves for both decedent groups and for survivors with CVD. In contrast, non-CVD survivors

T-Score





CVD = cardiovascular disease; TLW = third last wave; SLW = second last wave; LW = last wave.

Figure 15

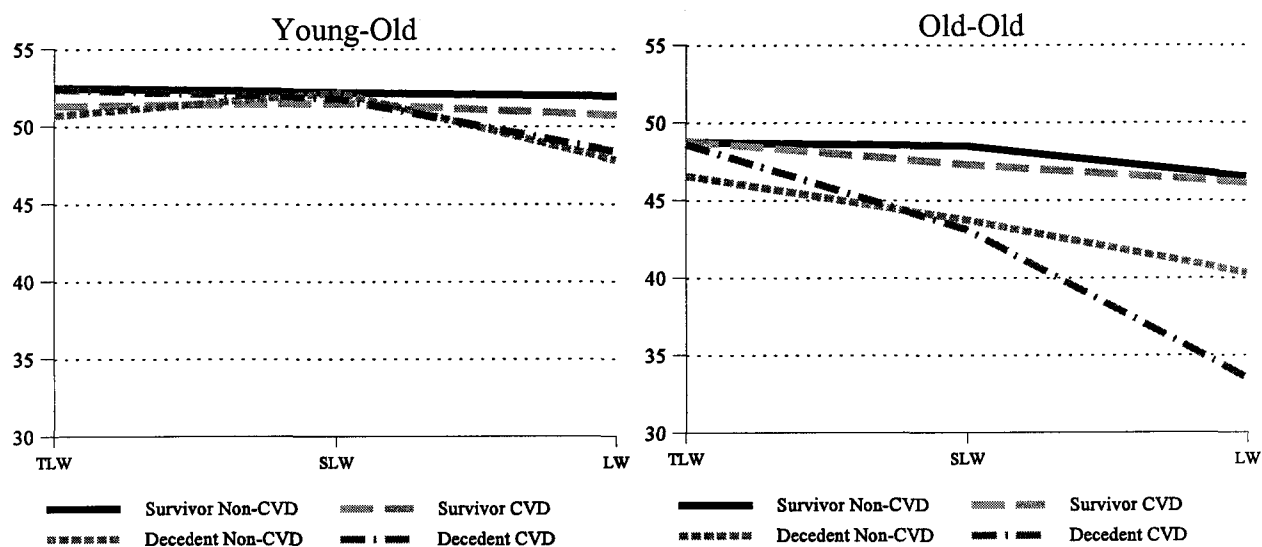
Cardiovascular Group Differences for 6-Year Cognitive Change

declined after the second last testing wave for word recall but not story recall. For story recall, demonstrated decline after the second last wave of testing. Finally, significant vocabulary declines were observed between the second last and last waves of testing for all four groups. Consistent with the 3-year decline analyses, decedents who died of CVD related causes declined more consistently across waves, exhibited larger magnitude declines, and exhibited lower mean performance relative to the other groups (see Figure 15).

Several significant Cardiovascular Group x Wave interactions were further modified by Age Group. Significant three-way interactions were observed for: semantic verification, $F(6, 1018) = 2.34$, $p < .05$, $\eta^2 = .01$; sentence construction, $F(6, 1018) = 2.32$, $p < .05$, $\eta^2 = .01$; and vocabulary, $F(6, 1018) = 3.87$, $p < .01$, $\eta^2 = .02$. Figure 16 outlines 6-year cognitive change, separately for each age group, as a function of cardiovascular group differences.

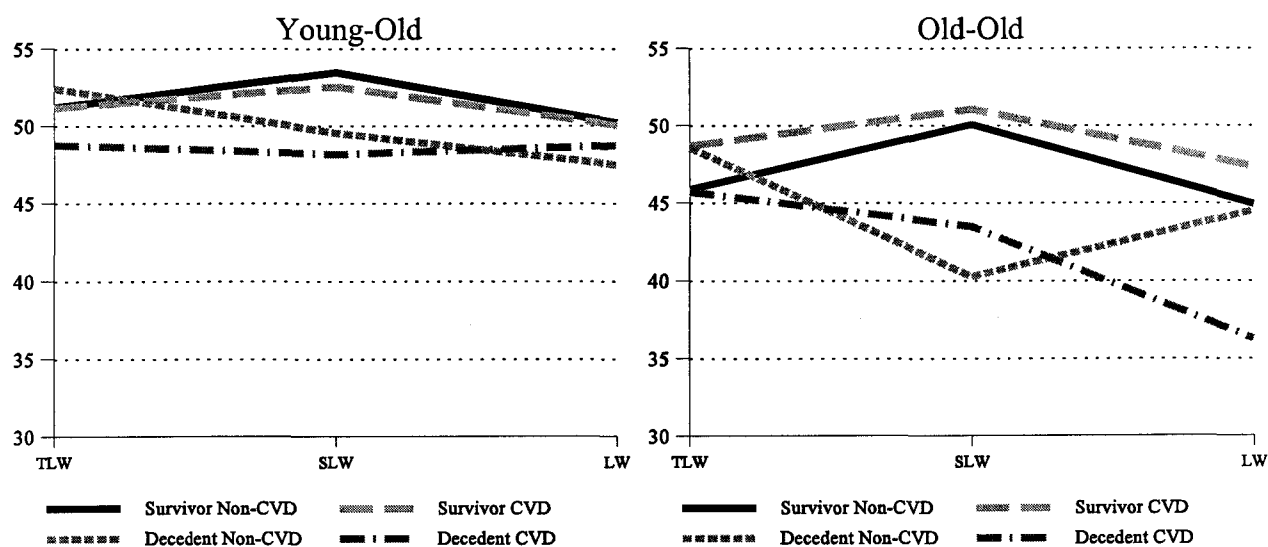
For semantic verification, pairwise comparisons for the Young-Old revealed significant declines for non-CVD decedents between the second last and last waves of testing; no significant change was observed for any other groups. In contrast, significant 6-year declines were magnified for the Old-Old with both CVD survivors and non-CVD decedents significantly declining between the third and second last waves of testing. Notably, CVD decedents declined significantly between all adjacent waves, with the rate of decline accelerating between the second last and last testing waves. Significant cardiovascular group differences were also observed within level of age group and testing wave. For the Old-Old age group on the semantic verification task, decedents with CVD performed significantly worse than both survivor groups at the second last testing wave.

Semantic Verification

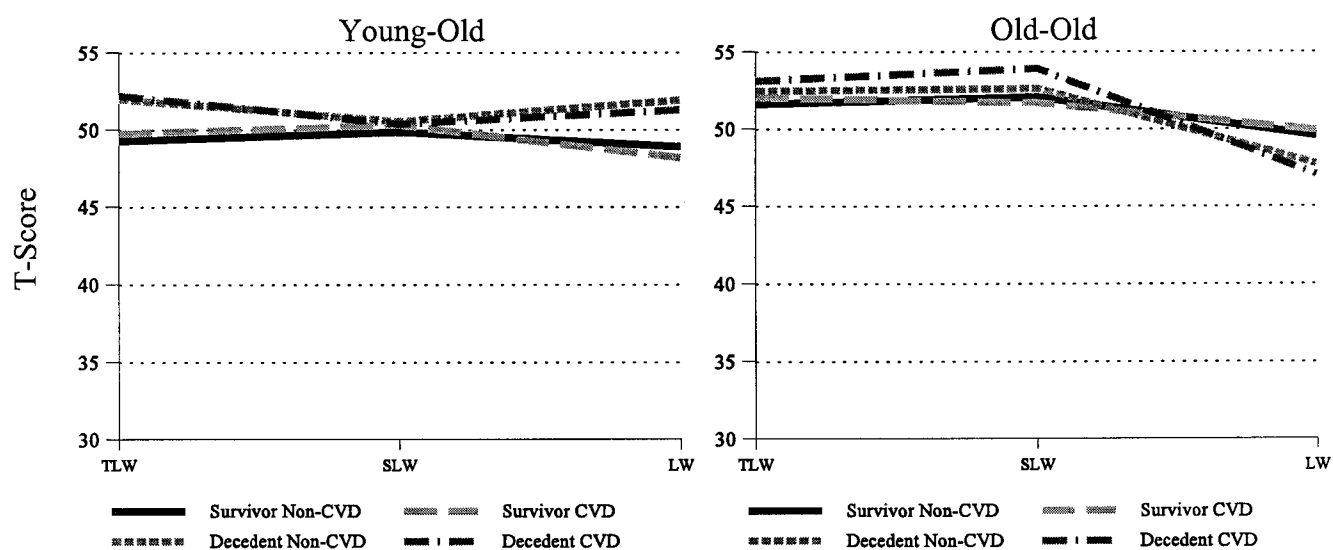


T-Score

Sentence Construction



Vocabulary



CVD = cardiovascular disease; TLW = third last wave; SLW = second last wave; LW = last wave.

Figure 16

6-Year Cognitive Change as a Function of Cardiovascular and Age Group Differences

By the last wave, decedents with CVD performed significantly worse than all groups.

Pairwise comparisons for sentence construction revealed significant declines after the second last testing wave for both the Young-Old survivor groups; no significant declines occurred for decedents' adjacent waves. For the Old-Old, both survivor groups declined after the second last wave of testing. Non-CVD decedents declined between the third and second last waves of testing prior to leveling off. In contrast, Old-Old decedents with CVD declined precipitously after the second last wave of testing. Examining cardiovascular group differences within level of age and wave, both Young-Old decedent groups performed significantly worse than non-CVD survivors for the second last testing wave. In comparison, both Old-Old decedent groups scored below the two survivor groups for the second last testing wave. For the last wave, Old-Old decedents with CVD performed significantly worse than all other groups.

For the third and final interaction, significant vocabulary declines were observed between the second last and last testing waves for both the Young-Old and Old-Old survivor groups (with and without CVD). Similarly, both Old-Old decedent groups declined after the second last wave. Cardiovascular groups were not significantly different when compared within level of age and wave. Of mention, although the Cardiovascular Group x Age Group x Wave interactions were not significant for other cognitive measures, observed marginal means were consistent with patterns observed in Figure 16. Mortality-related cognitive declines were further amplified by older age and presence of cardiovascular disease.

Predicting mortality status. Cox regression (Cox, 1972) represents an alternative approach for examining associations between mortality status, cardiovascular disease,

and cognitive performance for the last wave of testing. Using a Cox proportional hazards analysis, survival status (decedents vs. survivors) across a follow-up period of measurement was predicted using a variety of covariates (e.g., demographic variables, cognitive measures). For present purposes, there are several advantages of the Cox regression approach. First, given that decedents were in the study for varying periods of time, Cox analyses are apropos in that they model the hazard rate (i.e., the probability of dying for a given time interval) rather than mortality status as the dependent outcome. Second, actual years to death from the last respective wave of testing for decedents ($\underline{M} = 5.18$, $\underline{SD} = 3.74$) and years to last known date alive for survivors ($\underline{M} = 2.79$, $\underline{SD} = 0.44$) are incorporated into the prediction model. Mortality status assessment was discontinued as of May 31, 2002, so right censoring (i.e., cases for which the death event had not yet occurred) was applied for the remaining 442 survivor cases. Cox models assume that the population mortality rate depends upon this continuous time variable. Finally, Cox analyses control for confounds present in the preceding cause of death MANCOVA findings (e.g., those who died of CVD were older than those who died of non-CVD conditions). A chi-square analysis indicated significant differences between the four cardiovascular groups. Relative to expected frequencies, there were a greater number of survivors with CVD ($\underline{n} = 262$) and fewer decedents with or without CVD ($\underline{n} = 118$ and 147 respectively); these differences reflect the smaller number of overall decedents relative to survivors.

Cox analyses yielded individual regression coefficients representing the association between each covariate (age and other demographic variables, cardiovascular disease, and cognitive performance for the last wave of testing) and the likelihood of

dying. Associated odds ratios were computed to provide the percentage difference in mortality risk per unit difference in covariate score. For example, an observed Odds Ratio of 1.05 reflects a 5% increased mortality risk per unit increase in the covariate independent of all other variables in the equation. As for previous analyses, cognitive predictors were entered as standardized T-scores to facilitate comparison of coefficients and odds ratios across cognitive measures. Although MANCOVA findings for the last wave of testing analyses yielded a significant cause of death effect (i.e., decedents with cardiovascular disease exhibited poorer cognitive performance), it is important to examine these influences controlling for other confounding factors.

Table 12 presents the Cox regression results predicting mortality status as a function of demographic variables, cardiovascular disease, and cognitive performance. Covariates were entered in 3 hierarchical blocks. Age, education, and self-reported health measures were entered on Block 1. Several cardiovascular disease measures were entered on Block 2 including CVD conditions (self-reported presence of heart disease, stroke, arteriosclerosis, and high blood pressure for any wave of testing) and CVD medications (self-reported use of heart, chest, high blood pressure, and anticoagulant medications). For each CVD predictor, the total score ranged from 0 (no CVD conditions or medications for any wave) to 4 (all four of the CVD conditions or medications for any wave). Cognitive performance measures were entered individually for the last block (Block 3). Separate models were computed for each individual indicator of verbal speed, working memory, episodic memory, semantic memory, and verbal ability.

The findings summarized in Table 12 demonstrate that age, absolute self-reported health, and CVD medications all shared significant associations with mortality status.

Table 12

Cox Regression Analyses Predicting Mortality Status

as a Function of Age, Cardiovascular Disease, and Cognitive Performance (n = 707)

Covariates	β	Odds Ratio	95% CI	<i>p</i>
Block 1				
Age	0.029	1.030	1.011 - 1.049	0.002
Education	0.026	1.026	.991 - 1.062	0.146
Relative health	0.196	1.216	.958 - 1.543	0.108
Absolute health	-0.230	0.795	.636 - .992	0.042
Block 2				
CVD conditions	-0.003	0.997	.834 - 1.193	0.976
CVD medications	0.265	1.304	1.105 - 1.539	0.002
Block 3				
Semantic verification	-0.001	0.999	.979 - 1.018	0.881
Lexical decision	-0.009	0.991	.974 - 1.009	0.341
Computation span	0.004	1.004	.986 - 1.023	0.631
Listening span	-0.005	0.995	.976 - 1.014	0.589
Sentence construction	-0.003	0.997	.986 - 1.009	0.615
Story recall	-0.002	0.998	.981 - 1.015	0.832
Word recall	0.002	1.002	.987 - 1.017	0.815
Fact recall	-0.001	0.999	.986 - 1.013	0.935
Vocabulary	0.009	1.009	.997 - 1.021	0.134

Mortality status was coded as either 0 (survivor) or 1 (decedent). CI = confidence interval; CVD = cardiovascular disease. On a 5-point scale (0 = very good to 4 = very poor), absolute health reflects health relative to perfect; relative health reflects health relative to same-aged peers. 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 reflects self-reported use of medications prescribed to treat cardiovascular symptoms and disease (heart, chest, high blood pressure, and anticoagulants).

Older participants were at an increased risk of mortality over the follow-up interval; the mortality risk increased by 3% per year or 30% per decade of increased age. In contrast, each additional unit increase in self-reported absolute health (reflecting poorer health) was associated with a 20.5% decreased mortality risk. This finding likely reflects inaccuracy of self-reports. Finally, controlling for all other covariates, each additional medication (from 0 to 4) used to treat cardiovascular disease for any wave of testing was associated with a 30.4% increased mortality risk. Those individuals taking all four medications to treat heart disease, high blood pressure, and arteriosclerosis were 121.6% more likely to die during the follow-up interval. Notably, when entered separately after the demographic variables, both CVD conditions and medications were associated with increased mortality risk. Each additional self-report chronic condition for any given wave was associated with an 18.7% increased mortality risk (74.8% for those reporting all four conditions at some point during their participation). That the partial regression coefficient for CVD conditions was no longer significant when entered with CVD medications indicated that the latter self-report was a more accurate predictor of mortality. Finally, in contrast to the MANCOVA findings indicating decedents exhibited poorer performance for their last wave of testing, no cognitive measures significantly predicted mortality across the follow-up interval after controlling for demographic measures and cardiovascular conditions. The odds ratios for all cognitive measures were approximately 1 indicating that mortality risk was unrelated to last wave performance. In light of these null findings, interaction terms (e.g., age or CVD x cognitive performance) were not entered for subsequent blocks to test whether cognitive influences varied as a function of cardiovascular disease or age.

Summary: Does Cause of Death Influence Mortality-Related Cognitive Deficits?

In summary, evidence was found suggesting that specific cause of death differentially influenced cognitive performance. Those who died of cardiovascular disease exhibited poorer cognitive performance relative to those who died of non-cardiovascular causes. Moreover, patterns indicated that cause of death effects were more pronounced for cognitive process and episodic memory measures with lesser differences for semantic memory and verbal ability. Corresponding 3-year longitudinal analyses for the last two measurement waves did not reveal significant cause of death by wave or age group by cause of death by wave interactions. Although these interactions were not significant at conventional levels, all patterns of marginal means were in the expected direction (i.e., greater cognitive decline associated with those who died of cardiovascular disease). Limited cell size precluded examination of 6-year change for these decedents.

Additional analyses were computed to examine cardiovascular vs non-cardiovascular conditions for both survivors and decedents. At cross-section, decedents with CVD performed significantly worse than all other groups for verbal speed, working memory, and episodic memory measures. Trends indicated that terminal decline effects (i.e., impending death) were further amplified for those with cardiovascular disease and for Old-Old adults. Similarly, longitudinal analyses across 3- and 6-year intervals revealed significant cardiovascular disease by wave interactions for verbal speed, working memory, and episodic memory measures. For decedents but not survivors, cardiovascular disease was associated with accelerated cognitive declines and poorer mean performance relative to the other three groups. Age group by cardiovascular disease by wave interactions provided evidence that Old-Old decedents with CVD started

to decline prior to the other three groups.

Finally, Cox regression was used to examine survival status between last wave of testing and death as a function of demographic characteristics, cardiovascular conditions, and cognitive performance. Older individuals and those taking medications to treat cardiovascular symptoms and disease were at an increased risk for mortality. However, in contrast to the MANCOVA findings indicating decedents exhibited poorer performance for their last wave of testing, mortality risk was unrelated to cognitive predictors.

Multilevel Analyses: Intraindividual Cognitive Decline as a Function of Time to Death

For the preceding section, repeated measures MANCOVA was used to examine the influence of cause of death and cardiovascular disease on patterns of cognitive decline. These aggregate change repeated measures analyses represent precursors to the multilevel analyses examined in this section. Although previous research has examined the impact of impending mortality on cognitive performance, few investigations have examined longitudinal cognitive declines as a function of precise time to death estimates (e.g., number of years). The present analyses employed HLM to examine intraindividual cognitive change in relation to time to death. These multilevel analyses are unique and represent a significant improvement in precision as compared with the less reliable proximity to death categorical variable (died 3-years or less after the last testing wave vs. died greater than 3 years later). The Level 1 time to death predictor is actually a hybrid variable reflecting the number of years between date of testing for each completed wave and: (a) date of death for each decedent (i.e., years to death); or (b) a right censoring date of May 31, 2002 for all survivors (i.e., years remaining in the study prior to May 31, 2002). May 31 represents the last date surviving participants were known to be alive based on VLS project records or BC Vital Statistics information.

Multilevel Analysis of Intraindividual Cognitive Decline Profiles

Using HLM, within-subject trajectories of cognitive decline were examined as a function of years to death/years remaining in the study. There are several primary foci of these analyses. The first concerns examining longitudinal associations between intraindividual cognitive performance slopes, mortality status, age, and time to death/time

remaining in the study for both survivors and decedents. For decedents alone, a second analysis examined whether cognitive decline varied as a function of cause of death, age, and time to death. Questions of interest included: (a) whether those facing impending death declined more quickly than survivors; (b) whether terminal decline was limited to a narrow interval preceding death or was it observed across longer intervals (e.g., Bosworth & Schaie, 1999); and (c) whether proximity to death was better characterized by steady terminal decline or more precipitous terminal drop. Multilevel modeling represents a powerful statistical technique for differentiating terminal decline vs. terminal drop as a function of time to death.

Intraindividual cognitive decline as a function of mortality status. For the first multilevel analysis, intraindividual cognitive change was examined as a function of: (a) number of years between each testing wave and death/time remaining in the study (Level 1 variable); (b) age group at last wave of testing (Level 2 variable); (c) mortality status (Level 2 variable); and (d) the age group x mortality status interaction (Level 2 variable). For all analyses in this section, age was entered as a Level 2 predictor because of model saturation issues. Specifically, most participants completed three or fewer measurement waves and only $k - 1$ Level 1 predictors can be entered into a model. In addition to linear rate of cognitive change as a function of time to death, Level 1 quadratic trends were examined for subsequent analyses absorbing the remaining Level 1 degree of freedom. Relevant Level 1 and 2 equations were constructed to specifically test whether cognitive performance differentially declined as a function of these predictors.

For each cognitive measure, the Level 1 model (see equation 4) examined individual rates of change with increasing proximity to death across measurement

occasions. Separate regression slopes and intercepts were calculated for each individual in the study.

$$\text{Level 1: Cognitive Performance}_{it} = B_{0i} + B_{1i} * (\text{Time to Death}_{it}) + R_{it} \quad (4)$$

In interpreting Equation 4, cognitive performance for a given individual (i) at a given time (t) was modeled as a function of that individual's performance at the centered midpoint on time to death (B_{0i} : the intercept), plus her/his average individual rate of change across waves with increasing proximity to death (B_{1i} : the slope), plus a random within-subject error term (R) representing prediction error holding intercept and slope constant. For all models analyzed, the Level 1 time to death predictor was grand-mean centered (initial $M = 7.24$ years; range .12 to 15.88 years); findings reflect cognitive performance at the sample midpoint for time remaining in the study.

Level 2 HLM equations permit between-individual analyses of change treating the Level 1 within-individual intercepts and slopes as dependent variables (i.e., testing whether there are between-group differences in within-person change). Specified Level 2 equations considered whether mortality and age group differences (or their interaction) were associated with different trajectories of individual change with increasing proximity to death.

$$\begin{aligned} \text{Level 2: } B_{0i} = & \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Mortality Status}_i) \\ & + \gamma_{03} * (\text{Age Group} \times \text{Mortality Status}_i) + U_{0i} \end{aligned} \quad (5)$$

$$B_{1i} = \gamma_{10} + \gamma_{11}*(\text{Age Group}_i) + \gamma_{12}*(\text{Mortality Status}_i) + \gamma_{13}*(\text{Age Group} \times \text{Mortality Status}_i) + U_{1i} \quad (6)$$

Equation 5 models a given individual's cognitive performance (B_{0i}) as a function of performance for Young-Old Survivors of average time to death (γ_{00}), the average difference in performance between age (γ_{01}), mortality groups (γ_{02}), and the age by mortality status interaction (γ_{03}), plus a random effect (U_{0i}) estimating variability about the intercept holding all model predictors constant. When multiple Level 2 predictors are entered as in Equation 5, resulting coefficients are interpreted as partial regression coefficients (i.e., significant mortality group differences independent of age and the interaction). Equation 6 examines the predicted linear rate of change for each individual (B_{1i}) as a function of average change in cognitive performance for Young-Old Survivors per year difference in proximity to death (γ_{10}), the average difference in change per year closer to death 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 cognitive performance slopes holding these predictors constant (i.e., do between-group differences in within-person cognitive change remain to be explained).

As was the case for previous HLM analyses, these analyses were computed using raw scores. Although useful for comparing across domains, data were not standardized due to unequal listwise n and differing patterns of complete data across individuals, tasks, and measurement occasions. Semantic verification and lexical decision coefficients reflect milliseconds, sentence construction coefficients reflect values on a 0 to 6 scale, listening and computation span coefficients reflect values on a 0 to 7 scale, story recall

coefficients reflect percent correct, and word recall, fact recall, and vocabulary coefficients reflect raw scores recalled out of 30, 40, and 54 respectively.

Table 13 summarizes multilevel findings associated with equations 4 to 6. For each of the cognitive measures, coefficients in the intercept column (γ_{00}) reflect performance for Young-Old Survivors at the sample midpoint for time to death/time remaining in the study. Not surprisingly, all intercepts were significantly different from 0 ($p < .01$). Significant mortality and age group differences in cognitive performance were observed for all measures ($p < .01$) except vocabulary (no age differences). For all measures, the Young-Old and Survivors outperformed the Old-Old and Decedents.

Greater theoretical interest was reserved for examining intraindividual rates of cognitive change as a function of age and impending mortality. There were three slopes for each cognitive measure. The first slope (γ_{10}) assessed the average rate of cognitive change for Young-Old Survivors per additional year of time remaining in the study. The second slope (γ_{11}) tested the average difference in cognitive change (per year change in time to death) between Young-Old and Old-Old adults controlling for mortality group differences. This slope is tantamount to an Age Group x Time interaction in repeated measures ANCOVA; it tests how much more the Old-Old decline relative to the Young-Old with increasing proximity to death. Similarly, the third slope (γ_{12}) examined the average difference in cognitive change per year change in time to death between Survivors and Decedents controlling for age (i.e., a Mortality Status x Time interaction). Of particular interest, this slope examines terminal decline at the within-person level. To facilitate interpretation, it is important to recognize what the coefficient sign (negative or positive) reflects in the slope column of Table 13. Negative slope coefficients were

Table 13

Cognitive Change as a Function of Time to Death, Age, and Mortality Status

Variable	Intercept γ_{00}	Slope $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t
Semantic verification ^a	3090.19	-1.59	4.06	-0.39
		-43.92	7.15	-6.14**
		-37.44	7.89	-4.75**
Lexical decision ^a	990.26	2.64	1.70	1.56
		-19.33	2.97	-6.50**
		-15.49	3.20	-4.84**
Computation span	3.34	0.01	0.007	0.90
		0.04	0.013	3.08**
		0.01	0.017	0.17
Listening span	3.24	0.02	0.006	2.86**
		0.02	0.011	1.95
		-0.01	0.014	-0.27
Sentence Construction ^a	5.26	0.01	0.006	1.50
		0.03	0.011	2.81**
		0.02	0.012	1.64
Story recall	38.49	-0.01	0.038	-0.11
		0.22	0.067	3.26**
		0.14	0.076	1.86
Word recall ^a	19.00	0.04	0.016	2.60**
		0.16	0.029	5.48**
		0.06	0.033	1.93
Fact recall ^a	21.91	0.10	0.018	5.39**
		0.14	0.032	4.54**
		0.16	0.037	4.36**
Vocabulary ^a	44.76	0.04	0.016	2.58*
		0.11	0.029	3.77**
		0.01	0.009	0.26

^a Significant mortality x age group interactions were observed for these variables;

associated mortality coefficients are reported separately for each age group in Table 14.

Table 13 con't

γ_{00} = cognitive performance for Young-Old Survivors at the sample midpoint for time remaining in the study ($\underline{M}$ = 7.24 years); γ_{10} = average rate of cognitive change per additional year of time remaining in the study for Young-Old Survivors; γ_{11} = average difference in rate of change between Young-Old and Old-Old adults partialling the influence of mortality status; γ_{12} = average difference in rate of change between Survivors and Decedents controlling for age group differences; SE = standard error; t = t-ratio.

*p < .05

**p < .01

associated with the two reaction time latency variables whereas positive slopes were associated with the remaining cognitive measures. The sign of these slopes is a function of the scaling of the time to death variable (i.e., years between each wave and death or May 31) with higher values reflecting greater distance from death. Negative coefficients for the latency variables indicate that, per year closer to death below the sample midpoint, participants' RT latencies were increasing. Similarly, positive coefficients for the other cognitive measures indicated that each additional year closer to death was associated with a corresponding decline in cognitive performance. Alternatively phrased, each additional year further away from death was associated with better cognitive performance.

Young-Old Survivors performed more poorly with increasing proximity to May 31 for 4 of 9 cognitive measures (see Table 13, slope γ_{10}). Significant performance declines were observed for listening span, word recall, fact recall, and vocabulary for each additional year closer to this date. As expected, performance for the Old-Old declined at significantly faster rates per year closer to death for all measures except listening span (see coefficients associated with slope γ_{11}). Using semantic verification as an example, a Young-Old Survivor at the midpoint on the time to death variable had a response latency of 3090.19 msec and slowed a further 1.59 msec (γ_{10}) for each additional year closer to May 31. The γ_{11} slope coefficient for semantic verification indicated that the average difference between rates of change for the Young-Old and Old-Old was significant. Old-Old adults exhibited significantly slower response latencies per year closer to death ($-1.59 + -43.92 = -45.51$ msec per year). This amounts to a 329.49 msec increase in RT over the course of the interval between 7.24 years and death/May31.

Significant differences in rates of cognitive change were also observed between

survivors and those facing impending death for semantic verification, lexical decision, and fact recall (see coefficients associated with slope γ_{12}). With increasing proximity to death, Decedents declined at significantly faster rates. Again, using semantic verification as an exemplar, the γ_{12} slope coefficient indicated that Decedents exhibited significantly slower response latencies ($-1.59 + -37.44 = -39.03$ msec) per year closer to death. Whereas Survivors RT latencies increased 11.51 msec between the midpoint and terminus of the time to death interval, latencies for Decedents increased 282.58 msec. Random effect coefficients were examined for each corresponding intercept and slope. All were found to be significant ($p < .01$), indicating that, even after controlling for the predictors examined, significant between-group differences in intercepts (average performance) and slopes (individual rates of change per year change in proximity to death) remained to be explained.

As indicated in Table 13, significant Age Group x Mortality Status interactions were observed for all but the listening span, computation span, and story recall measures. To probe these significant interactions, simple regression slopes examining the association between cognitive change, time to death, and mortality were calculated separately for each age group (consistent with simple effects analysis in ANOVA). There were 337 Young-Old survivors, 182 Young-Old decedents, 105 Old-Old survivors, and 83 Old-Old decedents.

Table 14 summarizes the results for these significant interactions. Consistent with expectations, marked differences in patterns were observed between the Young-Old and Old-Old. Beginning with the top of Table 14, Young-Old Survivors performed more poorly with increasing proximity to May 31 for semantic verification, sentence

Table 14
Cognitive Change as a Function of Time to Death and Mortality Status
for Young-Old ($n = 519$) and Old-Old ($n = 188$) Adults

Variable	Intercept γ_{00}	Slope γ_{10}/γ_{11}	SE	t
Young-Old				
Semantic verification	3153.85	-7.08	3.55	-2.00*
		-8.31	8.64	-0.96
Lexical decision	1012.10	0.76	1.05	0.50
		-5.73	3.52	-1.63
Sentence Construction	5.24	0.01	0.006	2.41*
		0.00	0.013	-0.02
Word recall	18.91	0.05	0.016	3.17**
		0.02	0.039	0.45
Fact recall	21.74	0.11	0.017	6.12**
		0.11	0.046	2.34*
Vocabulary	44.63	0.05	0.015	3.44**
		-0.07	0.041	-1.68
Old-Old				
Semantic verification	3546.12	-28.59	9.50	-3.01**
		-90.34	16.55	-5.46**
Lexical decision	1129.50	-10.74	4.22	-2.55*
		-35.79	7.04	-5.09**
Sentence Construction	4.86	0.03	0.014	2.07*
		0.06	0.024	2.34*
Word recall	16.77	0.18	0.033	5.42**
		0.15	0.060	2.43*
Fact recall	19.15	0.21	0.035	6.06**
		0.25	0.065	3.88**
Vocabulary	45.25	0.12	0.034	3.49**
		0.13	0.064	2.02*

Table 14 con't

γ_{00} = cognitive performance for Survivors at the sample midpoint for time remaining in the study ($\underline{M}$ = 7.24 years); γ_{10} = average rate of cognitive change for Survivors per additional year of time remaining in the study; γ_{11} = average difference in rate of change between Survivors and Decedents; SE = standard error; t = t-ratio.

* $p < .05$

** $p < .01$

construction, word recall, fact recall, and vocabulary (see Table 14, Young-Old slope γ_{10}). However, intraindividual trajectories of performance decline for Young-Old Decedents relative to Survivors showed significant differences for the fact recall task alone (see coefficients associated with slope γ_{11}). Decedents declined at approximately twice the rate ($0.11 + 0.11 = 0.22$ fewer facts recalled per year closer to death) of survivors (0.11 facts per year). In contrast, reported coefficients for the Old-Old revealed a different story. Old-Old Survivors performed more poorly per year closer to death for all six cognitive measures (see Table 14, Old-Old slope γ_{10}). Moreover, unlike the Young-Old findings, significant differences in rates of cognitive change were observed between survivors and decedents for all measures (see coefficients for Old-Old slope γ_{11}); decedents declined at least twice the rate (e.g., $0.21 + 0.25 = 0.46$ fewer facts recalled per year closer to death) and as much as triple the rate ($-28.59 + -90.34 = 118.93$ msec slower on the semantic verification task per year closer to death) for these cognitive tasks. These findings underscore that mortality differences in cognitive decline are further amplified with increasing age.

Intraindividual cognitive decline as a function of cause of death. A follow-up multilevel analysis was computed for decedents only. Longitudinal change for each cognitive measure was examined as a function of: (a) years to death/years remaining in study (HLM Level 1 variable); (b) age group (HLM Level 2 variable); and (c) specific cause of death (HLM Level 2 variable). The associated HLM equations are interpreted in the same manner as those just presented. Together, equations 7, 8, and 9 test whether cognitive performance declines more precipitously as impending death draws nearer and whether time to death trajectories vary as a function of age or cause of death.

$$\text{Level 1: Cognitive Performance}_{it} = B_{0i} + B_{1i} * (\text{Time to Death}_{it}) + R_{it} \quad (7)$$

$$\begin{aligned} \text{Level 2: } B_{0i} = & \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Cause of Death}_i) \\ & + \gamma_{03} * (\text{Age Group} \times \text{Cause of Death}_i) + U_{0i} \end{aligned} \quad (8)$$

$$\begin{aligned} B_{1i} = & \gamma_{10} + \gamma_{11} * (\text{Age Group}_i) + \gamma_{12} * (\text{Cause of Death}_i) \\ & + \gamma_{13} * (\text{Age Group} \times \text{Cause of Death}_i) + U_{1i} \end{aligned} \quad (9)$$

Table 15 summarizes the results for these multilevel analyses. Expected differences were observed between the Young-Old and Old-Old. Controlling for cause of death, Old-Old adults exhibited significantly greater cognitive decline on all cognitive measures per additional year closer to death (see Table 15, Slope γ_{11}). In contrast, expected increases in cognitive decline for those who died of CVD-related causes were only observed for two measures: semantic verification and word recall (see Table 15, Slope γ_{12}). For semantic verification, those who died of CVD compared with those who died of non-CVD causes slowed an additional 37.83 msec per additional year closer to death (e.g., from 7.24 years to 6.24 years). Similarly, word recall performance for those who died of CVD declined an additional .18 units per additional year closer to death relative to those who died of non-CVD causes. Significant age group by cause of death interactions were not found for any cognitive measures. These findings highlight that decedents exhibit increased cognitive decline with increasing proximity to death, particularly for the Old-Old. However, intraindividual trajectories of cognitive decline do not appear to share strong associations with CVD compared with non-CVD causes of death (although absolute performance was lower for those with CVD).

Table 15

Cognitive Change as a Function of Time to Death, Age Group, and Cause of Death

Variable	Intercept γ_{00}	Slope $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t
Semantic verification	3407.73	-6.43	13.12	-0.49
		-90.29	18.10	-4.99**
		-37.83	17.80	-2.13*
Lexical decision	1067.23	-6.06	5.24	-1.16
		-35.28	7.39	-4.77**
		-5.80	7.19	-0.81
Sentence Construction	4.76	0.01	0.019	0.65
		0.06	0.026	2.24*
		0.01	0.025	0.57
Listening span	3.01	0.01	0.018	0.49
		0.05	0.023	2.12*
		-0.01	0.024	-0.06
Computation span	3.09	0.01	0.027	0.43
		0.08	0.036	2.19*
		-0.04	0.036	-1.11
Story recall	33.34	0.04	0.105	0.39
		0.35	0.143	2.48*
		0.17	0.141	1.22
Word recall	17.18	0.02	0.045	0.35
		0.18	0.060	2.94**
		0.18	0.059	2.94**
Fact recall	19.74	0.18	0.055	3.28**
		0.20	0.074	2.71**
		0.08	0.074	1.10
Vocabulary	43.37	-0.02	0.054	-0.28
		0.23	0.073	3.08**
		0.04	0.072	0.55

Table 15 con't

γ_{00} = cognitive performance for Young-Old adults who did not die of CVD-related causes at the sample midpoint for time remaining in the study ($\underline{M} = 7.24$ years); γ_{10} = average rate of cognitive change per additional year of time remaining in the study for Young-Old adults without CVD; γ_{11} = average difference in rate of change between Young-Old and Old-Old adults partialling the influence of cause of death; γ_{12} = average difference in rate of change between CVD and non-CVD cause of death controlling for age group differences; SE = standard error; t = t-ratio.

*p < .05

**p < .01

Examining the Shape of Cognitive Change: Terminal Decline or Drop?

In addition to examining linear rates of change, HLM permits assessment of nonlinear decline. Examining nonlinear functions facilitates comparison of terminal decline vs. terminal drop patterns of cognitive change as a function of time to death. Several HLM models were fit to specifically contrast a slow-but-steady vs. precipitous drop account of cognitive decline.

Quadratic acceleration. First, a Level 1 quadratic time to death variable was included as an additional predictor in equation 4 to examine the acceleration or deceleration for each linear growth trajectory. Significant quadratic effects were observed for semantic verification ($\gamma_{20} = 1.87, t = 3.01$), story recall ($\gamma_{20} = -0.01, t = -2.10$), and vocabulary ($\gamma_{20} = -0.01, t = -2.39$). As polynomial analyses are sequential, it was first necessary to assess whether quadratic trends significantly improved prediction over linear effects prior to interpreting the coefficients. To determine whether the model including the quadratic effect yielded a significant improvement in fit to the data, a simple test was performed by subtracting the deviance statistic in the linear and quadratic model from the deviance statistic in the linear only model (the difference between deviance statistics of two nested models has an associated chi-square distribution). Performing this calculation yielded a change in deviance (Δ deviance) between the two nested models of 147.8, 33.52, and 17.49 (respectively for semantic verification, story recall, and vocabulary) with a corresponding change of 3 degrees of freedom ($\Delta df = 3$). Comparing these estimates to the critical value of chi-square with 3 df at the $\alpha = .05$ level (7.81), the change in deviance values exceeded the critical value indicating that the linear and quadratic model fit the data significantly better than the linear model alone.

Thus, consideration of the fixed and random effects associated with the quadratic model are warranted.

For semantic verification, the mean quadratic acceleration was positive indicating that increasing response latencies were accelerated for each additional year closer to death. Moreover, there were significant mortality group differences for the semantic verification quadratic effect. Slower latencies were significantly accelerated a further 4.52 msec ($t = 3.09$) per year over and above the linear rate for decedents relative to survivors. This is consistent with a terminal drop pattern of cognitive decline with increasing proximity to death. The quadratic trend did not exhibit age differences nor an age x mortality status interaction.

For both story recall and vocabulary, the quadratic term was negative indicating a deceleration of the positive linear time to death slope. This implies that each year further from death is associated with increasing cognitive performance, but that this function is negatively accelerated (i.e., dampened) for larger values of time to death (i.e., more years from death). The converse was true for the shape of cognitive change in closer proximity to death; the rate of cognitive decline was larger for smaller values of the time to death predictor (i.e., fewer vs. more years to death). Significant age and mortality group differences were observed for the story recall quadratic effect. Over and above the linear rate, the percentage decline in story recall performance per additional year closer to death was accelerated for the Old-Old relative to the Young-Old (.03 percent per year, $t = -2.58$) and for Decedents relative to Survivors (.03 percent per year, $t = -2.15$). There were no group differences for vocabulary or significant interactions for either measure. The random effects coefficient (U_{2i}) associated with the quadratic effect for each cognitive

measure was significant ($\chi^2(515) = 926.23, 871.65, \text{ and } 947.62, p < .001$) indicating between-person differences about the average quadratic slope respectively for semantic verification, story recall, and vocabulary. The slopes for some individuals were characterized by more curved acceleration than others. For the remaining six cognitive measures, individual quadratic effects were not significant.

Piecewise acceleration. Although informative, the preceding quadratic analyses provide a conservative estimate of terminal decline vs. terminal drop. With fewer occasions of measurement, a quadratic function does not represent the most powerful approach for examining nonlinear trends. An alternative analysis using piecewise regression coefficients was conducted to more precisely pinpoint the locus of change. Consider the individual trajectories of cognitive change in Figure 17. Many survivors displayed relatively stable response latencies across as many as five waves (12 years) of measurement; comparatively few survivors showed large RT increases. In contrast, decedents exhibited cognitive deterioration that appeared to accelerate toward their respective last wave of testing with a much larger number of individuals demonstrating large increases in RT latency. Observed differences in cognitive change trajectories between survivors and those who would die suggest it is important to test whether acceleration of change differs between the mortality groups.

To test a two-piece linear growth model, a multilevel piecewise regression analysis was computed examining individual growth rates for two separate periods. First, a linear growth model was fit examining change in cognitive performance per additional wave of measurement. In addition to examining change across waves, between-group age and mortality group differences in rates of change were considered (see equations 10,

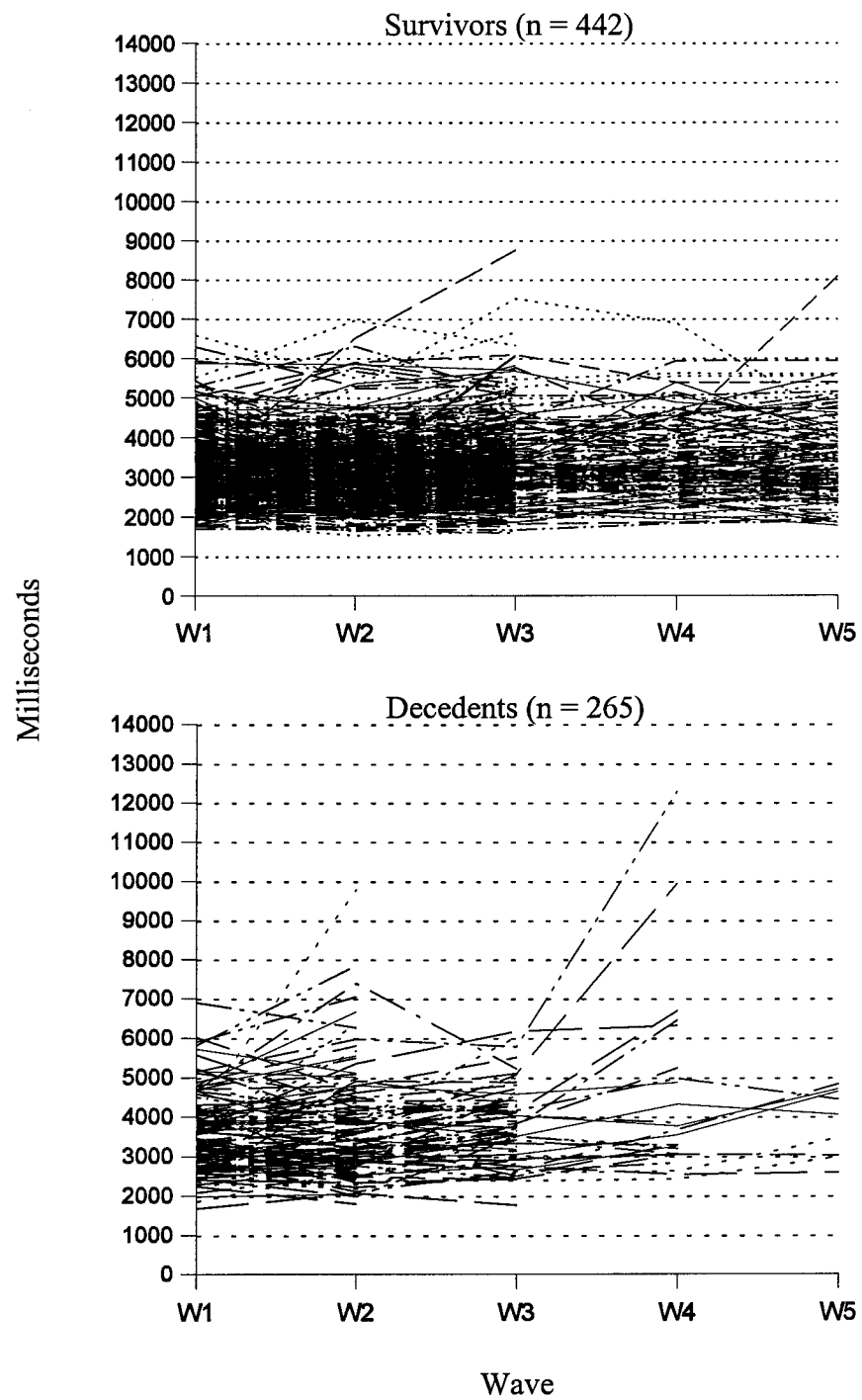


Figure 17

Intraindividual Profiles for Semantic Verification
by Measurement Wave and Mortality Group

11 and 12).

$$\text{Level 1: Cognitive Performance}_{it} = B_{0i} + B_{1i} * (\text{Wave}_{it}) + R_{it} \quad (10)$$

$$\text{Level 2: } B_{0i} = \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Mortality Status}_i) + U_{0i} \quad (11)$$

$$B_{1i} = \gamma_{10} + \gamma_{11} * (\text{Age Group}_i) + \gamma_{12} * (\text{Mortality Status}_i) + U_{1i} \quad (12)$$

Next, a two-piece multilevel regression was computed entering both the base linear rate of change (coded as 0, 1, 2, 3, and 4) as well as a second variable reflecting the increment (or decrement) to the linear growth rate (coded as 0, 0, 1, 2, and 3) following the second measurement wave (see equations 13, 14, and 15).

$$\begin{aligned} \text{Level 1: Cognitive Performance}_{it} = & B_{0i} + B_{1i} * (\text{Wave}_{it}) + \\ & B_{2i} * (\text{Change after Wave 2}_{it}) + R_{it} \end{aligned} \quad (13)$$

$$\text{Level 2: } B_{0i} = \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Mortality Status}_i) + U_{0i} \quad (14)$$

$$B_{1i} = \gamma_{10} + \gamma_{11} * (\text{Age Group}_i) + \gamma_{12} * (\text{Mortality Status}_i) + U_{1i} \quad (15)$$

In contrast to the quadratic predictor, the second piecewise predictor used in these analyses examined whether the rate of change significantly increased (or declined) for a specified period (i.e. after the second wave of testing). The nature of the trends outlined in Figure 17 (and for many of the other cognitive measures) suggested the interval between waves 2 and 3 may represent an important transition point. Moreover, this interval was chosen as it reflects the average distance to death (7.24 years) for decedents (and time remaining in the study for survivors). Age group and mortality status were

entered as additional predictors to assess between-group differences for this secondary rate of change. Examination of this two-piece growth model tests whether change in cognitive performance is differentially accelerated for decedents relative to survivors across waves (i.e., with increasing proximity to death).

Both the linear and piecewise models (equations 10 through 12 and 13 through 15 respectively) were fit. The linear growth model estimated four parameters whereas the more complex linear plus piecewise growth model estimated seven parameters. For each cognitive task, change in model deviance values were computed to compare against a critical value of chi-square with 3 df (7.81, $p = .05$). In all cases, the reduction in deviance for the two-piece model exceeded the critical value indicating that the more complex model fit the data significantly better than the linear model alone. Thus, Table 16 displays fixed effect coefficients (in raw score metric) associated with the piecewise change model.

As expected, all intercepts (γ_{00}) were significantly different from 0 ($p < .01$). For Young-Old Survivors, the average rate of linear cognitive change per additional wave of measurement (γ_{10}) was significant for lexical decision, sentence construction, and fact recall; slight improvements were observed for the first two tasks whereas decline was observed for the latter. Of greater interest, Old-Old adults exhibited significantly greater linear decline across waves than the Young-Old for semantic verification, lexical decision, sentence construction, word recall, and fact recall (see Table 16, Wave Slope B_1 column, γ_{11} coefficient). Similarly, relative to Survivors, Decedents declined more across waves for tests of semantic verification, lexical decision, and sentence construction; linear declines approached significance for computation span, word recall, and fact recall

Table 16

Piecewise Cognitive Change as a Function of Measurement Wave, Age Group, and Mortality Status

Variable	Intercept γ_{00}	Wave Slope B_1 $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t	Change after Wave		
					2 Slope B_2 $\gamma_{20}/\gamma_{21}/\gamma_{22}$	SE	t
Semantic verification	3097.30	-55.54	30.71	-1.81	81.01	35.80	2.26*
		186.89	50.73	3.68**	-157.15	59.13	-2.66**
		104.10	46.30	2.25*	-125.49	53.97	-2.33*
Lexical decision	1008.79	-48.55	11.90	-4.08**	60.30	13.78	4.38**
		78.97	19.65	4.02**	-68.68	22.75	-3.02**
		73.83	17.93	4.12**	-86.89	20.77	-4.18**
Computation span	3.36	0.06	0.054	1.06	-0.13	0.067	-1.87
		-0.14	0.093	-1.52	0.11	0.115	0.91
		-0.15	0.091	-1.64	0.25	0.112	2.20*
Listening span	3.33	-0.07	0.045	-1.60	0.04	0.058	0.66
		0.01	0.078	0.14	-0.07	0.099	-0.68
		-0.03	0.076	-0.38	0.09	0.096	0.88
Sentence Construction	5.25	0.12	0.052	2.38*	-0.19	0.061	-3.07**
		-0.19	0.086	-2.24*	0.18	0.101	1.75
		-0.25	0.078	-3.17**	0.33	0.092	3.55**

Table 16 con't

Variable	Intercept	Wave Slope B ₁	SE	t	Change after Wave 2 Slope B ₂	SE	t
	γ_{00}	$\gamma_{10}/\gamma_{11}/\gamma_{12}$			$\gamma_{20}/\gamma_{21}/\gamma_{22}$		
Story recall	38.69	-0.14	0.312	-0.44	0.12	0.371	0.32
		0.02	0.516	0.05	-0.43	0.613	-0.71
		-0.65	0.471	-1.38	0.77	0.559	1.38
Word recall	19.14	0.10	0.132	0.74	-0.25	0.155	-1.61
		-0.44	0.212	-2.04*	0.25	0.257	0.96
		-0.32	0.199	-1.63	0.48	0.234	2.04*
Fact recall	22.34	-0.33	0.148	-2.24*	0.19	0.174	1.11
		-0.49	0.245	-1.99*	0.26	0.287	0.91
		-0.41	0.224	-1.85	0.61	0.262	2.34*
Vocabulary	44.90	0.01	0.140	0.10	-0.13	0.167	-0.77
		-0.27	0.232	-1.15	0.13	0.275	0.48
		-0.10	0.212	-0.50	0.22	0.251	0.86

γ_{00} = cognitive performance for Young-Old Survivors at the first wave of testing; γ_{10} = average rate of linear cognitive change per additional wave of measurement (approximately 3.22 year intervals) for Young-Old Survivors; γ_{11} = average difference in linear change between Young-Old and Old-Old adults partialling the influence of mortality status; γ_{12} = average difference in linear change between Survivors and Decedents controlling for age group differences; γ_{20} = average increment or decrement to the linear change base rate per additional measurement occasion after wave 2 for Young-Old Survivors; γ_{21} = average difference in change increment/decrement between Young-Old and Old-Old adults partialling the influence of mortality status; γ_{22} = average difference in change increment/decrement between Survivors and Decedents controlling for age group differences; SE = standard error; t = t-ratio.

* $p < .05$ ** $p < .01$

(see Table 16, Wave Slope B_1 column, γ_{12} coefficient). Overall, trends indicated greater linear declines for Decedents and the Old-Old.

Coefficients associated with the second slope (see Table 16, Change after Wave 2 Slope B_2 column) tested whether the rate of linear change was accelerated or decelerated across waves. These coefficients speak to the issue of terminal decline vs. terminal drop. The γ_{22} coefficient is of particular interest; it examines whether differences in accelerated cognitive decline exist between Survivors and Decedents. Consistent with the terminal drop hypothesis, significant acceleration of cognitive decline was observed for sentence construction, computation span, word recall, and fact recall. The γ_{22} coefficients for each of the four measures was positive indicating an acceleration of the negative linear slopes (i.e., accelerated decline) across waves. Performance for these measures yielded fewer correct responses per additional wave of measurement (over and above linear decline) beginning after the second wave. Decline for Decedents was accelerated an additional 0.33, 0.25, 0.48, and 0.61 units per wave for sentence construction, computation span, word recall, and fact recall respectively. To illustrate the impact of this acceleration, consider decline in sentence construction for Decedents across a five-wave period. If only the linear rate were taken into account, the average decedent would decline 1.25 sentence construction units across waves ($-0.25 \text{ units per wave} \times 5 \text{ waves}$). Adding to this decline, the significant piecewise coefficient for sentence construction indicated a further acceleration of 0.33 units for Decedents (relative to survivors) after the second wave of measurement. Decedents declined .50 units across the first two waves followed by an increased rate of decline ($1.74 \text{ units} = .25 \times 3 + .33 \times 3$) across the final three waves (total decline of 2.24 units across five waves). Similarly, Decedents' rates of decline for

the other cognitive measures increased from: (a) .30 units across the first two waves to 1.20 units across the final three waves for computation span; (b) .64 units across the first two waves to 2.40 units across the final three waves for word recall; and (c) .82 units across the first two waves to 3.06 units across the final three waves for fact recall.

Significant piecewise coefficients were also observed for semantic verification and lexical decision. In addition to significant linear increases in response latency across waves, significant differences in rates of decline after Wave 2 emerged for both age group and mortality status. For semantic verification, Old-Old adults slowed an additional 157.15 msec per wave relative to the Young-Old (see Table 16, Change after Wave 2 Slope B_2 , γ_{21}). Similarly, over and above linear decline, response latencies slowed for Decedents relative to Survivors by 125.49 msec per wave (see Table 16, Change after Wave 2 Slope B_2 , γ_{22}). Figure 17 demonstrates the mortality status differences in rate of change after Wave 2. Similar trends were observed for lexical decision with both the Old-Old and Decedents exhibiting increasingly slower response latencies following Wave 2 performance. Older adults slowed by an additional 68.68 msec per wave whereas Decedents slowed 86.89 msec per wave over and above the linear rate. Finally, the random effects coefficient (U_{2i}) associated with the second piecewise predictor was significant for each cognitive measure ($p < .001$) indicating between-person differences (independent of age and mortality) in rates of change post second wave.

Summary: Intraindividual Cognitive Decline as a Function of Time to Death.

The focal point of the fourth research question concerned the shape (i.e., linear vs. curvilinear) of cognitive decline in relation to years to death. Consistent with expectations, cognitive decline trajectories varied as a function of proximity to death, age,

and mortality status. Old-Old adults declined at significantly faster rates per additional year closer to death for all cognitive measures except listening span. Significant differences in cognitive change were also observed between survivors and decedents (for three measures). With increasing proximity to death, decedents declined at significantly faster rates. Significant age by mortality interactions were observed for six measures. For Old-Old adults, significant differences in rates of cognitive change were observed between survivors and decedents for all measures. In contrast, mortality differences for the Young-Old were only present for one measure. This dissociation indicates that mortality-related effects on cognitive performance are amplified with increasing age. A separate multilevel analysis, for decedents alone, considered the role of global vs. specific causes of death on cognitive decline. Cause of death significantly influenced change for semantic verification and word recall. For both measures, those who died of CVD-related causes declined more per additional year closer to death.

Several nonlinear analyses were computed to consider whether change over time was better characterized as terminal decline or terminal drop. Significant quadratic trends were observed for three cognitive measures. Trends indicated that the shape of cognitive decline was accelerated in closer proximity to death, particularly for decedents. A second piecewise regression analysis was conducted to more precisely describe the shape of change, and to consider whether change varied between age and mortality groups. Findings indicated greater linear declines across testing waves for Decedents and Old-Old adults. Of particular import, significant acceleration of cognitive decline after the second wave of testing (i.e., in closer proximity to death) was observed for six cognitive measures. The rate of decline for decedents relative to survivors was accelerated; this

pattern is consistent with a terminal drop trajectory.

Performance Variability as a Correlate of Terminal Decline

Having demonstrated consistent patterns of terminal decline, a logical next step involved examining plausible correlates of mortality-related cognitive deficits to identify potential mechanisms. Intraindividual performance variability (i.e., inconsistency) represents a promising correlate. Previous investigations have documented relationships between inconsistency across RT trials, age, and neural integrity (e.g., Hultsch et al, 2000; Hultsch et al., 2002). As a behavioural indicator, cognitive inconsistency is thought to reflect neurobiological irregularities due to factors including aging and disease. Given potential associations of mortality status and terminal decline with general CNS disturbances (e.g., Berg, 1996), and the suggestion that neurobiological irregularities may underlie both neural integrity (e.g., Hultsch et al, 2000) and age-related increases in performance inconsistency (Hultsch et al., 2002), the Figure 1 path (see page 33) between performance inconsistency and mortality status warrants further investigation. Both cross-sectional and longitudinal MANCOVAs were employed to examine RT inconsistency in relation to age, mortality status, and time to death.

Performance Variability as a Function of Age and Mortality Status

Measuring inconsistency. Findings from earlier sections support the hypothesis that decedents exhibit greater performance inconsistency. For example, standard deviations for most cognitive performance measures were larger for decedents relative to survivors, particularly for the Old-Old (e.g., see Table 6, page 63). However, prior to drawing any conclusions based on these observations between RT variability and cognitive performance, several confounds were addressed. Although multiple measures

have been used to index RT inconsistency (Ratcliff, 1979; Slifkin & Newell, 1998), the simplest indicator is the intraindividual standard deviation (ISD) computed across trials. However, ISDs based on raw scores are potentially misleading: mortality group differences in response latency and systematic changes in performance across trials (or over time) may influence inconsistency estimates. For example, decedents may exhibit higher ISDs relative to survivors based solely on their slower latencies (e.g., Hale, Myerson, Smith, & Poon, 1988). Similarly, mortality group differences for practice effects may also influence ISD estimates (e.g., Hultsch et al., 2000, 2002).

To dissociate systematic versus transient changes in performance, ISDs were calculated by partialing group differences and systematic within-person changes from the raw data. Using a person by time data matrix, a split-plot ANOVA was used to partial age group, mortality status, cause of death, trial and occasion effects, and all their higher-order interactions by regressing each verbal speed measure on these potential confounding variables: only the lexical decision and semantic verification tasks were examined because calculation of ISDs requires a large number of trials. This procedure produced residual scores uncontaminated by group differences in speed or accuracy of performance, or systematic variation across occasions due to influences such as practice, learning to learn, or materials effects associated with alternate task forms. Residual trial scores were converted to T-scores (to permit same-metric comparisons across tasks) followed by the computation of ISDs for each participant. Higher ISDs reflect relatively inconsistent performance across trials, whereas lower scores indicate relatively consistent performance. As a graphical illustration, Figures 18 and 19 show residual T-scores for all participants for lexical decision and semantic verification latency graphed as a function of

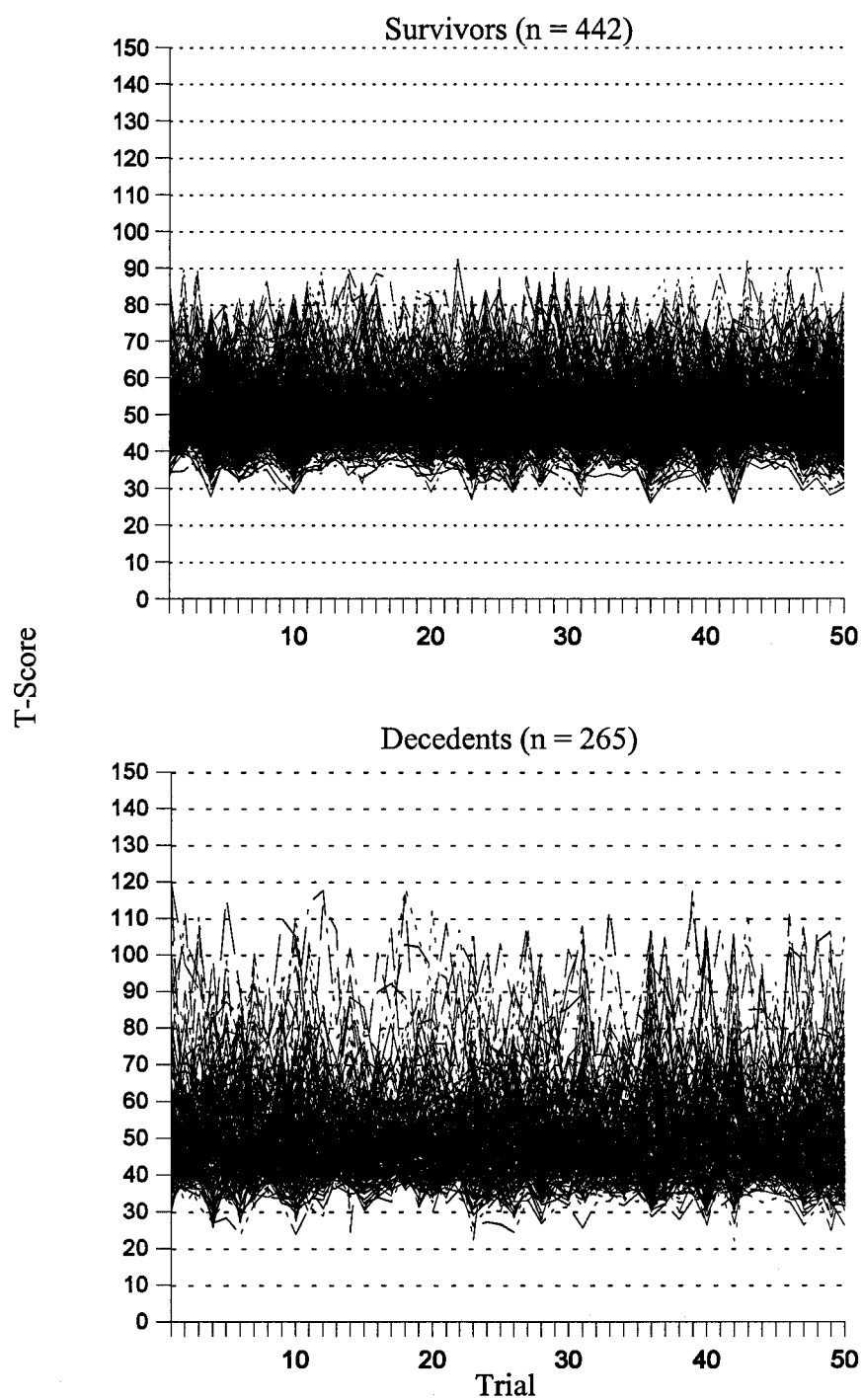


Figure 18
Intraindividual Variability Profiles: Residualized T-scores
for Semantic Verification by Trials and Mortality Group

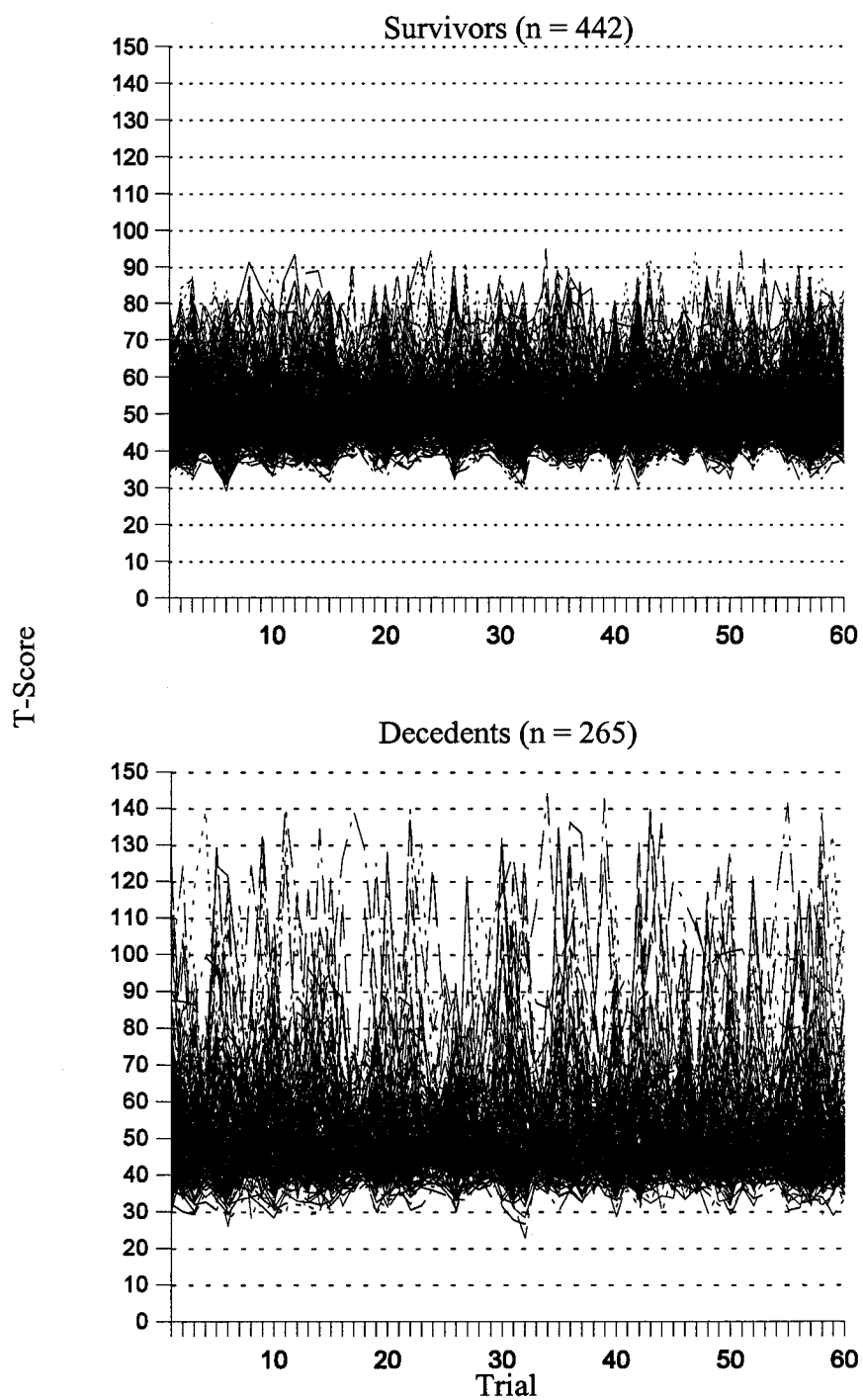


Figure 19
Intraindividual Variability Profiles: Residualized T-scores
for Lexical Decision by Trials and Mortality Group

trials and mortality group. The figures demonstrate that marked individual differences in performance inconsistency remain even subsequent to partialing select systematic effects from the data (i.e., for both survivors and decedents, group $\underline{M} = 50$ and $\underline{SD} = 10$). Patterns of inconsistency were conspicuously amplified for decedents.

Prior to computing the ISDs, distributions of raw latency scores were examined for outliers. Extremely fast or slow responses might reflect various types of errors (e.g., accidental key presses or task interruptions). Outliers were trimmed to minimize such influences. For each task, a lower bound for legitimate responses was set based on minimal response times suggested by prior research (lexical = 400 ms; semantic = 1000 ms): trial scores below these limits were dropped. For each occasion of measurement, upper bounds were established by computing means and standard deviations as a function of age group and dropping any trials exceeding the mean by three or more standard deviations. The total number of trials dropped across the entire Persons x Trials x Occasions data matrix was relatively small (Wave 1: lexical = 1.65%; semantic = 2.02%; Wave 2: lexical = 1.59%; semantic = 2.86%; Wave 3: lexical = 1.68%; semantic = 2.65%) and unrelated to mortality status. To avoid statistical problems associated with missing data, values for outlier trials were imputed using a regression procedure in which missing value estimates were based on relationships among responses across trials and occasions. Missing values were imputed using data from all available individuals, trials, and occasions. Because dropping outlier scores and imputing corresponding missing values reduces variability, these data preparation strategies represent a conservative approach to examining the phenomenon.

Age and mortality differences in performance inconsistency. To statistically

analyze the intraindividual profiles depicted in Figures 18 and 19, several cross-sectional MANCOVAs were employed. First, for decedents alone ($n = 265$), a 2 (age group) $\times$ 2 (cause of death) MANCOVA (partialing education) was computed to examine potential global vs. specific mortality influences on inconsistency. Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .640$, $F(2, 259) = 73.00$, $p < .001$, $\eta^2 = .36$; and Cause of Death, Wilks' $\lambda = .965$, $F(2, 259) = 4.74$, $p < .05$, $\eta^2 = .04$. Neither the Education covariate nor the Age Group $\times$ Cause of Death interaction were significant.

As expected, univariate between-subjects effects for Age Group indicated that the Old-Old were significantly more variable than the Young-Old for both lexical decision ($F(1, 260) = 117.86$, $p < .001$, $\eta^2 = .31$) and semantic verification ($F(1, 260) = 103.14$, $p < .001$, $\eta^2 = .28$). Moreover, the cause of death main effect for inconsistency was also significant for lexical decision ($F(1, 260) = 7.34$, $p < .01$, $\eta^2 = .03$) and semantic verification ($F(1, 260) = 7.04$, $p < .01$, $\eta^2 = .03$). Relative to non-CVD causes, decedents with CVD exhibited significantly greater performance inconsistency across verbal speed RT trials for their respective final measurement waves. Figure 20 shows the means and standard deviations for performance inconsistency by age group and cause of death. Although not significant, the interaction was in the expected direction. These inconsistency findings are relevant to previous investigations that have addressed associations between mean cognitive performance levels and mortality status (see Figure 1, page 32). Relative to other causes, decedents who died of cardiovascular disease exhibited poorer mean cognitive performance for the last wave of testing (see page 115) as well as greater performance inconsistency. Indeed, these patterns were expected in light of previously documented associations between cognitive deficits and mortality

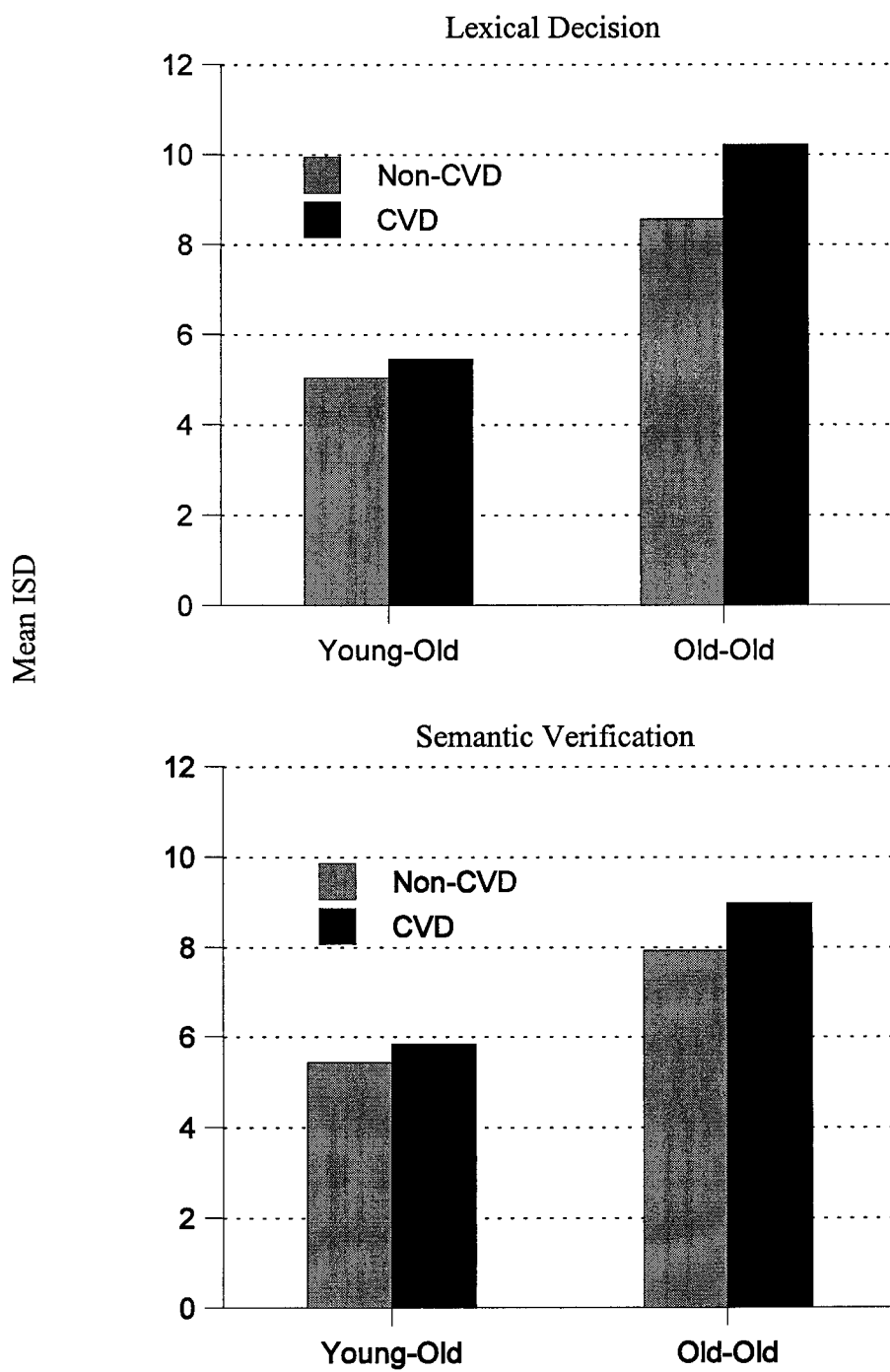


Figure 20

Intraindividual Standard Deviations (ISDs) for Last Wave of Testing
by Age Group and Cause of Death

status on the one hand and relationships between cognitive deficits and inconsistency on the other.

Supplementing the decedent only analysis, a second MANCOVA was computed to examine performance inconsistency for both survivors and decedents as a function of mortality status and cardiovascular disease. A 2 (Age Group) x 4 (Cardiovascular Disease: survivor non-CVD, survivor CVD, decedent non-CVD, decedent CVD) MANCOVA (listwise $n = 707$), covarying education, was computed. Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .752$, $F(2, 697) = 114.73$, $p < .001$, $\eta^2 = .25$; Cardiovascular Disease, Wilks' $\lambda = .780$, $F(6, 1394) = 30.81$, $p < .001$, $\eta^2 = .12$; and the Age Group x Cardiovascular Disease interaction, Wilks' $\lambda = .887$, $F(6, 1394) = 14.32$, $p < .001$, $\eta^2 = .06$. The Education covariate was not significant.

Significant univariate age effects indicated that Old-Old adults were significantly more variable across RT trials than the Young-Old for lexical decision ($F(1, 698) = 191.41$, $p < .001$, $\eta^2 = .22$) and semantic verification ($F(1, 698) = 141.74$, $p < .001$, $\eta^2 = .17$). Building on the earlier decedent analyses, significant univariate cardiovascular group differences in inconsistency were observed for both lexical decision, $F(3, 698) = 47.51$, $p < .001$, $\eta^2 = .17$, and semantic verification, $F(3, 698) = 45.58$, $p < .001$, $\eta^2 = .17$. Post-hoc LSD contrasts ($p < .01$) were computed to compare performance inconsistency for survivors and decedents both with and without CVD. For lexical decision, decedents with CVD ($M = 9.03$ $SD = 5.30$) were significantly more inconsistent than each of the other 3 groups: non-CVD decedents ($M = 7.13$ $SD = 3.34$), CVD survivors ($M = 5.95$ $SD = 1.97$), and non-CVD survivors ($M = 5.82$ $SD = 2.04$). Non-CVD decedents were significantly more inconsistent compared with both survivor groups who did not differ.

Equivalent cardiovascular group differences were observed for semantic verification. Decedents with CVD ($M = 8.32$ $SD = 3.32$) were significantly more inconsistent than non-CVD decedents ($M = 6.99$ $SD = 2.55$), CVD survivors ($M = 6.24$ $SD = 1.76$), and non-CVD survivors ($M = 5.72$ $SD = 1.66$). Decedents without CVD exhibited greater across-trial inconsistency than both survivor groups for the last wave of testing. In addition, survivors with CVD had higher ISDs relative to non-CVD survivors. These patterns suggest that both cardiovascular disease and mortality interactively influenced across-trial performance inconsistency for the last wave of testing.

Both aforementioned main effects were qualified by significant Age Group x Cardiovascular Disease interactions. Univariate tests of this 2-way interaction yielded significant differences for lexical decision, $F(3, 698) = 24.74$, $p < .001$, $\eta^2 = .10$, and semantic verification, $F(3, 698) = 16.61$, $p < .001$, $\eta^2 = .07$. Figure 21 shows cardiovascular group differences for both Young-Old and Old-Old adults. These significant interactions were decomposed using post-hoc simple effects analyses. As the figure indicates, performance inconsistency varied markedly as a function of group. For the Young-Old, across-trial inconsistency on the lexical decision tasks was relatively comparable across mortality status and cardiovascular disease. LSD post-hocs indicated that the Young-Old decedents with CVD were significantly more variable than both survivor groups; no other pairwise comparisons for the Young-Old were significant. In contrast, Old-Old group differences for lexical decision indicated all pairwise comparisons were significant ($p < .01$) save for between the two survivor groups. Similarly, for semantic verification, Young-Old decedents exhibited increased across-trial inconsistency relative to survivors. In addition, Young-Old decedents without CVD were

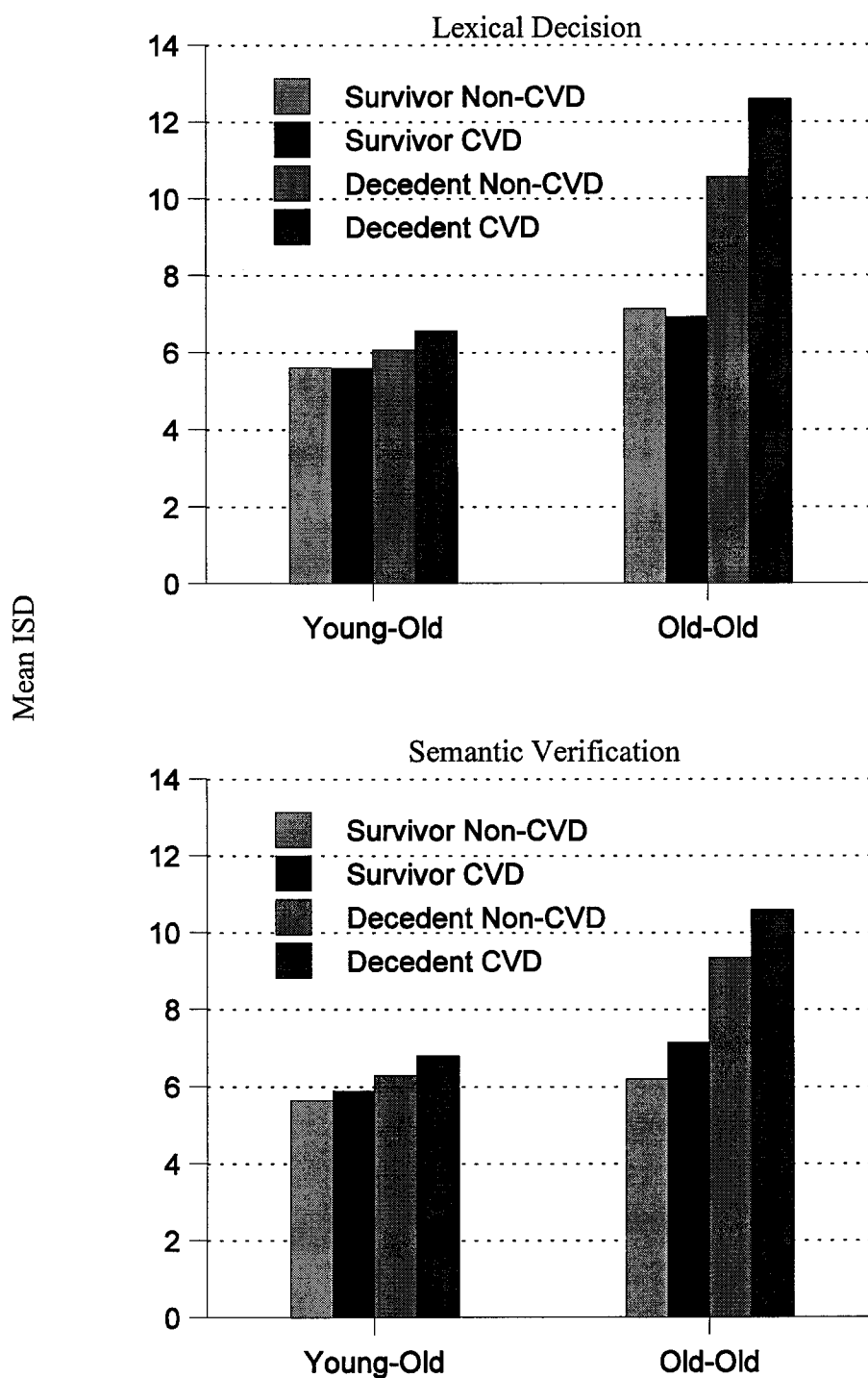


Figure 21

Intraindividual Standard Deviations (ISDs) for Last Wave of Testing
by Cardiovascular Group, Age, and Mortality Status

more inconsistent than non-CVD survivors. For the Old-Old, semantic verification inconsistency differences were accentuated. Decedents with CVD were significantly more variable than all other groups. Indeed, all pairwise group comparisons were significant for the Old-Old ($p < .01$) except between survivors ($p < .05$).

Change in performance inconsistency as a function of age and cause of death. To further inform the last wave of testing analyses, several repeated measures MANCOVAs partialing education were computed to examine 3- and 6-year change in performance inconsistency. First, for decedents alone, a 2 (age group) x 2 (cause of death) x 2 (wave) RMANCOVA examined change in inconsistency as a function of age group and cause of death. Significant omnibus effects for this 2-wave decedent analysis ($n = 114$) were observed for: Age Group, Wilks' $\lambda = .628$, $F(2, 138) = 40.86$, $p < .001$, $\eta^2 = .37$; Cause of Death, Wilks' $\lambda = .956$, $F(2, 138) = 3.18$, $p < .05$, $\eta^2 = .04$; Wave, Wilks' $\lambda = .954$, $F(2, 138) = 3.33$, $p < .05$, $\eta^2 = .05$; and the Age Group x Wave interaction, Wilks' $\lambda = .820$, $F(2, 138) = 15.16$, $p < .001$, $\eta^2 = .18$. No additional omnibus main effects or interactions were significant.

Univariate age group differences indicated that the Old-Old were significantly more variable than the Young-Old for both lexical decision ($F(1, 139) = 64.98$, $p < .001$, $\eta^2 = .32$) and semantic verification ($F(1, 139) = 56.51$, $p < .001$, $\eta^2 = .29$). Decedents exhibited greater inconsistency than Survivors for semantic verification, $F(1, 139) = 5.67$, $p < .05$, $\eta^2 = .04$, with the same pattern approaching significance for lexical decision ($p = .06$). Univariate wave effects were also in the expected direction; performance inconsistency significantly increased across the 3-year interval for semantic verification, $F(1, 139) = 6.64$, $p < .05$, $\eta^2 = .05$. Although not significant, mean performance

inconsistency for lexical decision increased from 6.44 to 7.63 ISDs across waves.

Qualifying the wave main effect, significant univariate Age Group x Wave interactions were observed for both lexical decision, $F(1, 139) = 26.37, p < .001, \eta^2 = .16$, and semantic verification, $F(1, 139) = 15.34, p < .001, \eta^2 = .10$. Relative to the Young-Old decedents, the Old-Old exhibited marked increases in performance variability across the 2 waves for lexical decision ($YO_{wave1} = 5.53$; $YO_{wave2} = 5.65$; $OO_{wave1} = 7.34$; $OO_{wave2} = 9.61$) and semantic verification ($YO_{wave1} = 5.70$; $YO_{wave2} = 6.24$; $OO_{wave1} = 7.14$; $OO_{wave2} = 9.01$). Non-significant patterns for the Age Group x Cause of Death and Cause of Death x Wave interactions were in the expected direction. Those who died of CVD exhibited greater RT inconsistency at cross-section and showed differentially increasing variability with increasing age and over time. The null findings for the cause of death interactions may reflect a power issue; only 114 decedents completed 2 waves of testing. The consistency of cause of death trends should not be overlooked; decedents who died of CVD were systematically more variable than those who died of other causes.

The 3-year change in inconsistency analyses were followed by an examination of 6-year change; a 2 (age group) x 2 (cause of death) x 3 (wave) RMANCOVA controlling for education was computed for decedents alone. Significant omnibus effects for this 3-wave decedent analysis ($n = 76$) were observed for: Age Group, Wilks' $\lambda = .723, F(2, 70) = 13.41, p < .001, \eta^2 = .28$; and the Age Group x Wave interaction, Wilks' $\lambda = .834, F(4, 68) = 3.93, p < .05, \eta^2 = .17$. No additional omnibus main effects or interactions were significant. Univariate age group differences indicated that the Old-Old were significantly more variable than the Young-Old for both lexical decision ($F(1, 71) = 25.95, p < .001, \eta^2 = .27$) and semantic verification ($F(1, 71) = 8.44, p < .01, \eta^2 = .11$).

The univariate effect for the Age Group x Wave interaction yielded a significant difference for lexical decision: $F(2, 142) = 10.15, p < .001, \eta^2 = .13$. Relative to the Young-Old decedents, the Old-Old exhibited marked increases in performance variability across the 3 waves ($M = 5.42, 6.18, \text{ and } 6.05$ vs. $M = 6.52, 8.10, \text{ and } 9.86$ respectively). Although not significant, those who died of CVD exhibited more RT performance variability at cross-section and differentially increasing variability over time. Relative to the 3-year analyses, the null findings for all cause of death main effects and interactions may reflect a further reduction in sample size (265 decedents participated for at least one wave whereas only 76 completed all 3 waves) and power. However, once again the consistency of cause of death trends represents an important observation: indeed, all non-significant univariate findings for these decedent-only analyses were in the expected direction.

Change in performance inconsistency as a function of age and mortality status.

For both survivors and decedents, separate repeated measures MANCOVAs were computed to examine change in performance inconsistency as a function of mortality status and cardiovascular disease. Three-year change was examined using a 2 (Age Group) $\times$ 4 (Cardiovascular Disease: survivor non-CVD, survivor CVD, decedent non-CVD, decedent CVD) $\times$ 2 (Wave: second last and last respective testing occasions) repeated measures MANCOVA (listwise $n = 586$) controlling for education. Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .773, F(2, 576) = 84.56, p < .001, \eta^2 = .23$; Cardiovascular Disease, Wilks' $\lambda = .873, F(6, 1152) = 13.46, p < .001, \eta^2 = .07$; Wave, Wilks' $\lambda = .984, F(2, 576) = 4.69, p < .05, \eta^2 = .02$; the Age Group $\times$ Cardiovascular Disease interaction, Wilks' $\lambda = .944, F(6, 1152) = 5.62, p < .001, \eta^2 = .03$;

the Age Group x Wave interaction, Wilks' $\lambda = .939$ $F(2, 576) = 18.69$, $p < .001$, $\eta^2 = .06$; the Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .916$, $F(6, 1152) = 8.64$, $p < .001$, $\eta^2 = .04$; the Age Group x Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .942$, $F(6, 1152) = 5.85$, $p < .001$, $\eta^2 = .03$; and the Education covariate, Wilks' $\lambda = .989$, $F(2, 576) = 3.19$, $p < .05$, $\eta^2 = .01$.

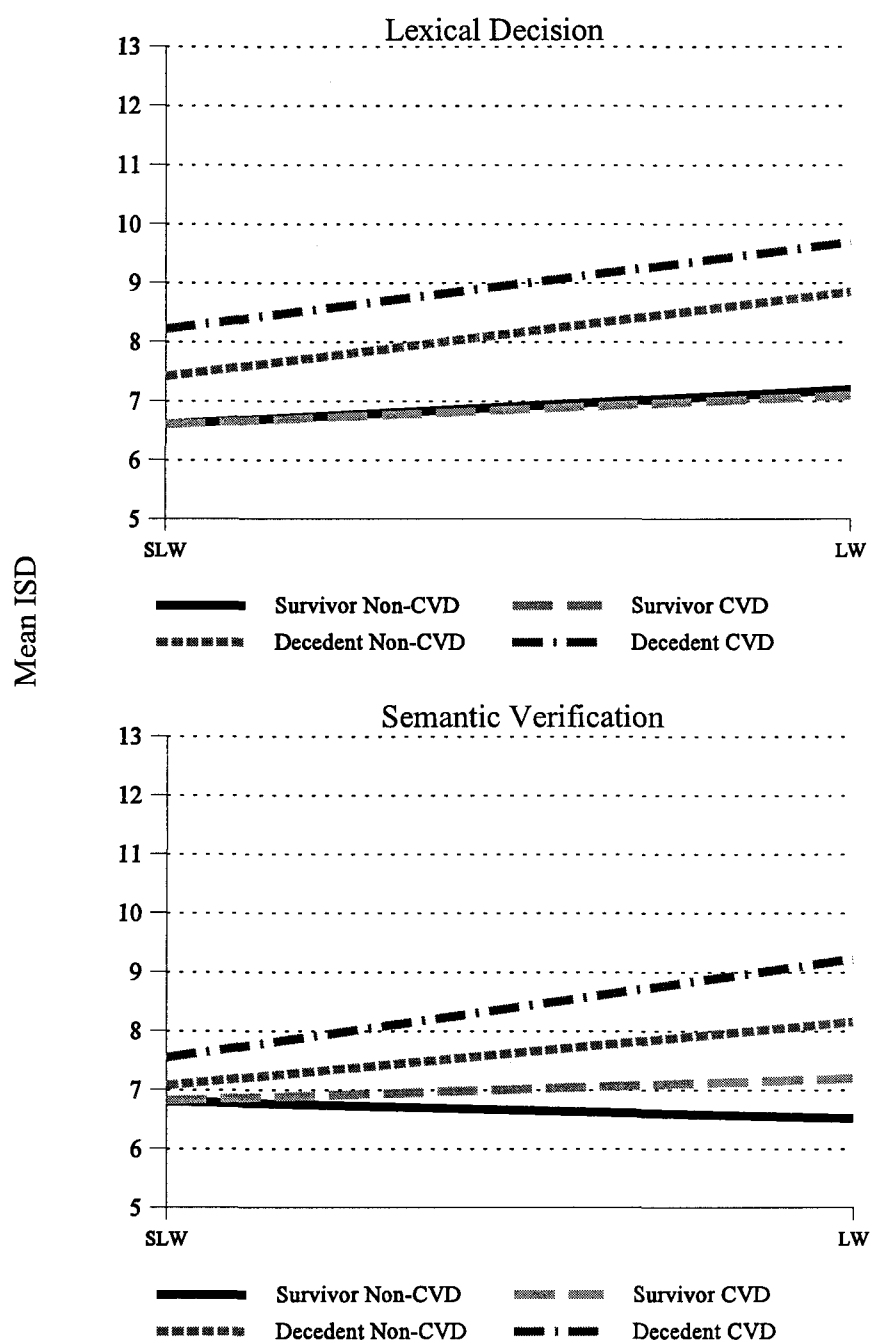
Univariate age and cardiovascular disease effects were consistent with group differences reported for last wave of measurement analyses. In brief, Old-Old adults exhibited increased inconsistency relative to the Young-Old for both lexical decision, $F(1, 577) = 142.51$, $p < .001$, $\eta^2 = .20$, and semantic verification, $F(1, 577) = 100.25$, $p < .001$, $\eta^2 = .15$. Cardiovascular group differences (as a function of mortality) were significant for lexical decision, $F(3, 577) = 21.64$, $p < .001$, $\eta^2 = .10$, and semantic verification, $F(3, 577) = 17.02$, $p < .001$, $\eta^2 = .08$. Post hoc LSD comparisons ($p < .01$) indicated that Decedents with CVD were significantly more inconsistent than non-CVD decedents and both survivor groups for lexical decision ($\text{Decedents}_{\text{CVD}} = 8.95$; $\text{Decedents}_{\text{non-CVD}} = 8.12$; $\text{Survivors}_{\text{CVD}} = 6.85$; and $\text{Survivors}_{\text{non-CVD}} = 6.90$) and semantic verification ($\text{Decedents}_{\text{CVD}} = 8.38$; $\text{Decedents}_{\text{non-CVD}} = 7.61$; $\text{Survivors}_{\text{CVD}} = 7.00$; and $\text{Survivors}_{\text{non-CVD}} = 6.66$). Non-CVD decedents were more inconsistent than the survivor groups who did not differ. For the final omnibus main effect of Wave, significant univariate differences were observed for lexical decision ($F(1, 577) = 7.28$, $p < .01$, $\eta^2 = .01$) and semantic verification ($F(1, 577) = 4.45$, $p < .05$, $\eta^2 = .01$). Performance inconsistency significantly increased over the 3-year interval ($\text{Lexical}_{\text{Wave1}} = 7.21$; $\text{Lexical}_{\text{Wave2}} = 8.20$; $\text{Semantic}_{\text{Wave1}} = 7.06$; and $\text{Semantic}_{\text{Wave2}} = 7.77$).

The age main effect was further qualified by significant age group by wave and

age group by cardiovascular disease interactions. Significant univariate Age Group x Wave interactions were observed for lexical decision, $F(1, 577) = 30.65, p < .001, \eta^2 = .05$, and semantic verification, $F(1, 577) = 15.71, p < .001, \eta^2 = .03$. Relative to the Young-Old, Old-Old adults exhibited marked increases in performance variability across the two waves for lexical decision ($YO_{wave1} = 6.23$; $YO_{wave2} = 6.54$; $OO_{wave1} = 8.18$; $OO_{wave2} = 9.87$) and semantic verification ($YO_{wave1} = 6.39$; $YO_{wave2} = 6.69$; $OO_{wave1} = 7.72$; $OO_{wave2} = 8.85$). Univariate tests of the Age Group x Cardiovascular Disease interaction were also significant for lexical decision, $F(3, 577) = 8.76, p < .001, \eta^2 = .04$, and semantic verification, $F(3, 577) = 7.44, p < .001, \eta^2 = .04$. Observed mean trends were identical to those reported for the Age Group x Cardiovascular Disease interaction for last wave of testing (see Figure 21, page 175). For the Young-Old, across-trial inconsistency for both verbal measures was relatively comparable across mortality status and cardiovascular disease. LSD post-hoc comparisons ($p < .01$) indicated that Young-Old decedents with CVD were: (a) significantly more variable than both survivor groups for lexical decision; and (b) significantly more variable than non-CVD Survivors for semantic verification. No additional pairwise comparisons were significant for the Young-Old. In conspicuous contrast, Old-Old group differences for lexical decision and semantic verification indicated all pairwise comparisons were significant except between the two survivor groups. Old-Old Decedents with CVD exhibited marked increases in performance inconsistency.

For the final omnibus two-way interaction, univariate tests of the Cardiovascular Disease x Wave interaction yielded significant differences for both lexical decision, $F(3, 577) = 5.00, p < .01, \eta^2 = .03$, and semantic verification, $F(3, 577) = 15.21, p < .001, \eta^2 =$

.07. Figure 22 displays cardiovascular group differences in verbal RT performance inconsistency over the 3-year longitudinal interval. Post-hoc LSD comparisons examined longitudinal change within each of the four cardiovascular groups as well as differences across groups within individual testing wave. For lexical decision, all four cardiovascular groups demonstrated accelerated increases in performance inconsistency over the 3-year period ($p < .05$ for non-CVD Survivors; $p < .01$ for the remaining groups). Similar significant increases in inconsistency were observed for semantic verification ($p < .01$) for all groups except the non-CVD Survivors. Figure 22 shows the magnitude of increased inconsistency for both verbal RT tasks was considerably higher for the two decedent groups. Contrasts within each testing wave for lexical decision revealed significant differences in inconsistency between all groups, except for survivors, for both the second last and last measurement waves. Decedents with CVD exhibited the most inconsistency followed by non-CVD decedents and finally the two survivor groups. Similar trends were observed within testing wave for semantic verification: (a) for the second last wave, Decedents with CVD were significantly more inconsistent than both survivor groups; and (b) for the last wave, significant differences in inconsistency were observed between all groups with CVD Decedents exhibiting the most inconsistency and non-CVD survivors showing the least. Mean ISD trends for the Cardiovascular Disease x Wave interaction suggest that Decedents exhibit earlier onset and greater magnitude increases in inconsistency relative to Survivors. Moreover, cardiovascular disease appeared to amplify these mortality differences, particularly for decedents. The significant Cardiovascular Group x Wave interaction was further modified by age group. A significant univariate Age Group x Cardiovascular Group x Wave interaction was



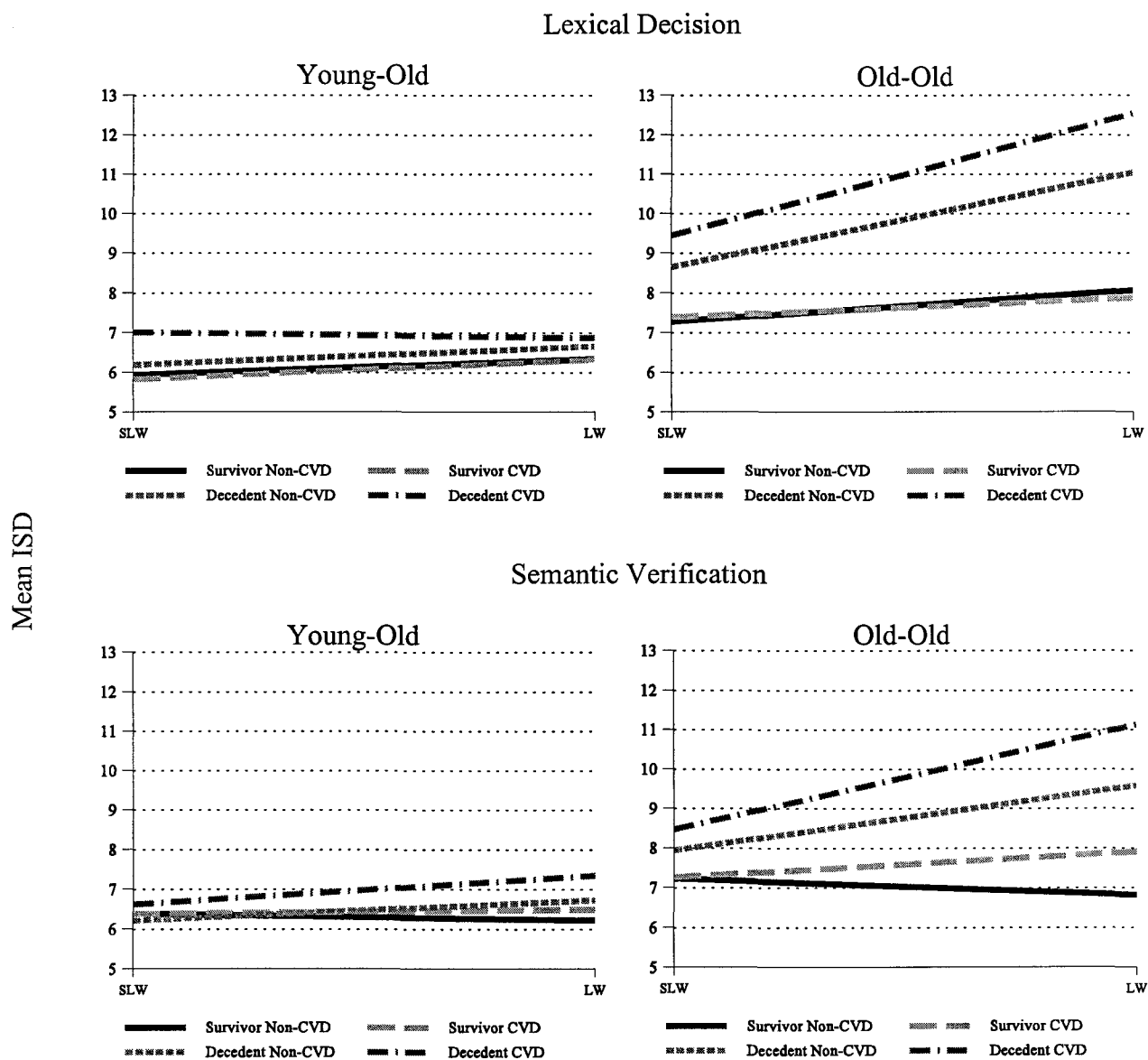
CVD = cardiovascular disease; SLW = second last wave; LW = last wave.

Figure 22

Cardiovascular Group Differences for 3-Year Change in Performance Inconsistency

observed for lexical decision, $F(3, 577) = 9.46$, $p < .001$, $\eta^2 = .05$, and semantic verification, $F(3, 577) = 4.32$, $p < .01$, $\eta^2 = .02$. Figure 23 outlines 3-year changes in RT inconsistency, separately for each age group, as a function of cardiovascular group differences. Pairwise LSD comparisons were used to decompose the interaction. For both verbal RT measures, changes in across-trial inconsistency for the Young-Old were relatively flat across waves for all cardiovascular groups. Both survivor groups showed modest increases in performance inconsistency ($p < .05$) for lexical decision with Young-Old Decedents with CVD exhibiting increases ($p < .05$) for semantic verification. No significant changes in inconsistency for Young-Old adults were observed for any other cardiovascular groups (see Figure 23). In contrast, marked cardiovascular group differences in rates of inconsistency change emerged for the Old-Old. Substantial increases in ISDs were observed for Old-Old Decedents with and without CVD. In comparison, only a single survivor group with CVD showed increasing inconsistency for the semantic verification task across waves.

Additional pairwise comparisons were computed to examine cardiovascular group differences within level of age group and measurement wave. For lexical decision, comparisons indicated that: (a) Decedents with CVD were more inconsistent than both survivor groups for Young-Old adults on the second last wave; (b) there were no cardiovascular differences for Young-Old adults on the last wave; (c) both Decedent groups were more variable than the survivor groups (with no significant differences within mortality groups) for Old-Old adults on the second last wave; and (d) all comparisons except those between survivors were significantly different for Old-Old adults on the last wave. Patterns for lexical decision indicated increasing inconsistency



CVD = cardiovascular disease; SLW = second last wave; LW = last wave.

Figure 23

Cardiovascular, Mortality, and Age Group Differences
for 3-Year Change in Performance Inconsistency

across waves, with increasing age, and as a function of impending death. Similar trends were observed for semantic verification. Within level of age group and wave, significant cardiovascular group differences were observed between: (a) no groups for Young-Old adults on the second last wave; (b) Decedents with CVD and the survivor groups for both Young-Old adults on the last wave and Old-Old adults on the second last wave; and (c) all groups for Old-Old adults on the last wave. Decedents with CVD showed the greater performance inconsistency with variability estimates increasing across waves.

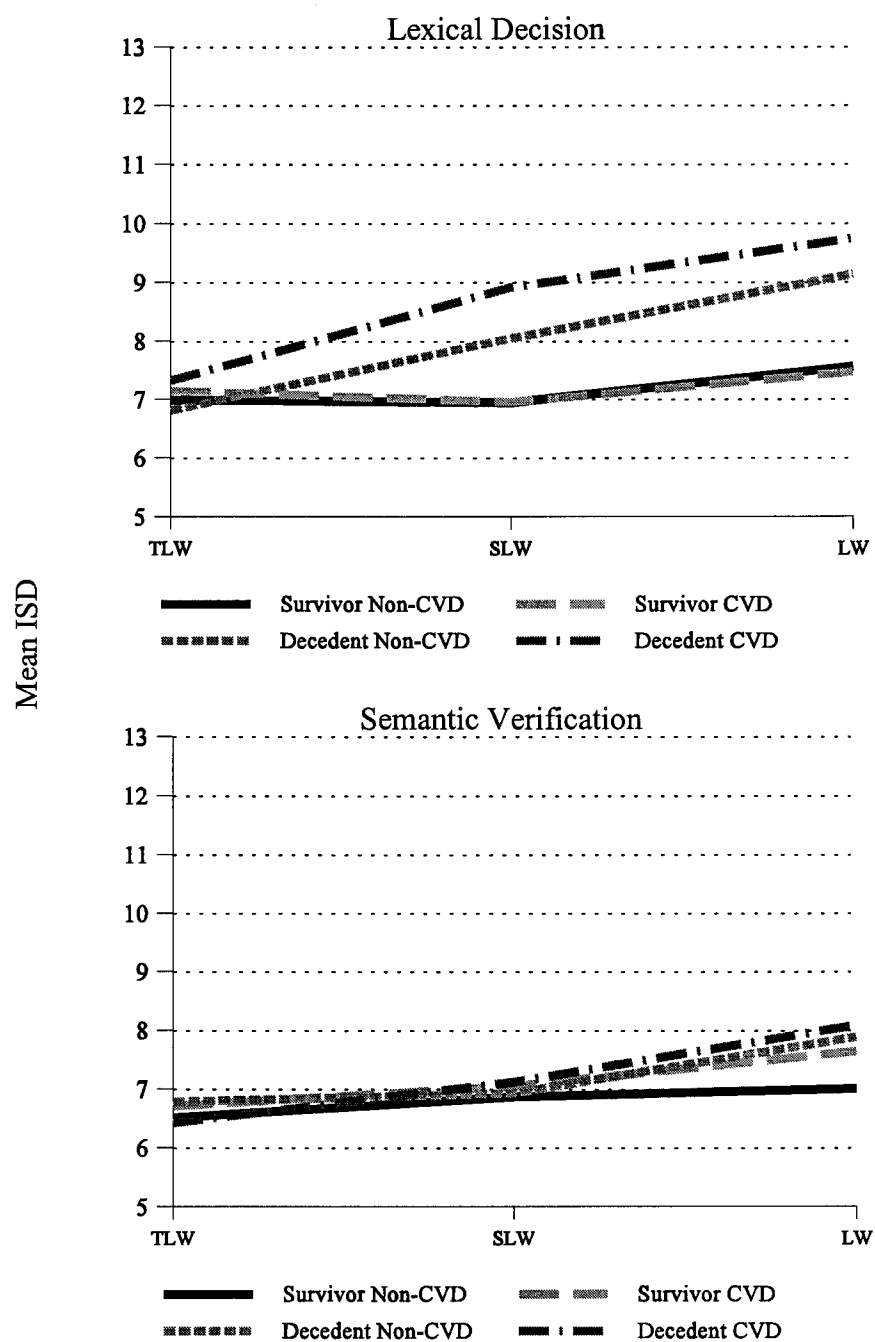
Complementing these two-wave analyses, 6-year change in inconsistency was examined using a 2 (Age Group) x 4 (Cardiovascular Disease) x 3 (Wave: third, second, and last respective testing occasions) repeated measures ANOVA (listwise $n = 518$). Significant omnibus effects were observed for: Age Group, Wilks' $\lambda = .884$, $F(2, 508) = 33.24$, $p < .001$, $\eta^2 = .12$; Cardiovascular Disease, Wilks' $\lambda = .945$, $F(6, 1016) = 4.89$, $p < .001$, $\eta^2 = .03$; Wave, Wilks' $\lambda = .972$, $F(4, 506) = 3.60$, $p < .01$, $\eta^2 = .03$; the Age Group x Wave interaction, Wilks' $\lambda = .925$, $F(4, 506) = 10.26$, $p < .001$, $\eta^2 = .08$; the Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .906$, $F(12, 1339) = 4.25$, $p < .001$, $\eta^2 = .03$; and the Age Group x Cardiovascular Disease x Wave interaction, Wilks' $\lambda = .953$, $F(12, 1339) = 2.04$, $p < .05$, $\eta^2 = .02$; and the Education covariate, Wilks' $\lambda = .984$, $F(2, 508) = 4.18$, $p < .05$, $\eta^2 = .02$. The omnibus Age Group x Cardiovascular interaction for the verbal speed measures approached significant ($p = .07$).

Significant univariate effects for both Age Group and Cardiovascular disease were consistent with those reported for the last wave of measurement analyses; they are briefly summarized here. For both lexical decision ($F(1, 509) = 66.37$, $p < .001$, $\eta^2 = .12$) and semantic verification ($F(1, 509) = 10.45$, $p < .01$, $\eta^2 = .02$), Old-Old adults were

significantly more variable across RT trials than the Young-Old. Cardiovascular group differences (as a function of mortality) for lexical decision ($F(3, 509) = 7.50, p < .001, \eta^2 = .04$) indicated that decedents with and without CVD did not significantly differ. Both decedent groups were more inconsistent than the survivor groups who did not differ. The univariate effect for semantic verification was not significant. Finally, the significant univariate Wave effects for lexical decision ($F(2, 1018) = 4.92, p < .01, \eta^2 = .01$) and semantic verification ($F(2, 1018) = 4.71, p < .01, \eta^2 = .01$) indicated that variability significantly increased over each interval of the 6-year period.

These main effects were further qualified by significant group interactions over the longitudinal interval. A significant univariate Age Group x Wave interaction was observed for lexical decision, $F(2, 1018) = 24.61, p < .001, \eta^2 = .05$. Relative to the Young-Old, Old-Old exhibited marked increases in performance variability across the 3 waves ($M = 6.58, 6.70, \text{ and } 6.87$ vs. $M = 7.54, 8.71, \text{ and } 10.08$ respectively). Albeit not significant, similar trends were observed for semantic verification.

Univariate tests of the Cardiovascular Disease x Wave interaction yielded significant differences for both lexical decision, $F(6, 1018) = 7.50, p < .001, \eta^2 = .04$, and semantic verification, $F(6, 1018) = 2.27, p < .05, \eta^2 = .01$. Figure 24 displays cardiovascular group differences in verbal RT performance inconsistency over the 6-year longitudinal interval. Post-hoc contrasts examined significant differences between waves of testing within each of the four cardiovascular groups as well as across groups within individual testing wave. For lexical decision latency, both decedent groups demonstrated accelerated increases in performance inconsistency following the third and second last testing waves. In contrast, the survivor groups did not exhibit increased inconsistency



CVD = cardiovascular disease; TLW = third last wave; SLW = second last wave; LW = last wave.

Figure 24

Cardiovascular Group Differences for 6-Year Change in Performance Inconsistency

until after the second last wave. As is evident in the figure, the magnitude of increased inconsistency was considerably higher for both decedent groups. Although no cardiovascular group differences were observed for Wave 1, decedents exhibited significantly more performance inconsistency than either survivor group for Waves 2 and 3. Similar, albeit attenuated, findings were observed for semantic verification.

Decedents with CVD demonstrated increased inconsistency for each additional wave of testing whereas non-CVD decedents only exhibited increases after the second last testing wave. A similar cardiovascular dissociation was observed for survivors (see Figure 23); those with CVD showed increased variability across both longitudinal intervals compared to non-CVD survivors who only exhibited increases between the third and second last waves. Contrasts within testing wave revealed no group differences in inconsistency for Waves 1 or 2, with significant Wave 3 increases observed for both decedent groups and survivors with CVD relative to non-CVD survivors. Consonant with the previous 3-year change analyses, decedents exhibited earlier onset and greater magnitude increases in inconsistency. Moreover, cardiovascular disease appears to exert a modest effect; both survivors and decedents with CVD showed trends for accelerated increases in inconsistency relative to their corresponding mortality status group.

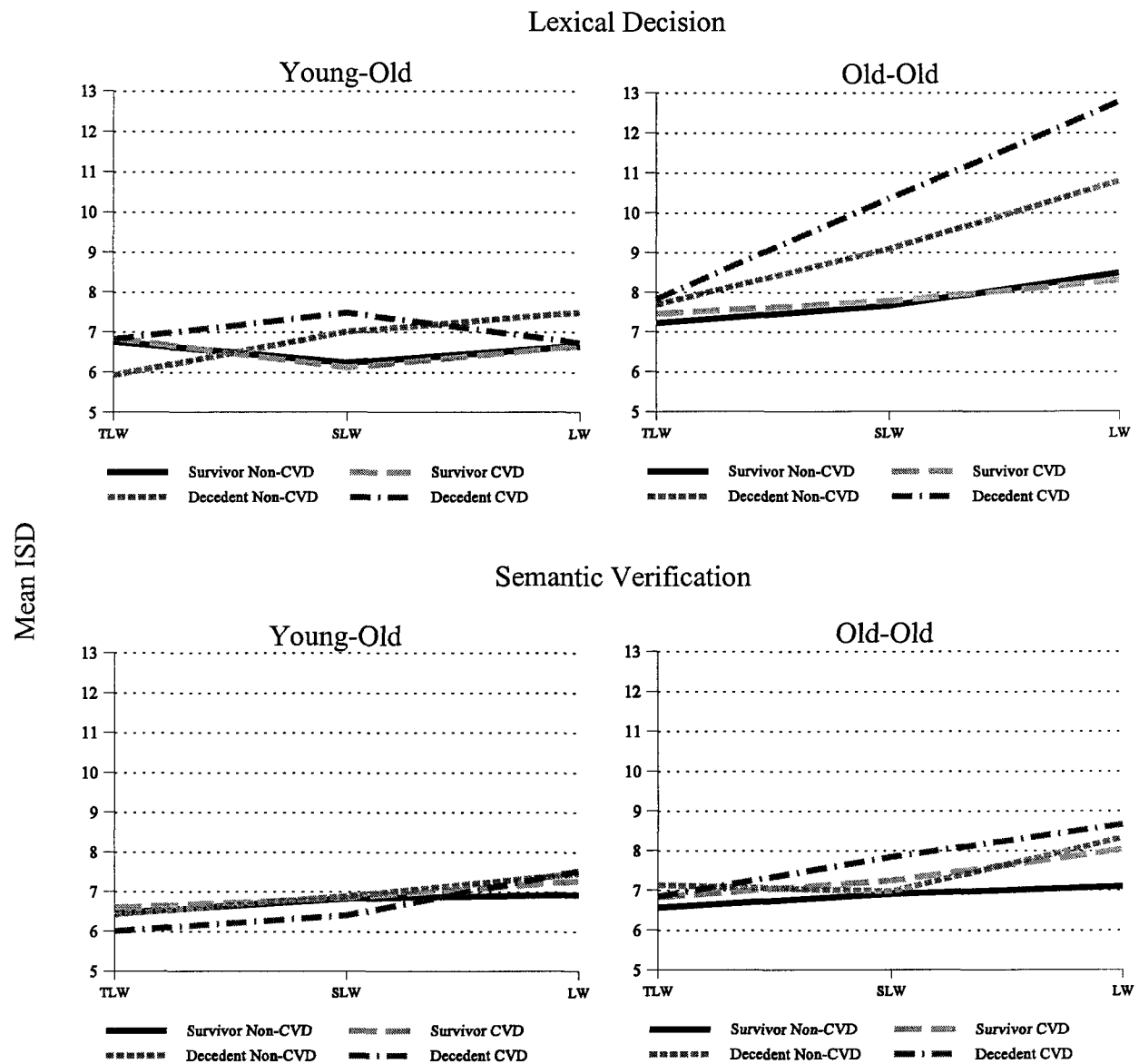
The significant Cardiovascular Group x Wave interaction was further modified by Age Group. A significant univariate Age Group x Cardiovascular Group x Wave interaction was observed for lexical decision, $F(6, 1018) = 3.86, p < .001, \eta^2 = .02$, but not semantic verification. Figure 25 outlines 6-year changes in RT inconsistency, separately for each age group, as a function of cardiovascular group differences. LSD post-hoc contrasts were used to probe the significant interaction for lexical decision.

Changes in across-trial inconsistency for the Young-Old were relatively comparable across mortality and cardiovascular groups. Both Young-Old survivor groups were less inconsistent between the third and second last waves of testing and then increased between the second last and last waves. Young-Old decedents without CVD exhibited increased performance inconsistency between the third and second last testing waves whereas decedents with CVD showed no significant increases. In contrast, findings across waves for the Old-Old indicated no significant changes in inconsistency for either survivor group, with marked increases in variability for decedents.

A second set of post-hoc contrasts examined cardiovascular group differences within level of age group and testing wave. Considering each wave separately, only one cardiovascular group difference emerged for Wave 2 (i.e., the second last wave); decedents with CVD exhibited greater inconsistency than survivors with CVD. In contrast, within-wave cardiovascular group comparisons for the Old-Old indicated: (a) no group differences for the third last wave; (b) significant increases in inconsistency for decedent relative to survivor status (but no differences within status) for the second last wave; and (c) significant increases in lexical RT variability for CVD decedents relative to all other groups for the last measurement wave (with non-CVD decedents also exhibiting increased inconsistency relative to both survivor groups). These patterns suggest that mortality, cardiovascular disease, and age interactively influence longitudinal change in across-trial performance inconsistency.

Inconsistency as Predictor of Survival Status

A separate set of analyses was computed to examine the association between performance inconsistency and mortality. Cox regression was used to examine whether



CVD = cardiovascular disease; TLW = third last wave; SLW = second last wave; LW = last wave.

Figure 25
Cardiovascular, Mortality, and Age Group Differences
for 6-Year Change in Performance Inconsistency

inconsistency predicted increased likelihood of mortality over the longitudinal interval spanning first wave of testing to: (a) death for decedents; or (b) the right censoring date of May 31, 2002 for survivors. The Cox analyses were structured to complement previously reported analyses examining cause of death and mean cognitive performance (see Table 12, page 138). Predictors in the present analysis included age and other demographic measures, cardiovascular conditions and medications, and performance inconsistency; all predictors reflected characteristics for the last wave of testing. Resulting individual regression coefficients represented associations between each covariate and the likelihood of dying over the longitudinal interval. As before, associated odds ratios were computed to provide the percentage difference in mortality risk per unit difference in covariate score.

Table 17 presents the Cox regression results predicting mortality status as a function of demographic variables, cardiovascular disease, and performance inconsistency across trials of the lexical decision and semantic verification tasks. Covariates were entered in three hierarchical blocks. Age, education, and self-reported health measures were entered on Block 1. Several cardiovascular disease measures were entered on Block 2 including CVD conditions (self-reported presence of heart disease, stroke, arteriosclerosis, and high blood pressure for any wave of testing ranging from 0 to 4) and CVD medications (self-reported use of heart, chest, high blood pressure, and anticoagulant medications ranging from 0 to 4). Block 3 incorporated last-wave performance inconsistency measures (separate models were computed for each predictor). The longitudinal interval for these analyses was calculated as the distance between the first wave of testing and death or censoring date (in contrast to the last wave interval used

Table 17
Cox Regression Analyses Predicting Mortality Status
as a Function of Age, Cardiovascular Disease, and Inconsistency (n = 707)

Covariates	β	Odds Ratio	95% CI	<i>p</i>
Block 1				
Age	-0.031	0.969	.948 - .990	.004
Education	-0.025	0.975	.940 - 1.012	.180
Relative health	0.473	1.606	1.257 - 2.050	.000
Absolute health	-0.990	0.372	.295 - .468	.000
Block 2				
CVD conditions	-0.007	0.993	.840 - 1.174	.934
CVD medications	0.167	1.181	1.003 - 1.392	.046
Block 3				
Semantic verification ISD	0.156	1.169	1.116 - 1.225	.000
Lexical decision ISD	0.097	1.102	1.067 - 1.138	.000

Mortality status was coded as either 0 (survivor) or 1 (decedent). CI = confidence interval; CVD = cardiovascular disease. On a 5-point scale (0 = very good to 4 = very poor), relative health reflects health relative to same-aged peers; absolute health reflects health relative to perfect. 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 reflects self-reported use of medications prescribed to treat cardiovascular symptoms and disease (heart, chest, high blood pressure, and anticoagulants). ISD = intraindividual standard deviation.

for the previous Cox regression analyses examining cause of death).

Findings summarized in Table 17 demonstrate that age, self-reported health, CVD medications, and performance inconsistency all share significant associations with mortality status. For the last wave of testing, increased chronological age was associated with decreased risk of mortality over the follow-up interval; the mortality risk decreased by 3.1% per year. In part, this unexpected finding likely reflects the advanced age and continued survival of participants who have completed as many as five measurement waves. Each unit increase in relative health, with larger values reflecting poorer health relative to same age peers, was associated with a 60.6% increased mortality risk. In contrast, each additional unit increase for self-reported absolute health was associated with a 62.8% decreased mortality risk. These dissociations may reflect inaccuracy of self-report. For Block 2, each additional medication (from 0 to 4) used to treat cardiovascular disease for any wave of testing was associated with an 18.1% increased mortality risk. Finally, increased inconsistency significantly predicted increased likelihood of death over the follow-up interval. For semantic verification, each additional ISD increase in performance inconsistency was associated with a 16.9% increased risk of mortality. The odds ratio for lexical decision indicated a 10.2% increased risk of mortality per unit increase in inconsistency scores.

These inconsistency findings are particularly interesting when compared with mean cognitive performance predictors reported in Table 12 (page 135). Although the longitudinal follow-up interval was different for the former analyses (distance between last wave of testing and death or censoring date), mean level performance did not significantly predict mortality for any of the cognitive measures (odds ratios were all

approximately 1 indicating no association between mortality risk and last wave performance). To provide a direct comparison with these former analyses, identical models using the same longitudinal interval were retested entering the two inconsistency predictors. Although the magnitude of the effects were slightly reduced due to reduction of the longitudinal interval, inconsistency for both semantic verification (odds ratio = 1.06, $p < .05$) and lexical decision (odds ratio = 1.035, $p < .01$) remained significant predictors of increased mortality risk. These findings are important because they: (a) demonstrate the link between inconsistency and mortality; and (b) provide evidence that inconsistency may be a more sensitive indicator of mortality risk than level of cognitive performance.

Multilevel Analysis of Change in Performance Inconsistency.

The ANCOVA and Cox regression analyses demonstrated significant associations between inconsistency and mortality. To more closely examine this association, multilevel analyses were computed to consider intraindividual associations between change in inconsistency and time (i.e., years) to death. Among other advantages, these analyses examine intraindividual change in inconsistency whereas corresponding repeated measures analyses considered only estimates of aggregate change. Moreover, HLM permits examination of time-varying covariation between change in inconsistency and proximity to death (analyses that have not been published elsewhere).

Change in inconsistency as a function of age and mortality status. For each measure of inconsistency, the Level 1 model (see equation 16) examined individual rates of change with increasing proximity to death across measurement occasions.

$$\text{Level 1: Inconsistency}_{it} = B_{0i} + B_{1i} * (\text{Time to Death}_{it}) + R_{it} \quad (16)$$

Inconsistency for a given individual (i) at a given time (t) was modeled as a function of that individual's performance at the centered midpoint on time to death (B_{0i} : the intercept), plus her/his average individual rate of change across waves with increasing proximity to death (B_{1i} : the slope), plus a random within-subject error term (R) representing prediction error holding intercept and slope constant. As for previous models, the Level 1 time to death predictor was grand-mean centered ($\bar{M} = 7.24$ years).

Associated Level 2 HLM equations were structured to examine between-group differences for change in inconsistency. Initial analyses examined change in inconsistency as a function of age and mortality group differences.

$$\begin{aligned} \text{Level 2: } B_{0i} = & \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Mortality Status}_i) \\ & + \gamma_{03} * (\text{Age Group} \times \text{Mortality Status}_i) + U_{0i} \end{aligned} \quad (17)$$

$$\begin{aligned} B_{1i} = & \gamma_{10} + \gamma_{11} * (\text{Age Group}_i) + \gamma_{12} * (\text{Mortality Status}_i) \\ & + \gamma_{13} * (\text{Age Group} \times \text{Mortality Status}_i) + U_{1i} \end{aligned} \quad (18)$$

Of most interest, equation 18 examines predicted linear rate of inconsistency change for each individual (B_{1i}) as a function of average change in inconsistency for Young-Old Survivors per year difference in proximity to death (γ_{10}), the average difference in change per year closer to death 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. Coefficients reflect ISDs

standardized as t-scores.

Table 18 summarizes multilevel findings associated with equations 16 to 18.

Taken together, the equations test whether performance inconsistency differentially changes with time to death or censoring date, age, or mortality status. Coefficients in the intercept column (γ_{00}) reflect mean ISDs for Young-Old Survivors at the sample midpoint for time to death/time remaining in the study. 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 death (see Table 18, slope γ_{10}).

The second slope (γ_{11}) examined age group differences in rates of inconsistency change. Consistent with expectations, performance inconsistency increased at a faster rate for Old-Old adults per year closer to death. For semantic verification, the γ_{11} slope coefficient indicated that the average difference between rates of change for the Young-Old and Old-Old was 0.06 ISD units per year. Over a 5-year period, performance inconsistency for Old-Old adults would increase by approximately 0.30 ISD units. Similarly, performance inconsistency also increased for the Old-Old relative to the Young-Old for lexical decision. The average difference between age groups was .22 units per year closer to death (a 1.1 ISD unit increase per 5 years closer to death).

The third slope coefficient (γ_{12}) assessed differences in rates of inconsistency change between survivors and those facing impending death. With increasing proximity to death, lexical decision inconsistency for decedents increased at significantly faster rates (a .29 ISD increase per additional year closer to death or 1.45 ISD units per 5 years). The slope for semantic verification was not significant but was in the expected direction.

Table 18

Change in Inconsistency as a Function of Time to Death, Age, and Mortality Status

Variable	Intercept γ_{00}	Slope $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t
Semantic verification	6.80	-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 A significant mortality x age group interaction was observed for this variable; associated mortality coefficients are reported separately for each age group in the text. γ_{00} = inconsistency for Young-Old Survivors at the sample midpoint for time remaining in the study ($M = 7.24$ years); γ_{10} = average rate of change in inconsistency per additional year of time remaining in the study for Young-Old Survivors; γ_{11} = average difference in rate of inconsistency change between Young-Old and Old-Old adults partialling the influence of mortality status; γ_{12} = average difference in rate of inconsistency change between Survivors and Decedents controlling for age group differences; SE = standard error; t = t-ratio.

* $p < .05$

** $p < .01$

Finally, random effect coefficients were examined for each corresponding intercept and slope. All were found to be significant ($p < .01$), indicating that, even after controlling for the predictors examined, significant between-group differences in intercepts and slopes remained to be explained.

A significant Age Group x Mortality Status interaction was observed for lexical decision (see Table 18). To decompose this interaction, it was necessary to calculate simple regression slopes examining the association between change in inconsistency, time to death, and mortality separately for each age group. For the Young-Old, significant differences in rates of inconsistency change were observed between mortality groups; Decedents exhibited an additional 0.16 ISD unit increase per year closer to death than Survivors ($t = -2.77$, $p < .01$). Consistent with expectations, results indicated that rates of inconsistency change per year closer to death were accelerated for Old-Old Decedents relative to Survivors. Performance inconsistency for Decedents increased by 0.40 ISD units (almost half a standard deviation) per year closer to death ($t = -4.33$, $p < .001$). Overall, these findings suggest that changes in performance inconsistency are amplified as a function of both age and mortality status.

Change in inconsistency as a function of age and cause of death. A separate multilevel analysis was computed for decedents alone to assess whether increases in performance inconsistency were related to cause of death (see equations 19 to 21).

$$\text{Level 1: Inconsistency}_{it} = B_{0i} + B_{1i} * (\text{Time to Death}_{it}) + R_{it} \quad (19)$$

$$\begin{aligned} \text{Level 2: } B_{0i} = & \gamma_{00} + \gamma_{01} * (\text{Age Group}_i) + \gamma_{02} * (\text{Cause of Death}_i) \\ & + \gamma_{03} * (\text{Age Group} \times \text{Cause of Death}_i) + U_{0i} \end{aligned} \quad (20)$$

$$B_{1i} = \gamma_{10} + \gamma_{11}*(\text{Age Group}_i) + \gamma_{12}*(\text{Cause of Death}_i) + \gamma_{13}*(\text{Age Group} \times \text{Cause of Death}_i) + U_{1i} \quad (21)$$

Table 19 summarizes multilevel findings associated with these equations. Of all analyses performed for the decedents, only two coefficients were significant. The first was the γ_{11} slope coefficient for lexical decision indicating that Old-Old adults exhibited greater inconsistency with increasing proximity to death (0.34 ISD units per year). The second coefficient was associated with the cause of death by age group interaction predictor. To further probe this interaction, separate regressions (examining associations between change in inconsistency, time to death, and mortality) were calculated for each age group. Results yielded no significant fixed effects associated with cause of death. The observed interaction was a function of differing associations between cause of death and performance inconsistency between age groups. For Young-Old Decedents, the cause of death slope was positive (0.16) indicating that those who died of CVD-related causes (relative to non-CVD causes) exhibited decreasing performance inconsistency per year closer to death. The opposite pattern was observed for Old-Old Decedents where performance inconsistency increased per year closer to death (0.26 ISD units per year) for those who died of CVD. Although cause of death was an important predictor for the last wave and aggregate change ANCOVA analyses, it did not emerge as a significant predictor of intraindividual change in inconsistency.

Summary: Performance Variability as a Function of Time to Death, Age, and Mortality

The primary focus of the fifth and final research question concerned associations between performance inconsistency (a potential indicator of neurological disturbance),

Table 19

Change in Inconsistency as a Function of Time to Death, Age, and Cause of Death

Variable	Intercept γ_{00}	Slope $\gamma_{10}/\gamma_{11}/\gamma_{12}$	SE	t
Semantic verification	6.73	-0.09	0.047	-1.84
		-0.03	0.060	-0.53
		-0.09	0.060	-1.53
Lexical decision ^a	5.75	-0.09	0.083	-1.13
		-0.34	0.105	-3.26**
		-0.07	0.105	-0.64

^a A significant cause of death x age group interaction was observed for this variable; associated cause of death coefficients are reported separately for each age group in the text. γ_{00} = inconsistency for Young-Old Decedents who did not die of cardiovascular disease (CVD) at the sample midpoint for time remaining in the study ($\bar{M}$ = 7.24 years); γ_{10} = average rate of change in inconsistency per additional year of time remaining in the study for Young-Old Decedents who did not die of CVD; γ_{11} = average difference in rate of inconsistency change between Young-Old and Old-Old adults partialling the influence of cause of death; γ_{12} = average difference in rate of inconsistency change between those who died of CVD vs. non-CVD causes controlling for age group differences; SE = standard error; t = t-ratio.

* $p < .05$

** $p < .01$

time to death, age, and mortality status. Consistent with expectations, Decedents exhibited greater performance inconsistency relative to survivors for the last wave of testing. Moreover, both cardiovascular disease and impending death interactively influenced across-trial performance inconsistency. Across 3- and 6-year intervals, Decedents with CVD exhibited systematically higher performance inconsistency with variability estimates increasing across waves relative to survivors. In general, longitudinal trends indicated that mortality, cardiovascular disease, and age interactively influenced change in performance inconsistency (with older Decedents exhibiting the greatest variability). Cox regression findings revealed that inconsistency for the last wave of testing significantly predicted increased likelihood of death. This finding is of particular interest in light of the fact that mean level cognitive performance did not significantly predict death. Finally, findings from multilevel analyses showed that performance inconsistency significantly increased per each additional year closer to death. Moreover, these increased rates of change for inconsistency were significantly higher for the Old-Old and for Decedents.

Chapter VI

DISCUSSION

The present investigation explored neglected and not well understood aspects of the terminal decline phenomenon. Primary foci were to replicate and extend previously observed mortality-cognition associations and to examine novel issues not previously addressed. Organized according to these two primary themes, this final chapter discusses the results and considers their implications. The first theme examines two terminal decline questions identified in Chapter 3 for which a fair amount of evidence exists: (a) is terminal decline evident across a broad age continuum? (b) do mortality-related deficits vary across cognitive domains? Results are compared with those already published and possible reasons for discrepancies are explored. The second theme explores the final three terminal decline questions from Chapter 3 for which there are few extant studies or answers: (a) does cause of death (CVD vs. non-CVD) differentially influence the magnitude of cognitive decline? (b) are performance declines accelerated for those in closer proximity to death (and are there group differences in the shape of decline)? and (c) is terminal decline related to performance inconsistency (an indicator of neurological compromise)? These novel findings represent new and interesting possibilities for examining the terminal decline phenomenon. The chapter concludes by addressing research limitations and considering directions for future research.

Replication and Extension of
Previous Terminal Decline Investigations

Associations between Age- and Mortality-Related Cognitive Deficits

The first research question of this investigation examined whether mortality-related deficits were similar across the latter part of the adult life span. Surprisingly, research on this facet of the terminal decline phenomenon remains inconclusive (Small & Bäckman, 1999). Evidence has been found in support of two views: (a) terminal decline effects are attenuated with age; and (b) terminal decline effects are magnified with age. Only a few investigations (e.g., Bosworth & Schaie, 1999; Bosworth et al., 1999) have examined the terminal decline phenomenon across the adult life span (34 to 93 years). Most recent studies have examined the phenomenon in participants aged 70 and older (e.g., Anstey et al., 2001; Johansson & Zarit, 1997; Small & Bäckman, 1997), and even for those 90 year and older (Maier & Smith, 1999). The present study supplemented this emerging evidence by examining terminal decline across middle and late adulthood (59 to 95 years at last wave of testing). Thorough consideration of the first research question was facilitated through examination of both cross-sectional age differences and longitudinal age change.

Overall, results from the analysis of cross-sectional differences, longitudinal aggregate change, and longitudinal within-person change converged on a similar story. Whether for last wave of testing or across multiple waves, Decedents performed more poorly than Survivors with these mortality differences magnified with increasing age. These findings are contrary to Riegel and Riegel's (1972) hypothesis that terminal decline

deficits are more pronounced for younger adults facing impending death.

Cross-sectional results analyzing VLS data showed that Survivors significantly outperformed Decedents for all nine cognitive measures (domains of verbal speed, working memory, episodic memory, semantic memory, and crystallized verbal ability). These findings examining mortality differences for the last wave of measurement mirror trends reported in a number of previous investigations. For example, Bosworth and Schaie (1999) found that Decedents had lower levels of crystallized ability, spatial ability, verbal memory, perceptual speed, and psychomotor speed. Similarly, Bosworth, Schaie, Willis, and Siegler (1999) reported lower levels of verbal meaning, spatial ability, reasoning ability, and psychomotor speed for Decedents relative to Survivors.

In direct contrast to Riegel and Riegel's (1972) prediction, observed mortality differences in the present investigation further increased with age for measures of verbal speed, semantic memory, and verbal ability with Old-Old Decedents exhibiting the poorest performance. Only one cognitive measure exhibited an effect size greater than $d = .5$ for the Young-Old whereas medium to large mortality differences were observed for 5 of 9 measures for the Old-Old. These more pronounced mortality-cognition associations with increasing age are consistent with previous findings. For example, similar survival status by age group interactions for the last wave of testing were reported by Bosworth and Schaie (1999). Decedents aged 75 years and older exhibited lower levels of crystallized ability and psychomotor speed relative to same-aged Survivors (with little difference observed between Survivors and Decedents for those younger than 75). Because increasing age is associated with increased likelihood of death, it stands to reason that mortality-related deficits should be magnified for the Old-Old.

In general, these cross-sectional findings underscore the sensitivity of last wave of measurement to terminal decline. As reported in previous investigations (e.g., Bosworth et al., 1999; Deeg, Hofman, & Van Zonneveld, 1990), one's level of performance at the conclusion of the study is more sensitive than baseline performance for examining terminal decline effects. Moreover, significant mortality status by age interactions suggest that crystallized verbal ability and semantic memory may be differentially sensitive to age. This finding is consistent with previous suggestions that cognitive abilities resistant to aging processes may share stronger relations with impending mortality (e.g., Bosworth & Schaie, 1999; Small & Bäckman, 1997; White & Cunningham, 1998). When cognitively-supported tasks such as vocabulary and world knowledge exhibit deficits, influences other than primary aging processes may be responsible including disease and impending death (e.g., Cooney et al., 1988). Although the verbal speed measures in the present investigation reflect process measures of cognition, observed mortality by age interactions could in part reflect emerging verbal deficits for Decedents. Following from this argument, one explanation why similar interactions were not observed for working and episodic memory tasks is that these measures are cognitively demanding (with little to no verbal support) and exhibit normative deficits at younger ages. Consequently, mortality differences were masked as both Survivors and Decedents already exhibited age-related declines for these measures (Cooney et al., 1988). The cognitive specificity of terminal decline is discussed further in the profile analysis summary.

For the repeated measures analyses, Decedents declined at faster rates than Survivors across the last occasions of measurement (3- and 6-year intervals) for verbal

speed, working memory, episodic memory, and semantic memory supporting the hypothesis that Decedents exhibited poorer cognitive performance and declined at a faster rate over time. Only vocabulary did not exhibit accelerated decline for Decedents across waves. Also consistent with the cross-sectional findings, mortality differences were further modified by age with Old-Old Decedents showing the greatest deterioration over time for verbal speed and verbal ability.

However, in contrast to present longitudinal findings, Bosworth and Schaie (1999) found only partial support for accelerated mortality-related decline across measurement waves. They reported that Old-Old Decedents' declined at faster rates than Survivors for measures of crystallized ability and psychomotor speed. One reason for this discrepancy may have been due to the number of measurement waves available for estimating change. For the Bosworth and Schaie (1999) investigation, only one measurement wave was available for 52% of total Decedents, thereby influencing observed rates of change in the study sample; they reported that attrition resulted in a more select group of Decedents and attenuated rates of change. In contrast, 45.7% of Decedents completed a single wave in the present investigation with all of these data points included in the multilevel analyses. A second possibility concerns differences in the time to death interval for each study. In the present investigation, mean years to death from baseline for Decedents was closer than the interval reported for Bosworth and Schaie ($M = 8.11$ years compared with $M = 14.6$ years). This difference reflects duration of retest interval within each study (3 years for the VLS compared with 7 years for the SLS), with the shorter interval potentially more sensitive to terminal decline.

Finally, the most stringent test of the association between terminal decline and age

was conducted using multilevel modeling. Relative to corresponding repeated measures MANCOVA analyses, HLM examined multiwave cognitive decline as a function of within-person (i.e., Level 1) increases in age. This investigation of the age-terminal decline relationship is unique and has not been reported elsewhere. For all measures except vocabulary, comparison of cognitive change slopes between mortality groups revealed significantly accelerated decline for Decedents relative to Survivors per additional year increase in age. Of particular importance, the significant interaction of slopes for the longitudinal multilevel analysis represents convincing support for the hypothesis that mortality differences vary as a function of age.

Multilevel analyses were likely more sensitive than the repeated measures approach (i.e., eight measures yielded significant age interactions compared with three) for several reasons including: (a) the examination of intraindividual as opposed to aggregate slopes; and (b) the inclusion of all available data for Decedents in the multilevel models. Indeed, a particular strength of such techniques is that all data points can be analyzed. The discrepancy between multilevel and repeated measures results is consistent with the argument that selective attrition influences terminal decline estimates. In contrast to cross-sectional studies that include more heterogeneous participants (including those very near death), repeated measures longitudinal studies often involve more select groups of returning participants (Hultsch et al., 1998; Schaie, 1996). Thus, listwise deletion employed in repeated measures analyses may underestimate terminal decline (particularly in relation to age). The present demonstration of age increases in terminal decline at the within-person level (as opposed to the between-person level or repeated-measures aggregate change level) represents a unique contribution to the

terminal decline literature. Future terminal decline investigations should examine participant attrition or use analytic techniques that circumvent listwise deletion over longitudinal intervals.

In summary, considering all analyses together, several additional possibilities may account for observed age-mortality discrepancies between present and previous findings. First, relative to Riegel and Riegel (1972), the present study considered a broader age range with more participants aged 75 years and older (thereby increasing power to detect mortality differences in the Old-Old). Significant mortality differences were observed between Old-Old Survivors and Decedents despite the fact that both groups had experienced more cognitive deterioration relative to Young-Old participants. Of particular importance, observed patterns were not solely a function of age-related differences; impending mortality clearly exerted an additive effect over and above that of age. One plausible explanation for this interaction is that magnified mortality-related deficits reflect protracted chronic disease processes for Old-Old Decedents. This is consistent with previous arguments citing the higher prevalence of chronic diseases among older adults (Cooney et al., 1998) and the greater likelihood that Old-Old participants (particularly those with chronic disease) were in a terminal decline phase during their last measurement wave. Supporting this notion, Bosworth (1996) found that removing individuals within 2 years of death (with arguably more acute chronic conditions) resulted in higher mean levels of cognitive performance. Potential interactions between impending death, age, and cardiovascular disease/cause of death are discussed in a later section.

A second reason for the discrepancy between studies reporting age-related

increases in terminal decline and the findings of Riegel and Riegel (1972) concerns the diversity and characteristics of cognitive measures assessed. Studies demonstrating mortality-related deficits well into old age (e.g., Anstey et al., 2001; Bosworth et al., 1999; Johansson & Zarit, 1997; Maier & Smith, 1999; Small & Bäckman, 1997) have employed multiple measures reflecting diverse cognitive domains (including both fluid and crystallized abilities). In contrast, Riegel and Riegel used five verbal achievement measures indexing the crystallized ability domain (synonyms, antonyms, selections, classification, and analogies). Given the importance of this topic, the second research objective of this investigation examined cognitive specificity of terminal decline.

Cognitive Specificity of Terminal Decline

A second question from the terminal decline literature concerns whether mortality-related deficits are uniform across cognitive domains (e.g., Berg, 1996; Small & Bäckman, 1999). Extant evidence regarding the global relative to specific nature of terminal decline is mixed. For example, some previous studies (e.g., Birren, 1968; Bosworth & Schaie, 1999; Small & Bäckman, 1997; White & Cunningham, 1988) have reported more pronounced mortality effects for abilities typically resistant to aging (e.g., crystallized abilities, cognitively supported memory tasks such as recognition vs. recall, etc.) as compared with tasks that show earlier onset age-related decrements (e.g., processing speed, fluid abilities). The argument is that performance decrements for typically age-resistant tasks reflect impending pathology (particularly for the Young-Old) as opposed to normative age-related differences in functioning (Small & Bäckman, 1997). In contrast, other studies have reported mortality effects for process measures alone (e.g., Maier & Smith, 1999) as well as for both process and product measures

(Anstey et al., 2001). In opposition to the cognitive support argument, these findings are consistent with the notion that tasks with increasing cognitive demands are more sensitive for detecting terminal decline.

Regardless of view, empirical support requires indexing performance for an assortment of cognitive domains. Surprisingly, many studies examining this issue have employed a limited number and diversity of cognitive measures (e.g., Riegel & Riegel, 1972), thereby restricting conclusions about the specificity of mortality-related cognitive deficits. Only recently have investigations included a diverse mix of fluid and crystallized tasks (e.g., Anstey et al., 2001; Bosworth et al., 1999; Bosworth & Schaie, 1999). Complementing these studies, the VLS also includes diverse measures spanning a broad age range. As advocated by Small and Bäckman (1999), the present study incorporated multiple tasks indexing different cognitive domains and exhibiting considerable variability in terms of the magnitude of normal age-related decrements typically observed. To assess whether impending mortality differentially influenced performance across various domains, cross-sectional differences and longitudinal changes in cognitive profiles were examined as a function of mortality status and age group. Profiles were ordered according to a process-to-products continuum of domains spanning verbal speed, working memory, episodic memory, semantic memory, and verbal ability.

To statistically test for mean differences between cognitive measures (i.e., across the nine-measure continuum), the second objective employed profile analysis. Profile analysis treats the nine cognitive tasks as a within-subjects factor and inferentially tests differences between cognitive measures within mortality group (as well as the conventional between mortality groups within cognitive task). Such statistical

comparisons extend previous analyses that have focused on comparing patterns of significance across independent tests of mortality group differences. Similar findings have not been reported elsewhere and represent a novel contribution to the terminal decline literature.

Results from the profile analysis for the last wave of testing indicated that mortality-related deficits were present across all cognitive domains except verbal ability. Profiles significantly differed by mortality status and age group with Old-Old Decedents demonstrating the poorest absolute performance as well as the largest variation across the continuum of cognitive tasks (i.e., more profile variability). Notably, Decedents' mean vocabulary performance was higher than their performance for all other cognitive indicators; verbal speed and episodic memory means were significantly below those of working memory. In contrast, for both Young-Old and Old-Old Survivor groups, profiles were relatively flat reflecting similar performance regardless of task demands. These findings suggest that performance differences between cognitive measures are amplified for Decedents. Specifically, on the same measurement metric, more demanding process and memory measures show greater deficits relative to crystallized verbal ability.

Longitudinal results established that both Survivors and Decedents declined over the 3-year interval on measures of verbal speed, working memory, episodic memory, semantic memory, and verbal ability. However, Survivors demonstrated little deviation in rates of proportional decline across all cognitive measures in the profile. In contrast, Decedents' 3-year declines were magnified for verbal speed and episodic memory with terminal decline effects narrowing toward the product end of the continuum. Consistent with the profile analyses for last wave of testing, more demanding process and memory

measures once again showed greater deficits and accelerated decline relative to crystallized verbal ability.

Overall, results from these profile analyses are inconsistent with previous suggestions that terminal decline deficits are magnified for cognitive abilities resistant to aging processes. Several studies have reported magnified mortality-related deficits for crystallized cognitive abilities (e.g., Bosworth & Schaie, 1999; Riegel & Riegel, 1972; White & Cunningham, 1988). For example, Reigel and Reigel (1972) reported greater mortality deficits for Young-Old Decedents relative to Old-Old Decedents on verbal ability measures suggesting differential influences of impending death as a function of age and cognitive domain. Similarly, Bosworth and Schaie (1999) reported that survival effects are age-specific and ability-specific. At cross-section, Survivors outperformed Decedents for both fluid and crystallized measures but consistent 7- and 14-year mortality differences in decline were observed for crystallized measures of verbal meaning alone.

The opposite pattern was observed for the present investigation where Old-Old Decedents exhibited their best performance for vocabulary (at last wave of measurement and over the 3-year interval) relative to the other cognitive domains. Cognitively demanding abilities including verbal speed, working memory, and episodic memory showed lower levels and greater declines relative to vocabulary. However, these patterns of mortality-related deficits for more cognitively demanding measures are not novel. For example, Botwinick and colleagues (1978) reported larger mortality differences for perceptual and psychomotor tasks. More recent studies have also reported terminal decline effects across a continuum of measures including verbal abilities, memory, and processing speed (e.g. Anstey et al., 2001; Small et al., in press).

Thus, the obvious question concerns what accounts for observed discrepancies in the terminal decline literature as a function of cognitive domain. There are several likely possibilities for divergence between the present results (showing mortality effects for process measures) and previously reported findings (demonstrating mortality effects for crystallized measures). The first explanation focuses on sample characteristics. Present findings showing greater decline for more cognitively-demanding tasks may be a consequence of sample selection. The VLS sample is a convenience sample with above average health, years of education, socioeconomic status, and cognitive performance relative to the Canadian population (Hultsch et al., 1998). Previous studies suggest that no clear mortality differences are observed for process or fluid measures because both Survivors and Decedents have experienced age-related declines on these tasks (e.g., Bosworth & Schaie, 1999; White & Cunningham, 1988). The argument is that changes in these process measures are more a function of normal aging than terminal drop.

In relative comparison, older adults in the VLS sample may be healthier than those in other studies. Consequently, mortality declines are still apparent for process measures with less pronounced differences for world knowledge and crystallized abilities. In contrast, more diverse samples may have already shown decline on process measures with mortality differences most pronounced for crystallized measures (among the last cognitive abilities to show mean differences and decline). Although crystallized abilities are typically resistant to age-related change, less healthy samples may show earlier deterioration for multiple reasons (e.g., greater proportion of sample with acute chronic illness). In effect, the relative health of the sample would affect which measures show terminal decline and for which age range mortality differences emerge.

A second possibility for observed differences in cognitive specificity of terminal decline relates to the nature of the measures used. Independent of sample differences, task demands represent a plausible explanation for the dissociation between Decedents' rates of change across the nine measures. Crystallized measures of verbal ability and world knowledge reflect accumulated experiences across the life span. That these abilities were among the last to decline for Old-Old Decedents in the present investigation is an intuitive pattern consonant with reported age-related memory differences in the cognitive aging literature (e.g., Hultsch et al., 1998; Schaie, 1996). Crystallized (e.g., vocabulary) and cognitively-supported tasks (e.g., recognition compared with recall memory) are more resistant to age-related change as compared with fluid-process tasks. These classic patterns of age differences appear to generalize to observed mortality differences in the present study; impending mortality resulted in poorer cognitive performance for process compared with product measures of cognition. Moreover, with increasing age and over time, mortality differences were magnified with greater variation observed across cognitive profiles for Decedents relative to Survivors. Notably, restricted scale range represents one potential reason why the working memory tasks did not show terminal decline effects statistically comparable to those of verbal speed and episodic memory.

The issue of sample differences in health also shares a direct association with varying task demands. Specifically, the relative health of the VLS sample may have reduced observed mortality differences for crystallized tasks compared with previous investigations. Indeed, consistent with this argument, it has been proposed that poor health mediates cognition with magnified deficits observed for abilities that are more

sensitive to aging processes including process and fluid measures (Christensen, Jorm, Henderson, Mackinnon, Korten, & Scott, 1994). This explanation would predict larger mortality deficits for crystallized abilities for follow-up waves of the VLS sample (as process measures have already declined).

A third possibility for cognitive-specificity discrepancies concerns differences in research design and statistical techniques employed. In addition to influencing observed age differences in terminal decline, the number of measurement waves and length of retest interval may also influence which cognitive measures exhibit mortality deficits. Present results were based on as many as five measurement waves with shorter retest intervals. The length of longitudinal retest interval likely shares an important association with magnitude of terminal decline effects. Although terminal decline has been demonstrated as much as 14-years prior to death (Bosworth & Schaie, 1999), both theory (e.g., Palmore & Cleveland, 1976) and empirical evidence (e.g., Berg, 1987) suggest the acceleration of terminal decline (i.e., terminal drop) with increasing proximity to death. Consequently, as shorter retest intervals are more likely to sample participants in a terminal decline phase, they may be more sensitive to mortality-related cognitive deficits. Consistent with present findings and the argument that more demanding cognitive tasks show increased deficits for older adults and those facing impending death, proximity to death may also magnify declines for process tasks.

As reported by Small and Bäckman (1999), specific statistical techniques may also be responsible for observed differences in the literature. Previous investigations have not employed profile analysis to directly compare performance across cognitive domains. Present findings indicated significant differences across cognitive profiles for

Decedents but not Survivors. Analogous to clinical interpretation of standardized Wechsler Adult Intelligence Scale (WAIS) profiles, increasing diversity in cognitive profiles may prove useful for differentiating those at increased risk for death. Similarly, results from the multilevel analyses demonstrated terminal decline at the intraindividual level for all available data. Consistent with the profile analysis patterns, uniform increases in terminal decline as a function of age were observed for all cognitive domains except crystallized verbal ability. For every year of age above the sample average, Decedents declined at significantly faster rates than Survivors. Although these findings suggest ability-specific terminal decline for process measures, null results for the crystallized ability measure may reflect above-average sample characteristics. Future studies should employ multilevel analyses to examine whether similar intraindividual patterns are observed across cognitive domains for less select samples.

Overall, first theme findings from this study (i.e., replication and extension of previous terminal decline investigations) support existing reports that suggest mortality-related deficits are magnified (as opposed to attenuated) with increasing age. Both cross-sectional and longitudinal results were quite consistent in this regard. In contrast, it remains unclear whether terminal decline is ability specific. Support for a strong version of the terminal decline hypothesis would require that impending mortality exert a pervasive (as opposed to specific) influence on all cognitive abilities. Although mortality deficits were consistently observed for process measures including verbal speed, working memory, and episodic memory, terminal decline was not observed for typically age-resistant cognitive tasks. However, as noted, the above average health of the VLS sample and the relative few crystallized ability and world knowledge measures may have

influenced observed patterns. In support of this argument, non-significant patterns demonstrated that impending death influenced well-preserved cognitive abilities but exerted greater impact on more cognitively demanding tasks. Indeed, trends observed in the multilevel analyses suggest that terminal decline influences all cognitive abilities.

Novel Contributions to The Terminal Decline Literature

This section focuses on the remaining three research questions for which little evidence is available. Terminal decline is considered in relation to cause of death and chronic disease, years to death, and performance inconsistency. Only a single investigation has directly examined cause of death as a possible source of terminal decline and findings have not been replicated. No published studies have examined time-varying covariation between longitudinal decline and number of years to death. Finally, terminal decline has not been examined in relation to other potentially interesting correlates such as markers of neurological disturbance (e.g., performance inconsistency).

Cause of Death and Terminal Decline

A third unresolved issue garnering little research focus in the terminal decline literature concerns whether specific chronic diseases or causes of death influence cognitive performance (Berg, 1996; Small & Bäckman, 1999). An obvious potential exists for diseases to be differentially present in Decedents relative to Survivors. Moreover, links between causes of death, cognitive functioning, and mortality are intuitive. For example, cardiovascular disease is of particular interest given its association with cognitive performance (e.g., Fahlander et al., 2000) and relationship to mortality risk (Haan et al., 1999). It follows that CVD-related causes of death should influence magnitude of mortality-related decline. However, based on existing empirical evidence, it is unclear whether observed deficits reflect specific disease processes or a more general biological deterioration (Berg, 1996).

Perhaps surprisingly, only a single investigation has directly examined

associations between cause of death (as a between-subjects factor) and magnitude of mortality-related cognitive deficits (Small et al., in press). Results indicated that magnitude of cognitive deficits did not vary as a function of CVD compared with non-CVD causes of death. These findings imply that mortality-related cognitive deficits are not disease specific. However, indirect evidence has been found in support of associations between specific causes of mortality and terminal decline. Anstey and colleagues (2001) reported significant attenuation of cognition-mortality associations after controlling for measures of self-reported disease and health implying a disease-specific role. In a separate study, controlling for cerebrovascular and cardiovascular indicators resulted in fewer cognitive measures significantly predicting mortality status (Hässelting et al., 2002). Thus, to further examine this third unresolved issue, cross-sectional differences and longitudinal changes in cognition were examined as a function of cause of death, age, and proximity to death.

Consistent with expectations, cross-sectional results for Decedents alone ($n = 265$) indicated that specific cause of death differentially influenced cognitive performance. Those who died of CVD-related causes exhibited poorer mean cognitive performance. Magnitude of cause of death effects were more pronounced for cognitively demanding tasks including verbal speed, working memory, and episodic memory. Complementing the previous profile analyses, the combination of CVD and increased cognitive demands resulted in deteriorating performance. However, cause of death did not interact with age; CVD was an important predictor of mortality-related cognitive deficits regardless of age. This finding does not support the hypothesis that death occurs more randomly for older compared to younger adults (e.g., Riegel & Riegel, 1972) or that disease-related terminal

decline may be more normative for the Young-Old (Berg, 1996). More random causes of death with increasing age should have resulted in diminished differences between Survivors and Decedents.

Proximity to death was also examined for the Decedents-only analysis. Word recall was the lone task to exhibit a significant cause of death by proximity to death interaction. Those who died of CVD exhibited poorer performance within 3 years of death. However, contrary to expectations, patterns for the remaining measures indicated that those who died of CVD and were closer to death (i.e., died within 3 years of their last wave of testing) did not exhibit markedly poorer cognitive performance. Similarly, corresponding 3-year longitudinal analyses for Decedents did not show magnified cognitive declines for those who died of CVD (although means were in the expected direction). These findings suggest that associations between cause of death and terminal decline are not further modified by proximity to death. However, the imprecision of the categorical proximity to death variable in relation to each individual's terminal decline phase may partly account for these findings. More precise analyses are discussed in the next section summarizing the shape of terminal decline.

Complementing the Decedent-only results, additional analyses compared Decedents with and without CVD to corresponding Survivor control groups. Cross-sectional results showed that magnitude of terminal decline increased as a function of age and cardiovascular disease. Across the 3- and 6-year intervals, Decedents who died of CVD exhibited accelerated cognitive declines relative to the other three groups. These trends were further modified by age group with Old-Old Decedents showing the earliest declines. CVD was not systematically associated with poorer cognitive performance for

Survivors. This finding is likely a consequence of the self-report coding for Survivors compared with the objective ICD-9 coding used for Decedents. Finally, results from the Cox regressions revealed that mortality risk shared significant associations with CVD (number of medications) but was unrelated to level of cognitive performance for the last wave of testing.

Considered together, these findings are the first to demonstrate systematic associations between cause of death and terminal decline. Notably, observed patterns are inconsistent with Berg's (1996) biological vitality hypothesis. Magnitude of terminal decline was differentially influenced by cause of death, reflecting the impact of specific disease processes relative to global compromised biological vitality. Support for a global influence account would have resulted in no significant differences between CVD and non-CVD causes of death. The disease specificity of terminal decline is consistent with previous findings suggesting that compromised health further reduces life expectancy (e.g., Christensen et al. 1994). As the present results demonstrated, the health-life expectancy association implies that mortality deficits for those with specific disease (e.g., CVD) should be more pronounced. Moreover, the differential influence of impending death and CVD on performance deficits for process measures (e.g., verbal speed, working memory, and episodic memory) is also compatible with the proposal that poorer health is associated with magnified deterioration for abilities more sensitive to aging processes (Christensen et al., 1994).

Generalized vascular disturbance, including compromised brain oxygenation and associated effects on the central nervous system, reflect one possible mechanism influencing the magnitude of terminal decline (e.g., Fahlander et al., 2000). Restricted

blood flow and oxygenation for those with CVD has both intuitive and documented influences on cognitive performance. Results from this study show that the presence of both CVD and impending death magnifies rates of terminal decline relative to non-CVD Decedents and Survivors. The distinction between terminal decline patterns for CVD and non-CVD Decedents is an important one. It underscores that mortality differences are further modified by cardiovascular disease.

Whereas Small and colleagues (in press) reported cause of death did little to modify mortality differences, CVD-related causes were consistently associated with compromised cognitive performance in the present investigation. Results likely differ for several reasons. First, the current study examined associations across a broader age range (beginning at 55 years) compared to the very old sample (75 to 95 years) examined in the Small et al (in press) investigation. It could be that causes of death for the oldest-old are more random, thereby attenuating any differential influences of specific causes.

Although results from this study do not support this interpretation, diminished brain reserve capacity for those above a certain age may result in cognitive deterioration regardless of cause of death. A second possibility is that the VLS sample includes a larger number of individuals who died of acute causes of death often associated with CVD (e.g., heart attack, stroke). This would serve to magnify mortality differences as a function of cause of death.

Neither the present nor several previous (e.g., Small & Bäckman, 1997) Cox regression analyses have found mean level of cognitive performance to predict mortality status. This finding is unexpected given the significantly poorer mean performance exhibited by Decedents relative to Survivors. In contrast, other investigations have

identified significant associations between cognitive performance and mortality in Cox models (e.g., Anstey et al., 2001; Bosworth, Schaie & Willis, 1999; Hässing et al., 2002). There are several possible reasons for these discrepant findings. The most obvious concerns sample size and available power. Whereas the present findings were based on a sample size of 707 (with 265 Decedents), Bosworth and colleagues (1999) examined 1210 participants with as many as 1947 participants included in other studies (Anstey et al., 2001). Moreover, studies documenting significant cognition-mortality associations using Cox regression have reported that cognitive change is a better predictor than level of performance (e.g., Anstey et al., 2001; Bosworth et al., 1999). Indeed, as suggested by Small and Bäckman (1999), some unresolved issues in the terminal decline literature may be a function of analytic technique employed. Nonetheless, consistent with other recent findings (e.g., Hässing et al., 2002), present results did underscore the importance of CVD conditions as predictors of mortality in the Cox model. Those who reported taking more CVD drugs for any given wave of testing were at increased risk of dying. Moreover, this was a conservative test given the above-average health of the VLS sample, the self-report data used, and the relatively mild (as compared with severe) nature of the conditions reported.

Overall, present findings are among the first to provide direct evidence for the disease specificity of terminal decline. Surprisingly, few studies have focused on potential mechanisms underlying associations between cognition and mortality (Small & Bäckman, 1999). Results from the present investigation suggest that specific disease processes represent an important avenue of future investigation. Replication of these findings is necessary to address whether terminal decline is a function of specific disease

processes or a more pervasive biological system breakdown due to basic aging processes (Berg, 1996). In fact, results from a study by Anstey and colleagues (2001) suggest that potential mechanisms underlying terminal decline may reflect both specific disease processes and general biological aging processes. After partialling self-reported health and disease variables, a number of cognitive measures no longer predicted mortality implying observed associations were due to specific disease processes. However, significant associations for other cognitive measures remained, perhaps reflecting general biological aging. Given the importance of general biological aging as a predictor of cognitive decline (e.g., Anstey & Smith, 1999; MacDonald, Dixon, Cohen, & Hazlitt, 2002), future research should examine associations between select biomarkers (e.g., sensory acuity, physiological function) and impending mortality. One distinct possibility is that compromised biological health is associated with increased mortality risk, with a further amplification of this risk due to specific disease processes such as CVD. Finally, the demonstration of magnified cognitive deficits for those who died of CVD-related causes underscores the importance of examining sample attrition. Although robust, the present findings likely underestimate actual magnitude of terminal decline as those who drop out of studies have proportionally more chronic diseases (Anstey et al. 2001; Bosworth & Schaie, 1999; Cooney et al., 1988).

Shape of Terminal Decline

A fourth unresolved issue in the terminal decline literature pertains to the rate and shape of terminal decline. Some evidence suggests that those closer to death show accelerated cognitive declines (e.g., Bosworth & Schaie, 1999; Bosworth et al., 1999; Small & Bäckman, 1997). Although terminal decline and terminal drop have been

introduced to refer to linear and accelerated curvilinear change respectively (Palmore & Cleveland, 1976), few investigations have directly examined this issue using longitudinal data (see Berg, 1996). For those studies that have (e.g., Berg, 1987), inclusion of few measurement occasions, long test-retest intervals, and use of aggregate change techniques may all reduce precision for identifying terminal drop. Thus, it remains unclear whether patterns of cognitive performance are best characterized by steady decline or more precipitous drop as death nears. The present investigation examined the shape of terminal decline as a function of years to death. Multilevel analysis of across-time covariation between cognitive decline and years to death has not been examined previously. The defining question asked whether individuals closer to death showed accelerated cognitive decline consistent with the definition of terminal drop.

Multilevel results showed that the shape of cognitive decline varied as a function of years to death, age, and mortality status. Older adults declined at significantly faster rates per additional year closer to death (or time remaining in the study for Survivors). This result is inconsistent with the suggestion that young elderly may actually show the greatest declines due to random causes of death (e.g., Berg, 1996; Riegel & Riegel, 1972). Of greater import, significant differences in rates of change were observed between mortality groups with increasing proximity to death. As hypothesized, decline was accelerated for Decedents. Also consistent with a terminal drop pattern, significant mortality differences in slopes were magnified for the Old-Old. Finally, for Decedents alone, evidence showed that those who died of CVD-related causes declined more on several measures per additional year closer to death. The contrast between this finding and the one reported in the previous section likely reflects the imprecision of the

categorical proximity to death variable.

To more precisely characterize terminal decline compared with terminal drop, additional nonlinear multilevel analyses were computed. Model fit was significantly improved incorporating either polynomial or piecewise change growth terms. Significant quadratic trends indicated an acceleration of cognitive decline in closer proximity to death, particularly for Decedents. Consistent with a terminal drop trajectory, piecewise regression findings showed: (a) greater linear decline for Decedents and Old-Old adults; and (b) significant acceleration of decline in closer proximity to death for Decedents. Accelerated decline was particularly pronounced after the second wave of testing, foreshadowing the large proportion of the Decedent sample who would die within the following 3-year interval (i.e., prior to Wave 3 testing).

Overall, these results support the terminal decline/terminal drop distinction proposed by Palmore and Cleveland (1976). As hypothesized, results yielded an interaction of mortality status by time to death. Independent of age group, steeper rates of cognitive decline were observed for Decedents relative to Survivors per additional year closer to death. Using a novel statistical approach, these results are compatible with previous findings (e.g., Berg, 1987; Bosworth & Schaie, 1999; Small & Bäckman, 1997) and make a unique contribution to the terminal decline literature by revealing within-person patterns of terminal drop. Relative to previous investigations, examining intraindividual change permitted greater statistical precision for examining whether individuals closer to death showed accelerated cognitive decline. Moreover, trends were relatively consistent across the continuum of cognitive measures.

As one plausible explanation, risk factors predisposing one to mortality may

shorten an individual's life span with terminal drop manifested for an interval immediately preceding death. Either specific diseases or more general compromised biological aging may precipitate such accelerated declines (e.g., Anstey et al., 2001), with more risk factors (e.g., age, health) increasing mortality risk. For example, terminal drop patterns may reflect CNS dysfunction and compromised brain reserve where, beyond a critical threshold, a terminal cascade effect is observed for cognitive performance. Indeed, Berg (1996) has suggested that compromised biological vitality due to basic aging processes may result in decreased reserve capacity and failure of homeostatic systems. Similarly, present findings are consistent with those of Bosworth and colleagues (1999) who found that distance to death uniquely explained variance for 5 of 6 intellectual performance measures. They employed Birren and Cunningham's (1985) Cascade Model to explain cognitive deterioration in relation to distance to death. Greater cognitive deterioration on process measures as a function of cause of death is consistent with the secondary aging (i.e., disease-related) facet of the Cascade Model. However, at odds with this explanation, cause of death was not related to accelerated decline for all process measures (although null findings were likely a function of limited sample size and fewer occasions of measurement for Decedents). Multilevel findings are also consistent with tertiary aging influences (e.g., closer proximity to death) with compromised performance emerging for both cognitive processes and products (Bosworth et al., 1999).

Important implications are associated with the present demonstration of terminal drop. Perhaps the most obvious concerns how to delay such precipitous decline. Present findings indicate that multiple risk factors including health can increase risk for terminal

drop. Thus, addressing even small health risk factors such as cardiovascular symptoms and signs (particularly with increasing age) may be of critical importance for staving off terminal drop. At least for the near term, facilitating longevity will be far more successful via preventative health maintenance (e.g., treating cardiovascular and circulatory diseases) as opposed to treating vascular dementia.

Similarly, patterns observed in these findings differentiating Survivors and Decedents imply that it may be possible to identify normal compared with pathological aging slopes. For example, interactions of age and mortality demonstrated that rate-of-decline slopes for Old-Old Decedents were between twice and triple the rate of Old-Old Survivors. Cohen's d , calculated as the mean difference between mortality group slopes divided by the slope standard deviation, yielded large mean effect size differences (i.e., $d > .80$) for all cognitive measures. These large mortality effects at the intraindividual level represent a particularly strong demonstration of the perils of treating elderly samples as homogeneous (Bosworth et al., 1999). Such large discrepancies in mortality-related cognitive change speak directly to how reported findings in the cognitive aging literature may overestimate normative aging effects due to inclusion of those in a terminal decline phase. Quadratic and piecewise results exhibiting the acceleration of cognitive decline for Decedents further underscores the need to distinguish between normative relative to pathological aging trends in future research. Not only do mortality slopes show large differences, these differences are magnified with increasing age and proximity to death. Thus, consistent with Riegel and Riegel's (1972) argument, controlling for impending death is associated with attenuated estimates of normative age decline.

Tertiary aging is associated with pervasive influences on cognitive performance

(e.g., Birren & Cunningham, 1985; Bosworth et al., 1999) and may reflect a broad underlying influence such as general biological aging. To consider potential associations between CNS integrity and terminal decline, the final section examined mortality-related cognitive deficits in relation to a potential marker of neurological disturbance (performance inconsistency).

Performance Inconsistency and Terminal Decline

A relatively unexplored issue in the terminal decline literature involves examining potential correlates of mortality-related cognitive deficits. Performance inconsistency is a promising correlate; it increases with age (Hultsch et al., in press), time (MacDonald et al., in press), and neurological disturbance (Hultsch et al., 2000) and is associated with poorer cognitive performance across various cognitive tasks. Inconsistency is posited to reflect neurobiological irregularities due to factors including aging and disease.

Associations between impending mortality and cognition as well as inconsistency and cognition have been demonstrated. An hypothesized association between impending mortality and inconsistency is expected if both phenomena are influenced by similar underlying sources. However, as suggested by Berg (1996), it is not clear whether sources of mortality-related deficits are global (e.g., general biological breakdown) or local (e.g., specific diseases).

Analyses examined performance inconsistency for two RT measures as a function of mortality status, time to death, age, and cardiovascular disease/cause of death. As hypothesized, results showed that Decedents were more inconsistent relative to Survivors for the last wave of testing and over time. Of particular interest, change in inconsistency varied as a function of impending death, cardiovascular disease, and age. Decedents were

more variable than Survivors with the magnitude of mortality differences in inconsistency increasing across waves of testing (Old-Old Decedents with CVD exhibited the most inconsistency). Of particular importance, multilevel findings showed that performance inconsistency significantly increased per each additional year closer to death; rates of change in inconsistency were significantly higher for the Old-Old and for Decedents. Finally, performance inconsistency for the last wave of testing significantly predicted increased likelihood of death.

The present results are the first to demonstrate an association between impending mortality and performance inconsistency. Overall, mortality differences observed for inconsistency are strikingly similar to those observed for cognitive performance (i.e., terminal decline). A common underlying source represents one possible explanation for the similar patterns. For example, previous research has argued that neurobiological irregularities may underlie age-related increases in inconsistency (Hultsch et al., 2002; Li et al., 2001). Similarly, general CNS integrity has been postulated as a mechanism underlying cognitive deficits (e.g., Berg, 1996; Lindenberger & Baltes, 1994). Both the biological vitality and common-cause hypotheses suggest that age-related cognitive declines may be associated with a global deterioration of CNS functioning. In light of the present findings, it is plausible that general CNS function may underlie Decedents' compromised cognitive performance and increased inconsistency. It is important to note, however, that magnitude of inconsistency was differentially influenced by CVD reflecting the potential importance of specific disease processes as well. As suggested for the observed cause of death effects, general vascular disturbance may further compromise CNS function resulting in even greater cognitive decline and increased inconsistency.

Indeed, a distinct possibility is that global neurological disturbances are associated with impending death and that specific risk factors further magnify observed deficits. Future research must examine whether global or specific influences account for mortality differences in both cognitive performance and inconsistency.

A second unique finding demonstrated that performance inconsistency significantly predicted increased mortality risk (independent of CVD) using Cox regression. This finding is particularly important when contrasted with analyses examining mean cognitive performance as a predictor of death. Results from both the present and past investigations (e.g., Small & Bäckman, 1997) have failed to demonstrate that mean performance level significantly predicts survival over a longitudinal interval. This outcome is inconsistent with both cross-sectional and longitudinal ANOVA findings showing reliable differences in cognitive performance between mortality groups.

Important implications follow from this finding. Perhaps most interesting, performance inconsistency significantly predicts mortality risk in situations where mean performance does not. Previous studies posit that inconsistency reflects a behavioural marker of dementia (e.g., Hultsch et al., 2000). In support of this, unpublished results from Project MIND and the VLS suggest that even mild irregularity in the neurological system (i.e., Cognitively Impaired Not Demented status) increases the amount of variability in cognitive functioning. Conceivably then, performance inconsistency might be used as a screening device to identify those at risk for developing dementia and subsequent death. Based on these findings, one approach would be to identify avenues of intervention such as life style adjustment (e.g., Botwinick et al., 1978). Moreover, measures of performance inconsistency could prove useful in routine health assessments

of the elderly. In addition to the many benchmarks currently used by general practitioners to index health (e.g., blood pressure), inconsistency estimates may prove useful as early indicators of dementia and impending death. As already noted, treating CVD conditions is far easier than treating vascular dementia. An important next step is to identify those at risk and to advocate prevention prior to advanced cognitive decline.

Considered together, second theme findings from this study (i.e., novel contributions to the terminal decline literature) provide interesting perspective on some unanswered questions. Specific cause of death was associated with terminal decline, patterns of terminal drop were identified, and performance inconsistency was differentially associated with mortality. Overall, results suggest that cognitive performance may share associations with impending mortality via several mechanisms. Mortality-related decline may reflect specific disease processes (e.g., Anstey et al., 2001) with CVD emerging as an important predictor in the present investigation. In addition, evidence was also found in support of terminal decline as a marker of general CNS integrity. Cognitive performance, inconsistency, and impending mortality shared similar associations perhaps reflecting the same underlying neurobiological influence. These associations suggest that inconsistency may be a potentially useful marker of terminal decline.

Research Limitations and Suggestions for Further Research

The chapter concludes by addressing several research limitations and proposing directions for future research.

Limitations and Future Directions

Several limitations were associated with the present study. Although at the same time a strength, perhaps the most limiting factor was available waves of data. In the present study, Decedents completed fewer waves of measurement than Survivors (last wave $n = 265$; second last wave $n = 144$; third last wave $n = 78$). This made it difficult to use multilevel modeling for examining curvilinear decline; for two waves of measurement, only linear effects could be examined. Examining terminal decline across three or more waves resulted in serious compromises of statistical power due to diminished sample size. However, despite the conservative design of the present multilevel analyses, consistent intraindividual trends were observed.

A second limitation was the completeness of the cognitive continuum. Additional measures of perceptual speed, fluid reasoning, and verbal ability would help round out the continuum of measures used in the present investigation. Although the VLS incorporates these measures in its battery, they were not administered for earlier measurement waves. In lieu of including additional measures from later waves, it was necessary to maximize the Decedent sample by selecting participants who completed earlier waves without these measures. More and multiple indicators of cognitive performance should be included in future studies. In particular, additional indicators of verbal ability and semantic memory would provide useful contrasts with the mortality

deficits observed for the more cognitively demanding tasks in the present study.

Moreover, results from the profile analyses suggest closer examination of mean cognitive differences across the continuum within mortality group as opposed to between-group differences within each task.

Present findings have identified other important avenues for future investigation. Arguably, the most important of these concerns the source of mortality-related deficits. For example, results from the profile analyses demonstrated that the magnitude of mortality deficits was considerably smaller for Young-Old compared with Old-Old Decedents. Cognitive decline accounted for only 1% of the variance in Young-Old Decedent scores. Among possible explanations, this may reflect sudden cause of death for the Young-Old and underscores the importance of considering sources of mortality-related deficits. Future research should examine differences between acute compared with protracted diseases as well as the role of disease severity in magnifying terminal decline effects.

Similarly, cause of death findings indicated that mortality deficits were further magnified by cardiovascular disease. Compromised brain oxygenation or regional cerebral blood flow represent possible explanatory mechanisms. Future research should examine this possibility using neurological imaging techniques (e.g., fMRI). At the most basic of levels, all future studies of terminal decline should report simple indices of health and disease status of participants (Berg, 1996). Present results suggest that inclusion of objective measures of CVD would also prove useful. In addition, multilevel modeling with larger samples and more occasions of measurement would provide for a more powerful test of terminal decline in relation to cause of death. Ultimate determination of

global relative to specific influences requires use of data from a population study including a broad age range and sufficient sample size to examine multiple specific causes of death. Such large samples would include a wider array of causes to death to adequately test this hypothesis (Small et al. in press). If specific diseases share stronger relations with terminal decline, then research focus could be directed to understanding how these pathologies influence cognitive performance. In contrast, if many causes of death are related to terminal decline, global influences related to CNS integrity should be considered (Berg, 1996).

Despite the promise of present findings, it is critical that future research consider diverse sources of terminal decline in addition to those already identified. In support of this statement, random coefficients were significant for all HLM analyses conducted in this investigation. This indicates that, even after controlling for predictors including mortality status, time to death, cardiovascular disease, and age, significant between-group differences in intercepts (average performance) and slopes (individual rates of change) remained to be explained. Promising future avenues of investigation should consider examining biological and physiological health as predictors of terminal decline (e.g., Anstey et al., 2001; MacDonald et al., 2002). Moreover, potential common-cause mechanisms underlying both terminal decline and inconsistency should be considered. Findings from this investigation suggest that inconsistency contributes unique information to the prediction of mortality independent of mean level performance. Inconsistency may prove useful as an early indicator of impending mortality; measures of inconsistency should be considered for inclusion in standard health screening measurement batteries.

Summary

A major confound in aging studies concerns whether age differences in cognitive performance are a function of age on the one hand, or proximity to death on the other. The inclusion of premorbid individuals (e.g., those within 3 years of death) may erroneously decrease reported levels of cognitive performance and increase estimated rates of age-related cognitive decline. With respect to studies of cognitive aging, it is important to resist treating elderly samples as homogeneous (Bosworth & Schaie, 1999; Bosworth et al., 1999). Typical samples include individuals close to death as well as healthy adults. Averaging the performance of both groups may lead to the faulty conclusion that all older adults exhibit age-related cognitive declines.

Findings from the present investigation make important contributions toward answering some of the unanswered questions in the terminal decline literature. Consideration of the five primary research questions served to: (a) provide a better understanding of terminal decline and its characteristic nature; and (b) identify potential sources underlying mortality-related cognitive deficits. First, mortality-related deficits increased with age. Second, patterns of cognitive deficits were consistent with an ability-specific account of terminal decline (more demanding tasks showed greater deficits). Third, cause of death moderated patterns of terminal decline with those who died of CVD showing greater cognitive deterioration. Fourth, accelerated decline for Decedents in closer proximity to death was found in support of the terminal drop hypothesis. Finally, increased performance inconsistency was associated with mortality status suggesting that a common mechanism may account for associations among impending death, cognitive decline, and inconsistency. Overall, terminal decline results were remarkably congruous

across analyses and predictors. Those with risk factors including increased age, impending mortality, and cardiovascular disease all exhibited increased cognitive deficits. Notably, the longitudinal design, objectives, and findings of this study go beyond simple prediction of mortality status and its relation to cognitive deficits at a single point in time. This research is of particular theoretical importance to the field given both documented and potential relations among age, chronic disease, mortality, performance inconsistency, and cognition.

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