Psychology and Aging
2001, Vol. 16, No. 1, 3–11

0882-7974/01/$5.00 DOI: 10.1037//0882-7974.16.1.3

Demographic, Health, Cognitive, and Sensory Variables as Predictors of Mortality in Very Old Adults

Kaarin J. Anstey
University of New South Wales

Mary A. Luszcz, Lynne C. Giles, and Gary R. Andrews
Flinders University

Cognitive and sensorimotor predictors of mortality were examined in the Australian Longitudinal Study of Ageing, controlling for demographic and health variables. A stratified random sample of 1,947 males and females aged 70 and older were interviewed, and 1,500 were assessed on measures of health, memory, verbal ability, processing speed, vision, hearing, and grip strength in 1992 and 1994. Analyses of incident rate ratios for mortality over 4- and 6-year periods were conducted using Cox hierarchical regression analyses. Results showed that poor performance on nearly all cognitive variables was associated with mortality, but many of these effects were explained by measures of self-rated health and disease. Significant decline in hearing and cognitive performance also predicted mortality as did incomplete data at Wave 1. Results suggest that poor cognitive performance and cognitive decline in very old adults reflect both biological aging and disease processes.

There is strong evidence that among older persons performance on cognitive tests predicts subsequent mortality over periods of 5 to 10 years (Baltes, Schaie, & Nardi, 1971; Berg, 1996; Bosworth, Schaie, & Willis, 1999; Korten et al., 1999; Maier & Smith, 1999; Small & Bäckman, 1997; Swan, Carmelli, & LaRue, 1995). This effect remains even when demographic variables and health status are controlled statistically (Korten et al., 1999; Maier & Smith, 1999; Small & Bäckman, 1997). It also appears that cognitive function and health are more important predictors than psychosocial factors such as socioeconomic status (SES), neuroticism, and social support (Korten et al., 1999; Maier & Smith, 1999).

Even though the relationship between level of cognitive function and mortality appears to be a robust finding (Berg, 1996), a number of related empirical questions remain unanswered. As noted recently by Maier and Smith (1999) and Small and Bäckman (1997), it is still unclear whether certain cognitive factors show stronger relationships with mortality compared with others. For instance, some authors have argued that there is a sudden decline in crystallized intelligence (abilities depending on the accumulation of knowledge in long-term memory such as general knowledge and vocabulary) prior to death (Birren & Cunningham, 1985), whereas other studies have found that fluid abilities (reasoning and problem-solving abilities that do not depend on prior education or experience) are stronger predictors of mortality. For example, Korten et al. (1999) found that after controlling for health, performance on the Mini-Mental State Examination (MMSE) and the Symbol Letter Modalities Test predicted mortality, but that performance on the National Adult Reading Test (NART; Nelson, 1982) and an episodic memory test did not. Maier and Smith (1999) found that fluid measures were stronger predictors of mortality than crystallized measures, although all of the cognitive factors (perceptual speed, reasoning, memory, knowledge, and fluency) examined in their study were associated with mortality after controlling for age, SES, and health. In the Seattle Longitudinal Study, crystallized abilities, visualization abilities, verbal memory, and perceptual speed all predicted mortality (Bosworth, Schaie, & Willis, 1999). It is possible that the range of findings from recent studies is due to methodological differences between the studies, such as length of follow-up, sample size, gender, and the psychometric properties of the cognitive tests used.

Another issue relating to cognition and mortality is whether cognitive decline predicts mortality (Berg, 1996). The terminal drop hypothesis (Riegal & Riegal, 1972) proposes that a decline in cognitive function occurs approximately 5 years prior to death. Palmore and Cleveland (1976) emphasized the distinction between terminal decline represented by a steady rate of decline and terminal drop represented by an accelerating rate of decline prior to death. Different cognitive abilities have shown terminal decline in different studies. For example, some authors have found decline on verbal tests to be predictive of death (Birren, 1965; Siegler, McCarty & Logue, 1982; White & Cunningham, 1988), whereas Botwinick, West, and Storandt (1978) found that paired-associate and speed tasks rather than verbal tasks were predictive of death. More recently, Bosworth and Schaie (1999) found that mortality was associated with increased rate of decline on measures of both

Kaarin J. Anstey, Prince of Wales Medical Research Institute, University of New South Wales, Sydney, Australia; Mary A. Luszcz, School of Psychology and Centre for Ageing Studies, Flinders University, Adelaide, South Australia; Lynne C. Giles, Computing Services, Flinders University; Gary R. Andrews, Centre for Ageing Studies, Flinders University.

This study was partly funded by National Health and Medical Research Council of Australia Grant 987100. We gratefully acknowledge the men and women who participated in this study; the Centre for Ageing Studies at Flinders University; and Konrad Petsuvis, Linnett Sanchez, Sabine Schreiber, and the Epidemiology Branch of Department of Health and Human Services in South Australia for their assistance.

Correspondence concerning this article should be addressed to Kaarin J. Anstey, Prince of Wales Medical Research Institute, Prince of Wales Hospital, Barker Street, Randwick, New South Wales 2031, Sydney, Australia. Electronic mail may be sent to k.anstey@unsw.edu.au.

verbal meaning and psychomotor speed. Although it is still unclear whether decline in any specific cognitive ability is a more salient predictor of mortality, a general association between cognitive decline and mortality appears to be a robust finding. A plausible explanation for this is that significantly increased rate of decline (i.e., terminal drop) in cognitive function is an indicator of either disease or accelerated biological aging, and should therefore be a predictor of mortality. Cognitive performance has been used as a biomarker in functional age studies (see Anstey, Lord, & Smith, 1996, for a review) in which it has been argued that certain variables are indicators of individual differences in aging rates.

A third question involving the relationship between cognitive performance and mortality is whether sensory function follows a similar pattern to cognitive function. Given the evidence from cross-sectional studies that sensory function is strongly associated with cognitive performance in old age (Anstey & Smith, 1999; Lindenberger & Baltes, 1994; Salthouse, Hambrick, & McGuthry, 1998), it is possible that both may operate similarly in relation to mortality. This would be expected if sensory and cognitive aging are both biomarkers or are caused by common age-related changes in neurophysiological integrity (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994).

A fourth issue involves the particular challenge encountered in the longitudinal study of sensory and cognitive performance in very old adults, whereby missing data occurs both within and across times of measurement. The causes of a large proportion of missing data are unlikely to be random but are likely to be related to disease and cognitive and sensory impairment. These factors are also likely to be significant predictors of mortality. It is therefore important to obtain some estimation of the extent to which missing data may bias results of studies that evaluate the association between cognitive performance and subsequent mortality.

The present study of cognitive predictors of mortality in a large representative sample of very old adults examines these four issues. We examined baseline performance on cognitive measures of memory, verbal ability, and processing speed and sensorimotor measures of vision, hearing, and grip strength as predictors of mortality over a 6-year period. We examined decline in performance on these measures over a 2-year period as a predictor of mortality in the subsequent 4 years of follow-up. In the present study, the analysis of decline involves classifying participants according to the amount of decline over a 2-year period, independent of initial score. A large amount of decline is therefore suggestive of an accelerated decline before death, or terminal drop (Berg, 1996; Palmore & Cleveland, 1976).

We used demographic variables including age, gender, and education as control variables. In this study, we also controlled for self-rated health and measures of disease to enable an evaluation of whether sensory and cognitive performance predicted mortality independent of the influences of self-rated health and disease. If this were the case, it could suggest that cognitive and sensory functions are reliable markers of biological-aging processes that occur independent of disease. Self-rated health and disease are known to affect cognitive function, sensory function, and life expectancy (Idler & Benyamini, 1997; McCallum, Shadbolt, & Wang, 1994; Van Doorn & Kasl, 1998; Wolinsky & Tierney, 1998). Therefore, the effects of these health variables may explain rather than confound the relationship between cognitive and sensory function, and mortality, in old age.

Method

Sample

The sample was drawn from participants in the Australian Longitudinal Study of Ageing (ALSA; see Luszcz, Bryan, & Kent, 1997; Luszcz, 1998; Van Doorn & Kasl, 1998, for more details). The South Australian Electoral Roll was used as a sampling frame to identify households with residents over 70 years of age (Hugo, Healy, & Luszcz, 1987). The sample was stratified by age and gender into three 5-year cohorts: 70–74, 75–79, 80–84, and a fourth cohort of individuals over 85 years of age. Randomly sampled individuals from within these cohorts were invited to participate in the ALSA on a voluntary basis. The participation rate for the baseline data collection (Wave 1) was 55%. The study comprises six waves of data collection: the baseline, between September 1992 and March 1993, four subsequent waves of data collected at approximately 12-month intervals, and a sixth wave in 1998. Waves 2, 4, 5, and 6 comprised telephone interviews and did not include a clinical or cognitive assessment. Therefore, data from Waves 1 and 3 are used in this study. In these waves, a comprehensive 2-hr home interview was followed by an optional individual assessment conducted approximately 2 weeks later. The home interviews yielded demographic data and information on self-rated health, depression, medical conditions, cognitive status, memory and subjective measures of vision, audition, and physical performance. Individual clinical assessments provided objective cognitive and sensory data. At Wave 1, 1,947 participants (1,039 men) were interviewed and 1,500 (828 men) underwent the clinical assessment. At Wave 3, 1,557 participants were interviewed (809 men) and 1,311 underwent the clinical assessment (694 men).

Cognitive Measures

The cognitive measures have been described more fully elsewhere (Luszcz et al., 1997). Verbal measures included similarities, NART, and picture naming. Memory measures included picture recall and symbol recall.

Similarities was a three-item (*apple–banana, boat–car, egg–seed*) test taken from the Wechsler Adult Intelligence Scale—Revised (WAIS-R; Wechsler, 1981), and assessed verbal reasoning (Christensen et al., 1994). Items were scored 0 (*incorrect*) or 1 (*correct*) to give a possible total of 3. Picture naming was a short form of the Boston Naming Test (Mack, Freed, Williams, & Henderson, 1992) used to assess confrontation naming. A score of 1 was given for each item correctly named without cues for a maximum possible total of 15. Observed scores on confrontation naming ranged from 6 to 15. NART (Nelson, 1982) measures verbal knowledge and comprises 50 infrequent words of irregular pronunciation that respondents are asked to read aloud. The number of items correctly named was used as the score. Processing speed was assessed using the Digit Symbol Substitution subscale (DSS) of the WAIS-R. The participant was required to substitute symbols corresponding to the numbers 1 through 9 into a randomly ordered array of 93 digits. Symbols to be used were available throughout the task on a code sheet illustrating the nine digit–symbol pairs. The participant was required to make substitutions as rapidly as possible. The score was the number of substitutions completed correctly in 90 s. Symbol memory was the total number of symbols recalled from the processing speed task. Participants were given a recall sheet with the numbers 1 through 9 minus the symbols and asked to draw as many of the symbols as they could remember with each number. Participants had not been informed at any time that they would be required to recall the symbols. Picture memory was the total number of pictures correctly recalled from the picture naming measure (Luszcz et al., 1997). Participants were asked (without prior warning) to recall the 15 pictures immediately after the test. The MMSE comprised 21 items from the test developed by Folstein, Folstein, and McHugh (1975) that assess orientation, registration, attention, calculation, and recall (Teng, Chui, Schneider, & Metzger

1987). Incomplete scores were prorated if participants had responded to 17 or more of the 21 items.

Sensorimotor Measures

Pure tone thresholds (hearing) in left and right ears at 0.5, 1, 2, 3, 4, 6, and 8 kHz were assessed with portable audiometers with standard earphones. Testing began in the better ear, and a standard bracketing technique was used. For this study, we created a composite for hearing variable comprising the average of the best of left and right ears, using frequencies with the least missing data that also were among the most sensitive to age changes. These included 2 kHz, 3 kHz, and 4 kHz. The 6-kHz and 8-kHz frequencies were not used for this study because of the large amount of missing data on these variables at Wave 1. Corrected distance visual acuity (vision) was tested at 3 m for each eye with a well-illuminated Snellen chart. If the participant wore glasses or contact lenses but did not have them at hand, a best estimate of refracted visual acuity was obtained using pinhole testing, whereby the eye being tested was covered with an occluder containing a pinhole (Casson, Giles, & Newland, 1996). Participants were encouraged to read the smallest line possible. The criterion for distance visual acuity was the smallest line read successfully, that is, at least half of the characters in the line correctly read. Vision scores were converted to logMAR units. LogMAR units have an interval scale and are equivalent to $\log_{10}$ of the Snellen fraction, where the Snellen fraction equals the size of the letter read as a proportion of the test distance. Grip strength (grip) was measured in the dominant hand with a dynamometer while the participant was standing. The best of 3 attempts at maximum force was recorded in kilograms.

Measures of Health and Disease

We used a measure of self-rated health and three measures of disease in the present study. Although we collected data on 60 medical conditions, we created variables reflecting the presence or absence of neurological and cardiovascular conditions because these medical conditions have been shown to predict both mortality and cognitive function (Haan, Shemanski, Jagust, Manolio, & Kuller, 1999). Measures included a 3-point scale of self-rated health (SRH; 1 = *very good/excellent,* 2 = *good,* 3 = *fair/poor*), a dichotomous measure of neurological conditions (neurological conditions; none vs. one or more), a dichotomous measure of cardiovascular conditions (CVD; none vs. one or more), and number of medications (medications).

Determination of Mortality Status

After Wave 1, we traced all study participants at each successive round of data collection. At each follow-up, information on deaths was recorded and this information was subsequently confirmed by searches of official death certificates conducted by the Epidemiology Branch of Department of Health and Human Services in South Australia. Confirmation of deaths were obtained from the South Australian Cancer Registry, which by law has direct access to the Births, Deaths, and Marriages Registration Office of South Australia. The Cancer Registry matched study participants' identifying data (full name, date of birth, address) with deaths. If an obvious direct match was not easily identified, the electoral role was checked for names and addresses, errors with dates of birth, and misspellings of names. A minority of deaths (n = 37) occurring in other states or overseas could not be confirmed using this method and in these cases participant histories traced through the friends or relatives listed as informants by study participants were relied on to provide final death status.

Classification of Participants Into Decliners and Nondecliners on the Cognitive and Sensory Variables

For each cognitive and sensory variable, participants were classified as having either declined significantly or not declined significantly. The lowest quintile of the distribution of change scores for each variable (in the direction of decline) was taken as the category representing significant decline and the remainder of the sample was classified as not having declined significantly. For each variable, cases with scores at Wave 1 that were too low to allow for measurable change according to this criterion were excluded from this classification and treated as missing in the analyses of decline.

Statistical Analysis

For all analyses, participants were classified as alive or deceased at the censoring date of August 31, 1998. Differences between survivors and the group who were deceased at the censoring date were assessed with t tests, analysis of variance, and chi-square tests of association.

Cox proportional hazard models (Cox, 1972) were fitted to estimate the importance of each predictor of mortality. The Cox regression model assumes that the death rate of the population depends on a continuous time variable, which in the present study was the interval between the Wave 1 assessment and date of death. In the present study, predictors included demographic, health, cognitive, and sensorimotor variables. The Cox regression model (Kelsey, Whittemore, Evans, & Thompson, 1996) may be represented by the formula,

$$\ln[I(t,x)/I(t,x^*)] = b_1(x_1 - x_1^*) + \cdots + b_p(x_p - x_p^*),$$

where $\ln[I(t,x)/I(t,x^*)]$ is the logarithm of the incident rate ratio (IRR) for an individual with values of predictor variables x relative to someone with baseline values of x^*; $x_1^*, \ldots x_p^*$ represents the values for the predictor variables at baseline; and $b_1, \ldots b_p$ are regression coefficients. The regression coefficients are estimated by comparing the risk factors of survivors and deceased individuals. The IRRs are calculated by exponentiating the beta weight for the predictor (Kelsey et al., 1996). An IRR of 1.10, where the incident is defined as death, as in the case of the present study, suggests a 10% increase in the death rate with each unit increase in the raw test score compared with the reference category, adjusting for the effects of all other variables in the equation.

In this study, the distributions of the cognitive and sensory variables were divided into quintiles for which this was possible (picture naming, NART, processing speed, symbol recall, picture recall, hearing, grip, and a cognitive composite), with the quintile of the top-performing group being chosen as the reference group. For example, on the measure of processing speed at Wave 1, IRR ratios were calculated for the bottom four quintiles compared with the top-performing quintile. When variables could not be divided into meaningful quintiles because of their distributional properties, they were divided into quartiles (MMSE, vision) or thirds (similarities).

A composite of the cognitive measures was also created by adding raw scores of the cognitive variables, to enable analyses of an overall effect of the cognitive measures. Raw scores were added in this composite as a means of weighting tests with varying numbers of test items. For example, similarities had three items, processing speed had a possible maximum of 93 items, and picture naming had 15 items, so that adding z scores of these tests would give undue weighting to the individual items of similarities. The method of adding raw scores was also consistent with all other analyses in this study in which no transformations of data were made.

Analyses were also conducted to determine whether individuals with no or missing cognitive and sensory data at Wave 1 had an increased risk of mortality. It was expected that much of the missing data would not be missing at random (Little & Rubin, 1987). Therefore, this analysis enabled an evaluation of how missing data may bias the results of the previous analyses. A categorical variable was created that classified participants according to whether they had completed all cognitive tests, one or two cognitive tests, or no cognitive tests at Wave 1. Another variable was created that classified participants according to whether they had complete sensory data, incomplete sensorimotor data (data for one or two of grip strength, vision, and hearing), or no sensorimotor data at Wave 1.

Results

Length of Follow-Up and Days Until Death

The average length of follow-up for participants in the study was 5.76 years (2,104.75 days; *SD* = 40.61 days), with a range of 2,010 to 2,168 days. Of those who died during the follow-up period, the average length of survival was 4.85 years (1,769.54 days; *SD* = 586.63), with a range from 3 to 2,175 days. Table 1 shows the number of participants who died each year according to 5-year age groups. Note that the proportion of the total number of deceased individuals is small for the sixth year because this year represents approximately 9 months, rather than 12 months of follow-up. Table 1 shows the mortality rates for each 5-year age cohort for each year of the follow-up. An estimate of the 12-month mortality rate for Year 6 was used to calculate the average yearly mortality rates for each group defined in terms of age at Wave 1.

Differences in Wave 1 Performance Between Mortality Status Groups

Table 2 shows the demographic characteristics of the participants who survived the 6-year follow-up and those who did not. At Wave 1, participants who did not survive lived to an average age of 82.05 (*SD* = 6.29), compared with 77.26 (*SD* = 5.53) for those who did survive, and this difference was statistically significant, $t(1945) = 17.376$, $p < .01$. Differences between groups were significant for gender, with men being more likely to die in the 6 years after Wave 1, $\chi^2(1, N = 1{,}947) = 33.25$, $p < .01$. Participants reporting a history of neurological disorder were also less likely to survive, $\chi^2(1, N = 1{,}942) = 42.26$, $p < .01$, as were participants who had worse self-rated health, $\chi^2(2, N = 1{,}941) = 74.53$, $p < .01$, had less than 14 years of education, $\chi^2(1, N = 1{,}921) = 6.72$, $p < .01$, and were taking more medications, $\chi^2(5, N = 1{,}934) = 71.74$, $p < .01$. Participants who reported having had cardiovascular disease were neither more nor less likely to survive.

Descriptive statistics for all cognitive and sensorimotor measures of the participants who were interviewed and assessed at Wave 1 and those who subsequently died are shown in Table 3. Survivors performed significantly better on all performance-based measures of sensory and cognitive function at Wave 1. Not surprisingly, those who did not survive for 6 years were initially older than those who did survive. When age differences were taken into

Table 2
Demographic Characteristics of Survivors and Deceased at Wave 1

Variable	Survivors (*n* = 1,267)	Deceased (*n* = 680)
Age		
M	77.26	82.05
SD	5.53	6.29
Gender		
Male	616	423
Female	651	257
Education		
0–14 years	681	401
15+ years	576	263
SRH		
Excellent/very good	538	195
Good	409	181
Fair/poor	320	298
CVD		
None	638	321
One or more	624	359
Neurological conditions		
None	1,048	477
One or more	214	203
No. of medications		
0	181	47
1	214	76
2	249	107
3	200	115
4	153	89
5+	263	240

Note. SRH = self-rated health; CVD = cardiovascular conditions. Numbers do not consistently add up to 1,267 survivors and 680 deceased because of missing data.

Table 1
Number and Percentage of Participants Deceased Per 5-Year Age Cohort During Each Year of Follow-Up

Year of follow-up	Age cohorts							
	70–74 (*n* = 562)		75–79 (*n* = 524)		80–84 (*n* = 429)		85+ (*n* = 432)	
	No.	%	No.	%	No.	%	No.	%
Year 1	13	2.31	17	3.24	23	5.36	41	9.51
Year 2	7	1.28	26	5.13	34	8.40	66	16.92
Year 3	12	2.21	26	5.41	27	7.26	45	13.89
Year 4	20	3.77	31	6.81	25	7.25	46	16.49
Year 5	21	4.11	28	6.60	32	10.00	47	20.17
Year 6[a]	20	4.09	19	4.79	30	10.42	24	12.90
Total[b]	93	13.7	147	21.60	171	25.10	269	39.60
Average[c]		3.19		5.53		8.55		15.70

[a] Year 6 was approximately 9 months. [b] Total number deceased in each age group and percentage of total number deceased (*n* = 680) at censoring date of August 31, 1998. [c] Average percentage of age group deceased each year based on 12-month equivalent for Year 6.

Table 3
Wave 1 Descriptives for Cognitive and Sensory Variables by Mortality Group

Variable	Survivors	Deceased
Similarities**	(*n* = 1,192)	(*n* = 603)
M	2.52	2.15
SD	2.15	1.02
Picture naming**	(*n* = 843)	(*n* = 365)
M	11.72	10.97
SD	2.36	2.67
NART (errors)*	(*n* = 807)	(*n* = 364)
M	22.16	23.24
SD	8.44	8.59
Processing speed**	(*n* = 819)	(*n* = 341)
M	30.98	24.44
SD	10.70	10.85
Symbol recall**	(*n* = 811)	(*n* = 332)
M	6.44	5.44
SD	1.81	2.40
Picture recall**	(*n* = 842)	(*n* = 363)
M	5.71	4.64
SD	2.30	2.54
MMSE**	(*n* = 1,244)	(*n* = 655)
M	19.19	17.89
SD	2.41	3.74
Vision**	(*n* = 900)	(*n* = 359)
M	0.33	0.30
SD	0.21	0.23
Hearing**	(*n* = 990)	(*n* = 450)
M	34.46	40.58
SD	17.53	17.13
Grip**	(*n* = 1,007)	(*n* = 457)
M	25.52	23.31
SD	8.87	8.62

Note. NART = National Adult Reading Test; MMSE = Mini-Mental State Examination.
* $p < .05$. ** $p < .01$. (For *F* tests comparing groups after controlling for age differences.)

account, survivors still performed better at Wave 1 on similarities, $F(1, 1792) = 24.55$, $p < .01$; processing speed, $F(1, 1159) = 22.05$, $p < .01$; symbol recall, $F(1, 1142) = 21.44$, $p < .01$; picture recall, $F(1, 1202) = 12.14$, $p < .01$; and MMSE, $F(1, 1898) = 31.74$, $p < .01$. However, after controlling for age, there was no difference between groups on vision, hearing, grip, the NART, or picture naming.

Incident Rate Ratios for Sensory and Cognitive Variables

Unadjusted IRRs were calculated for each cognitive and sensorimotor variable, and then IRRs were calculated after controlling for demographic variables and then after controlling for both demographic and health variables. Demographic variables included age, gender, and education. Health variables included SRH, medications, and neurological conditions. Cardiovascular conditions were not used as a control variable, as these did not show any association with increased risk mortality in the present study.

Table 4 shows the results of the Cox regression analyses for all cognitive and sensorimotor variables. The unadjusted analyses showed that poor performance on all measures was associated with increased IRRs of mortality over 6 years. After health and demographics were controlled for, poorer performance on similarities, processing speed, symbol recall, MMSE, and grip was associated with an increased IRR of mortality over 6 years. The association between level of performance at Wave 1 and the IRR of mortality was present for each of the three performance categories for similarities. It is possible that this finding was due to the smaller number of categories into which scores on this test were divided. In the case of processing speed and MMSE, a significant IRR was only found in the lowest quintile of these sample distributions. IRRs for symbol recall showed an irregular pattern of risk.

A composite of the cognitive variables (except for the NART) was also evaluated as a predictor of mortality (Table 4). Performance on the lower three quintiles of this composite was associated with increased IRR of mortality after demographic and health variables were controlled for. This pattern of results was unchanged when premorbid intellectual function was controlled for using the NART. This demonstrated that the effect of cognitive performance on mortality was independent of individual difference in cognitive abilities occurring prior to the effects of aging.

The data in Table 4 also show that poor performance on measures of hearing and grip was also associated with increased IRR of mortality after demographic characteristics were controlled for. The effects for hearing were virtually eliminated after health was also controlled for, whereas for grip each performance quintile maintained a significant increased risk of death, relative to the reference category.

Incident Rate Ratios for Decliners Versus Nondecliners

Table 5 shows the IRRs for mortality for participants who declined significantly (defined as the quintile of the distribution with the largest change scores in the direction of decline) from Wave 1 to Wave 3, compared with the remainder of the sample. To be included in these analyses, participants had to have survived until Wave 3 and completed the assessment yielding the cognitive or sensory data at both Wave 1 and Wave 3. The IRR for mortality in these analyses is therefore calculated over a 4-year interval rather than the 6-year interval shown in Table 4. The unadjusted results reported in Table 5 show that significant declines on similarities, MMSE, vision, and hearing, as well as the cognitive composite, were all associated with an increased IRR of mortality. However, significant declines on measures of picture naming, NART, processing speed, symbol recall, picture recall, and grip were not associated with an increased IRR of mortality in any analyses. After demographic and health variables were controlled for, significant decline in similarities, vision, and hearing represented significant IRRs for mortality. Significant decline on a composite of all the cognitive measures was associated with the largest IRR for mortality after demographic and health variables were controlled for. This result was unchanged when premorbid intellectual ability as measured by the NART was controlled for statistically.

Evaluation of Sample Bias Due to Missing Baseline Data

Finally, analyses were conducted to see if there were sample biases created by data missing from these analyses. For the cognitive variables, 56% of the sample at Wave 1 had complete data, 9% had incomplete data, and 35% had no data. For the sensory variables, 57% of the sample had complete data, 20% had incomplete data, and 23% had no data. Cox regression controlling for demographic variables was conducted to determine whether miss-

Table 4
Incident Rate Ratios (IRRs) for Levels of Performance on Cognitive and Sensory Variables

Predictor	Category approx. %	Unadjusted		After demographics		After health, demographics	
		IRR	95% CI	IRR	95% CI	IRR	95% CI
Similarities (items correct)							
0–1	0–16	**2.19**	**1.79–2.68**	**1.66**	**1.35–2.05**	**1.55**	**1.26–1.91**
2	17–38	**1.74**	**1.44–2.11**	**1.50**	**1.24–1.82**	**1.49**	**1.22–1.81**
3 (reference)	39–100	1.00		1.00		1.00	
Picture naming (items correct)							
2–8	1–20	**2.61**	**1.58–4.30**	**1.72**	**1.03–2.89**	1.56	0.93–2.63
9–10	21–40	**1.90**	**1.15–2.79**	1.31	0.78–2.18	1.14	0.68–1.90
11–12	41–60	**1.73**	**1.08–2.79**	1.47	0.91–2.38	1.24	0.77–2.02
13	61–80	1.17	0.69–2.00	1.24	0.72–2.12	1.11	0.65–1.89
14–15 (reference)	81–100	1.00		1.00		1.00	
NART (errors)							
30–48	1–20	1.35	0.97–1.88	1.04	0.73–1.42	0.94	0.66–1.34
25–29	21–40	1.30	0.94–1.82	1.12	0.80–1.57	1.00	0.71–1.41
20–24	41–60	0.94	0.94–1.66	0.94	0.66–1.34	0.85	0.59–1.22
15–19	61–79	1.24	0.87–1.76	1.14	0.80–1.63	1.05	0.73–1.49
0–14 (reference)	80–100	1.00		1.00		1.00	
Processing speed (items correct)							
0–19	1–20	**3.70**	**2.61–5.24**	**1.83**	**1.24–2.70**	**1.55**	**1.05–2.30**
20–25	21–40	**2.03**	**1.39–2.96**	1.28	0.87–1.90	1.15	0.80–1.71
26–31	41–60	1.38	0.93–2.06	1.08	0.72–1.61	0.95	0.63–1.42
32–38	61–80	1.13	0.75–1.70	0.99	0.66–1.50	0.93	0.62–1.40
39–67 (reference)	81–100	1.00		1.00		1.00	
Symbol recall (items correct)							
0–5	1–20	**3.47**	**1.96–6.14**	**2.32**	**1.30–4.14**	**2.01**	**1.13–3.59**
6	21–40	**2.00**	**1.00–3.62**	1.64	0.90–3.00	1.62	0.89–2.95
7	41–60	1.70	0.94–3.09	1.57	0.84–2.86	1.49	0.82–2.71
8	61–80	**2.10**	**1.15–3.82**	**1.95**	**1.07–3.56**	**1.85**	**1.02–3.79**
9 (reference)	81–100	1.00		1.00		1.00	
Picture recall (items correct)							
0–3	1–20	**2.61**	**1.87–3.63**	1.41	0.99–1.99	1.25	0.88–1.77
4–5	21–40	**1.46**	**1.04–2.04**	0.99	0.70–1.40	0.90	0.63–1.27
6	41–60	1.17	0.78–1.75	0.95	0.63–1.43	0.90	0.60–1.35
7	61–80	0.99	0.65–1.50	0.89	0.58–1.35	0.84	0.55–1.30
8–13 (reference)	81–100	1.00		**1.00**		1.00	
MMSE (items correct)							
0–17	1–20	**2.16**	**2.11–3.24**	**1.81**	**1.44–2.27**	**1.61**	**1.28–2.02**
18–19	21–40	**1.41**	**1.13–1.77**	1.12	0.90–1.41	1.04	0.82–1.30
20	41–60	**1.30**	**1.03–1.64**	1.19	0.92–1.50	1.11	0.87–1.40
21 (reference)	61–100	1.00		1.00		1.00	
Cognitive composite							
0–22	1–20	**3.48**	**2.64–4.57**	**2.06**	**1.54–2.75**	**1.80**	**1.34–2.40**
23–36	21–40	**2.36**	**1.74–3.21**	**1.73**	**1.26–2.37**	**1.61**	**1.17–2.20**
37–68	41–60	**2.92**	**2.20–3.89**	**1.63**	**1.21–2.19**	**1.42**	**1.05–1.92**
69–82	61–80	**1.51**	**1.10–2.07**	1.16	0.85–1.60	1.03	0.75–1.42
83–127 (reference)	81–100	1.00		1.00		1.00	
Vision							
6/18–6/60	1–25	**2.02**	**1.50–2.72**	1.10	0.80–1.53	1.01	0.72–1.39
6/12	26–50	**1.71**	**1.26–2.31**	1.16	0.84–1.59	1.10	0.80–1.52
6/9	50–75	1.16	0.83–1.62	0.95	0.67–1.33	0.89	0.63–1.25
6/6 (reference)	76–100	1.00		1.00		1.00	
Hearing (dB)							
52.6–80	1–20	**2.25**	**1.67–3.02**	1.01	0.73–1.39	0.98	0.71–1.35
40–52.5	21–40	**1.79**	**1.33–2.40**	1.09	0.80–1.48	1.08	0.79–1.47
31–39	41–60	**1.96**	**1.42–2.70**	**1.45**	**1.04–2.00**	1.33	0.96–1.84
21–30	61–80	1.26	0.90–1.76	1.09	0.77–1.52	1.50	0.82–1.61
0–20 (reference)	81–100	1.00		1.00		1.00	
Grip (kg)							
0–17	1–20	**1.86**	**1.37–2.52**	**3.66**	**2.41–5.57**	**2.81**	**1.84–4.28**
17.10–21.50	21–40	**1.52**	**1.11–2.08**	**2.42**	**1.70–3.45**	**2.06**	**1.44–2.94**
21.60–26.75	41–60	**1.53**	**1.11–2.09**	**1.77**	**1.27–2.46**	**1.56**	**1.12–2.18**
26.76–33.05	61–80	**1.47**	**1.07–2.02**	1.21	0.87–1.67	1.13	0.82–1.56
33.06–50.75 (reference)	81–100	1.00		1.00		1.00	

Note. Predictor = cognitive and sensory variables categorized into ranges of raw scores. Scores on each variable were categorized into quintiles or quartiles where possible. Significant results are shown in bold. Category approx. % = the approximate percentage of the sample with scores in each category at Wave 1; CI = confidence interval; reference = the reference category according to which the hazard ratio is calculated for each other category of a variable (e.g., on similarities, participants who scored 0 or 1 had a mortality hazard of 2.19 compared with participants who scored 3 on this test); NART = National Adult Reading Test; MMSE = Mini-Mental State Examination; Cognitive composite = sum of cognitive variables except for the NART. Demographics include age, gender, and education. Health includes self-rated health, medications, and neurological conditions.

Table 5
Incident Rate Ratios (IRRs) for 2-Year Decline on Cognitive and Sensory Variables

	Uncorrected		After demographics		After health, demographics	
Predictor	IRR	95% CI	IRR	95% CI	IRR	95% CI
Similarities	**1.77**	**1.31–2.24**	**1.42**	**1.08–1.87**	**1.40**	**1.06–1.84**
MMSE	**1.52**	**1.10–2.09**	1.26	0.90–1.76	1.23	0.88–1.72
Vision	**1.55**	**1.01–2.36**	**1.49**	**1.06–2.10**	**1.46**	**1.04–2.06**
Hearing	**1.71**	**1.25–2.50**	**1.52**	**1.07–2.16**	**1.44**	**1.01–2.05**
Cognitive composite	**1.93**	**1.45–2.57**	**1.68**	**1.22–2.19**	**1.66**	**1.24–2.22**

Note. The quintile of the sample who declined the most on each variable was compared with the reference group of the remainder of the sample. The cognitive composite was formed by summing all raw scores on cognitive tests. Significant results are shown in bold. CI = confidence interval; MMSE = Mini-Mental State Examination; Cognitive composite = sum of change scores of all cognitive variables except for the National Adult Reading Test (NART). No significant results were found for decline on picture naming, NART, processing speed, symbol recall, picture recall, or grip. Demographics include age, gender, and education. Health includes self-rated health, medications, and neurological conditions.

ing data on the cognitive and sensory assessments was a risk factor for mortality. Table 6 shows that not completing any of the cognitive tests at Wave 1 or only completing one or two cognitive tests at Wave 1 was associated with increased IRR for mortality. Importantly, compared with participants who completed 3 or more cognitive tests, participants who were interviewed but did not complete any of the cognitive assessment were 40% more likely to die in the next 6 years, even after demographic variables were controlled for. Compared with participants who completed the full visual and auditory assessment, participants who did not complete any of the sensory assessment were 50% more likely to die over the next 6 years. Interestingly, 11% of survivors had missing MMSE data at Wave 1, whereas only 5% of participants who died had missing MMSE data at Wave 1.

Discussion

In the present study, we aimed to evaluate the importance of performance on cognitive and sensorimotor variables as indicators of the risk of mortality in very old adults. We investigated both level of performance and decline in performance. An important consideration when interpreting the results of this study is that the participants in the sample were 70 years of age or older at Wave 1. This means that the level of measured cognitive performance at Wave 1 reflects the effects of aging and disease in addition to individual differences in cognitive abilities.

Table 6
Missing Cognitive and Sensory Data as Predictors of Mortality After Controlling for Demographic Variables

Predictor	IRR	95% CI
Cognitive data		
Completion of 0 tests at Wave 1	**1.44**	**1.23–1.67**
Completion of 1 or 2 tests at Wave 1	**1.01**	**1.23–1.68**
Completion of 3 or more tests at Wave 1 (reference)	1.00	
Sensory data		
Completion of 0 tests at Wave 1	**1.52**	**1.27–1.81**
Completion of 1 or 2 tests at Wave 1	**1.28**	**1.05–1.56**
Completion of 3 tests at Wave 1 (reference)	1.00	

Note. Demographic variables included age, gender, and education. Significant results are shown in bold. IRR = incident rate ratio; CI = confidence interval.

Consistent with results from previous studies (Baltes et al., 1971; Berg, 1996; Bosworth & Schaie, 1999; Maier & Smith, 1999; Korten et al., 1999; Small & Bäckman, 1997; Swan et al., 1995), we found that after demographic variables were controlled for, poor performance on several measures of cognitive performance was a predictor of mortality. The NART was the only cognitive variable that was not associated with increased IRR of mortality, replicating the results of Korten et al. (1999). This finding provides validation for the NART being a measure of cognitive function that is relatively robust against the effects of disease and decline in old age. Because other measures of verbal ability were predictive of mortality, these results suggest that the premorbid ability thought to be measured by the NART is distinct from verbal abilities measured by tests such as similarities and the Boston Naming Test. After we controlled for demographics, self-rated health, and measures of disease, some cognitive variables were no longer significant predictors of mortality. This is consistent with the view that cognitive performance in very old adults is to some extent a reflection of disease processes. However, poor performance on similarities, processing speed, symbol recall, and MMSE remained significant predictors of mortality, even after health and disease were controlled for. This may be interpreted to suggest that in addition to being a reflection of disease, poor cognitive performance is a predictor of biological aging processes. The wide range of cognitive variables showing significant relationships with mortality in this study does not support the view that a specific cognitive ability is related to mortality, but rather suggests that the association holds at a more general level.

Sensory variables predicted mortality after demographic variables were controlled for. However, this effect was largely explained by self-rated health and disease measures. This finding is of interest because it differs from the cognitive results. If sensory and cognitive functioning are similarly indicative of the aging brain, as suggested by the common cause hypothesis (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994), or equally reliable biomarkers (Anstey & Smith, 1999), then it could be expected that they would both show similar relationships to mortality. However, the results of the present study suggest that the relationship between level of sensory function and mortality is

explained by health, whereas several aspects of cognitive function retain a relationship with mortality that is independent from health.

There are at least three possible explanations for the different pattern of results between cognitive and sensory variables: (a) sensory function is more sensitive to disease than cognitive function, (b) sensory function is amenable to medical intervention, whereas cognitive function is not, (c) the psychometric properties of the sensory measures are different from the psychometric properties of the cognitive measures. The actual tests of hearing and vision have a log scale and therefore may not be as sensitive to individual differences as some of the cognitive measures. Even though the vision score may be converted to logMAR, which has an interval scale, the Snellen Chart we used in the present study did not have items presented at intervals of equal increases in difficulty. Furthermore, floor effects on sensory variables will be present because of the inclusion of participants with severe sensory impairments. It is also more likely that a participant with a sensory impairment will be able to complete cognitive tests than that a participant with a cognitive impairment will be able to complete sensory tests.

The three lower quintiles of performance on grip strength at Wave 1 predicted mortality after demographic variables, self-rated health, and measures of disease were controlled for. This is consistent with the view that grip strength is a reliable biological marker of aging (Anstey et al., 1996; Anstey, Lord, & Williams, 1997; Anstey & Smith, 1999), where biological aging is viewed as an inevitable aging process that is independent of disease. Unlike the measures of vision and hearing, the measure of grip strength had an interval scale and no ceiling or floor effects. It is possible that these measurement properties contribute to this variable being such a reliable predictor of mortality.

A strength of the present study is the longitudinal data available on all measures over a 2-year interval. Our measure of decline was independent of initial performance level and identified individuals who exhibited a significant rate of decline over a 2-year period. We chose this approach because of the relatively short period of follow-up for which change data were available during which many participants showed very small amounts of decline on the cognitive and sensorimotor variables. Measuring decline over two time points in longitudinal studies of very old adults is further complicated by the increased variability in performance among this age group (Christensen et al., 1999). Our analyses therefore did not evaluate whether decline per se predicted mortality, but rather whether significant decline relative to the remainder of the sample predicted mortality. Although significant decline on several individual cognitive measures did not lead to significant IRRs for mortality, decline on a cognitive composite indicated a substantial IRR for mortality even after health and demographic variables were controlled for, as was the case for similarities. This result supports the terminal drop hypothesis but also suggests that change in individual markers of cognitive performance in this study may lack the sensitivity required to show a significant association with mortality.

Interestingly, significant decline in performance on the vision and hearing measures represented significant risks for mortality after demographic and health variables were controlled for, even though poor performance on the vision and hearing measures were not independent risk factors for mortality after demographic and health variables were controlled for. It is possible that individuals with poor vision or hearing have had the same level of sensory function for many years because of specific occupational exposures or disease, whereas marked decline over a 2-year period is indicative of an active aging or disease process. In contrast to the vision and hearing variables, significant decline in grip strength was not a significant IRR for mortality, whereas grip strength score at Wave 1 was one of the most sensitive predictors of mortality. The difference in the pattern of results between vision, hearing, and grip strength illustrates the point that significant decline in performance on a variable may be a significant predictor of mortality even though poor performance on this variable is not, and vice versa. This is particularly relevant with such an old sample when there are no data on past performance so that we do not know the extent to which performance at Wave 1 represents a decline from each individual's developmental peak in cognitive and sensory function. However, control of premorbid intellectual function measured by the NART did not alter the pattern of results for the cognitive composite we used in the present study.

It is also possible that the differing results for sensory and cognitive decline result from developmental influences not measured in the present study in addition to the psychometric properties of the tests used to measure sensory and cognitive function. Intercorrelations of the same measures between Wave 1 and Wave 3 ranged from .45 for similarities to .88 for some of the hearing measures (Anstey, Luszcz, & Sanchez, 2000), and these values may be seen as representing the lower bound of reliability of the measures. However, there was no trend for the change scores of more reliable measures to be more sensitive predictors of mortality.

An important contribution of our study is the analysis evaluating the effects of missing data at baseline on the risk of mortality. Having incomplete cognitive or sensory data was associated with very large increased risk of mortality. This probably reflects disease, possibly including incipient dementia, preventing participants from completing the cognitive and sensory assessments (although this pattern did not hold for the MMSE). It also strongly suggests that missing data in very old samples is unlikely to be random. A probable consequence of missing data that is not missing at random (Little & Rubin, 1987) is an underestimation of the size of the observed association between cognitive performance and subsequent mortality in the present study. Such a bias is likely to apply to other studies linking cognitive performance and cognitive decline in very old adults to subsequent mortality.

Despite the fact that longitudinal studies of cognitive aging are complicated by missing data, the effects of disease, and differing psychometric properties of tests, the results of the present study suggest that cognitive function may be associated with mortality in two ways. First, cognitive performance and decline may reflect disease processes and preclinical dementia (Sliwinski & Buschke, 1997), and second, cognitive performance may in fact be a marker of biological aging that is independent of disease.

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Received October 1, 1999
Revision received March 13, 2000
Accepted May 8, 2000 ■