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Fluid intelligence is independently associated with all-cause mortality over 17 years in an elderly community sample: An investigation of potential mechanisms

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ABSTRACT

The long-term relationship between lower intelligence and mortality risk in later life is well established, even when controlling for a range of health and sociodemographic measures. However, there is some evidence for differential effects in various domains of cognitive performance. Specifically, tests of fluid intelligence may have a stronger association with mortality than do tests of crystallized intelligence. The present study examines the relationship between intelligence and mortality in a sample of 896 Australian community-dwelling males and females, aged 70–97 at recruitment and followed for up to 17 years. There were 687 deaths during the follow-up period. Cox proportional hazard regression models examined whether the relationship between intelligence and mortality might be mediated by socioeconomic status, by health behaviors, by health status, or a combination of these. Higher fluid intelligence — as measured by the Symbol–Letter Modalities Test — was strongly associated with lower mortality rates (Hazard ratio = 0.80; 95% confidence interval = 0.72–0.88), even after accounting for any combination of potential mediators and confounders. A significant association between crystallized intelligence, as measured by the National Adult Reading Test, and mortality (HR = 0.89; 95% CI = 0.80–0.99) was attenuated by the inclusion of socioeconomic, health status measures, and health behavior measures and when deaths from the first four years of the study were excluded. The findings show little support for the hypothesized mechanisms of the intelligence–mortality relationship.

Long-term studies of intelligence and mortality demonstrate that higher intelligence is associated with lower all-cause mortality. A recent review (Batty, Deary, & Gottfredson, 2007) examined nine studies investigating the relationship between early-life intelligence and later mortality risk. The studies followed cohorts for between 17 and 69 years. All found that higher IQ was associated with lower mortality. For example, one of the reviewed studies retrospectively traced the vital status of 2230 participants in the 1932 Scottish Mental Survey after 65 years (Whalley & Deary, 2001). The hazard of mortality over the 65 year follow-up period was decreased by 21% for each 15-point increase in intelligence as measured by the Moray House test. Studies reporting follow-up into old age have also reported consistent findings (Deeg, Hofman, & van Zonneveld, 1990; Rabbitt, Lunn, & Wong, 2006; Shipley, Der, Taylor, & Deary, 2006). However, the intelligence–mortality relationship may be dependent of the type of test administered, the age of the cohort and the length of the follow-up period.

Poor performance on *executive tests* such as the Mini-Mental State Exam (Bassuk, Wypij, & Berkman, 2000; Dartigues et al., 2007) or the Short Portable Mental Status Questionnaire (Blazer, Sachs-Ericsson, & Hybels, 2005; Liang, Bennett, Sugisawa, Kobayashi, & Fukaya, 2003) tends to be associated with higher

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mortality risk, however the relationship has not always been found to be significant (Ganguli, Dodge, & Mulsant, 2002; Ostbye et al., 2006) and may be dependent on the length of the follow-up period (Ganguli et al., 2002; van Gelder, Tijhuis, Kalmijn, Giampaoli, & Kromhout, 2007). Performance on tests of *crystallized intelligence*, such as the National Adult Reading Test (Abas, Hotopf, & Prince, 2002; Anstey, Luszcz, Giles, & Andrews, 2001) or Raven's Mill Hill Vocabulary Scale (Rabbitt et al., 2002) tends to be robust to the effects of aging and is less likely to exhibit an association with mortality after health and social status are taken into account.

Tests of *fluid intelligence*, such as Digit–Symbol Substitution (Anstey et al., 2001; Ghisletta, McArdle, & Lindenberger, 2006; Pavlik et al., 2003; Portin et al., 2001) or various learning tasks (Abas et al., 2002; Ghisletta et al., 2006; Rabbitt et al., 2002; Royall, Chiodo, Mouton, & Polk, 2007) tend to decline more with age and are more strongly associated with mortality than performance on tests of general intelligence or tests of executive functioning. However, the effect size may be greater for long-term (e.g., Ghisletta et al., 2006) rather than short-term (e.g., Bosworth, Schaie, & Willis, 1999) studies and for older rather than younger cohorts (Lyyra, Heikkinen, Lyyra, & Jylha, 2006; Shipley et al., 2006). The association between *short-term memory performance* and mortality among non-demented adults is also well documented (Ghisletta et al., 2006; Portin et al., 2001; Shipley et al., 2006). In addition, two reviews have reported an association between dementia or mild cognitive disorders and mortality (Dewey & Saz, 2001; Guehne et al., 2006; Guehne, Riedel-Heller, & Angermeyer, 2005). Indeed, it has been contended that the relationship between intelligence and mortality is largely mediated by dementia (Backman & MacDonald, 2006).

Given the evidence for the relationship between intelligence and mortality, potential mechanisms driving this association warrant further examination. In early research on the relationship between cognitive decline and mortality, Riegel and Riegel (1972) described the effect in terms of "terminal drop". While the relationship between childhood intelligence and mortality cannot be explained by terminal decline alone, two theories posited by Riegel and Riegel (1972) form the basis of contemporary understanding of the intelligence–mortality relationship. Firstly, a biological theory suggested that physiological mechanisms related to cell aging were responsible for the decline and also for death. Secondly, a sociological theory suggested that performance and chance of survival drops earlier in life for those who cope less well with their environment due to disadvantages in, for example, education, income, nutrition and medical assistance.

More recently, three potential mechanisms for the relationship have been detailed by Whalley and Deary (2001) and Deary (2005) and tested by Kuh, Richards, Hardy, Butterworth, and Wadsworth (2004) and Shipley et al. (2006). First, socioeconomic status (SES) may mediate the relationship between intelligence and mortality. This theory, advocated by Siegrist and Marmot (2004), is similar to the sociological theory of Riegel and Riegel (1972), suggesting that disadvantages in intelligence lead to burdens in occupation, which are linked to poorer health outcomes. Siegrist and Marmot (2004) elaborate on the relationship by taking into account the mediating effect of control on health outcomes. The demand–control model (Karasek, 1979) proposes that high work demands interact with low levels of perceived control to cause such outcomes as depression and exhaustion, which adversely effect health outcomes and consequent mortality. A second explanation is that the relationship between intelligence and mortality is mediated by health behaviors and knowledge, which include substance use, diet, physical activity, healthcare utilization, and accident and illness prevention (Deary, 2005). Gottfredson and Deary (2004) argued that a high level of cognitive resources is required to prevent disease and to ameliorate illness through behaviors such as health monitoring, screening, medication adherence, understanding health information and becoming health literate. Failure to adequately undertake these health behaviors can lead to illness or more severe illness, resulting in hospitalization and health costs, and consequently, greater risk of mortality (Gottfredson & Deary, 2004).

A third explanation is that the relationship between intelligence and mortality may be due to a common association with health status. There are two possible explanations for an association between intelligence and health (Deary, 2005): (i) intelligence may be viewed as a marker of biological "fitness" or of system integrity, or (ii) intelligence may be an indicator of developmental problems that impact on later health. The former explanation aligns with the biological theory proposed by Riegel and Riegel (1972), with evidence from studies of the common cause hypothesis linking sensory function, lung function, grip strength and other biological markers with performance on cognitive tests (Christensen et al., 2000; Christensen, Mackinnon, Korten, & Jorm, 2001; Salthouse, Hancock, Meinz, & Hambrick, 1996). The latter explanation suggests that development in early life, such as fetal events, birth weight and early nutrition, shape future patterns of health and disease, which confound the relationship between intelligence and mortality (Deary, 2005). A refinement of (i) is that intelligence is associated with mortality because it reflects basic or core information processing mechanisms reflected in measures such as RT and grip strength (Deary & Der, 2005; Shipley et al., 2006). These two studies demonstrated that SES and health factors affect the relationship but that core processes such as reaction time are critical in predicting mortality.

The three proposed explanations of the link between mortality and intelligence are testable. The first predicts that education, employment history and other measures of lifetime opportunity will be associated with both intelligence and mortality and will consequently reduce the effect of intelligence on mortality. The second predicts that health behaviors, such as substance use history and healthcare utilization measured both currently and retrospectively over the lifespan, will likewise mediate the association between intelligence and mortality. The third set of explanations is more complex but suggests that disease status and a range of health or biological markers may account for a large proportion of the variance in the relationship between intelligence and mortality. The refinement of the explanation proposed by Deary and Der (2005) is that after accounting for core biological processes (reflected in biological measures such as grip strength, sensory processing and reaction time), the relationship between intelligence and mortality should be reduced or eliminated.

Two tests that capture the construct of intelligence are used in the present study. The Symbol–Letter Modalities Test (SLMT), a task similar to Smith's (1973) Symbol–Digit

Modalities Test and Wechsler's (Wechsler, 1981) Digit–Symbol Substitution, is a perceptual speed test that provides a measure of fluid intelligence. SLMT measures the efficiency of visual search and memory for the symbols presented in the task (Gilmore, Spinks, & Thomas, 2006) and performance on the task is correlated with measures of general intelligence such as the Wechsler Adult Intelligence Scale (Waldmann, Dickson, Monahan, & Kazelskis, 1992). The National Adult Reading Test (NART; Nelson, 1982) is a test of vocabulary that provides a measure of crystallized intelligence. NART performance is also correlated with measures of general intelligence and, unlike SLMT, is resistant to the effects of dementia (Bright, Jaldow, & Kopelman, 2002). Having the two tests allowed us to formulate differential predictions of the effect of these cognitive variables on mortality. Based on past research, SLMT performance was hypothesized to be a better predictor of mortality than NART. We also predicted that the relationship between SLMT and mortality would be more strongly mediated by health status than the relationship between NART and mortality, since NART performance is more resistant to the effects of declining health.

1. Method

1.1. Participants

The Canberra Longitudinal Study (CLS) was a large epidemiological survey of mental health and cognitive functioning that began in 1990. The study design is more fully detailed by Christensen et al. (2004). Eight hundred and ninety-six participants (456 men and 440 women) aged 70 or older at the time of the baseline assessment were recruited for the baseline assessment. All participants were initially living in the community in the cities of Canberra or Queanbeyan, Australia. Participants were sampled from the compulsory electoral roll, with 69% responding. Approval for the research was obtained from the Ethics in Human Experimentation Committee of The Australian National University.

1.2. Survey procedure

Participants were interviewed on up to four occasions over 12 years. Interviews were sought from both the participant and an informant, although the present study only examines participant data. Baseline interviews lasted approximately 2 hours, incorporating a survey measuring a wide range of risk factors including socio-demographics, physical health and disease status, mental health status, cognitive performance and social support. Interviews also included physical assessments of blood pressure, lung function, grip strength, vision and reaction time. Trained professional interviewers conducted the interviews.

Of the original sample of 896 participants, 185 (20.6%) were deceased by four years, 363 (40.5%) were deceased by eight years, and 544 (60.7%) were deceased by 12 years. At the end of vital status collection in June 2007, 687 (76.7%) of the participants were deceased. Of the participants who remained in the study, 14.1% (100/711) refused or were unable to complete the first follow-up interview, 21.1% (100/474) for the second follow-up and 21.1% (57/270) for the third follow-up.

1.3. Assessment of mortality

Mortality status and date of death were established by contacting relatives, searching the National Death Index, and from death notices in the local newspaper. The National Death Index, a register of all deaths in Australia, was searched by name and date of birth. Missing death identifications from the National Death Index would most likely have been a rare occurrence, as the index provides nationwide coverage. The additional methods used for death reporting (contacting relatives, newspaper searches) provide further confidence in the mortality status data. Mortality status was followed for up to 17 years, from the start of baseline interviews in September, 1990 until June 30, 2007. Survival was calculated as the time from the baseline interview to death for deceased participants, or from baseline until June 30, 2007 for surviving (right-censored) participants. For six participants with unknown day of death, the day was set to the 15th of the month. For two participants with an unknown month of death, the month was set to June. Taking deaths into account, the mean follow-up time was 9.7 years – 16.4 years for surviving participants and 7.6 years for deceased participants.

1.4. Assessment of intelligence

Two tasks were used to assess different domains of intelligence. The Symbol Letter Modalities Test is a test of perceptual speed that has been used as a measure of fluid intelligence. This test is based on earlier tests of fluid intelligence such as Digit–Symbol Substitution (Wechsler, 1981) and the Symbol–Digit Modalities Test (Smith, 1973). Participants were provided with a key which linked 10 symbols with letters of the alphabet (A to I). They were given 90 s to call out to the examiner the letters of the alphabet that corresponded to symbols printed in rows on the page. The key to the symbol–letter pairings was printed above the array. The test measures both fluid intelligence and cognitive speed. However, it allows an oral response to be made by the participants, thereby limiting possible contamination from impaired psychomotor functioning. The number of correct symbol–letter pairs made in 90 s was summed and the scores were standardized to produce an IQ-type score (SLMT IQ).

The National Adult Reading Test (NART; Nelson, 1982) assessed crystallized intelligence by testing the vocabulary of participants. The NART is a list of 50 words that are not pronounceable phonetically. Participants read the words aloud and testing is discontinued whenever there were 14 failures out of 15 items. The number of correct pronunciations made was summed and the scores were standardized to produce an IQ-type score (NART IQ).

1.5. Control variables

All of the control variables were measured in the baseline interview, with the exception of subsequent dementia diagnosis which was made on each wave of measurement.

1.5.1. Socio-economic status

Educational status was based on responses to two questions regarding the number of years in school and the highest qualification obtained. These two questions were combined

into a single measure representing the number of years it took participants to attain their highest educational qualification. Work history was asked as an open-ended question that was then given a standard job classification coding. From these codings, participants were classified into one of six categories: unskilled, semi-skilled, skilled, white collar, lower professional, managerial/professional. However, given the advanced age of the sample (the categorizations are based on a contemporary coding system) and a lack of predictive power provided by these categories, they were collapsed into a binary measure reflecting manual or non-manual employment. Participants who were involved in home duties were classified based on their spouse's occupational status.

1.5.2. Locus of control

A 14-item locus of control scale was administered, with participants rating items such as "I am confident of being able to deal successfully with future problems" on a six-point Likert scale from "Strongly disagree" to "Strongly agree". The scale was based on the 17-item Locus of Control of Behavior scale (Craig, Franklin, & Andrews, 1984) with three symptom-related items removed. Eight items were negatively-worded and were reverse scored. The score was a mean of the ratings (range 1–6), with higher scores indicating that the participant saw themselves as the locus of control.

1.5.3. Health behaviors

Participants reported whether they were current smokers, past smokers who had quit, or had never smoked. Level of activity was based on a six-item scale asking participants how often they engaged in activities "these days", with possible scores ranging from 0–18. The activities were reading, some sort of physical activity, active involvement in interests or hobbies, sitting at home (inactivity, negatively scored), and planned activities such as household tasks and visiting people. (Christensen et al., 1996) While there is a relationship between the activity scale and the level of physical disability ($r = -.44$), participation in social and intellectual pursuits are not captured by measures of physical disability.

1.5.4. Physical health

A brief self-reported medical history for each participant was taken during the survey. Heart attack history, stroke history (combining strokes, mini-strokes and transient ischemic attacks) and hypertension history were measured as dichotomous variables. A disease count covering 14 other diseases (including diabetes and cancer) and a symptom count for 21 symptoms (including falls, dizziness and chest pain) were generated. To measure functional ability, eight Activities of Daily Living (ADLs) were rated for difficulty on a four-point scale (no difficulty, some difficulty but no help needed, need help, bedridden) and five Instrumental Activities of Daily Living (IADLs) were rated on a three-point scale (no help needed, need help, cannot do). Two scales of functional ability were generated from these items, with ADL scores ranging from 0 to 24 and IADL scores ranging from 0 to 8. Higher scores on these scales indicate greater functional disability. Self-rated health was measured by asking participants to rate their general health on a four-point scale from "Excellent" (1) to "Poor" (4).

Sensory indicators of physical health included reaction time, grip strength, visual and auditory function. To measure choice reaction time, participants were asked to press a button with their left or right hand depending on which of two stimulus lights were illuminated (interstimulus intervals ranged from 0.5 to 2.0 s) (Christensen et al., 2000). The trials were performed mid-way through the survey, and choice reaction time was measured as the mean response time over 20 trials. Grip strength was taken using a Smedley hand dynamometer which measures the force exerted in kilograms (Christensen et al., 2000). Visual impairment was self-rated, with participants reporting "poor" eyesight or blindness classified as visually impaired. Hearing impairment was also self-rated, with participants who used a hearing aid or reported poor hearing classified as hearing impaired.

Table 1
Descriptive statistics for predictor variables by vital status after 17 years ($n = 896$).

	N	Survivors ($n = 191$–209)	Decedents ($n = 566$–687)	*p* value
		Mean (SD) or freq (%)	Mean (SD) or freq (%)	
SLMT IQ score	853	103.80 (14.10)	93.91 (16.97)	<0.001
NART IQ score	835	113.16 (8.35)	111.32 (10.08)	0.001
Age	896	74.09 (3.38)	77.30 (5.09)	<0.001
Gender: male	896	83 (39.7%)	373 (54.3%)	<0.001
Socio-economic status				
Years of education	894	11.17 (2.29)	11.41 (2.66)	0.275
Work history: manual work	896	141 (67.5%)	551 (59.7%)	0.039
Self as locus of control	759	4.36 (0.61)	4.21 (0.60)	<0.001
Health behaviors				
Smoking status	877			0.004
Never smoked		110 (52.9%)	391 (42.0%)	
Previously smoked		78 (37.5%)	383 (45.6%)	
Currently smoke		20 (9.6%)	103 (12.4%)	
Activity score	875	12.72 (2.43)	11.51 (3.17)	<0.001
Physical health				
Self-rated health	874			<0.001
Excellent		59 (28.5%)	160 (15.1%)	
Good		120 (58.0%)	478 (53.7%)	
Fair		27 (13.0%)	192 (24.7%)	
Poor		1 (0.5%)	44 (6.4%)	
Heart attack history	885	23 (11.1%)	157 (19.8%)	<0.001
Hypertension history	887	80 (38.5%)	371 (42.9%)	0.553
Stroke history	887	45 (78.4%)	269 (67.0%)	0.002
Disease count	896	1.69 (1.35)	2.12 (1.47)	<0.001
Symptom count	896	2.93 (2.74)	3.58 (2.87)	<0.001
ADL score	877	0.98 (1.31)	2.14 (2.78)	<0.001
IADL score	877	0.28 (0.67)	0.86 (1.61)	<0.001
Choice RT (s)	798	0.46 (0.12)	0.48 (0.15)	<0.001
Grip strength (kg)	877	25.95 (9.91)	24.29 (9.44)	0.012
Visual impairment	887	163 (78.4%)	455 (67.0%)	<0.001
Hearing impairment	887	156 (75.0%)	475 (70.0%)	0.069
Subsequent dementia diagnosis	896	3 (1.4%)	41 (6.0%)	0.013
Mental health				
Goldberg depression score	865	1.71 (1.79)	2.13 (2.00)	<0.001
Goldberg anxiety score	870	2.49 (2.35)	2.46 (2.25)	0.761

Notes: *p* values are from *Z* tests (binary and continuous variables) and χ^2 tests (categorical variables) from univariate Cox regressions using imputed data; SLMT IQ: Symbol–Letter Modalities Test IQ score; NART IQ: National Adult Reading Test IQ score; ADL: Activities of Daily Living; IADL: Instrumental Activities of Daily Living; RT: reaction time.

1.5.5. Mental health

Mental health ratings were included in the analysis as supplementary measures of health status. The Goldberg Depression Scale and Goldberg Anxiety Scale each consist of nine yes/no items measuring symptoms of depression and anxiety (Goldberg, Bridges, Duncan-Jones, & Grayson, 1988). Scores on these tests reflect a symptom count ranging from 0 to 9.

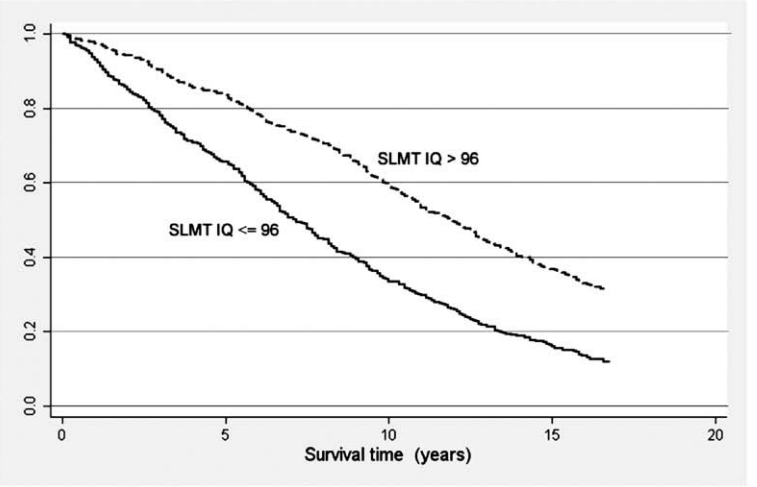
1.5.6. Dementia

To control for potential confounding by dementia, participants who were given an ICD-10 (World Health Organization, 1993) diagnosis of dementia or severe dementia later in the study (at waves 2, 3 and 4 4, 8 and 12 years after baseline) were identified. Diagnoses were made using the Canberra Interview for the Elderly (CIE) (Social Psychiatry Research Unit, 1992), which provides information from which a diagnosis of dementia can be made according to ICD-10 (World Health Organization, 1993) and DSM-III-R (American Psychiatric Association, 1987) by means of a computer algorithm.

1.6. Analyses

Descriptive analyses compared living participants to deceased participants to investigate which predictors were associated with death during the follow-up time. Survival time was graphed using Kaplan–Meier curves and modeled using Cox proportional hazards regression analyses. A series of six regression analyses included the intelligence measures with a combination of potential mediators or confounders, corresponding to the hypothesized mechanisms of the intelligence–mortality relationship. To facilitate interpretation of the Cox regression analyses, continuous variables (excluding age, years of education and disease and symptom counts) were standardized by subtracting the mean for the entire sample and dividing by the standard deviation. To account for potential confounding by end-of-life illness, specifically, sub-clinical disease states not captured by the health status measures, the analyses were repeated with the exclusion of participants who died

a) *Fluid intelligence: Symbol-Letter Modalities Test*



b) *Crystallized intelligence: National Adult Reading Test*

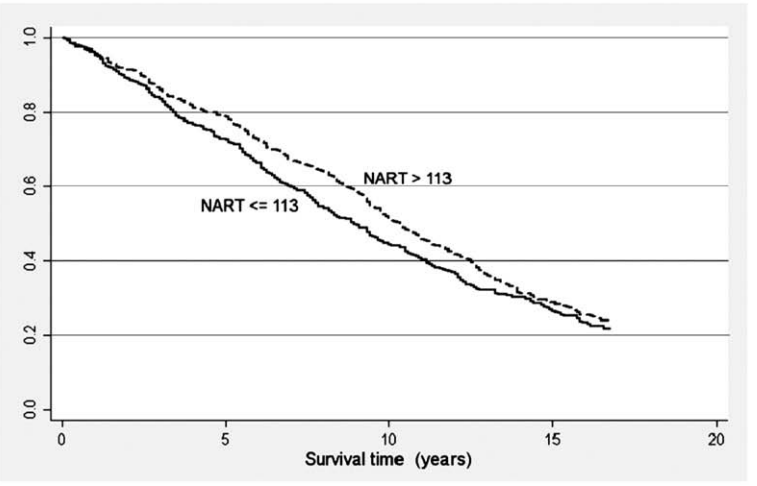


Fig. 1. Kaplan–Meier curves of cumulative survival over 17 years plotted separately for participants with IQ above and below the sample median ($n = 896$).

in the first four years of the study. The analyses were also repeated separately by gender to investigate whether the intelligence–mortality relationship differed for males and females.

Incomplete data for the survival analyses were imputed using the *ICE* procedure in Stata. Ten imputed data sets were generated by simultaneously modeling all of the independent variables from the baseline survey that were used in the analyses. The imputation procedure used linear regression to impute continuous variables, logistic regression to impute dichotomous variables and multinomial logistic regression to impute the two categorical variables (smoking status and self-rated health). Survival was not imputed, as complete data were available. Among the variables used in the analysis, 13% of the sample had one missing value and a further 10% of the sample had two or more missing values. The imputed data sets were combined using the *micombine* procedure in Stata, in conjunction with the *stcox* procedure that was used for the Cox proportional hazards regression models. SPSS version 15 was used for the descriptive analysis. Stata version 9 was used for the imputation and survival analyses.

2. Results

Descriptive statistics are shown in Table 1. These are based on the raw data, with *p*-values taken from Wald tests from univariate Cox regressions combined from analyses of the ten imputed data sets. Overall, the mean age was 76.6 years, with 11.4 years of education. With the exceptions of education, hypertension history, hearing impairment and Goldberg Anxiety score, all variables were significantly associated with mortality ($p<0.05$).

The effects of SLMT (Fig. 1a) and NART (Fig. 1b) on survival time were plotted using Kaplan–Meier curves. A median split was chosen to separate high and low performance on the two tasks. The figures show that the effect of SLMT on survival is much more pronounced than that of NART. There is a 20% difference in cumulative survival between high and low SLMT groups after approximately five years, and this difference is maintained until the end of the period of observation. The difference between high and low NART groups, however, is only apparent between approximately four and thirteen years after baseline.

Six models of mortality risk were tested using Cox proportional hazards regression models (Table 2). The univariate effect of SLMT was tested in Model 1, then age and gender were added to create Model 2. Model 3 built on Model 2, simultaneously testing the effect of SES (education and employment background) in conjunction with locus of control, a potential mediator of the effect of SES on mortality (Siegrist & Marmot, 2004). Health behaviors (smoking status and activity level) were entered into a separate model with SLMT, age and gender (Model 4). Health status, including physical health and mental health measures and dementia, was included simultaneously with SLMT, age and gender in Model 5. Finally, the significant predictors (at $p<.05$) from Models 2–5 were combined into a single model (Model 6). The six models were also fitted with the NART (Table 3).

Table 2
Cox proportional hazards regression models of mortality over 17 years using baseline fluid intelligence measure ($n=896$; 687 decedents).

Model	(1) Univariate	(2) With age & gender	(3) SES & locus of control	(4) Health behaviors	(5) System integrity	(6) Combined model
SLMT IQ score	0.69 (0.64, 0.75)***	0.76 (0.70, 0.82)***	0.74 (0.67, 0.81)***	0.81 (0.74, 0.89)***	0.81 (0.74, 0.89)***	0.80 (0.72, 0.88)***
Age		1.08 (1.07, 1.10)***	1.08 (1.06, 1.10)***	1.08 (1.06, 1.10)***	1.07 (1.05, 1.09)***	1.07 (1.05, 1.09)***
Gender = male		1.62 (1.39, 1.89)***	1.60 (1.37, 1.87)***	1.56 (1.31, 1.84)***	2.43 (1.87, 3.14)***	2.41 (1.89, 3.06)***
Yrs of education			1.06 (1.03, 1.10)***			1.06 (1.03, 1.09)***
Manual worker			1.02 (0.85, 1.22)			
Self as locus of control			0.91 (0.83, 1.00)*			0.93 (0.85, 1.02)
Smoking status						
Never				1.13 (0.95, 1.35)		
Previous				1.05 (0.81, 1.35)		
Current†				1.00		
Activity scale				0.83 (0.76, 0.90)***		0.91 (0.83, 1.00)*
Self-rated health						
Excellent					0.44 (0.27, 0.73)**	0.38 (0.24, 0.61)***
Good					0.53 (0.34, 0.83)**	0.46 (0.30, 0.69)***
Fair					0.71 (0.45, 1.13)	0.63 (0.41, 0.98)*
Poor†					1.00	1.00
Heart attack history					1.42 (1.16, 1.74)**	1.50 (1.24, 1.83)***
Hypertension history					1.24 (1.05, 1.45)*	1.28 (1.09, 1.51)**
Stroke history					1.13 (0.89, 1.43)	
Disease count					1.05 (0.99, 1.11)	
Symptom count					1.00 (0.97, 1.04)	
ADL score					1.11 (0.98, 1.25)	
IADL score					1.06 (0.94, 1.19)	
Choice RT					1.02 (0.90, 1.16)	
Grip strength					0.80 (0.70, 0.92)**	0.76 (0.67, 0.86)***
Visual impairment					1.12 (0.93, 1.34)	
Hearing impairment					0.99 (0.84, 1.18)	
Goldberg depression					1.03 (0.93, 1.14)	
Goldberg anxiety					0.89 (0.80, 0.99)*	0.91 (0.83, 0.99)*
Dementia diagnosis					1.14 (0.82, 1.58)	

†Reference category; *$p<.05$; **$p<.01$; ***$p<.001$.

SLMT IQ was significantly and substantially associated with mortality irrespective of which other variables were in the model. The effect of fluid intelligence on mortality was reduced but not fully accounted for when adjusting for age and gender, and including the 'competing' predictors measuring SES, health behavior, health status and sensory processing. A 15-point (one SD) disadvantage in SLMT IQ score was associated with between 23–45% increase in the risk of mortality (25% for the combined model), depending on which variables were included in the model. When age and gender were excluded from the models, attenuation of the SLMT hazard ratios from the inclusion of predictors representing the three proposed mechanisms ranged from 0–7%. In contrast, the effect of NART IQ on mortality was reduced when accounting for SES or health status and no longer significant after adjusting for health behaviors. A 15-point (one SD) disadvantage in NART IQ score was associated with between 8 and 14% increase in the risk of mortality, depending on the model. Attenuation for the NART hazard ratios ranged from 0–4%, after excluding the effects of age and gender. Subsequent dementia onset was not responsible for the association between intelligence and mortality.

Additional analyses were conducted to further account for potential confounding by baseline health status. Participants who died in the first four years of the study, prior to the first follow-up interview ($n = 190$ decedents), were excluded from the analysis. The effect of SLMT on mortality remained stable, as estimated by the six models presented in Tables 2 and 3. Hazard ratios remained between 0.72 and 0.84 with $p < .001$ for all models. However, the effect of NART was further attenuated, with only the univariate effect of NART significantly associated with mortality status (HR = 0.89, $p = .015$). When age and gender (HR = 0.91, $p = .055$), socioeconomic status (HR = 0.90, $p = .087$), health behaviors (HR = 0.94, $p = .223$) and health status (HR = 0.93, $p = .146$) were entered, the effect of NART became non-significant. A second supplemental analysis investigated whether the intelligence–mortality relationship was different for men and women. The effect of SLMT on mortality was consistent across males and females, with $p < .01$ for all six models across both genders and hazard ratios ranging from 0.64 to 0.84. However, the effect of NART tended to be weaker among men than women when adjusting for age ($HR_{female} = 0.86$, $p = .014$; $HR_{male} = 0.91$, $p = .078$) and when adjusting for health behaviors ($HR_{female} = 0.88$, $p = .039$; $HR_{male} = 0.95$, $p = .349$). The univariate effect of NART was significant for both genders, but after adjusting for socioeconomic variables or health status, the effect of NART did not reach significance for either males or females (HRs ranging from 0.88 to 0.90).

3. Discussion

The present study examined the intelligence–mortality relationship using data collected from the Canberra Longitudinal Study (CLS) over 17 years in an older cohort of community dwellers. Better SLMT performance was found to be significantly associated with lower mortality risk. This effect persisted after 17 years of follow-up, extending the findings

Table 3
Cox proportional hazards regression models of mortality over 17 years using baseline crystallized intelligence measure ($n = 896$; 687 decedents).

Model	(1) Univariate	(2) With age and gender	(3) SES and locus of control	(4) Health behaviors	(5) System integrity	(6) Combined model
NART IQ score	0.88 (0.81, 0.95)**	0.89 (0.82, 0.97)**	0.90 (0.81, 0.99)*	0.93 (0.85, 1.01)	0.90 (0.83, 0.98)*	0.89 (0.80, 0.99)*
Age		1.10 (1.08, 1.12)***	1.10 (1.08, 1.11)***	1.09 (1.07, 1.11)***	1.07 (1.05, 1.09)***	1.08 (1.06, 1.10)***
Gender = male		1.61 (1.38, 1.87)***	1.64 (1.40, 1.92)***	1.56 (1.32, 1.85)***	2.65 (2.05, 3.41)***	2.51 (1.97, 3.18)***
Yrs of education			1.05 (1.01, 1.08)*			1.05 (1.02, 1.09)**
Manual worker			1.09 (0.90, 1.31)			
Self as locus of control			0.87 (0.79, 0.95)**			0.92 (0.83, 1.01)
Smoking status						
Never				1.13 (0.94, 1.35)		
Previous				1.09 (0.85, 1.40)		
Current†				1.00		
Activity scale				0.78 (0.72, 0.85)***		0.86 (0.79, 0.94)**
Self-rated health						
Excellent					0.42 (0.26, 0.68)***	0.35 (0.23, 0.56)***
Good					0.51 (0.33, 0.80)**	0.43 (0.29, 0.64)***
Fair					0.69 (0.44, 1.10)	0.59 (0.38, 0.90)*
Poor†					1.00	1.00
Heart attack history					1.41 (1.15, 1.73)**	1.51 (1.24, 1.84)***
Hypertension history					1.22 (1.04, 1.44)*	1.27 (1.08, 1.50)**
Stroke history					1.14 (0.90, 1.44)	
Disease count					1.05 (0.99, 1.11)	
Symptom count					0.99 (0.96, 1.03)	
ADL score					1.12 (1.00, 1.27)	
IADL score					1.08 (0.96, 1.21)	
Choice RT					1.10 (0.97, 1.24)	
Grip strength					0.80 (0.70, 0.92)**	0.75 (0.66, 0.85)***
Visual impairment					1.09 (0.91, 1.31)	
Hearing impairment					1.02 (0.86, 1.21)	
Goldberg depression					1.04 (0.94, 1.16)	
Goldberg anxiety					0.89 (0.81, 0.99)*	0.91 (0.83, 0.99)*
Dementia diagnosis					1.16 (0.83, 1.61)	

†Reference category; *$p < .05$; **$p < .01$; ***$p < .001$.

of Korten et al. (1999) who examined the same cohort after four years. The relationship between SLMT and mortality remained when participants who died early in the study were omitted and was similar for men and women. NART performance was also significantly associated with mortality, although the effect was mitigated by controlling for health behaviors such as smoking status and activity level. Excluding participants who died in the first four years of the study diminished the effect of NART to non-significance. In addition, the effect of NART was stronger for women than men.

A set of six models tested three major proposed mechanisms for the relationship between intelligence and mortality. We tested for the effect of SES, as measured by education and type of employment. The effect of health behavior was tested using measures of smoking history and physical, mental and social activity. We tested for the effect of health status using measures of self-rated health, disease history, functional disability, grip strength and mental health status. In response to the findings of Deary and Der (2005), we controlled for sensory processing ability using measures of choice reaction time, visual impairment and hearing impairment. We also controlled for dementia diagnosis in response to the theory of Backman and MacDonald (2006) that the intelligence–mortality relationship is due largely to dementia-related deficits. The present study did not find strong support for any of the three explanations. Moreover, evidence for a particular explanation was contingent on the type of intelligence test used. For SLMT, although there was slight attenuation of the effect when controlling for SES, health behaviors or health status, the effect of SLMT remained significant, providing limited support for the three major mechanisms. The effect of intelligence on mortality when measured by the NART was also slightly attenuated by the effects of SES and health status. However, after adjusting for smoking status and activity level, the attenuation was sufficient that NART was no longer significantly associated with mortality.

The divergence in findings suggests the mediation of the relationship between intelligence and mortality by SES, health behaviors and health status is marginally stronger for fluid intelligence performance than for tests of crystallized intelligence. Fluid intelligence tasks such as the SLMT may reflect any initial and early adulthood effects of intelligence on mortality, combined with the effects of physical health decline and ageing processes not due to physical health. NART performance, on the other hand, is likely to reflect initial intelligence, education across the lifespan and consistent implementation of health behaviors, but is less susceptible to effects of independent or systemic disease and biological ageing processes.

Choice reaction time was associated with mortality, however, there was no effect of reaction time when models also included SLMT or NART score. Grip strength was associated with mortality but accounted for little of the variance in the intelligence–mortality relationship. Sensory impairments were not associated with mortality. Manual occupation was also not associated with mortality after controlling for intelligence. This finding is not due to the collapsing of occupational categories into a binary measure, as a six-category version of the measure also had no association with mortality. Smoking status was not associated with mortality, a finding divergent from past research (Tessier et al., 2000) which may be explained by the advanced age and low smoking prevalence of this sample. In the final model, which included measures of SES, health behavior and health status, a one standard deviation decrement in SLMT performance was associated with a 25% increase in mortality, while a one standard deviation decrement in NART performance was associated with an 12% increase in mortality

In this cohort, the intelligence–mortality relationship appears to be based on more than lower-level processing efficiency. There was a strong independent effect of SLMT even after adjusting for the hypothesized mechanisms of the relationship together with reaction time and sensory impairment. In addition to mental speed, SLMT measures the efficiency of visual search and memory for the symbols presented in the task (Gilmore et al., 2006). Since reaction time was controlled for, the aspects of SLMT that are associated with mortality would appear to be a combination of processing speed with memory and attention performance. Further research into which constituents of the SLMT task best predict mortality could adapt the frameworks used by Gilmore et al. (2006) and Salthouse and Kersten (1993) to modify the SLMT task into components that separately measure the processing speed, memory and attentional aspects of the task.

While the present findings were often in accordance with previous research, there were some important differences. Previous research of the relationship between poor SLMT performance and mortality risk was supported (Anstey et al., 2001; Ghisletta et al., 2006; Pavlik et al., 2003; Portin et al., 2001). Previous investigations have found little evidence for a relationship between NART performance and mortality (Abas et al., 2002; Anstey et al., 2001). While NART performance was significantly associated with mortality risk in the present study, the effect was tenuous after controlling for measures of SES, health behavior and health status. However, contrary to previous findings (Deary & Der, 2005), reaction time did not explain the effect of either of the intelligence tasks. The differences in findings may be attributable to the age of the cohort in the present study. All of the participants were 70 or older at baseline, averaging over 75 years of age, and were followed until they were in their late-80s or beyond. Previous research has shown that the effect of intelligence on mortality is most pronounced in older age groups (Lyyra et al., 2006; Shipley et al., 2006).

3.1. Limitations of the findings and directions for future research

The present study examined vital status over 17 years using baseline measurements as predictors. However, intelligence was assessed at the start of the study, when participants were already advanced in age and potentially in poor health. Participants who were close to death at the time of the baseline may have been in a state of terminal decline, leading to an overestimation of the effect of poor cognitive performance on mortality. While the follow-up analysis omitted participants who died in the early stages of the study, the baseline intelligence measurement may still have been influenced by sub-clinical health problems. Additional research into temporal variations in the intelligence–mortality relationship would further delineate the influence of time-to-death on cognitive performance, as would modeling cognitive

performance as an outcome, using time-to-death as a predictor. Another problem for the survival models is that there may have been cases where death occurred but was not recorded. Despite a thorough search protocol, the extent of missing death records could not be assessed in the present study, so all cases were treated as living if there was no evidence to the contrary. Having noted this, however, treating potential decedents as survivors would result in a more conservative estimate of the association between intelligence and mortality.

The models that were tested were operationalized using available measures from the baseline survey. Although it is difficult to articulate the models sufficiently well to test then more than in a general way, testing them is important, as it forces a theoretical articulation and reveals the difficulties of testing complex relationships among processes over long periods. Nevertheless, some of the measures that were used in this study could be further refined for the purposes of future research. Only a self-reported measure of vision impairment was included in the baseline interview, which may not accurately reflect visual functioning. Sensory function, particularly visual acuity, can influence performance on tasks like SLMT (Gilmore et al., 2006). Self-report measures of health status (disease history, functional ability) may also have been inaccurate, although objective health measures (grip strength, reaction time) were also included as predictors in the models. Additional measures of health behaviors would also have strengthened the analysis — the baseline interview did not include measures of alcohol and other substance use, diet, healthcare utilization or medication adherence. Reflecting the constraints of a large in-home epidemiological survey, the tests available to measure intelligence assessed only specific domains of cognitive performance. General intelligence tests, such as the WAIS and AH4, cover a broader array of abilities. However, simply examining the construct of general intelligence is not sufficient in investigating what aspects of intelligence are most strongly associated with mortality. Further research on the intelligence–mortality relationship should continue to examine a broad range of cognitive abilities, including memory, attention, reasoning, knowledge and executive function, in a variety of domains, including episodic, verbal and visuospatial.

Finally, examining all-cause mortality is a starting point for investigating the relationship between intelligence and mortality. There is strong evidence for a relationship between intelligence and cardiovascular mortality but less for the relationship between intelligence and cancer mortality (Hart et al., 2003; Shipley, Der, Taylor, & Deary, 2007). Examining modifiable mediators of both mortality and cognitive performance has the potential to guide future health interventions. Continuing to research the associations between various domains of cognitive performance and mortality will advance our understanding into the nature of the intelligence–mortality relationship.

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