歩行量を高く保つことが，前頭葉萎縮進行を抑制することが示された．また5,800歩が，前頭葉萎縮の進行を抑制する一日当たりの歩行量閾値として示され，認知機能低下の予防に繋がる身体活動量の目標値の一つとなる可能性が示唆された．

## 謝　辞

NILS-LSAにご協力頂いた参加者の皆様，ならびに研究スタッフの皆様に心より感謝申し上げます．また本研究の遂行にあたり，助成を賜りました公益財団法人石本記念デサントスポーツ科学振興財団に心より感謝申し上げます．

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Japanese Psychological Research THE JAPANESE PSYCHOLOGICAL ASSOCIATION

*Japanese Psychological Research*
2013, Volume 55, No. 4, 378–389

doi: 10.1111/jpr.12028

# Does high educational level protect against intellectual decline in older adults?: A 10-year longitudinal study[1]

YUKIKO NISHITA*, CHIKAKO TANGE, and MAKIKO TOMIDA *National Center for Geriatrics and Gerontology*

FUJIKO ANDO *Aichi Shukutoku University*

HIROSHI SHIMOKATA *Nagoya University of Arts and Sciences*

Abstract: This study examined the relation between educational level and intellectual change in Japanese older adults. Participants (age = 65–79 years, $n$ = 593) comprised the first-wave participants of the National Institute for Longevity Sciences-Longitudinal Study of Aging (NILS-LSA). They were followed for 10 years and were tested six times. Educational levels were divided into two groups (low-educated or high-educated), and intellectual changes for the 10 years were assessed using the Japanese Wechsler Adult Intelligence Scale-Revised Short Forms (JWAIS-R-SF); subtests included Information, Similarities, Picture Completion, and Digit Symbol. General linear mixed-model analyses revealed that education had not affected 10-year changes of the Information, Similarities, and Picture Completion subtest scores. In contrast, education was significantly associated with a change in the Digit Symbol subtest score; individuals with higher levels of education showed greater decline than those with less education, although they had higher ability at every time point. These findings suggest that higher education does not protect against intellectual decline in late life, although it is associated with long-term individual differences in intelligence.

Key words: intelligence, education, older adults, longitudinal study.

Many studies have suggested that early-life educational level is associated with better intellectual abilities in late life (e.g., Kaufman & Lichtenberger, 2006; Schaie, 2005; Wechsler, 1981). However, recent articles based on longitudinal data have shown conflicting results with respect to the relation between educational level and intellectual changes in old age. Some longitudinal studies have reported that educational attainment moderates intellectual decline in samples of older adults (e.g., Alvarado, Zunzunequi, Del Ser, & Beland, 2002; Arbuckle, Maag, Pushkar, & Chaikelson, 1998; Evans, Beckett, Albert, Hebert, Scherr, Funkenstein, & Taylor, 1993; Farmer, Kittner, Rae, Bartko, & Regier, 1995; Koster, Penninx, Bosma, Kempen, Newman, Rubin, Satterfield, Atkinson, Ayonayon, Rosano, Yaffe, Harris, Rooks, Van Eijk, & Kritchevsky, 2005; Lee, Kawachi, Berkman, & Grodstein, 2003; Lyketsos, Chen, & Anthony, 1999). However, others disagree with these findings, suggesting

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*Correspondence concerning this article should be sent to: Yukiko Nishita, Section of NILS-LSA, Center for Gerontology and Social Science, National Center for Geriatrics and Gerontology, Gengo, Morioka-cho, Obu 477-8511, Japan. (E-mail: nishita@ncgg.go.jp)

[1] This work was supported by Japan Society for the Promotion of Science KAKENHI (Grant Number 23730640).

that higher education does not protect against intellectual decline (e.g., Seeman, Huang, Bretsky, Crimmins, Launer, & Guralnik, 2005; Tucker-Drob, Johnson, & Jones, 2009; Van Dijk, Van Gerven, Van Boxtel, Van der Elst, & Jolles, 2008; Wilson, Hebert, Scherr, Barnes, Mendes de Leon, & Evans, 2009; Zahodne, Glymour, Sparks, Bontempo, Dixon, Macdonald, & Manly, 2011), or that relations between education and intellectual change appear to differ by intellectual domain (e.g., Alley, Suthers, & Crimmins, 2007; Anstey & Christensen, 2000; Anstey, Hofer, & Luszcz, 2003).

From the perspective of the *cognitive reserve* hypothesis, it is noteworthy that previous longitudinal studies have reported the mixed results described above. The hypothesis of cognitive reserve asserts that older individuals with greater experiential resources exhibit better cognitive functioning and are able to tolerate brain pathology before displaying clinical symptoms (Scarmeas & Stern, 2004; Stern, 2002). Stern (2002) postulated that high cognitive reserve may allow individuals to cope more successfully with age-related brain changes, and that one of the most well-established proxy measures of cognitive reserve capacity in the elderly was educational attainment, which is thought to reflect more effective use of brain networks or cognitive paradigms.

Two competing cognitive reserve models could offer insight into the effect of education on the rate of cognitive change (Stern, 2002; Van Dijk et al., 2008). First, if high education was found to slow the rate of cognitive decline, this finding would support an *active* cognitive reserve hypothesis. In this case, individuals with higher education would be hypothesized to process tasks more efficiently. Further, because they make more efficient use of brain networks, the same amount of organic cognitive damage would result in a smaller decline in cognitive function relative to those with less education. Second and alternately, if educational level does not relate to the rate of cognitive change, this would support a *passive* cognitive reserve hypothesis. If aging individuals begin to lose cognitive function from a common cause, such as normal aging brains, people with higher education would change at a rate similar to the total population, but would continue to perform at a higher level at any age because of greater baseline brain reserve. These theories of *active* and *passive* cognitive reserve processes are often evaluated with respect to the implications for *moderation* versus *stability* (Salthouse, 2003; Tucker-Drob et al., 2009) or *differential-preservation* versus *preserved-differentiation* (Bielak, Anstey, Christensen, & Windsor, 2012; Salthouse, 2006).

Inconsistencies in previous longitudinal studies may be due to some methodological differences among the studies. For example, studies differed in the number of consecutive assessments, or the measures of intellectual abilities used.

In terms of the number of assessments, some studies (e.g., Alvarado et al., 2002; Arbuckle et al., 1998; Evans et al., 1993; Farmer et al., 1995; Koster et al., 2005; Lee et al., 2003; Lyketsos et al., 1999) examined intellectual change by calculating a difference between only two test occasions and then used traditional regression analysis or repeated measures analysis of variance techniques. However, ideally, to estimate a true change, intellectual ability should be assessed at multiple time points rather than using a simple difference in two test administrations (Alley et al., 2007; Wilson et al., 2009). The use of three or more assessments of longitudinal intellectual aging can reduce measurement error (Winkens, Schouten, Van Breukelen, & Berger, 2006) as well as avoid the regression toward the mean phenomenon (Dufouil, Fuhrer, Dartigues, & Alpérovitch, 1996; Zahodne et al., 2011). Moreover, the use of three or more assessments makes possible the use of more sophisticated analytic techniques, such as multilevel modeling or general linear mixed modeling (Laird & Ware, 1982; Morrell, Brant, & Ferrucci, 2009; Verbeke & Molenberghs, 1997).

A second methodological difference concerns which domain of intellectual ability was being measured; it remains possible that education may have different effects on the changes in different intellectual domains. For example, in their review of the literature, Anstey and Christensen (2000) found that education appears to be more predictive for crystallized ability, but less predictive for fluid ability or processing speed. Similarly, Wilson et al. (2009) pointed out that their results were based on overall global cognition, so they could not establish whether education was related to decline in some intellectual domains but not others. Additionally, some studies (e.g., Evans et al., 1993; Farmer et al., 1995; Lee et al., 2003) have used mental status measures that assess the most basic level of cognitive abilities (e.g., the Mini Mental State Examination; Folstein, Folstein, McHugh, Practical, & Patients, 1975). Such basic level measures may be insensitive to change among well-educated older adults due to ceiling effects that prevent detection of changes within the upper levels of functioning, resulting in spurious relations between initial performance and change (Tucker-Drob et al., 2009). Thus, multiple and more sensitive assessments that reflect greater variability in intellectual functions might better address educational differences in future research.

### *The present study*

The purpose of the present study was to determine whether educational level is associated with the rate of intellectual change in community-dwelling older Japanese. The important characteristics of this study included the following: (a) the participants were followed for 10 years, tested six times, and general linear mixed models were used to analyze the data; and (b) to measure intelligence in late life, we used neuropsychological tests to cover the multiple intellectual abilities of the adults: the Japanese Wechsler Adult Intelligence Scales-Revised Short Forms (JWAIS-R-SF; Kobayashi, Fujita, Maekawa, & Dairoku, 1993). The JWAIS-R-SF includes four standardized subtests (Information, Similarities, Picture Completion, and Digit Symbol). To our knowledge, this may be the first study that approaches the effect of educational levels on intellectual changes for Japanese older adults.

## Methods

### *Participants*

The data for the present study were collected as a part of the National Institute for Longevity Sciences-Longitudinal Study of Aging (NILS-LSA; Shimokata, Ando, & Niino, 2000). The NILS-LSA is a population-based prospective cohort study of aging and age-related diseases. The participants were sex- and age-stratified random samples of Japanese community-dwelling adults aged from 40 to 79 years at baseline (Wavel: 1997–2000). This baseline sample consisted of 2267 participants who were followed up every 2 years (Wave2: 2000–2002, Wave3: 2002–2004, Wave4: 2004–2006, Wave5: 2006–2008, Wave6: 2008–2010). Informed consent was obtained from each participant at the beginning of the study.

We selected an initial sample of individuals who were aged 65 years or older at baseline ($n = 816$). We excluded individuals who: (a) provided data only at baseline ($n = 210$) because longitudinal analyses required a minimum of two valid scores per individual, (b) had a history of dementia at baseline ($n = 1$), or (c) had missing data on all dependent variables at baseline or on the independent variables ($n = 12$). Based on these criteria, the data from 593 individuals were included at baseline. Mean age at baseline was 70.96 years ($SD = 3.90$ years, age range = 65–79 years), with 46.54% of the sample being women.

### *Measures*

*Intelligence.* The Wechsler Adult Intelligence Scale (WAIS) is one of the most popular tools for assessing intelligence (Wechsler, 1944). In this study, intelligence was assessed using the JWAIS-R-SF (Kobayashi et al., 1993). The trained testers (clinical psychologists or psychology graduate students) administered the test to each participant one on one. The JWAIS-R-SF consists of the following four subtests: Information, Similarities, Picture Completion, and Digit Symbol.

1 Information: Participants were asked general knowledge questions covering people, places, and events (29 items, possible range 0–29). This subtest measured the fund of factual knowledge.
2 Similarities: Participants were asked to tell what way two things are alike (14 items, possible range 0–28). This subtest measured logical abstract reasoning.
3 Picture Completion: Participants were asked to spot the missing element in a series of drawings (21 items, possible range 0–21). This subtest measured the long-term visual memory and the ability to differentiate essential from inessential details.
4 Digit Symbol: Participants were asked to write down the symbol that corresponded to a given number (as many as they could in 90 s, possible range 0–93). This subtest measured processing speed and visual-motor coordination.

*Educational levels.* Participants self-reported their level of education on a scale with four options (1 = *elementary school or junior high school*, 2 = *high school or junior high school under the former Japanese educational system*, 3 = *higher vocational school or junior college*, and 4 = *college or graduate college*). The baseline sample reported 47.39% ($n = 281$) elementary school or junior high school, 35.92% ($n = 213$) high school or junior high school under the former Japanese educational system, 10.96% ($n = 65$) higher vocational school or junior college graduates, and 5.73% ($n = 34$) college or graduate college graduates. Given this distribution, we divided educational levels into two groups: the low-educated group (level = 1) and the high-educated group (levels = 2–4).

*Covariates.* At baseline assessment, marital status (0 = unmarried, 1 = married), occupation (0 = inoccupation, 1 = having occupation), smoking (0 = nonsmoker, 1 = smoker) and past and present illness (stroke, hypertension, heart disease, and diabetes: 0 = none, 1 = having past or present illness) were examined using questionnaires.

## Results

Statistical analyses were performed using the SAS System version 9.1.3. A $p$-value of <0.05 was considered statistically significant.

### Sample characteristics

Table 1 presents the baseline sample characteristics by level of education. There were no significant differences in age, sex, and other covariates by educational levels. All baseline intelligence scores in the high-educated group were significantly greater than in the low-educated group (all $ps < .001$).

In addition, compared with the excluded group ($n = 210$), which provided data only at baseline, this study sample showed higher scores on all intelligence measures (Information, 10.30 vs. 13.13, $t(793) = 6.47, p < .001$; Similarities, 8.73 vs. 11.17, $t(792) = 5.41$, $p < .001$; Picture Completion, 7.40 vs. 9.74, $t(794) = 7.87$, $p < .001$; Digit Symbol, 34.17 vs. 39.62, $t(792) = 6.49, p < .001$). However, there were no significant differences between the level of education, $\chi^2(3) = 4.24$, *ns*.

### Participation in follow up

There was an average of 4.18 repeated measurements per participant (range 2–6). The mean duration of follow up from baseline to final assessment for each participant was 6.80 years (range 1.92–11.21 years). Information about follow-up participation is summarized in Table 2. The participation rates in Wave5 and Wave6 in the high-educated group were significantly higher than in the low-educated group (Wave5, $\chi^2(1) = 4.53$, $p = .033$; Wave6, $\chi^2(1) = 5.39, p = .020$). However, there were no significant differences between the level of education and the participation rate in other follow-up measures (Wave2, $\chi^2(1) = .50$, *ns*; Wave3, $\chi^2(1) = .04$, *ns*; Wave4, $\chi^2(1) = .46$, *ns*).

### Educational levels and intellectual change

General linear mixed models were used to evaluate the effects of the level of education on the rate of intellectual change over time. We obtained fixed effects (i.e., average effects for the group of educational levels) and random

**Table 1** Descriptive statistics of baseline sample by level of education

| Variable | High-educated ($n$ = 312) | Low-educated ($n$ = 281) | $t/\chi^2$ *test* | |
|---|---|---|---|---|
| Age at baseline, mean (*SD*) | 70.70 (3.96) | 71.26 (3.82) | $t(591) = 1.75$ | *ns* |
| Sex, women, *n* (%) | 138 (44.23) | 138 (49.11) | $\chi^2(1) = 1.41$ | *ns* |
| Marital status, married, *n* (%) | 253 (81.09) | 216 (76.87) | $\chi^2(1) = 1.59$ | *ns* |
| Occupation, having occupation, *n* (%) | 86 (27.56) | 79 (28.11) | $\chi^2(1) = 0.02$ | *ns* |
| Smoking, smoker, *n* (%) | 57 (18.27) | 49 (17.44) | $\chi^2(1) = 0.07$ | *ns* |
| Past and present illness, *n* (%) | | | | |
| Stroke | 17 (5.45) | 15 (5.34) | $\chi^2(1) = 0.00$ | *ns* |
| Hypertension | 130 (41.67) | 101 (35.94) | $\chi^2(1) = 2.04$ | *ns* |
| Heart disease | 66 (21.15) | 47 (16.73) | $\chi^2(1) = 1.88$ | *ns* |
| Diabetes | 36 (11.54) | 34 (12.10) | $\chi^2(1) = 0.04$ | *ns* |
| Intelligence at baseline, mean (*SD*) | | | | |
| Information | 15.32 (5.66) | 10.70 (4.21) | $t(591) = 11.17$ | *** |
| Similarities | 13.36 (5.27) | 8.73 (4.70) | $t(590) = 11.23$ | *** |
| Picture Completion | 10.51 (3.41) | 8.88 (3.73) | $t(591) = 5.57$ | *** |
| Digit Symbol | 44.09 (10.16) | 34.61 (8.32) | $t(589) = 12.32$ | *** |

*Note.* The final sample consisted of 593 participants who had at least two visits. Data were missing as follows: Similarities, $n = 1$; Digit Symbol, $n = 2$.
***$p < .001$. *ns* = not significant.

**Table 2** Follow-up participation information

| | Participants, *n* (high-educated/low-educated) | Follow-up years from baseline, mean (*SD*) |
|---|---|---|
| Baseline | 593 (312/281) | 0.00 |
| Wave2 | 566 (296/270) | 2.05 (0.11) |
| Wave3 | 443 (232/211) | 4.08 (0.18) |
| Wave4 | 363 (195/168) | 6.20 (0.25) |
| Wave5 | 289 (165/124) | 8.28 (0.28) |
| Wave6 | 223 (131/92) | 10.27 (0.28) |

*Note.* The final sample consisted of 593 participants who had at least two visits.

effects (i.e., individual deviation from the fixed effects) to model individual intellectual change. That is, intellectual change was assumed to follow the mean path of the group, except for person-specific random effects that cause the initial individual level of functioning to be higher or lower and the rate of change to be faster or slower. In addition, general linear mixed models can handle missing data more appropriately than traditional models (e.g., general linear models), so they can use all available data during follow up. Moreover, the correlation between the repeated measures is properly accounted for through the variance-covariance structure of the random effects. A general linear mixed model was chosen for the analysis of intellectual change in some recent studies (e.g., Alfaro-Acha, Snih, Raji, Kuo, Markides, & Ottenbacher, 2006; Crane, Gruhl, Erosheva, Gibbons, McCurry, Rhoads, Nguyen, Arani, Masaki, & White, 2010; Ganguli, Du, Dodge, Ratcliff, & Chang, 2006; Nishita, Tange, Tomida, Ando, & Shimokata, 2012a; Van Dijk et al., 2008; Wilson, Beckett, Barnes, Schneider, Bach, Evans, & Bennett, 2002). Further information on the application of general linear mixed models to repeated measures data is published elsewhere (e.g., Laird & Ware, 1982; Morrell et al., 2009; Verbeke & Molenberghs, 1997).

**Table 3** Educational levels and 10-year change in intelligence as estimated from linear mixed effects models

| Intelligence scale | Model terms | Parameter estimate | *SE* | *p*-value |
|---|---|---|---|---|
| Information | Education | 4.44 | 0.39 | *** |
| | Time | −0.09 | 0.03 | ** |
| | Education x time | −0.02 | 0.04 | *ns* |
| Similarities | Education | 4.36 | 0.38 | *** |
| | Time | −0.09 | 0.04 | * |
| | Education x time | 0.00 | 0.04 | *ns* |
| Picture completion | Education | 1.37 | 0.26 | *** |
| | Time | 0.10 | 0.03 | *** |
| | Education x time | −0.04 | 0.03 | *ns* |
| Digit symbol | Education | 8.66 | 0.71 | *** |
| | Time | −0.27 | 0.06 | *** |
| | Education x time | −0.22 | 0.07 | ** |

*Note.* Higher scores indicate better performance. Possible score for the Information is 0–29; Similarities 0–28; Picture Completion 0–21; Digit Symbol 0–93. Time = years since baseline; Education = 0 (low level education: reference) or 1 (high level education). In addition to the terms shown in the table, each model included terms to control for the fixed effects of age at baseline, sex, marital status, occupation, smoking, each past and present illness and practice effect were included as covariates, and the random effects of the intercept (baseline performance) and slope (change over time).

***$p < .001$.

**$p < .01$.

*$p < .05$. *ns* = not significant.

The model used in the current study included fixed terms for the Intercept (baseline performance for an individual with value zero on all predictors), Education (0 = low-educated group, 1 = high-educated group), Time (time in years since baseline), and an Education × Time interaction term. Age (at baseline), sex (0 = men, 1 = women), marital status (0 = unmarried, 1 = married), occupation (0 = inoccupation, 1 = having occupation), smoking (0 = nonsmoker, 1 = smoker) and each past and present illness (0 = none, 1 = having past or present illness) were included as covariates. In addition to controlling for practice effects, we added indicators of prior exposure to the tests. To do this, we followed the procedure described by Alley et al. (2007) to account for the effects of repeated test exposures, by assigning the respondents a 0 for baseline participation and then 1 at each subsequent administration of tests for intellectual assessment. Moreover, random effects of intercept (baseline performance) and slope (change over time) were calculated using an unstructured covariance matrix. The term of primary interest for this study was the Education × Time interaction, which reflects whether the high- or low-educational groups differ in the rate of change in intellectual performance over time. Table 3 shows the general linear mixed models estimates for each intelligence score as a function of educational levels over a 10-year period. Figure 1 gives visual representations of intellectual changes as a function of educational levels.

The term for Education was significant for all subtests (Information, $\beta = 4.44$, $p < .001$; Similarities, $\beta = 4.36$, $p < .001$; Picture Completion, $\beta = 1.37$, $p < .001$; Digit Symbol, $\beta = 8.66$, $p < .001$). As can be seen in Figure 1, participants in the high-educated group scored better compared with those in the low-educated group on all intellectual abilities. The term for Time was also significant for all subtests (Information, $\beta = -0.09$, $p = .003$; Similarities, $\beta = -0.09$, $p = .015$; Picture Completion, $\beta = 0.10$, $p < .001$; Digit Symbols, $\beta = -0.27$, $p < .001$). As can be seen in Figure 1, scores of Information, Similarities, and Digit Symbol subtests showed a trend for a decline over time as the study progressed. In contrast, there was a significant improvement in performance over time on the Picture Completion subtest.

Of particular importance to the current study was the test of the Education × Time interaction, which would show whether intellectual change over time varied with educational levels. The interaction was not statistically significant for the subtests of Information, Similarities, and Picture Completion. However, on the Digit Symbol subtest score, there was significant interaction ($\beta = -0.22$, $p = .002$), indicating that the rate of change of the Digit Symbol scores was significantly related to educational level. The direction of this association indicated that there was greater decline for individuals in the high-educated group (*slope* $= -0.49$, $p < .001$) than in the low-educated group (*slope* $= -0.28$, $p < .001$).

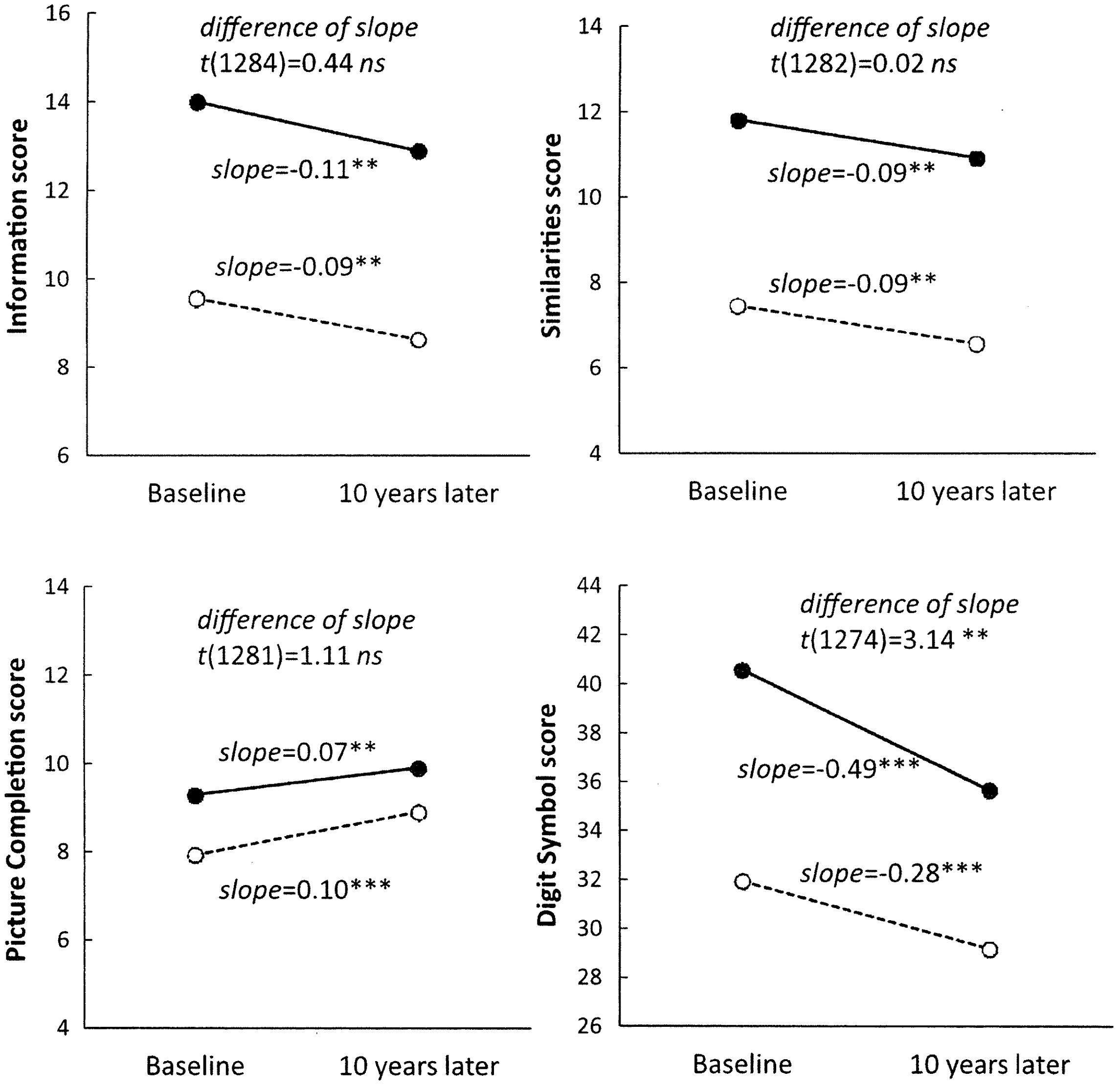

**Figure 1** Model-predicted 10-year change in intelligence by education levels. (a) The solid lines are estimated for individuals with a high education level; the dashed lines are estimates for individuals with a low education level. (b) Higher scores indicate better performance. Possible score for the Information is 0–29; Similarities 0–28; Picture Completion 0–21; Digit Symbol 0–93. (c) All models controlled for age at baseline, sex, marital status, occupation, smoking, each past and present illness and practice effect. (d) ***$p < .001$, **$p < .01$, *$p < .05$, *ns* = not significant.

## Discussion

### Summary and discussion of findings

The purpose of this study was to determine whether educational attainment was associated with the rate of intellectual change among a community sample of Japanese 65 years of age and over. The participants were followed for approximately 10 years and tested a maximum six times (at baseline and 2, 4, 6, 8, and 10 years after baseline).

As expected, level of education had a large cross-sectional effect on all intellectual performances. Some insist that education may improve brain function. That is, because enriched environments result in a greater number of synapses, individuals with higher education may enter old age with a greater synaptic density (Jacobs, Schall, & Scheibel, 1993). Additionally, education early in life is related to adult occupation or lifestyle, so higher education may result in greater intellectual activities in occupations or leisure pursuits throughout life (Kramer, Bherer, Colcombe, Dong, & Greenough, 2004). Thus, educational attainment early in life might have direct or indirect association with intellectual functions later in life.

In contrast, the relation between education and intellectual change differed by intellectual domain. However, on the whole, there was no statistically significant protective effect of higher education on changes in intellectual abilities over time, although education was associated with long-term individual differences in intelligence. Therefore, the *active* cognitive reserve hypothesis was *not* supported by the results of this study.

*Information, similarities, and picture completion subtests.* The Education × Time interaction was not statistically significant for the Information, Similarities, and Picture Completion subtests, suggesting that there were no effects of educational attainment on intellectual change over time. These findings are consistent with some recent studies that have observed no effects of education on intellectual change with aging (e.g., Seeman et al., 2005; Tucker-Drob et al., 2009; Van Dijk et al., 2008; Wilson et al., 2009; Zahodne et al., 2011). Thus, these results support a *passive* cognitive reserve hypothesis, in which individuals with higher educational attainment continue to perform at a high level compared with similarly aged individuals with less education, but change at a similar rate (Stern, 2002; Van Dijk et al., 2008).

Regardless of the educational level, the Information and Similarities subtest scores displayed a slightly declining trend with the progress of the study. These subtest scores consist of the verbal scales of the WAIS-R-SF, assessing the fund of factual knowledge and logical abstract reasoning that are reflective of crystallized intelligence (Cattell & Horn, 1978; Kaufman & Lichtenberger, 2006). Crystallized intelligence is strongly influenced by culture and experience, and is considered to remain relatively intact through adulthood. Longitudinal data from a previous study suggest that there is an increase in crystallized intelligence (measured using the "verbal meaning" test) until the age of 60 years, with little decline thereafter (Schaie & Willis, 2002). Our study sample with a baseline age of greater than 65 years was likely to show a similar intellectual decline over a 10-year period. However, the influence of education on this aging-associated change was not observed.

In contrast, the Picture Completion subtest is a performance scale in the WAIS-R-SF that measures fluid intelligence, which is more reflective of aging (Cattell & Horn, 1978). However, contrary to our expectations, trajectories of individual change showed improvements that were reflected in the Picture Completion test score, after adjusting for practice effects. The Picture Completion subtest consists of a basically simple task of finding missing parts of familiar pictures with a simple motor, or vocal output (pointing, or one-word responses). Therefore, it has been suggested that the Picture Completion subtest might be resilient to the impact of brain damage (Kaufman & Lichtenberger, 2006), and to hold up with age better than most other performance tests (Wechsler, 1944). In addition, habituation to the test situation or attrition might be important factors in the increased Picture Completion test score. It is suggested that the design of future studies should take these factors that may confound the results of longitudinal studies into consideration.

*Digit symbol subtest.* The digit symbol subtest measures processing speed, which is considered to be highly reflective of aging (Kaufman & Lichtenberger, 2006; Wechsler, 1944). Therefore, it is noteworthy that education in early life, which is a marker of cognitive reserve, contributed to the 10-year persistence of earlier differences in the Digit Symbol score, even after 65 years of age. Moreover, the Education × Time interaction was statistically significant for the Digit Symbol subtest. Surprisingly, individuals with higher levels of education actually experienced a greater decline in the Digit Symbol subset scores than those with less education, although they had a higher ability at every point in time.

There are two possible explanations for this finding. First, it is possible that highly educated adults might make the most of their high quality crystallized ability to supplement the declining fluid ability, or processing speed (Alley et al., 2007). As a result, when highly educated adults get to be aged 65 years or older, they eventually begin to lose their crystallized abilities (for example, measured by the Information and Similarities subtest) to draw on educational attainment, and as a result experience a faster rate of decline in processing speed than those with lower education. This phenomenon could be explained in terms of the *compensation* hypothesis, in which intact domains compensate for declines in other cognitive abilities until they, too, begin to deteriorate, leading the way for more rapid decline (Alley et al., 2007; Reuter-Lorenz & Mikels, 2006; Zahodne et al., 2011).

Second, it may be possible that the low-educated group showed a greater rate of decline in intellectual performance before age 65 years, prior to baseline. That is, for low-educated older adults, the rate of decline in processing speed could have been greater earlier in life, but slower after age 65 years. In contrast, it is possible that the high-educated group had very small or little decline in processing speed earlier in life (before age 65 years), but that their greatest rate of decline was observable after baseline. Thus, our findings may reflect a difference in the "onset of degeneration" (Alley et al., 2007) in processing speed between higher- and lower-educated older adults.

Some studies have reported that education was not related to the rate of change in processing speed (Christensen, Hofer, Mackinnon, Korten, Jorm, & Henderson, 2001; Tucker-Drob et al., 2009; Van Dijk et al., 2008). However, these previous studies had shorter durations (5–7-year periods) than our investigation (10-year period), or were restricted to the range of educational level in the samples (not enough low-education or high-education participants were included). Considering the better methods used in the present study, our results may reflect a true null finding of education on the change in processing speed; however, further studies on the matter are still needed. In order to confirm the abovementioned explanations, longer study observation periods (including age groups younger than 65 years) and the analyses of inflection points or nonlinear trajectories in processing speed will be required in future research.

### Limitations and future directions

Our study has some important limitations. First, it should be noted that this study included participants who were, on the whole, at higher baseline levels of intellectual functioning. Therefore, our findings may only be relevant to healthier aging patterns among community-residing older adults. Second, the statistical models used in this study did not include several factors that may influence intelligence, such as the visual and auditory senses, or motor functions. Third, it remains possible that cognitive reserve mechanisms may act differently in intellectual domains outside of this study (e.g., episodic memory and working memory). Therefore, we must limit our conclusion to the levels and rates of change in intelligence, as measured using the JWAIS-R-SF (Information, Similarities, Picture Completion, and Digit Symbol). Fourth, the measure of educational attainment in this study assumes an equivalence of educational quality across persons and time. However, this is unlikely to be true. Other measures, such as literacy or acquired knowledge, may better address the quality of education as the marker of cognitive reserve. In addition, we assessed the level of educational attainment by using a categorical scale with only four options. However, it is likely that individuals with very low education (fewer than 6 years) may experience the greatest intellectual declines in late life (Lyketsos et al., 1999). Further studies are required to measure educational experience, defined as the number of years of schooling, in order to examine the possibility of steeper decline among older adults with very low educational attainment.

Additionally, differences in adult lifestyle may increase cognitive reserve by making the individual more resilient (Scarmeas, Zarahn, Anderson, Habeck, Hilton, Flynn, Marder, Bell, Sackeim, Van Heertum, Moeller, & Stern, 2003). So the relation between education in early life and intellectual change in late life may be mediated by participation in leisure activities or an occupation throughout adulthood. Moreover, there are several psychological factors that may modulate or mediate the relation between education and intelligence. For example, Nishita, Tange, Tomida, Ando, and Shimokata (2012b) suggest that the personality trait of openness to experience, highly correlated with education, influences intellectual change in later adulthood. Van Dijk et al. (2008) asserts that physical or mental health factors have important effects on intellectual change in late life, and that even higher educated individuals, who may initially have a benefit of greater cognitive reserve, are not protected against the effects of intellectual aging once they acquire certain physical or mental disease. An understanding of the precise nature and mechanisms of the relation between education in early life and intellectual change in late life invite further studies, including lifestyle and personal traits.

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(Received August 31, 2012; accepted May 4, 2013)

RESEARCH ARTICLE

International Journal of
Geriatric Psychiatry

# Global cognition and 8-year survival among Japanese community-dwelling older adults

Hajime Iwasa[1,2], Ichiro Kai[1], Yuko Yoshida[2], Takao Suzuki[3], Hunkyung Kim[2] and Hideyo Yoshida[2]

[1]School of Public Health, Graduate School of Medicine, University of Tokyo, Tokyo, Japan
[2]Tokyo Metropolitan Institute of Gerontology, Tokyo, Japan
[3]National Center for Geriatrics and Gerontology, Aichi, Japan
*Correspondence to:* H. Iwasa, PhD, E-mail: hajimei-tky@umin.ac.jp

**Objective:** We sought to examine the longitudinal relationship between cognitive function and all-cause mortality among Japanese community-dwelling older adults, using an 8-year prospective cohort study design with mortality surveillance.

**Methods:** A total of 454 men and 386 women, aged 70 years and older, participated in the study. The Mini Mental State Examination (MMSE) was administered to assess global cognition. The total MMSE score and subscale scores were used as independent variables, and age, gender, education level, chronic disease, sensory deficit, depressive symptoms, and instrumental activities of daily living were used as covariates.

**Results:** During the follow-up period, 191 subjects (139 men and 52 women) died, and 64 subjects (31 men and 33 women) moved to a different region of Japan and were lost to follow-up. Use of the multivariate Cox proportional hazards model, adjusted for potential confounders, showed that global cognition was significantly and independently associated with mortality (hazard ratio [HR] = 1.59, 95% confidence interval [CI]: 1.14–2.23 and HR = 2.81, 95% CI: 1.77–4.36 for the middle [24–27 points] and lowest [0–23 points] categories, respectively). Among the MMSE subscales, *place orientation* (HR = 1.57, 95% CI: 1.09–2.25), *calculation* (HR = 1.67, 95% CI: 1.18–2.35), and *delayed recall* (HR = 1.42, 95% CI: 1.03–1.96), were also significantly and independently associated with mortality.

**Conclusions:** Our study suggests that among older individuals, those with lower levels of cognitive function are more likely to have a shorter lifespan compared with those with higher cognitive functioning.

**Key words:** all-cause mortality; cognition; community older adults; Mini Mental State Examination (MMSE)
**History:** Received 16 February 2012; Accepted 7 September 2012; Published online 25 September 2012 in Wiley Online Library (wileyonlinelibrary.com)
**DOI:** 10.1002/gps.3890

## Introduction

Cognitive function is an important contributor to health among older adults. A number of recent studies have examined the longitudinal association between cognitive function and mortality among older adults. Among the different domains of cognitive function, episodic memory (Small and Backman, 1997; Portin *et al.*, 2001), executive function (Johnson *et al.*, 2007; Lavery *et al.*, 2009), and information processing speed (Smits *et al.*, 1999; Rosano *et al.*, 2008; Lavery *et al.*, 2009) have been shown to predict mortality among older adults living in a community setting. Global cognition measured by the Mini Mental State Examination (MMSE) (Folstein *et al.*, 1975) also predicts mortality (Kelman *et al.*, 1994; Bruce *et al.*, 1995; Gussekloo *et al.*, 1997; Fredman *et al.*, 1999; Korten *et al.*, 1999; Andersen *et al.*, 2002; Nguyen *et al.*, 2003). The MMSE is the most widely used test for objectively measuring cognitive function, and its validity and reliability have been confirmed (Tombaugh and McIntyre, 1992).

Only a few studies have examined the association between MMSE subscales and mortality. A study (Villarejo *et al.*, 2011) reported a longitudinal association between the three-word delayed recall task of the MMSE and all-cause mortality among non-demented older individuals. However, longitudinal associations between the other MMSE subscales and mortality are unclear. A careful examination of the relationship between MMSE subscales and mortality should help facilitate the prediction of early death among older adults in epidemiological surveys in the community and in clinical settings.

In this study, we examined the longitudinal relationship between cognitive performance, based on the MMSE, and all-cause mortality among Japanese community-dwelling older adults, using an 8-year surveillance of mortality.

## Methods

### Participants

The data for the present study were acquired from mass health checkups for community-dwelling older adults (*Otasha-Kenshin*) (Iwasa *et al.*, 2007; Suzuki *et al.*, 2008), conducted in 2002 by the Tokyo Metropolitan Institute of Gerontology. The Japanese term *Otasha-Kenshin* translates into "health checkups for accomplishing successful aging." The study was conducted in Itabashi ward in northern Tokyo, Japan, and we were granted access to the municipal resident registration files by the Itabashi ward authorities. Participants took part in a face-to-face interview, to establish the baseline, with trained research assistants. The study was approved by the Ethics Committee of the Tokyo Metropolitan Institute of Gerontology. As of 2002, a sample of 1945 residents (aged 70–84 years) was randomly obtained from the municipal resident registration files. We sent a letter asking them to participate in checkups. Eight hundred forty-seven individuals agreed to participate (43.5% participation rate). There was a lower proportion of women compared with men who participated in the baseline survey (46.2% *vs.* 51.9%, respectively, $p = 0.012$). Participants and non-participants were almost identical in age (76.2 *vs.* 76.4 years, respectively, $p = 0.233$).

Of those who participated in the baseline survey, seven were excluded from the analysis: one subject had missing educational information and six had missing MMSE scores. In total, 840 participants (454 men and 386 women, mean age of $76.2 \pm 3.6$ years at baseline) with complete data sets were included, and their data were used for the 8-year mortality surveillance (Figure 1).

### Mortality follow-up

Because the survey was completed at the end of 2002, we defined January 1, 2003 as the baseline for the follow-up period in the present study. Thus, we carried out an 8-year mortality surveillance, from January 1, 2003 to January 1, 2011.

Current residency in Itabashi ward on January 1, 2011 was determined using the municipal resident registration files for Itabashi ward. The dates on which residents moved away or died were identified from the registration files and used to calculate survival times. The certifications and dates of all decedents and those moving away were obtained from the Itabashi ward authorities.

The dependent variable in the analyses was survival time, calculated as the number of days between the baseline (i.e., January 1, 2003) and the date of death or censoring (including survivors and dropouts due to migration from Itabashi ward). Survivors were censored on January 1, 2011. Dropouts were censored on the date of migration from Itabashi ward.

We used all-cause mortality as the dependent variable because we did not have any data regarding the cause of death among the decedents.

### Measurements of cognitive performance

We used the MMSE (Folstein *et al.*, 1975) to assess cognitive function among older adults. The MMSE is the most widely used test of global cognitive function and has been used in numerous studies (Tombaugh and McIntyre, 1992; Dewey and Saz, 2001; Xu *et al.*, 2002; Inagaki *et al.*, 2009). The MMSE includes 12 test items that objectively assess different cognitive domains: (1) *time orientation* (5 points); (2) *place orientation* (5 points); (3) *registration* of three words (3 points); (4a) *calculation* (mentally subtracting seven iteratively from 100, 5 points); (4b) *reverse spelling* (mentally spelling backwards a word presented auditorily); (5) *delayed recall* of the three words presented earlier (3 points); (6) *naming objects* (2 points); (7) *repeating a sentence* (1 point); (8) *listening and obeying* (following a three-stage mmand, 3 points); (9) *reading and obeying* (following a message printed on a card, 1 point); (10) *writing sentences* (1 point); and (11) *copying figures* (copying figures on a sheet of paper, 1 point). Item scores were summed to give the total MMSE score (ranging between 0 and 30), with higher scores reflecting a higher level of global cognitive performance. Although in the original MMSE procedure (Folstein *et al.*, 1975), item 4b, *reverse spelling*, was conducted only if participants refused to perform item 4a, *calculation*, both items were implemented in this study. As previously reported (Holtsberg *et al.*, 1995),

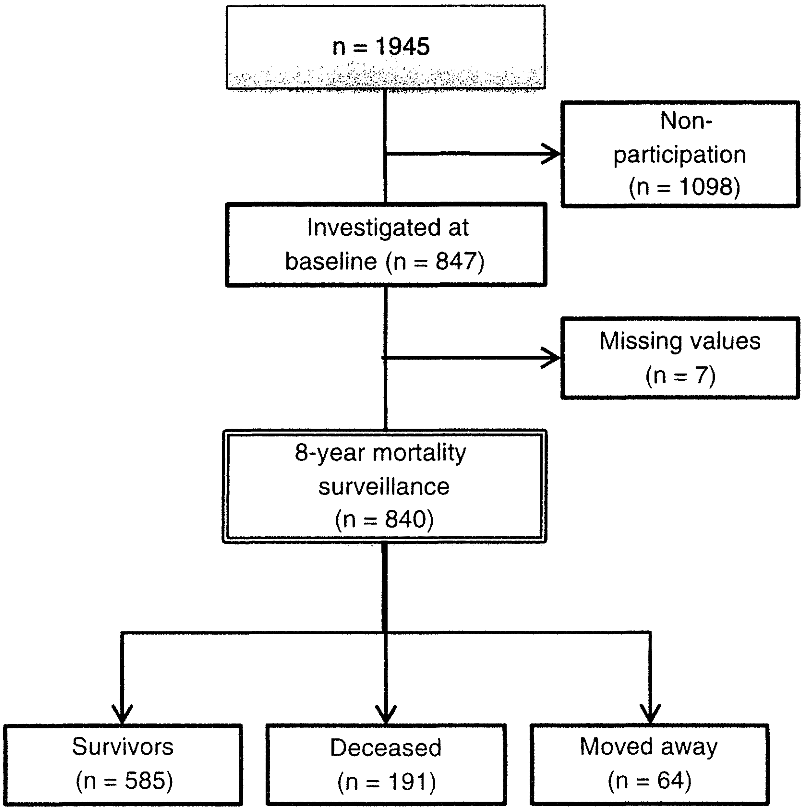

**Figure 1** Study sample in the analysis.

the higher of the two scores from item 4a, *calculation*, and item 4b, *reverse spelling*, was used for computing the total MMSE score. Thus, in this study, the number of participants for item 4a differed from those of other items because some subjects refused to perform this task ($n = 25$).

We divided the MMSE total score into the following three categories: low (0–23 points), middle (24–27points), and high (28–30 points), based on a previous protocol (Gussekloo *et al.*, 1997; Xu *et al.*, 2002). Subjects obtaining 0–23 points were considered to possibly have a cognitive impairment (Tombaugh and McIntyre, 1992).

When analyzing the MMSE subscales, all subscale scores were dichotomized as either completely correct or incorrect according to the score in each subscale. This method was based on a previous protocol (Ishizaki *et al.*, 1998). For example, for the *time orientation* subscale (max = 5 points), participants who scored 5 were considered to have responded correctly and were given 1 point. Those who scored 4 or lower were classified as being incorrect and were given 0 point.

## Other measurements

Data for baseline characteristics were used as covariates in the analysis to identify independent associations between cognitive performance and mortality, and to describe the characteristics of the study participants. Data for age, gender, education level, chronic disease, sensory deficit, depressive symptoms (Sheehan *et al.*, 1998), instrumental activities of daily living (IADL, measured according to the Tokyo Metropolitan Institute of Gerontology Index of Competence [Koyano *et al.*, 1991]), and self-rated health were included. Chronic disease was self-reported by the participants. Chronic disease was defined as experiencing at least one of the following diseases: history of stroke, heart disease, or diabetes mellitus. Sensory deficit was self-reported by the participants and was defined as experiencing at least one of the following: hearing loss or eyesight problems. To assess IADL, participants were asked to judge whether they were independent with respect to the five daily IADL tasks (e.g., using public transportation and preparing meals) (Koyano *et al.*, 1991). Higher scores reflect a higher level of functioning in IADL. In this study, a cut-off score of 4/5 (meaning that scores of 4 and below were classified as IADL dependent) was used to judge whether participants were dependent with respect to IADL (Ishizaki *et al.*, 2006).

### Statistical analysis

We carried out $\chi^2$ tests for categorical variables and analysis of variance for continuous variables to examine differences in baseline characteristics between groups (i.e., survivors *versus* deceased *versus* dropouts). We also carried out $\chi^2$ tests to examine differences in the MMSE subscale scores between the groups. Cox proportional hazards models, controlling for age, gender, education level, chronic disease, sensory deficit, depressive symptoms, and IADL, were used to test the independent relationships between each cognitive performance and all-cause mortality. All statistical procedures were performed using SPSS for Windows (version 17.0; SPSS, Inc., Chicago, IL, USA).

## Results

During the 8-year follow-up, of the 840 adults, 191 (139 men and 52 women) died and 64 (31 men and 33 women) moved to a different region of Japan and were lost to follow-up.

Table 1 shows the characteristics of the members in the follow-up cohort, collected in 2002, including age, gender, education level, chronic disease, sensory deficit, depressive symptoms, IADL, the distribution of MMSE scores, and mean duration of follow-up. Deceased individuals were more likely to be older ($p < 0.001$), men ($p < 0.001$), and have chronic diseases ($p = 0.012$). They were also more likely to have a lower IADL score ($p < 0.001$), exhibit lower health status ($p = 0.002$), display lower sensory function ($p < 0.001$), and have lower MMSE scores ($p < 0.001$), compared with survivors/dropouts.

Table 2 shows the MMSE subscale scores. Deceased individuals had lower scores in the seven tasks—*time orientation, place orientation, calculation, reverse spelling, delayed recall, repeated sentences*, and *copying figures.* Because the number who answered items incorrectly in subscales 6 (*naming objects*) and 9 (*reading and obeying*) was so small, we did not conducted any analysis on those subscales.

Figure 2 shows the Kaplan–Meier survival curves corresponding to the relationship between global cognition and mortality. The risk of mortality was significantly higher for lower functioning individuals than for higher functioning individuals (log-rank test: $p < 0.001$).

Table 3 shows the independent association between global cognition and mortality. Following multivariate Cox regression analysis, adjusted for the potential confounders cited earlier, global cognition (hazard ratio [HR] = 1.59, 95% confidence interval [CI] = 1.14 to 2.23 and HR = 2.81, 95% CI = 1.77 to 4.36 for the middle [24–27 points] and lowest [0–23 points] categories, respectively) was significantly and independently associated with mortality.

Table 4 shows the independent associations between MMSE subscale scores and mortality. Following multivariate Cox regression analysis, adjusted for the potential confounders cited earlier, *time orientation* (HR = 1.56, 95% CI: 1.12 to 2.18), *place orientation* (HR = 1.87, 95% CI: 1.37 to 2.56), *calculation* (HR = 1.81, 95% CI: 1.30 to 2.52), *reverse spelling* (HR = 1.42, 95% CI: 1.06 to 1.90), *delayed recall*

Table 1 Distribution of participants' characteristics at baseline ($N = 840$)

| | Survivors ($n = 585$) | Deceased ($n = 191$) | Dropouts ($n = 64$) | $p$-value[b] |
|---|---|---|---|---|
| Age, mean ± SD (years) | 75.6 ± 3.4 | 77.6 ± 3.8 | 76.5 ± 4.0 | <0.001 |
| Gender (women), $n$ (%) | 301 (51.5) | 52 (27.2) | 33 (51.6) | <0.001 |
| Number of years of education, mean ± SD (years) | 10.6 ± 2.9 | 10.9 ± 3.4 | 10.1 ± 3.7 | 0.235 |
| Chronic disease (present), $n$ (%)[a] | 206 (35.2) | 86 (45.0) | 31 (48.4) | 0.012 |
| Instrumental activities of daily living (dependent), $n$ (%) | 72 (12.3) | 52 (27.2) | 9 (14.1) | <0.001 |
| Self-rated health (fair/poor), $n$ (%) | 108 (18.5) | 58 (30.7) | 15 (23.4) | 0.002 |
| Sensory deficit, $n$ (%) | 62 (10.6) | 42 (22.0) | 4 (6.3) | <0.001 |
| Depressive symptoms, $n$ (%) | 12 (2.1) | 7 (3.7) | 1 (1.6) | 0.404 |
| MMSE, mean ± SD (scores) | 28.3 ± 2.1 | 26.9 ± 3.2 | 27.9 ± 2.5 | <0.001 |
| Duration of follow-up, mean ± SD (years)[c] | 8.0 | 4.7 (2.0) | 3.8 (2.2) | – |

MMSE, Mini Mental State Examination.

[a] Chronic disease was defined as having at least one of the following diseases: stroke, heart disease, or diabetes mellitus.

[b] $\chi^2$ tests for categorical variables and analysis of variance for continuous variables were used to examine differences in baseline characteristics between groups (survivors *versus* deceased *versus* dropouts).

[c] All survivors were followed up for 8 years (i.e., from January 1, 2003 to January 1, 2011).

Table 2 Number of participants who answered incorrectly on each subscales of the MMSE ($N = 840$)[a]

| | Survivors ($n = 585$) | Deceased ($n = 191$) | Dropouts ($n = 64$) | $p$-value[b] |
|---|---|---|---|---|
| 1. Time orientation, $n$ (%) | 81 (13.8) | 50 (26.2) | 13 (20.3) | <0.001 |
| 2. Place orientation, $n$ (%) | 103 (17.6) | 63 (33.0) | 16 (25.0) | <0.001 |
| 3. Registration (immediate recall), $n$ (%) | 10 (1.7) | 7 (3.7) | 2 (3.1) | 0.256 |
| 4a. Calculation, $n$ (%)[c] | 330 (57.8) | 134 (73.2) | 32 (52.5) | <0.001 |
| 4b. Reverse spelling, $n$ (%) | 205 (35.0) | 89 (46.6) | 25 (39.1) | 0.017 |
| 5. Delayed recall, $n$ (%) | 257 (43.9) | 120 (62.8) | 41 (64.1) | <0.001 |
| 6. Naming objects, $n$ (%) | 3 (0.5) | 1 (0.5) | 3 (4.7) | – |
| 7. Repeating a sentence, $n$ (%) | 40 (6.8) | 29 (15.2) | 5 (7.8) | 0.002 |
| 8. Listening and obeying, $n$ (%) | 5 (0.9) | 5 (2.6) | 1 (1.6) | 0.174 |
| 9. Reading and obeying, $n$ (%) | 3 (0.5) | 3 (1.6) | 1 (1.6) | – |
| 10. Writing sentences, $n$ (%) | 32 (5.5) | 15 (7.9) | 6 (9.4) | 0.289 |
| 11. Copying figures, $n$ (%) | 30 (5.1) | 19 (9.9) | 6 (9.4) | 0.041 |

MMSE, Mini Mental State Examination.

[a] When analyzing the MMSE subscales, all MMSE subscale scores were dichotomized as either correct or incorrect, according to the score for each subscale.

[b] $\chi^2$ tests were used to examine differences in number (%) of participants who answered MMSE subscale items incorrectly. Because the number who answered items incorrectly in subscales 6 and 9 was so small, we did not conducted any analysis on those subscales.

[c] The number of participants for item 4a (*calculation*) ($n = 815$) differed from those for other items because some subjects refused to perform this task ($n = 25$).

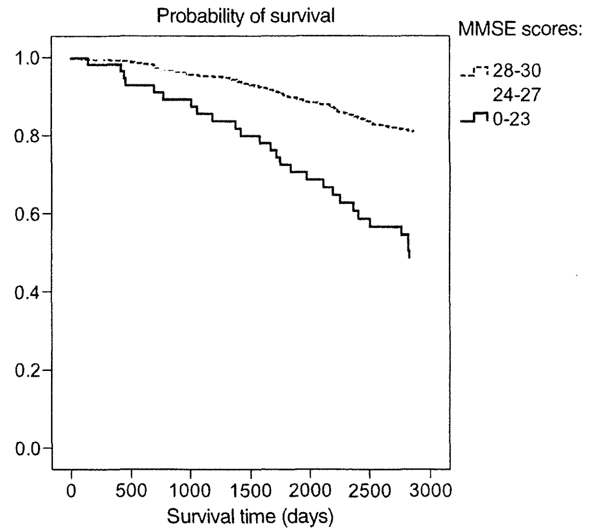

**Figure 2** Unadjusted Kaplan–Meier survival curves exploring differences in all-cause mortality between levels of global cognition (measured by Mini Mental State Examination [MMSE]) at baseline over the 8-year follow-up period. Risk of mortality significantly varied according to the level of global cognition (log-rank test: $p < 0.001$). The vertical axis indicates probability of survival. The horizontal axis indicates survival time (days).

(HR = 1.65, 95% CI: 1.22 to 2.25), *repeating a sentence* (HR = 1.53, 95% CI: 1.02 to 2.30), and *copying figures* (HR = 1.95, 95% CI: 1.20 to 3.15) were significantly and independently associated with mortality. Among the subscales, *registration*, *naming objects*, *listening and obeying*, *reading and obeying*, and *writing sentences* were not analyzed because these subscale scores were not associated significantly with mortality in the univariate analyses (Table 3).

To examine whether the relationships between cognitive performance on the MMSE subscales and mortality were affected by cognitive impairment at baseline, we performed the aforementioned analysis excluding possible cases of cognitive impairment based on the MMSE total score. We used a cut-off score of 24, meaning that scores of 23 and below were classified as possible cognitive impairment (Tombaugh and McIntyre 1992) ($n = 57$). The results revealed that, after excluding subjects with a score of 23 or below, the association of *place orientation* (HR = 1.57, 95% CI: 1.09 to 2.25), *calculation* (HR = 1.67, 95% CI: 1.18 to 2.35), and *delayed recall* (HR = 1.42, 95% CI: 1.03 to 1.96) with mortality remained significant.

Table 3 Adjusted hazard ratios of all-cause mortality by MMSE total score ($N = 840$)[a]

| MMSE total score | $N$ | Deceased | Hazard ratio (95% confidence interval) | $p$-value |
|---|---|---|---|---|
| 28–30 (ref.) | 588 | 107 | 1 | |
| 24–27 | 195 | 57 | 1.59 (1.14–2.23) | 0.007 |
| 0–23 | 57 | 27 | 2.81 (1.79–4.36) | <0.001 |

MMSE, Mini Mental State Examination.

[a] Adjusted for baseline characteristics (including age, gender, education level, chronic disease, sensory deficit, depressive symptoms, and instrumental activities of daily living).

Table 4 Adjusted hazard ratios of all-cause mortality for each subscale of the MMSE[a,b]

| | Total ($n = 840$) | | | | Individuals with no cognitive impairment ($n = 783$)[c] | | | |
|---|---|---|---|---|---|---|---|---|
| | *N* | Deceased | Hazard ratio (95% confidence interval) | *p*-value | *N* | Deceased | Hazard ratio (95% confidence interval) | *p*-value |
| 1. Time orientation | | | | | | | | |
| Correct (ref.) | 696 | 141 | 1 | | 679 | 135 | 1 | |
| Incorrect | 144 | 50 | 1.56 (1.12–2.18) | 0.009 | 104 | 29 | 1.24 (0.82–1.87) | 0.303 |
| 2. Place orientation | | | | | | | | |
| Correct (ref.) | 658 | 128 | 1 | | 637 | 122 | 1 | |
| Incorrect | 182 | 63 | 1.87 (1.37–2.56) | <0.001 | 146 | 42 | 1.57 (1.09–2.25) | 0.013 |
| 4a. Calculation[d] | | | | | | | | |
| Correct (ref.) | 319 | 49 | 1 | | 314 | 47 | 1 | |
| Incorrect | 496 | 134 | 1.81 (1.30–2.52) | <0.001 | 456 | 111 | 1.67 (1.18–2.35) | 0.004 |
| 4b. Reverse spelling | | | | | | | | |
| Correct (ref.) | 521 | 102 | 1 | | 511 | 96 | 1 | |
| Incorrect | 319 | 89 | 1.42 (1.06–1.90) | 0.002 | 272 | 68 | 1.34 (0.97–1.87) | 0.079 |
| 5. Delayed recall | | | | | | | | |
| Correct (ref.) | 422 | 71 | 1 | | 417 | 71 | 1 | |
| Incorrect | 418 | 120 | 1.65 (1.22–2.25) | <0.001 | 366 | 93 | 1.42 (1.03–1.96) | 0.034 |
| 7. Repeating a sentence | | | | | | | | |
| Correct (ref.) | 766 | 162 | 1 | | 733 | 145 | 1 | |
| Incorrect | 74 | 29 | 1.53 (1.02–2.30) | 0.037 | 50 | 19 | 1.44 (0.88–2.38) | 0.148 |
| 11. Copying figures | | | | | | | | |
| Correct (ref.) | 785 | 172 | 1 | | 742 | 154 | 1 | |
| Incorrect | 55 | 19 | 1.95 (1.20–3.15) | 0.007 | 41 | 10 | 1.31 (0.68–2.50) | 0.416 |

MMSE, Mini Mental State Examination.

[a] Adjusted for baseline characteristics (including age, gender, education level, chronic disease, sensory deficit, depressive symptoms, and instrumental activities of daily living).

[b] All MMSE subscale scores were dichotomized into either completely correct or incorrect, according to the score in each subscale.

[c] Individuals with possible cognitive impairment (MMSE < 24) were excluded.

[d] The number of participants for the item 4a (*calculation*) differed from those for other items because some subjects refused to perform this task. Total sample analysis, $n = 815$; analysis excludes individuals with cognitive impairment ($n = 770$).

## Discussion

In this study, we examined the relationship between cognitive performance and all-cause mortality among community-dwelling older people in Japan. Our findings indicate that global cognitive function (measured using the MMSE) predicts mortality after adjusting for potential confounders, such as age, gender, education level, chronic disease, sensory deficit, depressive symptoms, and IADL. In addition, among the MMSE subscales, *place orientation*, *calculation*, and *delayed recall* were significantly and independently associated with mortality. Our study suggests that older individuals with lower levels of cognitive function are more likely to have shorter lives, based on the 8-year follow-up, compared with those with higher cognitive functioning.

Our findings are similar to the results of previous studies showing a relationship between global cognition (measured using the MMSE) and mortality among community-dwelling older adults (Kelman *et al.*, 1994; Bruce *et al.*, 1995; Gussekloo *et al.*, 1997; Fredman *et al.*, 1999; Korten *et al.*, 1999; Andersen *et al.*, 2002; Nguyen *et al.*, 2003). The findings of these studies and our present report suggest that global cognition is a predictor of mortality among community-dwelling older adults. In addition, the association between global cognition and mortality was significant not only in individuals with possible cognitive impairment (i.e., MMSE total score of 0–23 points) but also in individuals exhibiting a mild deficit in global cognition (i.e., MMSE score of 24–27 points), which is consistent with the previous study (Gussekloo *et al.*, 1997). Previous studies indicate that individuals with cognitive impairment are more likely to have shorter lives, compared with those who are not cognitively impaired (Dewey and Saz, 2001). Our study also confirmed that older individuals who exhibit a mild deficit in global cognition are more likely to have a shorter lifespan, compared with those who are cognitively intact.

We speculate that there are four possible reasons why individuals who exhibited a mild deficit in global cognition (i.e., MMSE score of 24–27 points) were more likely to have shorter lives in this study. The first possible reason is the presence of potential cases of dementia. We divided participants into two groups, using a cut-off score of 24 in the MMSE, and regarded