Longitudinal Cognition-Survival Relations in Old and Very Old Age

13-Year Data from the Berlin Aging Study

Paolo Ghisletta[1], John J. McArdle[2], and Ulman Lindenberger[3]

[1]Center for Interdisciplinary Gerontology and Faculty of Psychology and Educational Sciences, University of Geneva, Switzerland, [2]Department of Psychology, University of Southern California, USA, [3]Center for Lifespan Psychology, Max Planck Institute for Human Development, Berlin, Germany

Abstract. We use a statistical model that combines longitudinal and survival analyses to estimate the influence of level and change in cognition on age at death in old and very old individuals. Data are from the Berlin Aging Study, in which an initial sample of 516 elderly individuals with an age range of 70 to 103 years was assessed up to 11 times across a period of up to 13 years. Four cognitive ability domains were assessed by two variables each: perceptual speed (Digit Letter and Identical Pictures), episodic memory (Paired Associates and Memory for Text), fluency (Categories and Word Beginnings), and verbal knowledge (Vocabulary and Spot-a-Word). Longitudinal models on cognition controlled for dementia diagnosis and retest effects, while survival models on age at death controlled for age, sex, socioeconomic status, sensory and motor performance, and broad personality characteristics. Results indicate: (1) Individual differences in the level of and in the linear change in performance are present for all cognitive variables; (2) when analyzed independently of cognitive performance, all covariates, except broad personality factors, predict survival; (3) when cognitive performance is accounted for, age, sex, and motor performance do predict survival, while socioeconomic status and broad personality factors do not, and sensory performance does only at times; (4) when cognitive variables are analyzed independently of each other, both level and change in speed and fluency, as well as level in memory and knowledge predict survival; (5) when all cognitive variables are analyzed simultaneously using a two-stage procedure, none of them is significantly associated to survival. In agreement with others, our findings suggest that survival is related to cognitive development in old and very old age in a relatively global, rather than ability-specific, manner.

Keywords: structural equation modeling, survival analysis, cognitive aging, terminal decline, joint analysis of longitudinal and survival models

The seminal papers of Kleemeier (1962), Riegel and Riegel (1972), and Siegler (1975) on the association between cognitive performance and mortality have provided the motivation for a substantial body of psychological literature on the theme of mortality prediction. It is now relatively well established that cognitive functioning predicts survival reliably in old and very old populations, even over very long periods (e.g., Rabbitt et al., 2002; Whalley & Deary, 2001) or when accounting for several physical and medical conditions in large epidemiological studies (e.g., Fried et al., 1998). Nonetheless, and as also pointed out by Small and Bäckman (1999), there are a number of relevant research questions yet to be resolved to further qualify the cognition-survival association. The most important of these is the pervasiveness versus specificity dilemma (Riegel & Riegel, 1972; White & Cunningham, 1988). According to some, the predictive power to foretell death is limited to specific intellectual domains. White and Cunningham further hypothesized that abilities typically well preserved in old age (e.g., verbal knowledge) are more affected by impending death than those that start declining in younger portions of the lifespan (e.g., perceptual speed). Therefore, older individuals who experience considerable declines in crystallized abilities are the ones with the greatest probability of imminent death.

The empirical findings, however, do not invariably confirm the specificity hypothesis. While some scholars obtained results in line with this hypothesis (e.g., Rabbitt et al., 2002), others found that several different cognitive abilities decline with approaching death (e.g., Bäckman, Jonsson Lauka, Wahlin, Small, & Fratiglioni, 2002; Hassing, Johansson, et al., 2002; Johansson & Berg, 1989; Johansson et al., 2004; Ljungquist, Berg, & Steen, 1996; Small, Fratiglioni, von Strauss, & Bäckman, 2003). Others still found that, when controlling for various health, socioeconomic, and psychological covariates, only abilities that start declining relatively early on in life are affected (e.g., Maier & Smith, 1999), confirming, in a sense, the specificity hypothesis, while disconfirming White and Cunningham's hypothesis (1988). Thus, at present, it remains to be

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clarified whether performance in all or only some cognitive abilities predicts survival. However, we are, at least, able to assert with relative confidence that cognitive performance is related to imminent death.

The confidence of this conclusion is much greater when the *level* of, rather than the *change* in, cognitive performance is investigated. Indeed, and somewhat surprisingly given the growing interest in cognition-mortality associations, longitudinal studies on terminal change continue to be scarce. Bosworth and Siegler (2002) reviewed longitudinal studies that were published in English, addressed human subjects only, were not uniquely focused on dementia, and had sufficiently large cognitive batteries to address, at least partially, the pervasiveness versus specificity dilemma. Only nine empirical articles were retained in their review. The general conclusions were that while the decrease in crystallized measures was found to be related to mortality, the findings concerning episodic memory and fluid reasoning measures were mixed. The authors proposed a number of possible design features that could explain some of the discrepancies in the results. These included the consideration of the sample's health status, the time interval between cognitive assessment and mortality as well as between repeated cognitive assessments, the role of dementia, and, of course, the age of the sample.

Methodological Considerations

In general, two major methodological approaches have been used to investigate the cognition-survival association (Maier & Smith, 1999; Small & Bäckman, 1999). The first relies on a single (i.e., cross-sectional) assessment of cognitive performance, often with measures of health status, sociological profiles, or psychological functioning. In this design, the participants' survival status is ascertained a number of months or years after the general assessment, and the goal of the analyses is to test the validity of the baseline variables in predicting age at death. The second methodology takes advantage of a repeated-measures design, where cognitive performance has been measured at least twice. This also enables changes in cognition to be estimated. Hence, with longitudinal data it is possible to contrast the predictability of level information with that of change. It is worth noting that level information is to some extent an index of earlier changes, especially in old age. This approach addresses the question of whether people who generally experience decrease in cognitive performance are more likely to die. The terminal-decline hypothesis, however, may also be interpreted as addressing a slightly different question, namely, whether death is imminent for those whose cognitive performance declines abruptly. The first question is at the sample level and focuses on overall trajectories in cognitive performance. The second question is more concerned with particular individuals who undergo greater changes compared to the overall sample trend. The vast majority of literature discussing terminal decline is actually concerned with the first question.

Both the cross-sectional and the longitudinal methodological approaches usually analyze participants' cognitive performance and their age at death with survival analyses. In survival analyses (also-called event-history analyses), both an event of interest and the time elapsed to the occurrence of the event are considered in a regression-based framework, allowing for the inclusion of covariates (e.g., Singer & Willett, 2003). Basically, this model predicts the probability of experiencing an event at a specific time point, given that up to that time point the event has not occurred. In this research application, the event is death and the time elapsed to its occurrence is usually the age at death. Hence, this type of time-based analysis aims at testing the effect of the covariates on age at death. Unlike standard regression analysis, survival analysis also includes information about cases that did not yet experience the event. These are the participants that did not die before survival assessment (they are termed "right-censored"). Rather than ignoring these data (in particular, their cognitive trajectories), survival analyses take into account their information when estimating the survival parameters (Kalbfleisch & Prentice, 1980). The survival model used most frequently by far has been the Cox Proportional Hazards model (Cox, 1972), which is nonparametric (consequently, it is not recommended with small sample sizes; Yamaguchi, 1991). Other frequently used survival models are parametric and include the Weibull and the exponential function (Allison, 1995).

In sum then, it seems that extant studies on the relation between cognitive change and mortality rely on appropriate analyses to estimate the survival process. However, the change process in this literature is rarely estimated with the most suitable analytical tools. Indeed, the studies investigating the relation between cognitive change and mortality (e.g., as reviewed in Bosworth & Siegler, 2002) either compared subgroups of the original samples that differed with respect to their survival status or timing of death, or included estimated cognitive change by computing simple difference scores (e.g., Time-2 minus Time-1 measures). The difference-scores estimation method, however, (1) includes only participants that returned at Occasion 2, (2) confounds level and change information, and (3) assumes that change is linear and that individuals differ reliably in how they change (i.e., that variance in change is significant).

There is a profusion of studies focusing on cognitive change in late adulthood (e.g., Baltes & Labouvie, 1973; Cattell, 1971; Horn, 1968; Hultsch, 2004; Schaie, 1983; Schaie & Hofer, 2001; Singer, Verhaeghen, Ghisletta, Lindenberger, & Baltes, 2003). The majority of these studies estimate change information by applying state-of-the-art analyses based on latent growth models or, analogously, longitudinal multilevel models (e.g., Bryk & Raudenbush, 1987; Collins & Horn, 1991; Laird & Ware, 1982; McArdle, 1986; Rogosa, Brandt, & Zimowski, 1982). This approach allows (1) analyzing all data (i.e., also those of par-

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ticipants that did not return after Occasion 1), (2) formally distinguishing level from change information, and (3) testing for interindividual differences in change. Very few of these studies, however, have made a direct link with survival information.

Henderson, Diggle, and Dobson (2000) proposed an analysis merging a longitudinal and a survival process via a random Gaussian process (more details follow in the Method section). Their method allowed integrating longitudinal and survival models into a unique joint (or shared) analysis to test the validity of change information in predicting the time to the occurrence of a focal event. Guo and Carlin (2004) showed how this method can be estimated with standard statistical software (e.g., SAS, WinBUGS). McArdle, Small, Bäckman, and Fratiglioni (2005) recently applied this methodology in the psychological literature to predict early diagnosis of Alzheimer disease with individual trajectories of episodic memory performance. In this paper we intend to apply this joint methodology to study the effects of cognitive level and change on mortality in the Berlin Aging Study (BASE; Baltes & Mayer, 1999; Baltes, Mayer, Helmchen, & Steinhagen-Thiessen, 1993).

Cognition and Mortality in the BASE

The BASE is an ongoing interdisciplinary aging study involving the fields of psychology, sociology and social policy, psychiatry, and internal medicine and geriatric medicine, which started in 1990. The study of psychological and, more specifically, cognitive functioning in very old individuals has been a focal interest of BASE (cf. Lövdén, Ghisletta, & Lindenberger, 2004; Singer et al., 2003), and as such, the BASE includes a large assessment battery of cognition (for details see the Method section and Lindenberger, Mayr, & Kliegl, 1993). Three papers have previously analyzed and documented psychological predictors of mortality in the BASE. We now consider each in turn.

Maier and Smith (1999) analyzed Time-1 measurements (collected between 1990–1993) in relation to mortality assessed in 1996. They found that cognitive functioning in all BASE cognitive domains and low extraversion, low openness, and high emotional loneliness were directly predictive of mortality. Further statistical control for age, sex, socioeconomic status SES, and health (objectively assessed by a physician) reduced the set of significant predictors to perceptual speed and dissatisfaction with aging (although SES played a very minor role).

Lindenberger, Singer, and Baltes (2002) assessed experimental and mortality selectivity of the Time-3 participants with respect to the Time-1 parent sample (at Time-2 a reduced measurement protocol was administered, cf. the Method section). Several medical, sensorimotor, personality, and socioeconomic variables were considered, together with overall cognitive functioning. The main conclusions were that, across all examined variables, mortality accounted for two thirds of the total selectivity. In all domains considered the selectivity effects were quite small, except for age and intellectual functioning, which were subject to medium effect sizes. Furthermore, through applications of the Pearson-Lawley selection formulae (Lawley, 1943; Pearson, 1903) the authors estimated selectivity effects of Time-3 nonparticipating survivors. They concluded that, after statistical control for age, there was small mortality selectivity because of dementia diagnosis and intellectual functioning. Overall, education was not subject to mortality selection.

Finally, Ghisletta and Lindenberger (2000) analyzed Time-1 and Time-3 BASE cognitive data in relation to survival assessed in 1999. Longitudinal multilevel models were used to estimate cognitive level and change as a function of age for each participant. This estimation procedure was contrasted to the alternative difference-score method discussed above, that is, level = Time-1 and change = Time-3 – Time-1. For both alternative methods, estimated level and change scores were next added to age in a Cox survival model to predict mortality. It was found that after controlling for age, only level in fluency and change in perceptual speed predicted mortality. Moreover, the multilevel strategy of estimating level and change scores had more predictive power than the difference-score method.

Objectives

The purpose of this paper is to extend these previous findings by (1) relying on additional longitudinal assessments of cognitive performance in the BASE and (2) applying new statistical methodologies that directly integrate longitudinal models with survival models. Thus, the rationales of this paper are both substantive and methodological. More precisely, we investigate the associations between eight cognitive variables, assessed up to 11 times over a period of 13 years, and the probability of survival in the BASE. We apply longitudinal models based on multilevel models to assess participants' level of and change in cognitive performance and merge this information with survival analyses to predict the age at death. First, joint longitudinal and survival analyses are computed on each cognitive variable in turn. Then, all cognitive variables are considered together to test the unique contribution of each variable to the predictability of death.

The longitudinal models statistically control for the likelihood of dementia diagnosis and possible retest effects. Indeed, both these variables represent threats to the internal validity of cognitive assessment and have been shown to affect aging estimates (Ferrer, Salthouse, Stewart, & Schartz, 2004; Ghisletta & de Ribaupierre, 2005; Lövdén et al., 2004; Sliwinski, Lipton, Buschke, & Stewart, 1996). As a number of variables are related to both survival probability and cognitive performance, we statistically disentangle the effects of cognitive functioning on survival by controlling for sex, initial age, SES, sensory and motor performance (vision, hearing, balance, and gait), and global

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personality characteristics (cf. Maier & Smith, 1999 and Lindenberger, et al., 2002) in the survival models.

Method

Participants

The initial sample of the BASE was stratified by age (ranging from 70 to 103 years) and sex, and included 516 participants. All six waves of measurement except the second included an initial assessment (IA) followed by a broader intensive protocol (IPr). The second wave did not include an IPr. The top portion of Table 1 includes the longitudinal sample size, the average sample age, and the average duration in the study since inception.

The sample's survival status was last obtained from the German State Registry office in September 2004. At that time, of the 516 initial participants, 404 were deceased (193 women, 211 men) and 112 were alive. The average age at death was 92.53 years for women and 91.17 years for men (given the large initial age range imposed by the BASE design, the respective standard deviations were quite large – 7.47 and 6.48).

Cognitive Functioning

The longitudinal cognitive battery of the BASE was motivated by two-component lifespan theories of cognitive development, in particular, the crystallized-fluid theory of Cattell (1971) and Horn (1968) and the pragmatic-mechanic theory of Baltes (1987; cf. Lindenberger, 2001, for a review). Four major cognitive domains were investigated, each assessed by two cognitive variables. A detailed description of these variables can be found elsewhere (Lindenberger et al., 1993) and we will only provide a brief description here. The bottom portion of Table 1 presents the longitudinal design of the cognitive variables in the BASE.

Perceptual Speed

This domain was measured by the Digit Letter and the Identical Pictures tests. The Digit Letter (DL) task is very similar to the Digit Symbol test of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955) and asked that participants name letters (instead of writing symbols). The number of correct responses provided within 3 min was analyzed. The Identical Pictures (IP) task was adapted from the homonymous test of the Educational Testing Services (Ekstrom, French, Harman, & Derman, 1976). Participants were shown a target picture on a computer screen and were asked to touch the picture identical to the target among five pictures shown below the target on the same screen. The number of correct responses provided within 80 s was analyzed.

Episodic Memory

This domain was assessed by the Memory for Text (MT) and the Paired Associates (PA) tasks. In the MT task, participants first listened to a short story read aloud (that was simultaneously shown on a computer screen) and were then asked six questions concerning various propositions about the story. The task was adapted from Engel and Satzger (1990) and the number of correct responses was analyzed. In the PA task, eight pairs of concrete nouns were presented twice for 40 s, after which only the first word was shown, in an order different from that of encoding. The number of correct paired nouns was analyzed.

Table 1. Sample characteristics and longitudinal design of the BASE for cognitive variables

	Measurement occasion										
	1		2	3		4		5		6	
	IA	IPr	IA	IA	IPr	IA	IPr	IA	IPr	IA	IPr
N	516	516	361	244	208[a]	164	132	88	82	48	47[b]
Mean age (*SD*)	84.92 (8.66)	85.04 (8.68)	85.26 (8.41)	84.34 (7.30)	83.87 (6.91)	84.07 (6.33)	84.30 (5.90)	85.87 (4.36)	85.86 (4.48)	89.36 (4.58)	89.47 (4.60)
Mean time (*SD*)	0.00 (0.00)	0.13 (0.09)	1.95 (0.71)	3.76 (0.66)	3.99 (0.69)	5.53 (0.79)	6.03 (0.80)	8.94 (0.84)	9.00 (0.86)	13.00 (0.87)	13.04 (0.88)
Cognitive domain											
Perceptual speed	DL	DL, IP	DL	DL	DL, IP	DL	DL, IP	DL	DL, IP	DL	DL, IP
Episodic memory		PA, MT			PA, MT		PA, MT		PA, MT		PA, MT
Fluency		CA, WB	CA	CA	CA, WB	CA	CA, WB	CA	CA, WB	CA	CA, WB
Verbal knowledge		VO, SW			VO, SW		VO, SW		VO, SW		VO, SW

Note. IA = Initial assessment. IPr = Intensive protocol. DL = Digit Letter. IP = Identical Pictures. PA = Paired Associates. MT = Memory for Text. CA = Categories. WB = Word Beginnings. VO = Vocabulary. SW = Spot-a-Word. [a] = for entire IP_3, $n_3 = 206$. [b] = for entire IP_6, $n_6 = 46$.

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Fluency

The Categories (CA) and the Word Beginnings (WB) tasks were used to measure fluency. In the CA task, participants had 90 s to name as many animals as possible, and the total number of different animals was analyzed. In the WB task participants were again allowed 90 s to name as many words starting with the letter S as possible. The total number of different words was analyzed.

Verbal Knowledge

This domain was assessed with the Vocabulary (VO) and the Spot-a-Word (SW) tasks. In the VO task participants were asked to define 20 words, presented sequentially on the screen, from the German version of the WAIS (Wechsler, 1982). In the SW task participants were asked to find the existing word from a list also containing four distracting but pronounceable nonwords. Twenty lists were presented on a computer screen and the total number of correct responses was analyzed.

The DL, IP, and SW tasks required that participants see the computer screens, because information could not be provided auditorily. Hence, the available data for these three tasks are fewer. More generally, in large studies it is rarely the case that participants provide valid answers to all questions (especially in very young or very old samples). The analytical models used adjusted for this unbalanced data situation. We transformed all cognitive performance scores to *t*-scores ($M = 50$, $SD = 10$), anchored at the first IPr.

Covariates in Longitudinal Models

To estimate aging effects in cognition with the smallest possible bias, we accounted statistically for possible dementia diagnosis and retest effects.

Dementia Diagnosis

Given that dementia, even at its preclinical stages, biases estimates of aging effects in nondemented populations (Sliwinski et al., 1996), we included markers of dementia diagnosis. Based on age cohort-specific cutoffs of the Short Mini Mental State Examination, Gerstorf, Herlitz, and Smith (in press) defined a dichotomous indicator of dementia diagnosis at each wave of the BASE. These cutoffs were shown to ensure high specificity (ranging from 72% to 98%) and sensitivity (ranging from 62% to 88%) of dementia classification when compared to independent clinical diagnoses of dementia based on standard clinical interviews and assessment procedures (Helmchen et al., 1999). This appraisal was performed at the initial assessment of each wave and was not recomputed at the respective IPr (given that the intrawave measurements were only a few months apart from each other). From Wave 1 to 6, there were 148, 117, 55, 32, 16, and 6 individuals considered demented by these standards.

It is worth noting that this diagnostic measure, though quite specific and sensitive, is used here (as well as in Gerstorf et al., in press) merely as a statistical control, and does not represent the variable of focal interest. We created a wave-specific (i.e., time-varying) dummy code that assigned a 1 to those who were considered diagnosed at a given wave of assessment and a 0 to the others. This allowed us to estimate the effects of dementia diagnoses on the parameter estimates of the longitudinal models. We preferred this to the more common strategy of simply excluding demented individuals (which drastically reduces statistical power and increases sample selectivity).

Retest Effects

A long-recognized threat to internal validity in longitudinal studies is represented by retest effects (e.g., Salthouse, 2000; Schaie, 1988). These may occur because prior exposure to testing material may influence participants' behavioral outcomes. In particular, participants may learn features about the tasks that facilitate the endorsement of correct answers, thereby underestimating the effects attributable to the chief variable of interest (e.g., chronological age). Several studies on cognitive change have shown evidence of retest effects (Ferrer, Salthouse, McArdle, Stewart, & Schwartz, 2005; Ferrer et al., 2004; Ghisletta & de Ribaupierre, 2005; Ghisletta, Schaie, & Willis, 2003; Lövdén et al., 2004; Rabbitt, Diggle, Holland, & McInnes, 2004), and often these effects bias aging estimates to the point of interacting with age (such that the benefits from repeated exposure on testing performance correlate with participants' age).

Retest effects may be estimated within adequate designs. For instance, the Seattle Longitudinal Study (Schaie, 1983, 2005) makes use of refreshment samples, whereby at each occasion of measurement new participants of the same age as ongoing participants are introduced to the study. Retest effects can then be estimated by comparing the performance of the new participants to that of the existing participants assessed at the same time. The only assumption is that the two groups of participants differ only with respect to experience with the testing material. McArdle, Prescott, Hamagami, and Horn (1998) have shown how retest effects may be statistically estimated in longitudinal analyses based on latent growth models, and Rabbitt, Diggle, Smith, Holland, and McInnes (2001) have shown how they may be statistically estimated in analogous longitudinal analyses within the multilevel modeling framework. Statistical estimations of retest effects are possible when the time basis of interest (e.g., age) is not collinear with the number of repeated exposures. In a truly longitudinal study, where one birth cohort is as-

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sessed repeatedly in time, age and time of testing are totally confounded. However, a cross-sequential design (Schaie, 1965) like that of the BASE, where multiple birth cohorts are sampled and measured longitudinally at the same time points, typically avoids multicolinearity between age and times of testing, thereby allowing the estimation of retest effects. At the same time, in this type of design it is impossible to disentangle cohort and retest effects, and assumptions about the lack of cohort effects must be made to statistically estimate retest effects. Generally, the more heterogeneous in age the sample, the lower the correlation between age and the number of previous testings. McArdle and Woodcock (1997) showed that this correlation is zero when the time interval between repeated testings is randomly assigned to participants.

Following earlier work, we created for each cognitive variable a specific set of dummy codes that marked, starting at the second testing, the number of previous testings. For instance, the DL variable was assessed at most 11 times (at each initial assessment and IPr). The participants who were administered the DL at each occasion are assigned 10 dummy codes, one for each repeated exposure (from the second to the eleventh measurement). This strategy allows estimating occasion-specific and variable-specific retest effects. We have shown in previous work that BASE participants exhibit retest effects of different magnitude on several cognitive variables (Lövdén et al., 2004).

Covariates in Survival Models

Before assessing the power of cognitive variables in predicting mortality, we tested several individual-specific covariates. In choosing them, we were guided by (1) general findings from previous research on mortality predictors, (2) specific BASE findings from previous work on mortality predictors (Lindenberger et al., 2002; Maier & Smith, 1999), and (3) practical limitations dictated by empirical computational issues encountered during the most complex analyses. Indeed, the use of several single predictors proved problematic in the joint longitudinal and survival analyses. We, hence, created aggregate scores when theoretically justified. Although all covariates were assessed longitudinally in the BASE, we considered their initial measurement only, because this greatly facilitated interpreting their influence (Singer & Willett, 2003).

Sex

In most Western countries women have longer life expectancies than men (although this gap is slowly narrowing) and consequently sex has proven to affect survival (Bosworth & Siegler, 2002; Rabbitt et al., 2004). We estimated this effect by contrast-coding sex (–1 for women, 1 for men).

Initial Age

We also tested the effects of age at study inception on mortality. This is especially important for samples with wide age ranges. Age is entered as a continuous variable.

Socioeconomic Status

This composite variable was constructed by merging information on the participants' (1) income, (2) occupational prestige, (3) social class, and (4) number of years of education. The final composite is considered continuous (for more detail, see Lindenberger & Baltes, 1997).

Sensory and Motor Performance Composites

Previous work on the BASE data has shown strong associations between sensorimotor variables and cognitive performance, both in cross-sectional (Baltes & Lindenberger, 1997; Lindenberger & Baltes, 1994; Lindenberger, Scherer, & Baltes, 2001) and longitudinal (Ghisletta & Lindenberger, 2005; Lindenberger & Ghisletta, 2006) analyses. Moreover, sensory and motor variables are subject to both mortality and experimental selection (Lindenberger et al., 2002). To understand the unique contributions of cognitive factors to mortality prediction better, we created two composite scores by averaging close visual acuity, distance visual acuity, and hearing to represent sensory performance, and an assessment of balance and of postural sway to represent motor performance. All measures were assessed by trained professionals with standard equipment (for more detail, see Baltes & Mayer, 1999). This bidimensional organization, although somewhat simplistic, described the five variables in a two-factor model very well, $\chi^2_{(N = 516,\ df = 5)} = 7.24$, RMSEA = 0.031 (90% confidence interval = [0.000–0.074]), p-value of close fit = 0.72, SRMR = 0.022, GFI = 0.99.

Positive and Negative Personality

The BASE assessed an extensive battery of personality and self variables (cf. Baltes & Mayer, 1999). Nine personality and self dimensions, each measured by several indicators, were analyzed. The personality/self domains were neuroticism, extraversion, openness, positive affect, negative affect, internal positive control, powerful others, future optimism/orientation, and loneliness. Details about these variables can be found in Smith and Baltes (1997, 1999) and Staudinger, Freund, Linden, and Maas (1999). Initial analyses were considered separately for each domain, but led to computational problems. We, therefore, reduced the personality/self space by aggregating variables in two factors. It is worth noting that our intention at this point was not to obtain a factor model that described with the interrelations

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of the nine personality/self scores with great precision, but rather to collapse the latent space representing personality and self variables to simplify the overall interpretations of their effects on mortality. After extensive exploratory and confirmatory factor analyses, we settled on a solution in which extraversion, openness, positive affect, internal control positive, and future optimism/orientation loaded on a factor we called Positive personality. On the other hand, neuroticism, negative effect, powerful others, and loneliness loaded on a second factor we labeled Negative personality. Although the overall model fit was not good, $\chi^2_{(N = 516,\ df = 26)} = 209.416$, RMSEA = 0.112 (95% confidence interval = [0.098–0.127], *p*-value of close fit = 0.000, SRMR = 0.099, GFI = 0.922), this model allows for a simple, albeit not precise, representation of the personality/self domains.

Longitudinal Models

Multilevel models were calculated for each cognitive variable separately. Change was defined over chronological age and the intercept was defined at age 70 years (by subtracting 70 from all ages) for nondemented individuals, prior to any retest effect. Equation (1) depicts the specification of a multilevel model applied to the cognitive variable *Y* of individual *j* assessed at age *i*.

$$Y_{i,j} = \beta_{0j} + \beta_{1j} \cdot \overline{A}_{i,j} + \beta_{2j} \cdot (\overline{A}_{i,j}^2 \mid \overline{A}_{i,j}) + \beta_3 \cdot DD_{i,j} + \beta_{RYk} \cdot R_k + r_{i,j} \quad (1)$$

$$\begin{aligned} \beta_{0j} &= \gamma_{00} + u_{0j} \\ \beta_{1j} &= \gamma_{10} + u_{1j} \\ \beta_{2j} &= \gamma_{20} + u_{2j} \end{aligned} \quad (2)$$

β_0 represents the intercept that is the predicted cognitive performance when all other covariates have the value 0. β_1 captures the linear effect of age centered around 70 years ($\overline{A}_{i,j}$). β_2 estimates the effect of the quadratic component of age, controlling for its dependence on the linear component of age ($\overline{A}_{i,j}^2 | \overline{A}_{i,j}$). The effect of participants' dementia diagnosis at time *i* ($DD_{i,j}$) on the intercept is estimated by β_3. The retest effect at time *k*, where *k* represents an occasion of measurement after the first (i.e., number of occasion – 1, hence, not defined at Occasion 1), is captured by the β_{RYk} parameters. These are the regression weights of the variable-specific dummy codes defined at each occasion after the first (i.e., for DL $k = 1, \ldots, 10$, for CA $k = 1, \ldots, 9$, and for the other variables $k = 1, \ldots 4$, that is, the maximum number of repeated exposures to testing material). Finally, the residual component $r_{i,j}$ defines the error of prediction.

Equation (1) includes participants' subscript *j* for the parameters estimating age effects (β_{0j}, β_{1j}, β_{2j}). Each of these parameters is defined in Equation (2) as the sum of a sample averaged value (also-called fixed effects; γ_{00}, γ_{10}, γ_{20}) and individual-specific variations (also-called random effects; u_{0j}, u_{1j}, u_{2j}), which are assumed to be Gaussian-distributed around 0. In practice, only the variances of the Gaussian-distributed random effects are estimated. With this kind of data, the random effects of the intercept are typically very high and significantly different from zero. However, the power to detect random effects of linear and quadratic age effects is typically low (Hertzog, Lindenberger, Ghisletta, & von Oertzen, 2006). We systematically tested these sources of individual-specific variance, and when significant, we added dementia diagnosis (individual-specific information) as a predictor (by adding the interactions between, on the one hand, $DD_{i,j}$ and on the other hand $\overline{A}_{i,j}$ and $[\overline{A}_{i,j}^2 | \overline{A}_{i,j}]$). The individual-specific random effects represent the contribution of the longitudinal model to the joint modeling of longitudinal and survival data.

Survival Models

As the joint longitudinal and survival models rely on a general maximum likelihood estimator, they require a formal specification of both processes. Therefore, we could not integrate nonparametric models. More specifically, the Cox model could not be used because it does not formally specify the survival function (a partial, rather than a full, likelihood algorithm is used as estimator; see Kalbfleisch & Prentice, 1980; Kleinbaum, 1996). We considered two parametric survival models, the Weibull and the exponential. The event of interest is death, and time (*t*) is the number of years at which the event (death) occurs after age 70 years. That is, we again centered age at 70 years and defined the risk of death for those having survived up to age 70 years (age of the youngest BASE participant at study inception).

The formula of the Weibull survival model is presented in Equation (3).

$$\lambda_j(t) = (r \cdot t^{r-1}) \cdot \exp(\alpha_0 + \alpha_1 \cdot sex_j + \alpha_2 \cdot IA_j + \alpha_3 \cdot SES_j) + W_j(t) \quad (3)$$

$r.t^{r-1}$ is the Weibull hazard function, and $r > 0$. The predictors of the survival model may or may not be those of Equation (1). In our case, for substantive reasons, we did not include the same predictors. The effects of sex (sex_j), initial age (IA_j), and socioeconomic status (SES_j) of individual *j* on his/her hazard rate at time *t* ($\lambda_j(t)$) are captured by the parameters α_1, α_2, α_3, respectively.

In the exponential model $\lambda_j(t) = 1$, for all *t*'s. Hence, when $r = 1$, the Weibull model reduces to the exponential model. The exponential is, thus, statistically nested within the Weibull model, and a likelihood ratio test based on 1 degree of freedom (i.e., *r*) can be applied to compare their fits when a general maximum likelihood algorithm is the estimator. The interpretation of the scale parameter *r* is straightforward. When *r* equals 1 (i.e., in the exponential model), the risk that the event occurs stays constant across

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time. When $r > 1$, the risk of failure increases as time goes by, and when $0 < r < 1$, the risk decreases. In our application, it would seem more sensible that the risk of death does not stay constant in time, but that it actually diminishes as time increases, as a result of increasing sample selectivity. That is, those that survived to age 70 represent an already selected sample (Lindenberger et al., 2002), and, as time increases, the risk of dying at the sample level diminishes.

Finally, $W_j(t)$ includes individual-specific covariate effects and an intercept, usually called frailty. Again, these effects are assumed to be Gaussian-distributed and centered at 0. The $W_j(t)$ term represents the crucial link between the longitudinal and the survival model in the joint modeling.

Two-Stage Longitudinal and Survival Models

The simplest integration of the longitudinal information in a survival model consists of applying a two-stage procedure, similar to the classical analyses of hierarchically organized data prior to multilevel models (cf. Chou, Bentler, & Pentz, 1998; de Leeuw & Kreft, 1986). This approach consists of a first stage, during which random effects of the longitudinal model (equations (1) and (2)) are estimated. In the second stage, the estimated random effects are added as usual covariates in the survival model (equation (3)). This was the method used by Ghisletta and Lindenberger (2000), discussed above.

Joint Longitudinal and Survival Models

Recent statistical advances allow the simultaneous estimation of longitudinal and survival information in a single joint model. Both the longitudinal model in Equation (1) and the survival model in Equation (3) contain individual-specific predictors and allow the estimation of individual-specific random effects around sample-averaged fixed effects. The joint modeling occurs by specifying the longitudinal and the survival models as two submodels where the random effects of the former are included in the term $W_j(t)$ of the latter, as shown in Equation (4).

$$W_j(t) = \gamma_0 \cdot u_{0j} + \gamma_1 \cdot u_{1j} + \gamma_2 \cdot u_{2j} + \gamma_3 \cdot \{u_{0j} + u_{1j} \cdot \overline{A}_{i,j} + u_{2j} \cdot (\overline{A_{i,j}^2} | \overline{A}_{i,j})\} + u_{3j} \quad (4)$$

The parameters γ_0, γ_1, γ_2 define the effects of the individual variations in intercept, linear age, and (residualized) quadratic age of cognitive performance, while γ_3 defines that of the fitted longitudinal value. Finally, u_{3j} represent individual independent frailty terms (cf. Guo & Carlin, 2004). It is important to note that the joint modeling occurs by solving for equations (1) through (4) simultaneously and not incrementally.

Analytical Plan

For all analyses we used the SAS software, version 8 (SAS-Institute, 1999). We first analyzed the individual trajectories in cognitive performance by applying the longitudinal model to each cognitive variable separately[1]. Then we obtained the Bayesian estimates of random effects[2]. Survival models were first computed nonparametrically to enable us to make an educated choice of parametric survival specifications. Initially, only sex was tested in nonparametric survival models[3]. The Weibull and exponential parametric survival models were then computed, estimating the effects of all covariates[4].

Joint analyses require that both longitudinal and survival models be specified and solved with a general likelihood function. Before computing the joint analyses, both longitudinal and survival submodels were estimated with a general likelihood function and these results were compared to those of the previous analyses[5]. We consistently obtained full agreement, justifying the application of the joint analysis. However, we had to check the robustness of results carefully when inserting additional covariates in the survival submodel of the joint analysis. This motivated collapsing the sensory, motor, and self/personality dimensions into aggregate scores.

Finally, we considered all cognitive information together in the same survival model. Indeed, the level and (linear plus quadratic) change components of the cognitive trajectories in BASE share considerable amounts of variance (Lindenberger & Ghisletta, 2006). It is, therefore, of interest to test the cognitive variables' predictability of mortality against each other. As the estimation of multivariate multilevel models is not straightforward in SAS, we ran a two-stage analysis with all cognitive variables simultaneously. We, hence, added the Bayesian estimates of the random effects of all cognitive variables in a unique survival model[4]. Throughout all survival and joint analyses, the Weibull and the exponential distributions were continuously compared, and the Weibull consistently provided a better fit to the data.

1 The longitudinal analyses were computed with the MIXED procedure in SAS.
2 The Bayesian estimates were computed with the NLMIXED procedure in SAS.
3 The nonparametric survival analyses were computed with the LIFETEST procedure in SAS.
4 The Weibull and exponential survival analyses were computed with the LIFEREG procedure in SAS.
5 The NLMIXED procedure in SAS is very flexible and allows the estimation of both longitudinal and parametric survival model with a general likelihood function. These results were then compared to those of the MIXED and LIFEREG procedures, respectively. Consistently, the same outcome was obtained

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Results

The results are laid out in four sections. First, we present survival analyses with noncognitive covariates. These analyses test (1) demographic information, (2) indicators of sensory-motor functioning, and (3) global personality facets. Second, we show the main results of the longitudinal models applied to each cognitive variable separately. Here we focus on the random effects parameters that are subsequently introduced in the survival models. Third, we present joint longitudinal and survival models, where again each cognitive variable was analyzed separately in relation to mortality. Finally, the fourth section presents the results of two-stage analyses, in which longitudinal information from all cognitive indicators was simultaneously tested in survival analyses. Throughout, the Weibull survival model has proven to be highly superior to the exponential, so only Weibull analyses are presented. We adopt a general 0.01 significance criterion for interpreting results.

Baseline Survival Analyses

The first set of analyses served as a baseline comparison for later survival models that included cognitive predictors. The survival models were first computed nonparametrically to obtain the estimated survivor functions for females and males. These curves are displayed in Figure 1 and plot the probability of death as a function of time (i.e., years since age 70). Two conclusions can be drawn from Figure 1. First, as expected, most censored individuals are displayed in the left half. This means that younger individuals (those closer to age 70) are less likely to have experienced the event during the study. In other words, fewer younger than older participants died during the observation period. Second, sex differences are not evident prior to about age 87. Thereafter, however, women are likely to live about two more years than men. The average sex difference in life expectancy in Germany is higher, and this reduced gender gap is probably a result of the selective nature of the BASE sample, which overrepresents older men. The plot of the log of the negative log of the estimated survivor function is shown in Figure 2. This plot can be used to diagnose the appropriateness of an exponential or Weibull survival mod-

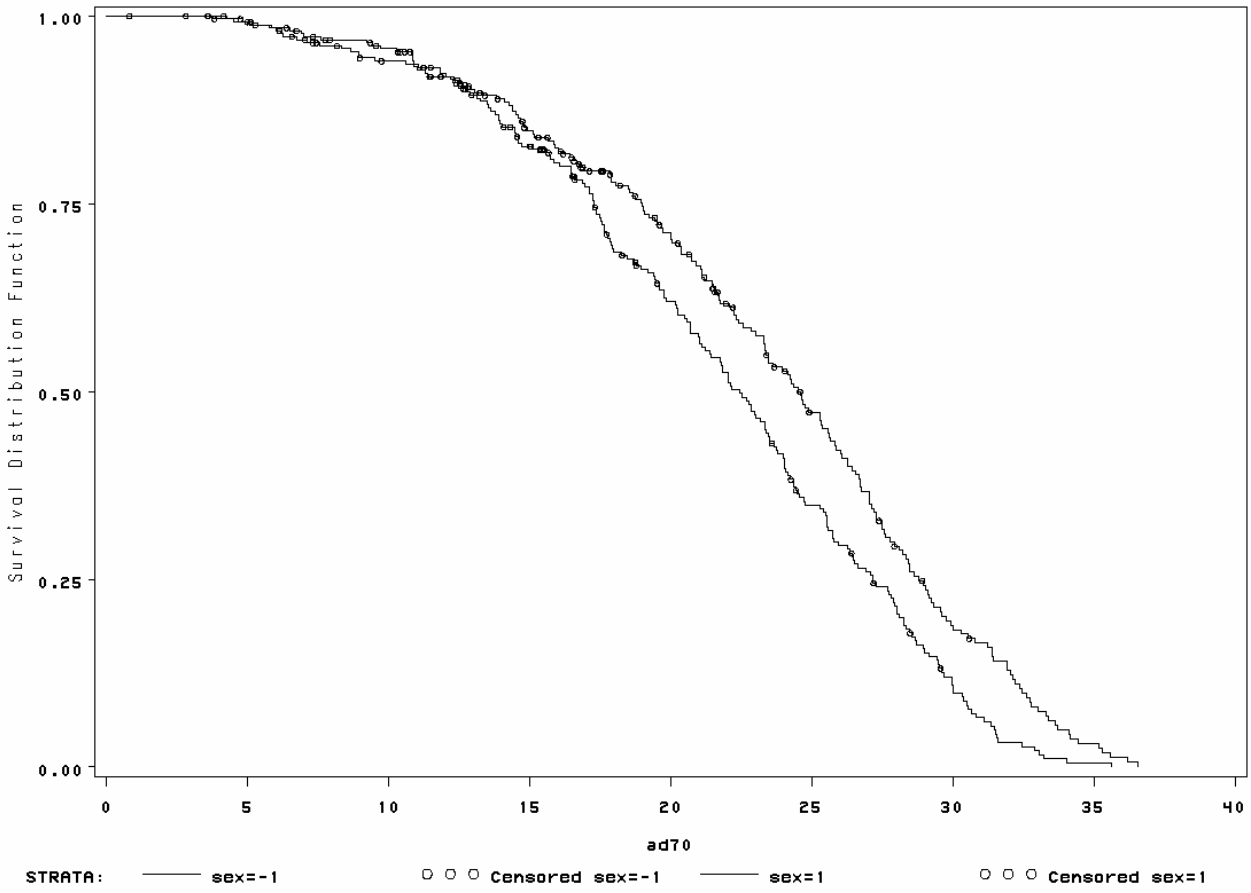

Figure 1. Estimated survival curves starting at age 70 years for men (on the left) and women (on the right). Circles represent right-censored observations.

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Table 2. Effects of age, sex, SES, sensory-motor functioning, and global personality characteristics on age of death in Weibull survival analyses

	Age, sex, SES		Age, sex, SES sensory, motor		Age, sex, SES sensory, motor pos. + neg. pers.	
	Parm. Est. (*SE*)	$p > t$	Parm. Est. (*SE*)	$p > t$	Parm. Est. (*SE*)	$p > t$
Intercept	0.821 (0.093)	<.001	0.286 (0.124)	.021	0.27 (0.13)	.03
Age	0.027 (0.001)	<.001	0.033 (0.001)	<.001	0.03 (0.00)	<.01
Sex	–0.028 (0.008)	<.001	–0.034 (0.001)	<.001	–0.035 (0.008)	<.001
SES	0.013 (0.009)	.156	0.001 (0.010)	.930	–0.001 (0.009)	.931
Sensory	–	–	0.037 (0.012)	.002	0.035 (0.012)	<.001
Motor	–	–	0.045 (0.010)	<.001	0.043 (0.010)	<.001
Pos. pers.	–	–	–	–	0.008 (0.009)	.364
Neg. pers.	–	–	–	–	–0.05 (0.009)	.540
r	0.166 (0.007)	<.001	0.159 (0.006)	<.001	0.159 (0.006)	<.001
–2LL	2,462		2,418		2,402	
# parms	5		7		9	

Note. SES = socioeconomic status. r = Weibull parameter (cf. equation 3). Pos. Pers. = positive personality factor. Neg. Pers. = negative personality factor. –2LL = deviance. # parms. = number of parameters estimated. Parm. Est = parameter estimate. *SE* = standard error. $p > t$ = probability of parameter estimate.

el (Allison, 1995). The lines for men and women are approximately linear and for the most part parallel, which indicates that a Weibull should describe the survival process well. This is confirmed by formal model comparisons for all survival models tested, where the Weibull result was consistently superior to the exponential function (all p's < .001).

Table 2 shows the parameter estimates, standard errors, and p-values of three Weibull survival analyses. Each model estimated the intercept and the r scaling parameter and incrementally tested the covariates. The general conclusions of this table are straightforward. Age and sex are significant, even when tested vis-à-vis sensory and motor performance, and global personality characteristics. SES status is consistently not related to mortality. Sensory and motor performance is predictive of survival, and, finally, the global personality characteristics are not related to mortality, although the sign of their effects are in the expected direction.

Longitudinal Models on Cognitive Data

Table 3 shows all the parameters of eight longitudinal models, one for each cognitive variable. The three major components of the longitudinal model were the level, that is, the predicted performance at age 70, the linear change, equivalent to the yearly linear decline in performance, and the quadratic change, which quantifies the amount of quadratic (curvilinear) decrease in cognitive performance that is independent of the linear change. For each component, the longitudinal models estimated sample averages (fixed effects) and individual variations (random effects). At first, we tested the full longitudinal model of equation (1). However, estimating the variance in the quadratic change component often caused computational problems. We hence tested a reduced form of the model in equation (1) where quadratic change was associated to a fixed effect only. This specification converged to an admissible solution for all cognitive variables.

As expected, all variables displayed variance in level scores. Furthermore, all variables had significant fixed (ranging from –0.801 for DL to –0.219 for SW) and random (ranging from 0.171 for CA to 0.325 for DL) effects for linear change. For all variables, quadratic change was not significant for either fixed or random components, except for DL where the fixed effect resulted significant (–0.029, indicating a negative curvature). In sum, cognitive performance decreased at the sample level across all indicators, but differential linear change appeared on all indicators. Digit Letter was the only indicator whose sample trajectory deviated from linearity.

All individual-specific random effects were statistically controlled for the effects of dementia diagnosis and retest effects. Dementia diagnosis had strong effects on all

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Table 3. Parameters of univariate multilevel models with control for dementia diagnosis and retest effects

	Cognitive variable							
	DL	IP	PA	MT	CA	WB	VO	SW
Fixed effects								
Lev.	61.226 (0.757)	60.793 (0.854)	57.851 (0.954)	57.160 (0.911)	59.155 (0.800)	56.826 (0.946)	56.183 (0.860)	54.780 (0.902)
L.C.	–0.801 (0.047)	–0.652 (0.061)	–0.385 (0.060)	–0.378 (0.058)	–0.505 (0.047)	–0.340 (0.059)	–0.289 (0.055)	–0.219 (0.062)
Q.C.	–0.029 (0.004)	–0.003* (0.005)	0.001* (0.005)	–0.003* (0.004)	–0.003* (0.004)	–0.001* (0.005)	0.003* (0.004)	–0.002* (0.005)
DD	–2.555 (0.976)	–4.537 (1.501)	–8.817 (1.651)	–8.150 (1.662)	–3.474 (1.311)	–6.778 (1.651)	–5.752 (1.376)	–6.333 (1.576)
DD*t	–0.028* (0.053)	–0.043* (0.087)	0.088* (0.089)	0.140* (0.090)	–0.131* (0.068)	0.046* (0.088)	–0.036* (0.074)	0.005* (0.088)
R.-IPr$_1$	0.632 (0.201)	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
R.-IA$_2$	0.341* (0.239)	n.d.	n.d.	n.d.	2.375 (0.347)	n.d.	n.d.	n.d.
R.-IA$_3$	0.533* (0.305)	n.d.	n.d.	n.d.	2.853 (0.432)	n.d.	n.d.	n.d.
R.-IPr$_3$	0.872 (0.315)	2.395 (0.445)	0.422* (0.466)	0.263* (0.501)	0.903* (0.439)	1.156* (0.467)	0.348* (0.384)	1.300 (0.464)
R.-IA$_4$	0.713* (0.370)	n.d.	n.d.	n.d.	3.923 (0.524)	n.d.	n.d.	n.d.
R.-IPr$_4$	1.412 (0.382)	2.709 (0.554)	1.815 (0.573)	1.586 (0.612)	2.000 (0.529)	2.227 (0.589)	3.349 (0.475)	1.379* (0.577)
R.-IA$_5$	0.853* (0.470)	n.d.	n.d.	n.d.	2.107 (0.645)	n.d.	n.d.	n.d.
R.-IPr$_5$	1.526 (0.481)	5.806 (0.759)	3.848 (0.769)	4.406 (0.790)	1.360* (0.656)	1.693* (0.775)	5.021 (0.654)	0.261* (0.770)
R.-IA$_6$	= 0	n.d.	n.d.	n.d.	3.124 (0.831)	n.d.	n.d.	n.d.
R.-IPr$_6$	= 0	1.450* (0.947)	1.099* (0.902)	2.484 (0.870)	= 0	0.642* (0.904)	3.437 (0.820)	2.424 (0.939)
Random effects								
Lev.	99.701 (14.277)	65.434 (13.179)	120.390 (16.595)	104.240 (16.985)	78.774 (11.725)	108.530 (19.661)	91.077 (13.130)	79.423 (15.176)
L.C.	0.325 (0.047)	0.249 (0.050)	0.310 (0.048)	0.307 (0.052)	0.171 (0.035)	0.255 (0.050)	0.190 (0.036)	0.252 (0.055)
Lev-LC	–3.811 (0.734)	–2.578 (0.739)	–4.750 (0.841)	–4.659 (0.894)	–2.282 (0.583)	–3.816 (0.862)	–2.615 (0.638)	–2.878 (0.859)
Resid.	9.547 (0.386)	16.129 (1.119)	20.376 (1.181)	25.513 (1.520)	23.275 (0.972)	21.223 (1.265)	11.463 (0.699)	18.454 (1.136)
–2LL	12,592	7,228	8,342	8,376	12,196	8,419	7,867	7,758
#parms.	17	13	13	13	17	13	13	13

Note. Nonsignificant parameters at $p = .01$ are marked with an asterisk (*). n.d. = not defined. = 0 means parameter fixed at zero (i.e., not estimated). Lev = level. L.C. = linear change. Q.C. = quadratic change (residualized for linear change). DD = prediction of dementia diagnosis on level variance. DD*t = prediction of dementia diagnosis on linear change variance. R. = occasion specific retest effect. IA = Initial assessment. IPr = Intensive protocol. Resid. = residual variance. –2LL = deviance. # parms. = number of parameters.

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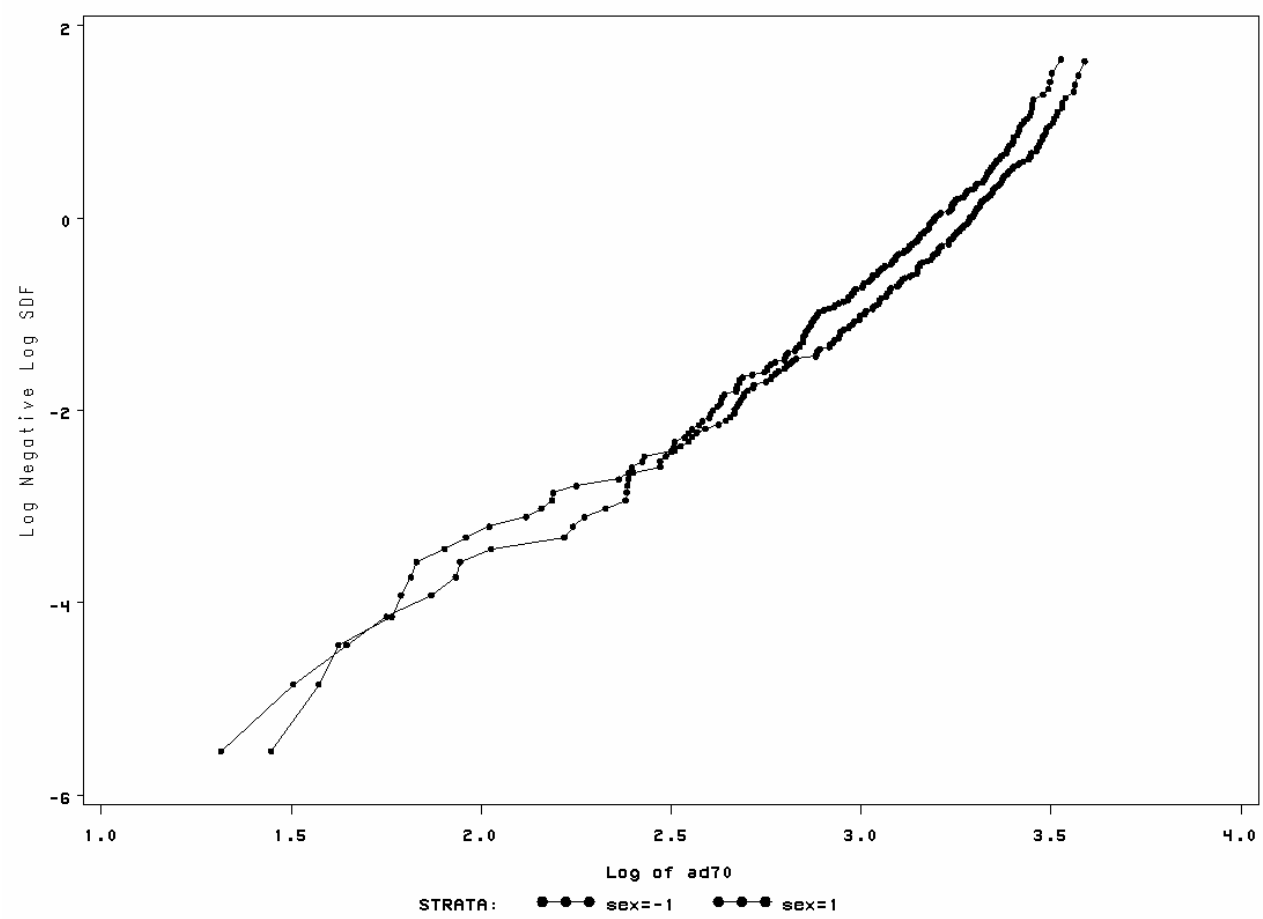

Figure 2. Log of the negative log of the estimated survival curves for men (above) and women (below).

variables' level component, varying in size from –2.555 in DL to –8.817 in PA. This means that when participants were diagnosed with dementia, their performance decreased substantially. Retest effects were found across all variables, but not on all occasions. Generally, the sign of the estimated retest effects were positive as expected (in 40 out of 43 cases), meaning the repeated exposure to the testing material increased one's score. Only in 3 cases were retest effects estimated to be negative. This occurred on the last two testing occasions for DL and on the last for CA. These two variables were measured most intensively (11 and 10 times, respectively, versus 6 repeated measures of the other cognitive variables; cf. Table 1). As a consequence, the age and the retest information end up overlapping to a greater extent for DL and CA. In this situation, it is best not to estimate the retest effect, because of its colinearity with age. We therefore fixed 3 parameters (of a total of 43), estimating retest effects to zero (for DL at the sixth initial assessment and for DL and CA at the sixth IPr). In sum, from these models we can conclude that each cognitive variable contributes level and linear change information to mortality prediction (u_{0j} and u_{1j} in equation (4)). None of the variables adds information in quadratic change (u_{2j}).

Joint Longitudinal and Survival Models

The joint analyses estimated the individual-specific random effects of the longitudinal process simultaneously and specified them as covariates of mortality in the survival process. This was done independently for each cognitive variable, and results are shown in Table 4. Similarly to the analyses displayed in Table 2, three types of survival models were specified. In the first model we controlled for demographic predictors (cf. second column), in the second model we further accounted for sensory and motor performance (cf. third column), and in the third model we also considered broad personality characteristics (cf. fourth column). The three survival models were merged with eight longitudinal models as specified above (one for each cognitive variable), so that in the end we tested 24 joint longitudinal and survival models. Table 4 contains only the predictability of the cognitive variables (for simplicity).

Throughout all models, age, sex, and motor performance (balance and gait) were consistently significant predictors of mortality. Participants who were older, male, and with lower motor functioning, showed an increased risk of death. Males had a risk of death 1.5% to 4.9% greater to that of females. The effect sizes of age and motor perfor-

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Table 4. Effects of cognitive performance on age of death in joint longitudinal and Weibull survival analyses (each cognitive indicator analyzed independently)

	age, sex, SES		age, sex, SES sensory, motor		age, sex, SES sensory, motor pos. + neg. pers.	
	Parm. est. (SE)	$p > t$	Parm. est. (SE)	$p > t$	Parm. est. (SE)	$p > t$
DL_L	0.008 * (0.002)	<.001	0.006 * (0.002)	<.001	0.006 * (0.002)	<.001
DL_{LC}	0.144 * (0.028)	<.001	0.122 * (0.030)	<.001	0.121 * (0.032)	<.001
IP_L	0.011 * (0.002)	<.001	0.008 * (0.002)	<.001	0.007 * (0.002)	<.001
IP_{LC}	0.114 * (0.033)	<.001	0.087 (0.034)	.101	0.082 (0.035)	.020
PA_L	0.005 * (0.002)	.001	0.004* (0.001)	.009	0.004* (0.001)	.009
PA_{LC}	0.074 (0.035)	.032	0.061 (0.033)	.068	0.059 (0.034)	.083
MT_L	0.006* (0.002)	.003	0.005 (0.002)	.011	0.005 (0.002)	.015
MT_{LC}	0.075 (0.041)	.064	0.068 (0.038)	.076	0.065 (0.039)	.094
CA_L	0.009* (0.002)	<.001	0.007* (0.001)	<.001	0.007* (0.002)	<.001
CA_{LC}	0.126* (0.042)	.003	0.104 (0.041)	.011	0.098 (0.043)	.023
WB_L	0.007* (0.002)	<.001	0.006* (0.001)	<.001	0.006* (0.002)	<.001
WB_{LC}	0.110* (0.039)	<.001	0.092 (0.038)	.016	0.091 (0.038)	.017
VO_L	0.004* (0.001)	.003	0.003 (0.001)	.034	0.003 (0.001)	.034
VO_{LC}	0.076 (0.040)	.058	0.054 (0.038)	.155	0.049 (0.039)	.215
SW_L	0.004* (0.002)	.009	0.002 (0.002)	.167	0.002 (0.002)	.158
SW_{LC}	0.065 (0.034)	.056	0.041 (0.034)	.238	0.036 (0.036)	.308

Note. DL = Digit Letter. IP = Identical Pictures. PA = Paired Associates. MT = Memory for Text. CA = Categories. WB = Word Beginnings. VO = Vocabulary. SW = Spot-a-Word. The subscripts L, LC, and QC denote the level, linear change, and quadratic change longitudinal components, respectively. SES = socioeconomic status. Pos. + neg. pers. = positive and negative personality factors. Parm. est. = parameter estimate. *SE* = standard error. $p > t$ = probability of parameter estimate.* denotes parameters significant at the .01 level.

mance were very similar across all models. Each additional 1-unit change was associated with an increased risk of 2.5% to 3.6%. SES and the two broad personality factors were consistently not related to mortality. Sensory performance (vision and hearing) was predictive of mortality when analyzed with PA and MT (both with and without statistical control for personality factors) and with VO (only prior to adding the personality factors).

When we controlled only for age, sex, and SES, all cognitive variables predicted mortality in their level component. Furthermore, four variables, DL, IP, CA, and WB, predicted mortality in their linear change component. When we further added statistical control for sensory and motor performance, MT, VO, and SW were no longer predictive in the level component, and only DL was predictive in its change component. Adding the personality factors did not alter this picture. All significant parameter estimates were positive, meaning that higher cognitive scores delayed the age of death. However, the effects of level of performance in cognition on survival were meager. The weakest significant effect was for level in VO and SW, where each additional unit of performance was associated with a 0.1% to 0.9% increased risk of death. Effects were much stronger for linear change in cognitive performance. The strongest significant effect was for linear change in DL when only demographic variables were controlled for. The associated risk ranged from 9.3% to 22.1% for each additional unit of change. It is worth not-

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Table 5. Effects of cognitive performance on age of death in two-stage longitudinal and Weibull survival analyses (all cognitive indicators analyzed simultaneously)

	age, sex, SES		age, sex, SES sensory, motor		age, sex, SES sensory, motor pos. + neg. pers.	
	P.Est. (*SE*)	$p > t$	P.Est. (*SE*)	$p > t$	P.Est. (*SE*)	$p > t$
DL_L	0.068 (0.002)	.305	0.001 (0.002)	.503	0.002 (0.002)	.484
DL_{LC}	0.007 (0.039)	.086	0.068 (0.039)	.082	0.070 (0.040)	.080
IP_L	–0.017 (0.003)	.017	0.005 (0.003)	.074	0.005 (0.003)	.080
IP_{LC}	0.001 (0.041)	.679	–0.034 (0.041)	.413	–0.034 (0.042)	.412
PA_L	0.001 (0.002)	.609	0.001 (0.002)	.747	0.001 (0.002)	.758
PA_{LC}	–0.011 (0.035)	.755	–0.005 (0.035)	.881	–0.005 (0.035)	.888
MT_L	0.002 (0.002)	.374	0.002 (0.002)	.380	0.002 (0.002)	.369
MT_{LC}	0.014 (0.042)	.732	0.018 (0.042)	.672	0.019 (0.042)	.646
CA_L	0.005 (0.002)	.034	0.005 (0.002)	.032	0.005 (0.002)	.038
CA_{LC}	0.102 (0.046)	.028	0.087 (0.046)	.060	0.087 (0.048)	.070
WB_L	0.002 (0.002)	.383	0.002 (0.002)	.377	0.002 (0.002)	.383
WB_{LC}	0.039 (0.042)	.355	0.036 (0.042)	.387	0.036 (0.043)	.397
VO_L	–0.001 (0.002)	.532	–0.001 (0.002)	.655	–0.001 (0.002)	.651
VO_{LC}	–0.060 (0.042)	.157	–0.052 (0.042)	.213	–0.051 (0.043)	.229
SW_L	0.001 (0.002)	.666	0.001 (0.002)	.917	0.001 (0.002)	.936
SW_{LC}	0.025 (0.035)	.475	0.017 (0.036)	.640	0.015 (0.037)	.679
–2LL	2,074		2,060		2,045	
# parms.	21		23		25	

Note. –2LL = deviance. # parms. = number of parameters estimated. SES = socioeconomic status. Pos. Pers. = positive personality factor. Neg. Pers. = negative personality factor. P.Est = parameter estimate. *SE* = standard error. $p > t$ = probability of parameter estimate. * denotes parameters significant at the .01 level.

ing, however, that while there were big variations in level of performance (ranging from a *SD* of 8.089 – square root of 65.434, cf. Table 3 – for IP to a *SD* of 10.972 for PA), variability in linear change was much smaller (ranging from a *SD* of 0.414 for CA to a *SD* of 0.570 for DL).

Two-Stage Survival Models

The final analyses aimed at testing the predictability of mortality using all cognitive variables together. This is currently too difficult in a joint analysis, so we opted for a two-stage approach. In the first step of the two-stage longitudinal plus survival analysis, the random effects revealed in the longitudinal analyses were estimated via Bayesian procedures. In the second step, we included the estimated random effects of all cognitive variables together in three survival analyses. In the first model, we again controlled for demographic information only; then, in the second model we added sensory-motor performance, and in the third model we also included personality factors. These results are shown in Table 5.

We consistently found that age, sex, and motor performance were related to mortality (all *p*s < .01), with effects very similar to those of the univariate joint analyses. SES, sensory performance and both personality factors were not

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related to mortality. Across the three models, no cognitive variable predicted mortality, neither in its level nor in its change component. In short, when all cognitive variables were tested against each other, no one single marker of cognitive performance showed a strong relation to survival information. It is worth noting that these results are based on a two-stage analysis and should be interpreted with caution. A joint longitudinal plus survival analysis should result in a gain in statistical efficiency and power (Guo & Carlin, 2004).

Discussion

The goals of this paper were two-fold. First, we wanted to further explore mortality prediction in the BASE in relation to up to 11 individual longitudinal measurements of cognitive performance, across a period lasting over 13 years. Second, we wanted to discuss in this context a recent advance in data analysis that joins longitudinal and survival models in a common statistical framework to examine the likely dependencies between the two processes.

With respect to cognitive change, the indicators of perceptual speed (DL and IP) displayed the steepest average sample decline, while the crystallized knowledge measures (VO and SW) evinced the lowest average change, after controlling for dementia diagnosis and retest effects. Variance in linear change was found for all cognitive indicators. This finding replicates previous BASE results obtained with cross-sectional data (Baltes & Lindenbeger, 1997; Lindenberger & Baltes, 1994, 1997) and with fewer longitudinal observations (e.g., Ghisletta & Lindenberger, 2003, 2004; Lindenberger & Ghisletta, 2006; Lövdén et al., 2004; Singer et al., 2003) as well as results of several independent studies (e.g., McArdle, Ferrer-Caja, Hamagami, & Woodcock, 2002; McArdle, Hamagami, Meredith, & Bradway, 2000; Schaie, 2005).

With respect to mortality prediction, our analyses partially agree with the previous BASE results discussed in the Introduction. In particular, we found that when the cognitive variables were analyzed individually in the joint longitudinal and survival models with demographic information (age, sex, and SES), all of them were related to mortality in their level component. This result clearly disagrees with the specificity hypothesis. When we statistically controlled further for sensory and motor performance and broad personality characteristics, both indicators of verbal knowledge (VO and SW) and one indicator of episodic memory (MT) were no longer associated with mortality, neither in their level, nor in their change component. This finding is in clear disagreement with the hypothesis of White and Cunningham (1988) that variables resistant to age effects should be most vulnerable to impending death. Overall, we confirmed previous results that level information in speed (Birren, 1964; Bosworth, Schaie, & Willis, 1999; Maier & Smith, 1999), episodic memory (Hassing, Small, von Strauss, Fratiglioni, & Bäckman, 2002; Shipley, Der, Taylor, & Deary, 2006; Small & Bäckman, 1997), and verbal fluency (Small & Bäckman, 1997) predict mortality when controlling for a number of important covariates.

The results of the joint analyses provide information about the terminal-decline hypothesis. Besides level information, the longitudinal component of the joint model also formally related change in cognition to mortality. Independent of age, sex, and SES, changes in speed (DL and IP) and in verbal fluency (CA and WB) provide information about the age at death; individuals with steeper declines in cognitive functioning were more likely to die. However, despite the high number of repeated measurements available in the BASE, we did not obtain variance in quadratic change.

The present data-analytic set-up is well suited to test the hypothesis that individuals with steeper performance decrements as a function of chronological age tend to be closer to death. Note, however, that terminal decline has also been conceived as a more abrupt drop in performance that is preceding death more or less immediately (e.g., Figure 5 in Baltes & Labouvie, 1973, p. 174; Figure 1 of Johansson et al., 2004, p. 146). The present set of analyses is not optimally suited to examine this variant of the terminal decline hypothesis because developmental time is represented as a continuous function over chronological age (i.e., distance from birth). Analyses with years to death as the main developmental time axis may be more sensitive to abrupt drops in performance that occur a certain number of years prior to death, and may therefore turn out to be superior in modeling this aspect of terminal decline, if and when it exists (see Sliwinski, Hofer, Hall, Buschke, & Lipton, 2003, for a similar application).

The final two-stage analyses allowed us to examine all cognitive variables conjointly, such that the effect of each would be statistically controlled for by all other variables. We expected that the number of significant survival predictors would diminish, given their high degree of interdependence (i.e., positive manifold of cognitive performance, Spearman, 1927). Surprisingly, no cognitive variable was significantly related to survival, neither in its level, nor in its linear change component. Based on some of our previous work (Ghisletta & Lindenberger, 2000) we expected that at least the level in fluency performance and change in perceptual speed would be significant predictors of age at death. Perhaps this lack of results is a result of the lower efficiency of the two-stage analysis when compared to the joint analysis (cf. Guo & Carlin, 2004). This possible difference in statistical efficiency should be investigated in future work.

The cognition-mortality relations examined here were only partially mediated by the covariates we considered. As expected, age and sex proved to be important covariates (Bosworth et al., 1999; Rabbitt et al., 2002). On the other hand, education, a principal constituent of our SES score, was not essential. This replicates some previous findings, both in the BASE (Lindenberger et al., 2002; Maier & Smith, 1999) as well as in other studies (e.g., Hassing, Johansson, et al.,

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2002; Rabbitt et al., 2002). It is possible that individual differences in health outcomes in old and very old age become progressively more predictable from biological processes and less from cultural factors (for related arguments, see Baltes, Lindenberger, & Staudinger, in press; Lindenberger & Baltes, 1997). In part, these differences may be of genetic origin (e.g., McGue, Vaupel, Holm, & Harvald, 1993). In addition, stochastic aspects intrinsic to biological aging may also play a role (cf. Thaler, 2002).

Sensory information was predictive of mortality in the initial analyses that ignored cognitive functioning (Anstey, Luszcz, Giles, & Andrews, 2001; Lindenberger et al., 2002). However, when analyzed in conjunction with cognitive performance, sensory information lost some (in the joint analyses) or all (in the final two-stage analyses) predictive power. With all likelihood, this is because in very old age vision and hearing share considerable amounts of variance with cognitive functioning, especially with measures that are highly age sensitive, such as perceptual speed (Anstey, 1999; Anstey, Hofer, & Luszcz, 2003; Anstey & Smith, 1999; Ghisletta & Lindenberger, 2005; Lindenberger & Baltes, 1994; Stankov & Horn, 1980). Overall, motor performance was related to mortality independently of cognitive performance. Indeed, it retained its predictive power even in the final survival analyses that included all cognitive variables simultaneously. Personality characteristics have also been shown to relate to mortality (e.g., Berkman, 1988; Friedman et al., 1995; Maier & Smith, 1999). This was not the case in the present set of analyses. Constrained by the computational complexity of the joint model, we reduced the BASE personality battery to two global factors. Thus, we cannot exclude that some of the personality constructs, such as extraversion, openness, and emotional loneliness, may predict mortality within the present data-analytic framework when considered separately.

The joint analysis, although available in standard statistical software like SAS, is quite intensive and at present seems to limit the number of possible covariates. When we tried adding additional covariates in the analysis (e.g., single personality aspects rather than global characteristics), we often encountered convergence problems. Furthermore, analyses of small samples (e.g., young-old vs. old-old individuals) resulted in similar empirical problems. Hence, a number of important covariates were not tested in this study. These include cause of death (Hassing, Johansson, et al., 2002; Shipley et al., 2006; Small & Bäckman, 1999), existing pathologies (Anstey et al., 2001; Rabbitt et al., 2002), and depression or dysphoria assessment (Lindenberger et al., 2002; Rabbitt et al., 2002), all of which have been shown to be related to survival.

More extensive cognitive measures would have increased knowledge about the cognition-mortality association. For example, Deary and Der (2005) have shown reaction time measures to be more powerful predictors of mortality than a test of general intelligence. Shipley, Der, Taylor, and Deary (2006) showed further that level and change (variability) estimates of reaction time were significantly related to higher risks of death over a 19-year period. The results held after control for demographic, lifestyle, and physical variables and also in a group of young adults. Reaction time (level and variability) could indicate efficiency of information processing and aging-induced deterioration of the neural system (cf. Hultsch, MacDonald, & Dixon, 2002; Li, Lindenberger, & Sikström, 2001). In the present analyses, measures of perceptual speed (i.e., DL and IP) and of verbal fluency (CA and WB) showed the strongest predictive relations to mortality, and the only variable whose change was predictive of death after controlling for all covariates considered was DL. As psychometric constructs, perceptual speed tests and choice reaction-time tasks are highly related and at times undistinguishable. Thus, the present results are well in agreement with these earlier findings.

All preceding examinations of the cognition-mortality association have failed to account for possible retest effects. Several longitudinal studies have shown that estimates of aging effects can be biased by retest effects (Ferrer et al., 2004, 2005; Ghisletta & de Ribaupierre, 2005; Lövdén et al., 2004; McArdle et al., 1998; Rabbitt et al., 2001, 2004; Salthouse, Schroeder, & Ferrer, 2004). Furthermore, some research has shown age interactions with retest effects (Lövdén et al., 2004; Rabbitt et al., 2004). In the present context, one might imagine that individuals approaching death may benefit less from repeated test exposure than those in better health. As a result, ignoring retest effects could imply that cognitive performance of the survivors would be overestimated by a greater degree than that of the people near to death. Although we are not saying that statistically controlling for retest effects (as was done here) is the panacea to this issue, we do think that not doing anything about possible retest effects may bias the results and subsequently all theoretical inferences. Clearly, the planned inclusion of control groups by design should result in more precise estimates of retest effects than the statistical modeling of available variability in test exposure (cf. McArdle & Woodcock, 1997; Schaie, 1965). The design of the BASE, however, as in most large-scale psychological panel studies, did not include such groups.

In conclusion, we feel that investigating the cognition-mortality association remains an important aspect of gerontological research. Modern statistical methods can be helpful in this context, provided that they are applied to appropriate designs. For crucial outcomes such as death, research designs that are sensitive to within-person patterns of change for the individuals composing the sample are more appropriate than designs that optimize descriptions of the group average.

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About the authors

Paolo Ghisletta is a research assistant professor at the Center for Interdisciplinary Gerontology and at the Faculty of Psychology and Educational Sciences of the University of Geneva, Switzerland. His main research interests include cognitive aging processes, lifestyle of older individuals, inter- and intraindividual variability across the lifespan, and multivariate methodologies applied to lifespan developmental processes.

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John J. (Jack) McArdle, Ph.D., is currently Senior Professor of Psychology at the University of Southern California in Los Angeles, CA, USA. His research has been focused on age-sensitive methods for psychological and educational measurement and longitudinal data analysis including publications in factor analysis, growth curve analysis, and dynamic modeling of adult cognitive abilities. He is also director of the ongoing National Growth and Change Study (NGCS), a longitudinal study of cognitive changes over age in the USA.

Ulman Lindenberger is a professor of psychology and directs the Center for Lifespan Psychology at the Max Planck Institute for Human Development. He studies cognitive and sensorimotor development across the lifespan. Specifically, he is interested in mechanisms regulating links between domains of functioning (e.g., sensorimotor, cognitive) and levels of analysis (e.g., behavioral, neuronal) during learning and lifespan development.

Paolo Ghisletta

Center for Interdisciplinary Gerontology and Faculty of Psychology and Educational Sciences
University of Geneva
Route de Mon-Idée 59
CH-1226 Thônex / Geneva
Switzerland
Tel. +41 22 305-66-04
Fax +41 22 348-90-77
E-mail paolo.ghisletta@cig.unige.ch

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