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Conflict of Interest

None.

Abstract

Functional health declines with advancing age, which is supposedly the consequence of both normal, “primary aging” as well as of mortality-related, “tertiary aging” processes. To better understand the independent effects of both processes, we investigated how age and time to death relate to changes in functional health over up to 12 years.

Additionally, adopting the disablement process model for an end-of-life perspective, we investigated if both age and time to death moderate associations of information processing speed and subjective well-being with functional health. Data from the German Ageing Survey were used. Our sample consisted of 578 participants who had died between 2002 and 2016 (mean age at death: 76.59 years, range 45-95 years). Information Processing Speed was measured by the Digit Symbol Substitution Test. The well-being indicators assessed were positive affect and depressive symptoms. Based on longitudinal multilevel regression models, we found that functional health significantly decreased over time in study. Approaching death was a stronger predictor of functional health decline than was chronological age. Regarding moderation effects, controlling for gender, education, and multimorbidity, individuals closer to death at baseline revealed stronger associations of both depressive symptoms and information processing speed with baseline functional health, whereas these associations were not moderated by chronological age.

Our findings suggest that change in functional health is more strongly affected by time to death than by chronological age. Moreover, there may be a growing importance of cognitive resources and well-being for functional health with advanced “tertiary aging”, but not with progression of “primary aging”.

Key Words: Physical functioning, primary aging, tertiary aging, depressive symptoms, disablement process model

7,888 Words

Functional health (or everyday competence; M. M. Baltes, Maas, Wilms, Borchelt, & Little, 1999; Diehl, 1998) describes the extent to which an individual is able to independently execute important tasks of daily living (such as walking, stair climbing, bathing or dressing). Intact functional health is a key indicator of “successful aging” (Rowe & Kahn, 1997) and can be considered an important prerequisite for autonomy and quality of life (Schilling, Wahl, & Reidick, 2013; Schöllgen, Morack, Infurna, Ram, & Gerstorf, 2016).

There is strong evidence that functional health declines with advancing age (Berlau, Corrada, & Kawas, 2009; Jacobs et al., 2012; Wahl, Schmitt, Danner, & Coppin, 2010), particularly in old and very old age (P. B. Baltes & Smith, 2003; Wettstein, Schilling, & Wahl, 2016). However, age is, of course, not the causal mechanism driving functional health change, but acts as a proxy variable. As P. B. Baltes, Lindenberger, and Staudinger (2006) state, “chronological age carries a multitude of causal agents with different and intertwined temporal dynamics” (p. 600). Similarly, DeCarlo and colleagues point out when referring to functional changes that “age is not a causal mechanism underlying cognitive and functional decline, but rather a temporal dimension along which causal factors operate” (DeCarlo, Tuokko, Williams, Dixon, & MacDonald, 2014, p. 96).

The “intertwined temporal dynamics” mentioned above have been further specified as processes of primary, secondary, and tertiary aging (Birren & Cunningham, 1985; Ram, Gerstorf, Fauth, Zarit, & Malmberg, 2010): Whereas primary aging refers to “normal” aging and changes that occur as a consequence of biological and physical decline and of “processes that are intrinsic to aging (i.e., unfolding regularly and irreversibly within individuals at certain ages)” (Schilling, 2016), secondary aging describes pathological processes that occur due to disease and disability. Finally, tertiary aging is characterized by “accelerated functional deteriorations that manifest shortly (months, maybe years) before death” (Ram et al., 2010, p. 28).

Contrasting primary vs. tertiary aging, decline in functional health indeed seems to be more pronounced when considered over the time metric “time to death”, which can be considered a “global overall vitality index” (Thorvaldsson, Hofer, & Johansson, 2006, p. 202), rather than over

chronological age (Diehr, Williamson, Burke, & Psaty, 2002; Gerstorf, Ram, Lindenberger, & Smith, 2013; Klijs, Mackenbach, & Kunst, 2010). Such a “terminal decline” phenomenon (Hülür, Ram, & Gerstorf, 2016; Schilling, 2016) has also been observed across other major developmental domains, such as cognition (Bäckman & MacDonald, 2006; Riegel & Riegel, 1972) or subjective well-being (Gerstorf, Hülür, Wagner, Kunzmann, & Ram, 2018; Gerstorf et al., 2010). So far, the specific biological mechanisms leading to terminal decline are not well understood. “Physiological deterioration or damage” (Riegel & Riegel, 1972, p. 314), accumulation of morbidity and mortality-related processes (such as the onset and progression of terminal diseases), and “biological processes of deterioration that precede and will finally precipitate the death of the individual” (Schilling, 2016) have been discussed as possible mechanisms.

Functional Health Trajectories: The Role of Age vs. Time to Death

Indeed, given the accumulating empirical evidence for terminal decline processes in functional health, and given the fact that “aging itself is not the ‘clocks’ that time aging” (S. C. Li & Schmiedek, 2002, p. 8), observed trends of age-related decline in functional health in later life might be to a large extent due to mortality-related, tertiary aging processes rather than due to normal, primary aging processes. For a better and direct comparability of the role of primary aging, measured as time since birth, vs. tertiary aging, measured as time to death, for functional health changes, it might be useful to empirically consider them simultaneously in one single model. Such an approach has already been applied to better understand change dynamics in domains other than health, such as cognitive or sensory abilities (Lindenberger & Ghisletta, 2009; Ram et al., 2010; Thorvaldsson et al., 2006). However, to our knowledge, functional health has – apart from one exception (Wolf, Freedman, Ondrich, Seplaki, & Spillman, 2015) – not yet been investigated as a process that is simultaneously driven by age and time to death. This exception is a study by Wolf et al. (2015) who specified latent-class trajectory models of disablement, and they found that nearness to death was more consistently associated with disability across the different classes than was age.

Adopting the conceptual and methodological approach introduced by Ram et al. (2010), we will investigate changes in functional health over time in study in an age-heterogeneous sample of now deceased middle-aged and older adults. Specifically, following Ram et al. (2010) by understanding time not only as a within-person process (as represented by “time in study” in our analyses), we will also investigate if and to what extent age at baseline vs. time to death at baseline simultaneously predict these changes. Both age and time to death represent “time-as-a-resource/burden” (Heirich, 1964; Ram et al., 2010) variables that indicate “interindividual differences in the accumulation of experience” (Ram et al., 2010, p. 40). Our assumption is that shorter distance to death may be more closely associated with steeper functional health decline than higher chronological age.

Moreover, as outlined in more detail below, age and time to death considered from such a time-as-a-resource/burden perspective might augment or buffer associations of established risk and protective factors with functional health, resulting in “age moderation effects” and “time to death moderation effects”. Such moderation effects might be of differential strength for the time metrics age vs. time to death.

The Disablement Process Model from an End-of-Life Perspective

There is remarkable heterogeneity regarding changes in functional health (Wettstein et al., 2016; Willis, Jay, Diehl, & Marsiske, 1992), both when considered over chronological age/primary aging and when considered over distance to death/tertiary aging. Several distinct profiles of age-related and terminal functional health changes have been identified in prior studies (Leigh, Byles, & Mishra, 2017; Lunney, Lynn, Foley, Lipson, & Guralnik, 2003; Wolf et al., 2015). Whereas a variety of risk and protective factors with regard to functional health is known from prior research, the role of these factors with advancing age and with increasing nearness to death is less clear. As outlined in more detail below, different scenarios are possible, that is either an increase in the importance of these factors with advancing age and impending death, or a decrease. Finally, these

moderating trends could also be different for age vs. time to death, resulting in “age moderation effects” that do not necessarily mirror the respective “time to death moderation effects”.

One established theoretical framework that addresses factors associated with functional health in general (and not necessarily at the end of life) is the disablement process model (Verbrugge & Jette, 1994). This model suggests that different classes of factors predict the onset or progression of functional limitations. Specifically, a distinction is made between risk factors (such as demographic variables, e.g. gender and education), intra-individual factors (e.g., psychosocial attributes such as positive affect, but also cognitive abilities and depressive symptoms), and extra-individual factors (such as medications) that may either increase or reduce the impact of accumulating diseases on functional limitations in aging individuals.

Building on this model, we will focus on two key developmental domains that are intra-individual factors in the disablement process model and that may be particularly closely associated with functional health trajectories and with the disablement process (Braungart Fauth, Zarit, Malmberg, & Johansson, 2007; Caplan & Schooler, 2003; Femia, Zarit, & Johansson, 2001; Pérès, Verret, Alioum, & Barberger-Gateau, 2005), namely cognitive abilities (specifically, information processing speed) and subjective well-being (specifically, positive affect and depressive symptoms).

Although the disablement process model postulates that these factors are moderators of the impact of pathology and diseases on functional limitations, we will investigate their *direct* association with changes in functional health, controlling for chronic diseases as an indicator of “secondary aging”. By considering age and time-to-death as potential moderators of these associations in a sample of decedents, we set the disablement process in an end-of-life context. Apart from multimorbidity (i.e., number of chronic diseases) – which is, according to the disablement process model, the key risk factor for functional limitations – additional demographic risk factors specified in the model (gender and education) will be considered as control variables as they have been identified as important predictors of health changes in prior studies, particularly in

individuals who are close to death (Gerstorf et al., 2013; Syddall, Cooper, Martin, Briggs, & Aihie Sayer, 2003; Wolf et al., 2015).

Like functional health, cognitive abilities (Bäckman & MacDonald, 2006; Riegel & Riegel, 1972), positive affect (Schilling, Deeg, & Huisman, 2018; Vogel, Schilling, Wahl, Beekman, & Penninx, 2013), and depressive symptoms (Burns et al., 2013; Diehr et al., 2002; Fauth, Gerstorf, Ram, & Malmberg, 2014; Hülür, Ram, & Gerstorf, 2015) are developmental domains that reveal terminal change. These tertiary aging dynamics across different domains may to some extent be driven by the same mortality-related processes as functional health, so that the association of processing speed and well-being with functional health might be closer in individuals who are closer to death, as evidenced by a “time to death moderation effect”.

Of course, this argument can also be made with regard to primary aging: There is evidence for age-related change in information processing speed (McArdle, Hamagami, Meredith, & Bradway, 2000; Salthouse, 2009; Wettstein, Wahl, Siebert, & Schröder, 2019), positive affect (Charles, Reynolds, & Gatz, 2001), and depressive symptoms (Sutin et al., 2013), so that the same mechanisms behind primary aging may drive changes in these domains and in functional health. This, in turn, should result in an “age moderation effect” i.e. closer associations of information processing speed and well-being with functional health among individuals who are older.

There is one additional reason for our expectation of moderating effects by age and time to death: Both increasing age – particularly very old age (P. B. Baltes & Smith, 2003; Gerstorf & Ram, 2009) – and proximity to death (Hülür et al., 2016) are life phases of heightened general vulnerability with regard to health and other life domains. In such a vulnerable life situation, the role of individual resources, such as processing speed or well-being, for functional health might be different than in previous life phases (Mueller, Wagner, & Gerstorf, 2017): Given the overall heightened vulnerability, these resources get more important to buffer decline in functional health. However, the opposite scenario could be that their importance decreases because it is primarily accumulating morbidity and mortality-related processes that drive functional health changes, and

these processes might only to a small extent be counteracted by investment in individual resources. As Mueller et al. (2017) state, it is thus possible that “the growing importance of physiological constraints overshadows the health benefits conveyed by psychosocial resources” (p. 71), so that in consequence processing speed and well-being might be less strongly associated with functional health with advancing age and impending death.

In the following, we will provide an overview regarding empirical evidence on associations between information processing speed, well-being and functional health and on the potentially moderating role of age vs. time to death.

Information Processing Speed and Functional Health

The successful execution of everyday tasks requires intact cognitive functions. Indeed, cognitive abilities, and particularly information processing speed, which is a key marker of cognitive functioning (Finkel, Reynolds, McArdle, & Pedersen, 2007; Lindenberger, Mayr, & Kliegl, 1993; Salthouse, 1996), are associated with physical functioning (e.g., Atkinson et al., 2009; Desjardins-Crépeau et al., 2014) and with the disablement process (Braungart Fauth et al., 2007; Caplan & Schooler, 2003; Pérès et al., 2005). For instance, changes in information processing speed over 12 years in older adults have been found to be associated with changes in functional ability (Wahl et al., 2010). Similarly, Finkel, Ernsth-Bravell, and Pedersen (2016) observed meaningful dynamic couplings between processing speed and motor functioning over an observation period of 19 years in middle-aged and older adults. They found that decline in motor functions precedes decline in processing speed, whereas the opposite temporal dynamics have been reported with regard to terminal (rather than age-related) trajectories (Wilson et al., 2012) which shows that the underlying causality of associations between processing speed and functional health might be complex and potentially reciprocal.

These associations between processing speed and functional health might be additionally moderated by age and closeness to death. As long as functional health is intact, its association with information processing speed might be rather weak. In contrast, in later life, when health gets more

vulnerable, speed might become an increasingly important compensatory resource to buffer health decline. Associations between cognitive, sensory and sensorimotor functioning have indeed been found to be closer at older ages (P. B. Baltes & Lindenberger, 1997; K. Z. H. Li & Lindenberger, 2002). Different explanations have been offered for this phenomenon, including a potential “common cause” underlying age-related decline in these domains of functioning (Lindenberger & Baltes, 1994; Wahl & Heyl, 2003).

Alternatively, it may be the same mechanisms driving mortality-associated, rather than age-related changes in processing speed and functional health which lead to stronger associations between both domains. In this case, time to death could moderate these associations instead of – or in addition to – chronological age. Generally, there are indeed substantial relationships between functional health and cognitive abilities particularly at the end of life (Gerstorf et al., 2013). For instance, not only age-related changes (Zammit et al., 2019; Zammit et al., 2018), but also time-to-death-related changes in cognitive abilities and in grip strength were found to be interrelated (Praetorius Björk, Johansson, & Hassing, 2016). There is also a high correlation between the onset of terminal decline in cognitive abilities, including speed, and the onset of terminal decline in motor functions (Wilson et al., 2012) which are closely related to functional health.

To summarize, based on this empirical evidence, it can be expected that processing speed is more closely linked to functional health both with advancing age and with increasing proximity to death. As it may actually be closeness to death that accounts, at least to some extent, for the “age moderation effect”, we assume a stronger effect for the time-to-death moderation than for the age moderation.

Subjective Well-Being and Functional Health

With regard to well-being, prior studies have already shown that health and well-being are interrelated in later life (Kunzmann, Little, & Smith, 2000; Schöllgen et al., 2016), and particularly at the end of life (Diegelmann, Schilling, & Wahl, 2016; Gerlach et al., 2017; Gerstorf et al., 2013;

Schilling et al., 2018), including close associations of within-person, terminal changes in both domains (Burns et al., 2013; Burns, Mitchell, Shaw, & Anstey, 2014).

In this study, we will take the multidimensionality of well-being (Diener, Suh, Lucas, & Smith, 1999; Ryff et al., 2006) as well as its multidirectionality with regard to age-related and end-of-life change trends (Wettstein, Schilling, Reidick, & Wahl, 2015) into account by focusing on two major well-being indicators, namely positive affect and depressive symptoms. Whereas positive affect, which describes how frequently an individual has experienced a range of positive emotions (e.g., excited, inspired) over a certain period of time, represents a prototypical indicator of affective and hedonic well-being (Diener et al., 1999; Ryan & Deci, 2001), depressive symptoms represent a component describing negative well-being, or “ill-being” (Ryff et al., 2006).

Positive affect has been found to be associated with better health outcomes in general (Lyubomirsky, King, & Diener, 2005; Pressman & Cohen, 2005). Different causal pathways and mechanisms, such as a buffering effect of positive affect on the detrimental effect of stress on health (Pressman & Cohen, 2005) or motivational factors (such as better health behaviors in individuals with higher positive affect levels; Ong, 2010), may explain this association. However, there is also evidence for the opposite pathway, leading from functional health to subsequent positive affect (Wahl, Drapaniotis, & Heyl, 2014), pointing at a complex, potentially reciprocal association between both domains.

Similarly, depressive symptoms are an established risk factor for functional ability decline (Geerlings, Beekman, Deeg, Twisk, & Van Tilburg, 2001; Stuck et al., 1999) and predictors of the disablement process (Pérès et al., 2005). However, depressive symptoms seem to be driven by disablement as well, as they increase with approaching disability as well as at the onset of disability (Fauth, Gerstorff, Ram, & Malmberg, 2011; Fauth et al., 2014).

Both positive affect and depressive symptoms have been found to reveal age-related (Charles et al., 2001; Sutin et al., 2013) and terminal changes (Burns et al., 2013; Diehr et al., 2002; Fauth et al., 2014; Hülür et al., 2015; Schilling et al., 2018; Vogel et al., 2013), which may in turn

be linked with trajectories in functional health because of identical age- and mortality-associated mechanisms that drive these changes.

Regarding existing empirical evidence on the role of age vs. time to death for well-being-health associations, Schöllgen et al. (2016) report a weaker time-varying association between depressive affect and functional limitations with advancing age. However, they did not control for time to death, so that this moderating effect of age might be either due to primary aging or to tertiary aging (or even a combination of both). Moreover, they considered depressive affect as outcome, **rather than health**, and interpreted the effect as an age-related decrease in “health sensitivity”, that is older adults’ depressive affect is less influenced by worsening health.

It thus seems that the role of age and time to death with regard to associations between well-being and functional health has only rarely been empirically addressed so far. In analogy to processing speed, assuming a compensatory role of well-being to buffer functional health decline with increasing levels of general vulnerability, we predict that the association between well-being and functional health will get stronger with advancing age as well as with increasing proximity to death. Again, as time to death may be the better marker of vulnerability, we expect the time-to-death moderation effect on associations between well-being and functional health to be stronger than the age moderation effect.

Research Aims and Expectations

To summarize, the aims of this study are as follows:

- (1) To investigate the role **of** primary aging vs. tertiary aging from a “time as a resource/burden” perspective for changes in functional health. Specifically, we investigate whether functional health trajectories are more closely associated with chronological (baseline) age as primary aging indicator vs. with time to death (at baseline) as a marker of tertiary aging.
- (2) To investigate the extent to which the associations of information processing speed and subjective well-being with functional health are moderated by age vs. time to death.

As already outlined above, we assume that time to death may have a stronger impact on functional health change than chronological age. In addition, we assume that in individuals who are older and who are closer to death, associations of processing speed and well-being with functional health are closer than in younger individuals and in subjects who are less close to death. Individual resources **such as** information processing speed may play an increasingly important compensatory role for the maintenance of functional health once general vulnerability and proneness to health deterioration increases, either due to “primary aging” processes, including normative biological age-related changes, or to “tertiary aging” processes that comprise mortality-related accumulation of morbidity. We also expect to find a stronger “time to death moderation effect” compared to the “age moderation effect”: Overall vulnerability may increase more with impending death than with advancing age, thus requiring more investment **in** resources of cognition and well-being to reduce health deterioration.

Method

Study Population and Sample Description

Data from the German Ageing Survey (“Deutscher Alterssurvey”, DEAS) were used. The German Ageing survey is a cohort-sequential study, with a study sample of German community-dwelling adults aged 40 years and older at baseline (Klaus et al., 2017). Six measurement waves (T1: 1996, T2: 2002, T3: 2008, T4: 2011, T5: 2014; T6: 2017) have been completed so far; however, the mortality data available by now include dates of death ranging from 2002 to 2016, so the sixth measurement occasion (2017) was not included in the following analyses. A new sample has been drawn at each measurement wave, based on national probability sampling, and all samples were systematically stratified by age, gender, and region (former West or East Germany). Moreover, if they agreed, study participants were re-interviewed at the subsequent measurement occasions. In 2011, no new baseline sample was drawn and only “panel participants” who had already taken part at least once in the survey were re-assessed.

As the outcome measure of this study (functional health) was not assessed before 2002, we used data from 2002 and from subsequent measurement waves (2008, 2011, 2014). The sample consisted of all study participants who died between 2002 and 2016 ($n = 578$; mean age at death: 76.59 years, $SD = 9.90$ years, range 45-95 years) and who had at least one valid information regarding their functional health as well as regarding the included predictor variables. Among those participants who provided at least two observations (individuals with one observation: 382 [66.1%]; 2 observations: 140 [24.2%]; 3 observations: 47 [8.1%]; 4 observations: 9 [1.6%]), the mean length of follow up was $M = 6.28$ years ($SD = 2.75$, range 3-12 years). A sample description is shown in Table 1. The correlations between study variables are shown in Table 2.

(Insert Table 1 about here)

(Insert Table 2 about here)

In order to quantify potential sample selectivity, we compared the baseline scores of the study variables in our study sample with the respective scores of the “parent sample” ($n = 20,038$) that includes all study participants (for a similar approach, see Singer, Verhaeghen, Ghisletta, Lindenberger, & Baltes, 2003). With regard to socio-demographic measures, the proportion of women in the study sample of deceased participants (31.7%) was below the proportion in the parent sample (49.2%). Women might thus be underrepresented in this sample because they live longer than men (Klenk, Rapp, Büchele, Keil, & Weiland, 2007), so that more women than men of the parent sample were still alive in 2016. Moreover, and not surprisingly, baseline age in the sample of deceased participants was about 10 years higher than in the parent sample ($M_{\text{deceased}} = 71.34$ years, $M_{\text{parent}} = 61.99$ years). This difference corresponds to about $\frac{3}{4}$ of a standard deviation in the parent sample, thus indicating – according to Cohen (1992)– a large effect ($d = .78$). In contrast, the distribution of education levels was similar in the samples (low education: 12.1% in study sample, 12.2% in parent sample; medium education: 56.2% in study sample, 54.8% in parent sample; elevated education: 13.1% in study sample, 12.3% in parent sample; high education: 18.5% in study sample, 20.6% in parent sample). Differences in baseline positive affect ($d = .21$) and baseline

depressive symptoms ($d = .12$) were small, whereas differences in baseline functional health ($d = .48$), number of chronic diseases ($d = .43$), and information processing speed ($d = .54$) were of medium effect size: Individuals of the study sample had lower functional health, more chronic diseases and lower processing speed scores at baseline compared to the parent sample.

Measures

Outcome: Functional Health. Functional health was assessed based on the physical functioning subscale of the SF-36 instrument (Bullinger & Kirchberger, 1998). Participants had to indicate **their** degree of limitation (“limited a lot”, “limited a little”, or “not limited at all”) with regard to 10 typical everyday activities (e.g., walking several blocks, bathing and dressing). Answers were summed and transformed according to the manual so that a sum score ranging from 0 to 100 resulted, with higher values indicating better functional health.

Predictors

Time-as-a-Resource/Burden: Age and time to death. We included age and time to death as between-person predictors of functional health change and as potential moderators of the associations between information processing speed/well-being and functional health. Age and time to death (i.e. the difference between death date and interview date) from baseline (first measurement participation) were determined, with “years” as time unit.

Information Processing Speed. Speed was measured based on the established and widely used Digit Symbol Substitution Test from the Wechsler Adult Intelligence Scale (WAIS-R; Tewes, 1991). Higher test scores indicate higher information processing speed.

Well-Being. As well-being is a multidimensional construct with distinguishable components (Diener et al., 1999; Ryff et al., 2006), two well-being indicators that may be particularly relevant with regard to functional health and the disablement process (Lyubomirsky et al., 2005; Pèrès et al., 2005; Pressman & Cohen, 2005 ; Spalter, Brodsky, & Shnoor, 2013) were assessed. *Positive affect* was measured by the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988). The positive affect subscale of the PANAS consists of 10 positive emotion adjectives (e.g.,

excited, inspired). Respondents were asked how often they experienced each of the 10 emotions during the past months. They provided answers on a response format ranging from 1 (never) to 5 (very often). A mean score was computed for each individual, with higher mean scores indicating more positive affect.

Assessment of *depressive symptoms* was based on a 15-item German adaptation (Hautzinger & Bailer, 1993) of the Center for Epidemiological Studies Depression Scale (CES-D scale; Radloff, 1977). Study participants had to indicate how often they experienced each of 15 depressive symptoms during the past week (e.g., “I felt sad”, “My sleep was restless”), and they provided answers on a 4-point response scale ranging from “rarely or none of the time (less than 1 day)” to “most or all of the time (5 to 7 days)”. A sum score ranging from 0 to 45 was computed, with higher scores indicating more frequent depressive symptoms.

Control Variables. In addition, we included study participants’ gender, education, and the number of chronic diseases they had as further predictors. The assessment of education was based on the International Standard Classification of Education (ISCED) coding (UNESCO, 2012). For this coding, school and professional education were considered, resulting in a differentiation of four educational groups, namely individuals with low, medium, elevated, and high education. **Number of** chronic diseases was assessed as a proxy measure of secondary aging based on a list of 11 chronic conditions (e.g., diabetes, cancer, cardiovascular diseases). Participants indicated which of the listed illnesses they had, and a sum score of chronic diseases per participant was computed.

Statistical Analyses

We used Stata 15.0 (StataCorp., 2017) to compute longitudinal multilevel models (Hox & Kreft, 1994). We specified time in study as time metrics and as “time-as-process” variable (Ram et al., 2010), which we centered at the middle of each individual’s observation period. This is a common approach (e.g., Lindenberger & Ghisletta, 2009; Wagner, Ram, Smith, & Gerstorf, 2015) which facilitates the interpretation of estimates and which warrants a more robust estimation in the midpoint of the observation periods. To reduce model complexity, given the somewhat limited

proportion of individuals who had provided 3 or more observations prior to death, all predictors were specified as time-invariant predictors measured at each individual's first measurement occasion. Additionally, with the exception of the categorical predictors (gender and education), all predictors were grand-mean-centered to ease the interpretation of the estimates. **To facilitate interpretation, time to death was recoded, ranging from -14.5 years (i.e., 14.5 years prior to death at baseline) to -0.08 years (i.e., 0.08 year prior to death at baseline), so that higher scores indicate less distance to death.**

We also checked for non-linear trajectories, following the guidelines suggested by Kass and Raftery (1995). Specifically, we tested whether the curvilinear model (including a quadratic time component in addition to a linear one) resulted in a substantially better model fit than the respective linear model as indicated by a lower Bayesian Information Criterion value (BIC; $2[\Delta\text{BIC}] \geq 6$).

Results

Trajectory of Functional Health

When comparing the quadratic with a linear change model without considering additional predictors, the BIC difference (BIC quadratic model: 9,421.49; BIC linear model 9,384.97) was in favor of the linear change model. In the quadratic change model, the intercept-slope covariance could not be estimated, so that the linear and quadratic change model were not nested and thus not comparable based on Likelihood Ratio Test. Also, the fixed quadratic slope component did not reach statistical significance. Consequently, we decided on the linear change model, also for the sake of model parsimony and due to the limited proportion of individuals who provided 3 and more observations which might not be sufficient for reliable estimates of nonlinear changes.

The fixed linear change component was significant in the model, indicating an annual linear decline in functional health of about 3 points over time in study ($\beta = -2.99$, $p < .001$; see Figure 1). The random slope component was also significant ($p < .001$), implying significant interindividual variation around the mean-level trajectory of functional health.

(Insert Figure 1 about here)

Change in Functional Health: Effects of Age and Time to Death

In the extended model with additional predictors (see Table 3), the fixed linear slope component was significant, revealing an average annual decline in functional health of about 3.71 points. Age at baseline was significantly associated with lower functional health levels, whereas time to death was not. However, both age and time to death significantly predicted change in functional health, with steeper mean-level decline in functional health in individuals who were older and closer to death at baseline (see Figure 2). Notably, comparing both effects in size, the effect of time to death exceeded the effect of age by about factor 4, **though the confidence intervals of both point estimates overlapped**. Each year closer to death at baseline augmented the annual decline in functional health by -.28 points, whereas each additional year of chronological age at baseline increased the annual decline only by about -.07 points.

(Insert Figure 2 about here)

(Insert Table 3 about here)

Associations of Functional Health with Information Processing Speed and Well-Being: Moderated by Time to Death, but not by Age

Lower positive affect as well as more depressive symptoms at baseline were related with lower functional health levels, but they did not significantly predict change in functional health. Baseline information processing speed was neither significantly related with functional health levels nor with functional health changes.

However, these effects need further specification as there were significant interaction effects of processing speed and depressive symptoms with time to death: In individuals who were closer to death at baseline, both the positive association of processing speed with functional health levels and the negative association of depressive symptoms with functional health levels were stronger than in individuals who were less close to death (see Figure 3). In contrast, baseline age did not significantly interact with processing speed or any well-being indicator. Effects were thus limited to “time to death moderation”, whereas there was no empirical evidence for “age moderation”.

(Insert Figure 3 about here)

Regarding additional predictor effects, individuals with more chronic diseases at baseline had lower functional health levels. Women had lower functional health levels than men, but change in functional health was not significantly affected by gender.

Discussion

In this study, we built on the distinction between primary aging and tertiary aging (Birren & Cunningham, 1985; Ram et al., 2010) and on the conception of “time as a resource/burden” (Heirich, 1964; Ram et al., 2010; Wagner et al., 2015) by comparing the role of age versus time to death for functional health changes in a sample of middle-aged and older adults. Furthermore, by adopting and extending the core assumptions of the disablement process model (Verbrugge & Jette, 1994) within an end-of-life perspective, we also investigated the role of both time metrics as moderators of the associations between cognitive abilities (information processing speed), well-being (positive affect and depressive symptoms), and functional health. In the following, the major findings of this study are summarized and discussed.

Age and Time to Death Affect Functional Health Changes

Not surprisingly and in line with previous empirical results (Berlau et al., 2009; Jacobs et al., 2012; Wahl et al., 2010; Wettstein et al., 2016), we found that functional health decreased over time. Additionally, regarding the role of primary, normative aging vs. tertiary aging that describes mortality-related changes, older baseline age was associated with lower functional health levels, whereas time to death at baseline was not significantly related with functional health levels. In contrast, both age and time to death were significantly associated with functional health changes. As expected, decline in functional health was steeper in individuals who were older and closer to death. The additional decrease in functional health per year closer to death was larger than the additional decrease per additional year of chronological age. This stronger effect of tertiary aging, as measured by distance to death, than of chronological age is in line with various previous terminal decline findings on health (Diehr et al., 2002; Gerstorf et al., 2013; Hülür et al., 2016; Klijs et al., 2010;

Wolf et al., 2015) and other domains such as cognition (Bäckman & MacDonald, 2006; Riegel & Riegel, 1972) or subjective well-being (Gerstorf et al., 2018; Gerstorf et al., 2010). For instance, Thorvaldsson et al. (2006) concluded based on their findings that “a greater part of the sources that drive cognitive changes in old age are death-related rather than age-related” (p. 202), and the same seems to be true with regard to functional health changes.

In conclusion, both age and time to death indeed operate in parallel as between-person “time as a resource/burden” indicators (Ram et al., 2010), with older age **and less distance to death** predicting **an** increasing decline in functional health. That both time metrics can operate simultaneously as driving mechanisms of change (Thorvaldsson et al., 2006; Wolf et al., 2015) may be seen as empirical support of the conceptual distinction between primary and tertiary aging. Given that age per se cannot be interpreted as a causal mechanism that explains change, but rather acts “as a shorthand for the set of variables acting over time” (Wohlwill, 1970, p. 50), considering only age as a predictor of change might thus result in a mixture of operating primary, secondary and tertiary aging processes. Time to death might be a better “overall vitality index” (Thorvaldsson et al., 2006, p. 202) than chronological age, describing the extent of mortality-related change that has so far occurred in an individual at a certain point in time. However, chronological age is also of importance and cannot be fully replaced by time to death as it “indicates the amount of normative age-related resources or burdens an individual has accumulated thus far” (Ram et al., 2010, p. 31). Notably, age-related change in functional health does not seem to be entirely due to mortality-related processes as both age and time to death independently predicted functional health change in our study sample. This supports the assumption that “late-life development is driven by multiple time-related processes” (Ram et al., 2010, p. 28) and suggests a co-occurrence of primary and tertiary aging dynamics that drive changes in functional health.

Association of Processing Speed and Well-Being with Functional Health: Moderated by Time to Death, but not by Age

Our study aim was to investigate the disablement process model from an end-of-life perspective. Other studies which investigated predictors of functional health based on the disablement process model have identified cognitive abilities, particularly processing speed, as well as well-being, as important determinants of functional health (Braungart Fauth et al., 2007; Caplan & Schooler, 2003; Femia et al., 2001; Pérès et al., 2005). In analogy to these findings, we observed higher functional health levels in individuals with higher positive affect and fewer depressive symptoms. However, in our sample, neither information processing speed nor well-being reached statistical significance as predictors of change in functional health. In this specific sample, with all individuals being more or less close to death (all < 15 years prior to death at their first study participation), higher well-being might thus contribute to higher functional health levels, and this functional health difference in favor of individuals with higher well-being remains over time. This pattern corresponds to “preserved differentiation” according to Salthouse (2006), who originally introduced this term with regard to cognitive aging. But higher well-being does not additionally buffer functional health decline and thus does not lead to “differential preservation” of functional health over time. The compensatory effects of individual resources such as well-being to buffer the detrimental effect of increasing age- and mortality-related vulnerability on functional health change may thus come to their limits when a certain proximity to death is reached, as may have been the case for the majority of individuals within our study sample. With this proximity to death, general vulnerability (P. B. Baltes & Smith, 2003) and constraints in adaptive self-regulatory capacities increase (Gerstorf & Ram, 2009), resulting in a maybe unavoidable decline in functional health and other domains that cannot be easily be counteracted by investment in individual resources.

Alternatively, the reason why associations of well-being and cognitive abilities with functional health changes were not significant could be that *changes in these domains, rather than levels*, are meaningfully associated with functional health trajectories (Burns et al., 2014; Fauth et al., 2011, 2014; Wahl et al., 2014). However, investigating these time-varying associations would have required to specify within-person, time-varying predictors. To obtain reliable estimates for

such effects, a larger proportion of individuals with three and more repeated observations than was available in our study sample would have been needed.

As pointed out before, caution is required with regard to causality of the associations reported, which might be reciprocal as functional health might as well act as a predictor of well-being (Burns et al., 2014; Fauth et al., 2011, 2014; Wahl et al., 2014). This is particularly true for those individuals who provided one observation only so that their functional health intercepts and their baseline well-being levels refer to the same time point.

Finally, we investigated potential “age moderation” and “time to death moderation” effects with regard to the associations of processing speed and well-being with functional health. Assuming that, in this “time as a resource/burden” framework, older age represents a higher amount of accumulated primary aging change and less distance to death represents a higher amount of accumulated mortality-related, tertiary aging change, both indicators indicate a higher general vulnerability and a higher proneness to functional health deterioration. This increased vulnerability might require more individual resources – such as processing speed or well-being – to counteract or buffer such deterioration. Therefore, we assumed stronger associations of processing speed and well-being with functional health at older ages and with increasing nearness to death. Associations might also get closer both with advancing age and with approaching death because age-related and mortality-related changes in cognitive abilities, well-being, and functional health are to some extent driven by identical “common cause” mechanisms.

Whereas our age moderation assumption could not be confirmed, we found indeed stronger associations of processing speed and **depressive symptoms** with functional health levels in individuals who were closer to death. Investment in processing speed and well-being resources might thus be particularly crucial once a certain proximity to death is reached, whereas the role of these resources for functional health does not change with chronological age, possibly because proximity to death is indeed the better indicator of general vitality or vulnerability. However, again, caution is needed because the causal link might also lead from functional health to processing speed

and depressive symptoms (particularly in individuals with only one observation and without any time lag between the assessment of predictors and outcome).

For positive affect and its association with functional health, there was no evidence for either age moderation or time to death moderation. This association thus seems to remain stable over age and with increasing closeness to death. Positive affect seems to be less prone to terminal decline dynamics compared to other well-being indicators (Schilling, Wahl, & Wiegering, 2013), which might explain why its relation to functional health does not change with **decreasing distance to death**.

Associations of processing speed and well-being with functional health *changes* were not significantly moderated by time to death. Thus, independent of closeness to death, speed and depressive symptoms do not seem to affect change in functional health in our sample of decedents, suggesting again that there is no “differential preservation” (Salthouse, 2006) of functional health according to available cognitive and well-being resources.

Primary and tertiary aging are, of course, not the only aging dimensions conceptualized by Birren and Cunningham (1985). They additionally specified secondary aging as another dimension, which “encompasses changes that accrue with or are causally linked to disease and disability” (Ram et al., 2010, p. 28). Secondary aging was to some extent represented by our outcome variable, namely functional health, and its change over time in study. Additionally, we included the number of chronic diseases as baseline predictor which may be seen as a rough indicator of secondary aging. Not surprisingly, individuals with more chronic diseases had lower functional health levels. This finding corresponds to the core assumption of the disablement process model (Verbrugge & Jette, 1994) which postulates a main pathway from pathology (diseases) to disability. However, the number of chronic diseases did not predict changes in functional health. Again, it might be the accumulation of diseases over time as represented by a time-varying, within-person predictor, rather than the mere number of diseases at one point in time that shapes trajectories of functional health.

Limitations

This study has several strengths and limitations. Among the strengths of this study are the availability of an extended longitudinal observational period of up to 12 years, and the availability of different well-being indicators as well as of an established indicator of cognitive abilities, namely information processing speed. Moreover, the study sample comprised a broad and heterogeneous time to death range as well as an age range from middle adulthood to old age which enabled us to include time to death and chronological age simultaneously as “time as a resource/burden” predictors in our analyses.

One limitation of this study is that the majority of individuals provided only one to two observations, and many also dropped out of the study several years prior to death. This is maybe not surprising as closeness to death is typically related with study dropout (Rabbitt, Lunn, & Wong, 2008; Sliwinski, Hofer, Hall, Buschke, & Lipton, 2003). Moreover, the high proportion of single-observations has to some extent been caused by the specific study design of the German Ageing Survey. This survey provides large baseline samples that were first assessed either in 2002, 2008, or 2014, but not all of the individuals in these samples – particularly within our specific sample of deceased participants – entered the “panel sample” by participating at one or more of the follow-up measurement waves. This high proportion of individuals with only one single observation limited our possibilities of data analyses, e.g. with regard to specification of more complex, quadratic or cubic change patterns or of time-varying, within-person predictor effects. **Also, the statistical power for our analyses may have been limited, resulting in large standard errors of a few coefficient estimates and thus requiring cautious interpretation and comparison of these exact point estimates.** Data sets based on more measurement occasions per individual, and ideally also encompassing a more expanded observation interval, are therefore desirable for future research.

With regard to sample selectivity, our sample of decedents was older at baseline compared to the total sample it was derived from, and this subsample scored also lower on information processing speed at baseline and reported more chronic diseases as well as lower functional health at baseline than the parent sample. However, these differences in favor of the parent sample are not

surprising, as samples of survivors and of decedents can be expected to be different with regard to **various characteristics** of psychosocial resources and functioning (such as cognitive abilities; Small & Bäckman, 1999). Moreover, only the difference in baseline age was of large effect size. The proportion of women was smaller in the sample of decedents than in the parent sample, probably due to the fact that women have a higher life expectancy than men (Klenk et al., 2007), so that more female than male study participants were still alive in 2016. With regard to age at death, mean age at death of men in our sample (76.4 years) was close to the mean life expectancy of German men (75.6 years; Klenk et al., 2007). For women, however, mean age at death in our sample (76.9 years) was below the average of German women (81.3 years; Klenk et al., 2007). Therefore, women with “premature death” might be overrepresented in our study sample, which may somewhat limit the generalizability of our findings.

Moreover, the functional health measure we used was a self-report questionnaire. Although it is an assessment tool with good psychometric properties (Bullinger & Kirchberger, 1998) and frequently used in empirical research on functional health, self-reported functional health may be biased, e.g. due to social desirability. In addition, individuals who report more depressive symptoms usually have less favorable self-perceptions in general and may therefore tend to underestimate their functional health, so that the association between depressive symptoms and functional health might have actually been to some extent overestimated in this study.

Our study included prototypical indicators of primary and tertiary aging, namely chronological age and time to death. However, secondary aging was only partly covered by our outcome measure of functional health, and partly by including number of chronic diseases as between-person, time-invariant predictor. There may be more elegant approaches to include secondary aging, e.g. by specifying time-to-disability or time-from-disability (Fauth et al., 2014; Ram et al., 2010), and there may be additional “markers of aging” (Featherman & Petersen, 1986) besides the ones included in this study that drive developmental changes in functional health and other domains.

Finally, only one measure of cognitive abilities was available in this study. The Digit Symbol Substitution Test is an important, widely used indicator of processing speed with good psychometric properties (Tewes, 1991), and processing speed indeed seems to be a crucial cognitive function regarding associations with functional health (Desjardins-Crépeau et al., 2014; Finkel et al., 2016; Wahl et al., 2010). Still, the availability of an aggregated processing speed score, consisting of more than one single test, and of cognitive tests representing additional cognitive functions, would have been desirable and could be a promising approach for future studies.

Conclusion

In this study, we investigated the role of primary aging (i.e., chronological age) and of tertiary aging (i.e., time to death), considered as between-person “time as a resource/burden” constructs, for change in functional health, including number of chronic diseases as a secondary aging predictor. We also analyzed the potentially moderating role of age vs. time to death for associations of processing speed and well-being – which are established predictors of “disablement” – with functional health. Functional health was found to be driven by both primary and tertiary aging, with a stronger effect of the “tertiary aging burden” on functional health decline than of the “primary aging burden” predictor. With regard to the disablement process model by Verbrugge and Jette (1994), which is the conceptual framework we built on in this study and which we applied for an end-of-life perspective, we found that only time to death moderated associations of processing speed and depressive symptoms with functional health, whereas chronological age did not. Our findings suggest that change in functional health is a product of co-occurring primary and tertiary aging, and that remaining resources in terms of processing speed and absence of depressive symptoms become particularly closely linked with functional health in individuals who are closer to death and whose tertiary aging processes have thus already further progressed.

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Table 1

Sample Description (Baseline)

Variables	<i>M ± SD</i>
Baseline Age, <i>M±SD</i>	71.34±9.67
Age at Death, <i>M±SD</i>	76.59±9.90
Female Sex, <i>n (%)</i>	183 (31.7%)
Education:	
Low <i>n (%)</i>	70 (12.1%)
Medium <i>n (%)</i>	325 (56.2%)
Elevated <i>n (%)</i>	76 (13.1%)
High <i>n (%)</i>	107 (18.5%)
Functional Health (baseline), <i>M±SD</i>	71.03±28.21
Number of Chronic Diseases (baseline) <i>M±SD</i>	3.24±2.11
Information Processing Speed (baseline), <i>M±SD</i>	34.83±13.52
Positive Affect (baseline), <i>M±SD</i>	3.39±0.57
Depressive Symptoms (baseline), <i>M±SD</i>	7.79±6.71
Mean Number of Individual Observations, <i>M±SD</i>	1.45±0.71
Time-to-Death (baseline), <i>M±SD</i>	5.78±3.60

M = mean; *SD* = standard deviation.

Table 2

Correlations Between Study Variables (T1)

Variables	Age	Gender ¹	Education	Functional Health	Number of Chronic Diseases	Depressive Symptoms	Positive Affect	Information Processing Speed
Age								
Gender ¹	-.01							
Education	-.05	-.23***						
Functional Health	-.25***	-.20***	.11**					
Number of Chronic Diseases	.26***	.09***	-.06	-.47***				
Depressive Symptoms	-.06	.10*	-.08	-.44***	.27***			
Positive Affect	-.08	.05	.13***	.27***	-.15***	-.33***		
Information Processing Speed	-.28***	-.02	.28**	.27***	-.26***	-.18***	.17***	
Time to Death ²	-.11***	.08	-.03	.20***	-.11***	-.08	.02	.12***

Note. * $p < .05$; ** $p < .01$; *** $p < .001$.

¹ 0 = male, 1 = female. ² higher scores indicate higher distance to death.

Table 3

Predictors of Change in Functional Health

	Estimates
Fixed Regression Coefficients:	
Intercept [<i>SE</i>]	70.47*** [2.00]
Number of Diseases [<i>SE</i>]	-4.14*** [0.48]
Positive Affect [<i>SE</i>]	5.08** [1.77]
Depressive Symptoms [<i>SE</i>]	-1.01*** [0.16]
Information Processing Speed [<i>SE</i>]	0.05 [0.08]
Time to Death ¹ [<i>SE</i>]	-0.30 [0.26]
Age [<i>SE</i>]	-0.43 [0.11]
Information Processing Speed*Age [<i>SE</i>]	-0.00 [0.01]
Depressive Symptoms*Age [<i>SE</i>]	-0.00 [0.01]
Positive Affect*Age [<i>SE</i>]	0.07 [0.18]
Information Processing Speed*Time to Death ¹ [<i>SE</i>]	0.04* [0.02]
Depressive Symptoms*Time to Death ¹ [<i>SE</i>]	-0.08* [0.04]
Positive Affect*Time to Death ¹ [<i>SE</i>]	-0.04 [0.48]
Education	0.34 [1.08]
Gender	-7.42*** [2.06]
Linear Slope [<i>SE</i>]	-3.71*** [0.75]
Education*Linear Slope [<i>SE</i>]	-0.14 [0.35]
Gender* Linear Slope [<i>SE</i>]	-0.56 [0.62]
Number of Diseases * Linear Slope [<i>SE</i>]	-0.23 [0.14]
Positive Affect * Linear Slope [<i>SE</i>]	-0.56 [0.97]

Depressive Symptoms * Linear Slope [SE]	0.05 [0.08]
Speed * Linear Slope [SE]	-0.02 [0.04]
Time to Death ¹ * Linear Slope [SE]	-0.28* [0.11]
Age * Linear Slope [SE]	-0.07* [0.03]
Information Processing Speed*Age* Slope [SE]	-0.00 [0.00]
Depressive Symptoms*Age* Slope [SE]	-0.00 [0.00]
Positive Affect*Age * Slope [SE]	-0.06 [0.04]
Processing Speed* Time to Death ¹ * Slope [SE]	-0.00 [0.01]
Depressive Symptoms* Time to Death ¹ * Slope [SE]	-0.00 [0.02]
Positive Affect* Time to Death ¹ * Slope [SE]	- 0.21 [0.23]

Random Variances:

Variance Intercept [SE]	280.79*** [36.01]
Variance Slope [SE]	2.21 [1.80]
Residual Variance [SE]	259.30*** [32.94]

Note. Time unit is time in study, centered at the middle of each individual's observation period.

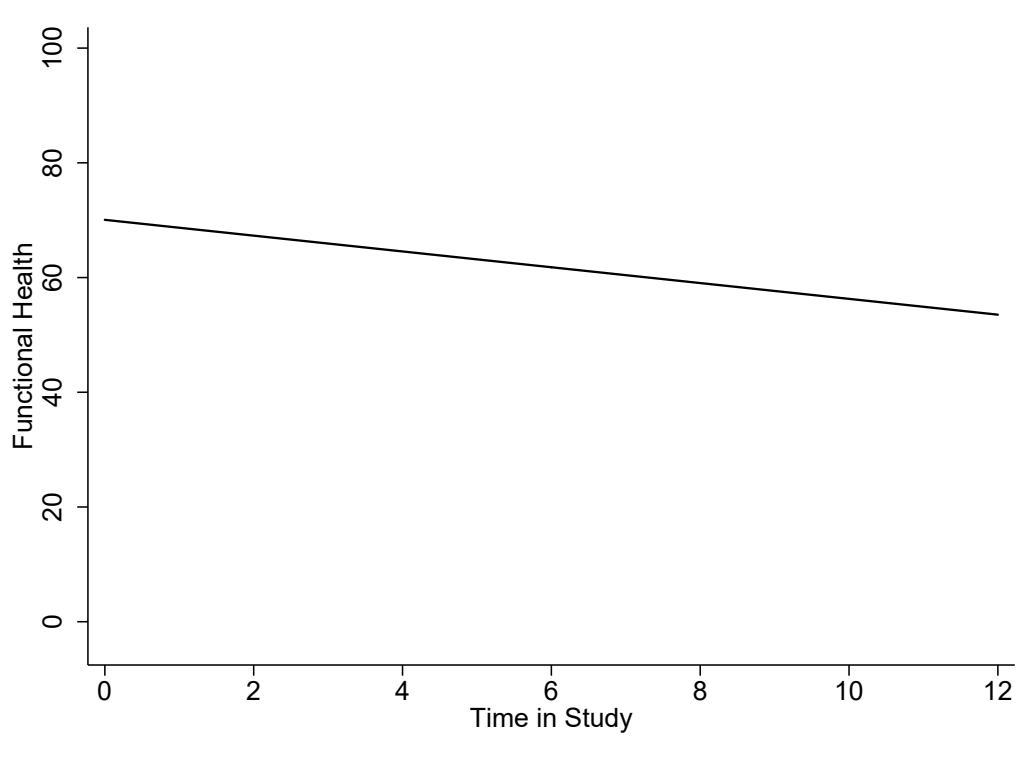
All predictors (except gender and education) were grand-mean-centered. Due to problems of estimability, the intercept-slope covariance was omitted.

* $p < .05$; ** $p < .01$; *** $p < .001$.

¹ higher scores indicate closer distance to death.

² 0 = male, 1 = female.

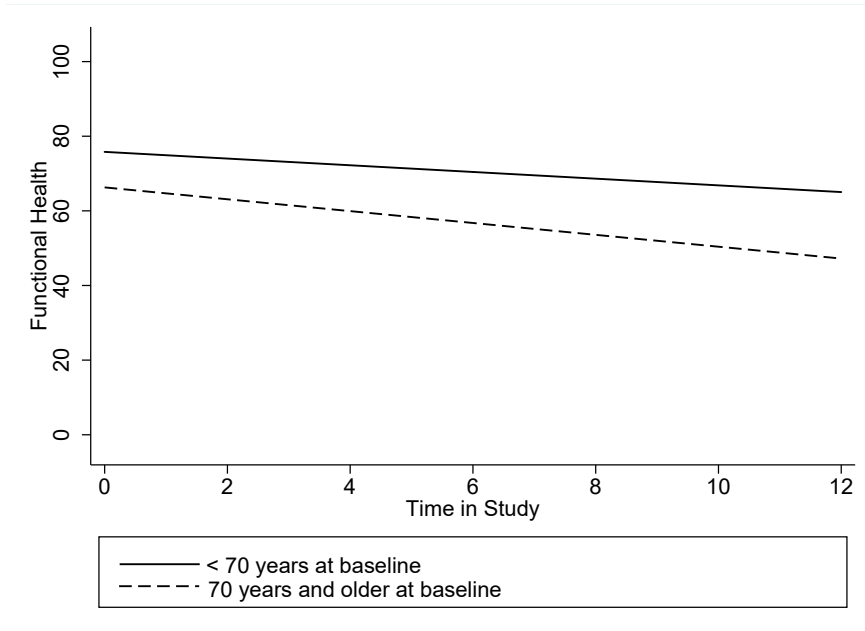
Figure 1
Mean-Level Trajectory of Functional Health



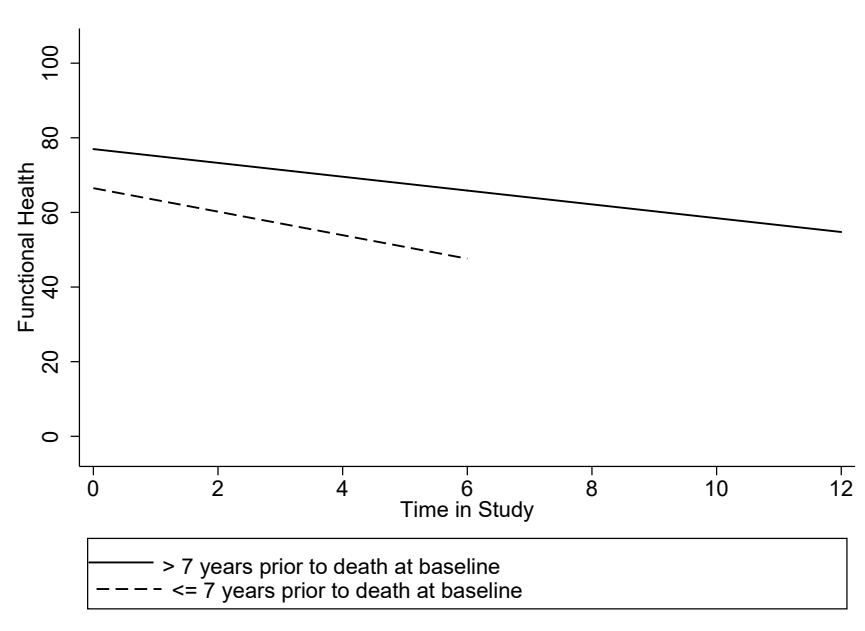
Note. Time unit is years.

Figure 2
Mean-Level Trajectory of Functional Health over Time in Study by Age and Time To Death

a) *Functional Health Trajectory by Age*



b) *Functional Health Trajectory by Time to Death*

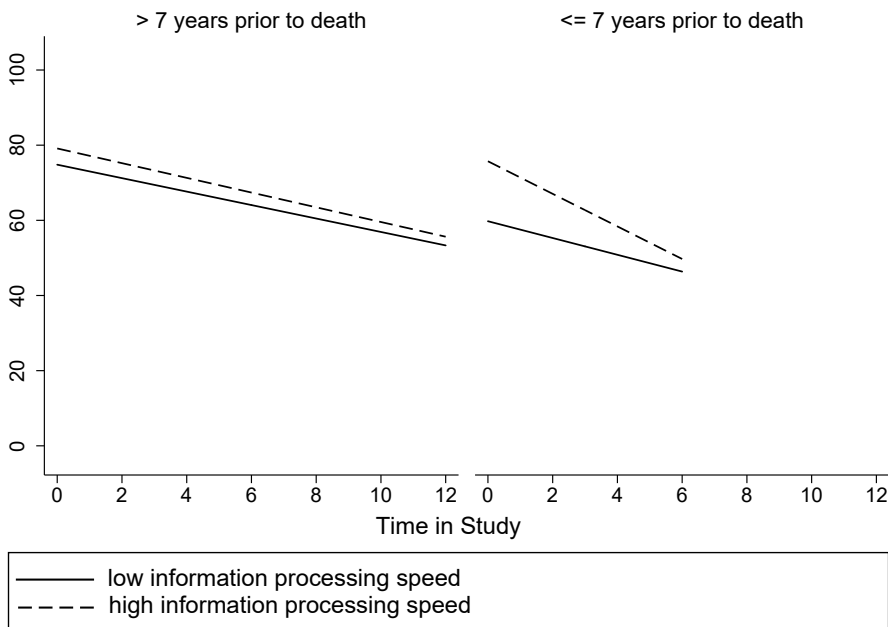


Note. Time unit is years.

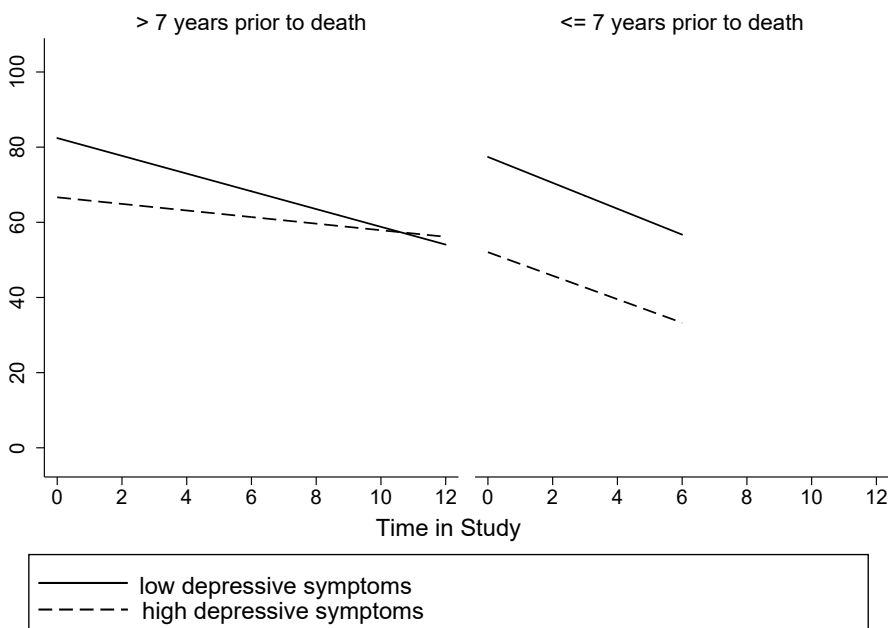
Figure 3

Associations of Information Processing Speed and Depressive Symptoms with Functional Health by Time to Death

a) *Information Processing Speed*



b) *Depressive Symptoms*



Note. Time unit is years. Groups with low vs. high information processing speed and with low vs. high depressive symptoms were built based on median split.