Social Determinants of Cognitive Functioning and Its Development in Old Age:

The Role of Education, Spousal Loss and Neighborhoods in a Longitudinal Perspective

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Chapter 1: Introduction

1.1 Background and aim of this dissertation

Populations are aging in many countries all over the world (United Nations, 2017). Worldwide, the number of persons aged 60 and older increased from about 400 million in 1980 to about 1 billion in 2017 and is projected to rise to 2 billion by 2050, corresponding to an increase in the population share of this age group from 9% to 21% between 1980 and 2050. In more developed regions of the world, one in four persons was aged 60 or older in 2017, and it will be one in three persons by 2050. Increases in old age life expectancy allow for more years spent in old age, as is evident in increasing shares of persons aged 80 and older among the older population. Although increased life expectancy can be considered a success in its own right (Oeppen & Vaupel, 2002), major concerns refer to the quality of life accompanying the quantitative gains in years. Very prominently, this idea is mirrored in the concept of Successful Aging which proposes that successful agers show a "low probability of disease and disease-related disability, high cognitive and physical functional capacity, and active engagement with life" (Rowe & Kahn, 1997, p. 433).

Against this background, this dissertation focusses on the cognitive functioning of older adults which is according to the concept of Successful Aging one of the main resources for active engagement with life besides physical capacities. Although medical, biological, and behavioral aspects play important roles in understanding cognitive functioning in old age, social factors are likely to determine successful cognitive aging given that they can be enduring or intense in nature and often shape experiences in many areas of life. Thus, I examine associations between social factors and cognitive functioning and shed light especially on the larger question whether social factors modify age-related cognitive decline. To this end, I combine two perspectives: On the one hand, a sociological approach that emphasizes the importance of social determinants in shaping individual behavior throughout the life course, and on the other hand psychological

theories that link individual behavior to cognitive functioning. Three social factors located at different levels are examined in this dissertation: At the individual level, I investigate the effect of education as a potential determinant that is attained early in life and shapes experiences throughout the life course. At the interpersonal level, I focus on the loss of the spouse as one of the most important social contacts. At the contextual level, socio-economic and spatial characteristics of the residential neighborhood – a major aspect of the living environment – are considered. Although previous research did not neglect these factors, especially questions regarding their role for the maintenance and decline of cognitive functioning in older adults remain unanswered: Are more (vs. less) educated persons more likely to maintain their cognitive functioning relative to a base level as they grow older? Will the loss of the spouse accelerate cognitive decline? Does the cognitive functioning of people in better off or urban neighborhoods differ from that of their counterparts in less well off or rural neighborhoods? Drawing on data from the Longitudinal Aging Study Amsterdam, this dissertation contributes to a better understanding of the role of social determinants by examining how some of them (i.e. education, neighborhood socioeconomic status and urbanity) are related to simple and more complex trajectories of cognitive development and by taking the dynamics of others (i.e. spousal loss) into account. Furthermore, the results of this dissertation also give insights into whether modifications of these determinants or interventions that alleviate their impact would be beneficial in promoting the cognitive functioning of older adults.

This first chapter gives an overview of the three studies presented in the subsequent chapters, which each address one of the potential social determinants of old age cognitive functioning. The chapter starts with an introduction to cognitive functioning, its development with age and its relevance. Subsequently, the core assumptions and theories are discussed, specifically the life course perspective and a life course model of cognitive functioning. Finally, a brief summary of each study is given and an overall conclusion is drawn.

1.2 Cognitive functioning and the relevance of its age-related decline

Due to their proneness to age-related decline, I especially focus on fluid cognitive abilities rather than on crystallized ones in this dissertation. This conceptual distinction is largely congruent with the distinction between cognitive mechanics and cognitive pragmatics, which develop differently over the lifespan (Baltes, 1987; Baltes, Staudinger, & Lindenberger, 1999; Craik & Bialystok, 2006; Harada, Natelson Love, & Triebel, 2013): Firstly, the fluid mechanics of cognition relate to "a person's innate ability to process and learn new information, solve problems, and attend to and manipulate one's environment" (Harada et al., 2013). They are related to the biological make-up of the brain and involve executive functioning, processing speed, memory, reasoning, and spatial orientation. Secondly, the crystallized pragmatics of cognition describe the context-dependent knowledge-based abilities that evolve through learning and experience, for example general knowledge, language skills, social intelligence or job-related expertise. Both aspects of cognitive functioning prototypically increase from childhood to early adulthood; after the first third of life, fluid abilities start declining while crystallized abilities remain rather stable or increase slightly throughout adulthood (Baltes, 1987; Baltes et al., 1999; Craik & Bialystok, 2006). Accordingly, it is the fluid abilities that decline more strongly in old age, while crystallized abilities decline less or remain stable (Lindenberger & Baltes, 1997; Singer, Verhaeghen, Ghisletta, Lindenberger, & Baltes, 2003).

According to Salthouse (2012, p. 217), "there may be very few situations at any age in which there is no advantage of high levels of novel problem-solving ability". Next to its instrumental value, cognitive functioning is a culturally valued resource in many contexts and worries about cognitive decline are common among middle-aged and older adults. It should however be emphasized that age-related decline in fluid abilities is qualitatively distinct from dementia as a summarizing term for cognitive impairment resulting from neurological disruption due to different medical conditions (Gavett & Stern, 2012). Because dementia also affects fluid

abilities – for example learning and memory, executive functioning, attention, and processing speed – it can be difficult to distinguish age-related cognitive decline from early stages of dementia (Alzheimer's Association, 2018; American Psychological Association, 2013; Hugo & Ganguli, 2014).[1] By definition however, cognitive decline that is so severe that it interferes with activities of daily living like managing money or household chores, taking medication, maintaining personal hygiene, or being able to eat independently is categorized as dementia (American Psychological Association, 2013; Hugo & Ganguli, 2014; Prince & Jackson, 2009). Notwithstanding, a significant share of persons experience cognitive decline that is not diagnosed as dementia, which becomes evident when comparing shares of cognitive decline to dementia prevalence rates: About 70% of older adults aged 70 years and older from community-based samples experience some form of decline in measures of cognitive status during periods of less than 10 years (Han, Gill, Jones, & Allore, 2016; Yaffe et al., 2009), compared to dementia prevalence rates in those over 60 years ranging from about 5% to 7% and increasing from about 3% to 8% between ages 70 to 79 to about 7% to 29% between ages 80 to 89 (Prince et al., 2013).[2]

While dementia strongly affects the individual, its ability to live independently, its social network, and the society via costs of care and lost productivity (Prince & Jackson, 2009), everyday functioning is largely maintained in normal age-related decline of fluid abilities due to multiple reasons (Salthouse, 2012): First, cognitive tests measure maximum performance capacity, while most people do not need to perform at their maximum in everyday situations. Second, with life experience, many everyday problems have become familiar and their solutions are integrated into the more stable crystallized abilities, such that being able to solve new

[1] Dementia is also referred to as major neurocognitive disorder in the Diagnostic and Statistical Manual of Mental Disorders (American Psychological Association, 2013). The first most frequent type of dementia is Alzheimer's disease with a share of about 50% to 75% of dementia cases, before vascular dementia, mixed forms, and dementia with Lewy Bodies (Knapp & Prince, 2007; Prince & Jackson, 2009)

[2] Note that some of the persons without dementia might fall into the category of Mild Cognitive Impairment. The concept describes persons with cognitive impairment that is more severe than that to be expected from age-related decline but less severe than that from dementia (Petersen, 2004) and affects about 15% to 20% of those aged 65 or older (Alzheimer's Association, 2018).

problems is less important than at younger ages. Third, it might be the case that age-related decline affects functioning in everyday situations, but that older adults avoid situations that would reveal these deficits by not exposing themselves to such situations or delegating tasks to more able persons.

Consequently, it is reasonable to assume that age-related decline in fluid abilities is highly relevant in situations in which the individual is required to perform at a high level, has to find solutions to new problems, or when it finds itself in challenging situations that it cannot – or does not want to – avoid (Institute of Medicine, 2015; Salthouse, 2012). Although more research is needed to identify the impact of age-related decline in fluid cognitive functioning on everyday activities, it is likely that the following situations are among them: Making complex financial and medical decisions, mastering digital technologies with their increasing relevance in everyday life, learning new tasks in professional contexts, reacting timely and smart to unexpected situations in traffic, or maintaining a large life space by being confident and able to move in or travel to unfamiliar places. Besides these more objective aspects, experiencing decline of one's own cognitive abilities might pose challenges to identity and well-being (Buckley, Saling, Frommann, Wolfsgruber, & Wagner, 2015; Wilson et al., 2013).

1.3 Core assumptions and theories

Research aiming to identify determinants of cognitive functioning and its maintenance or decline at higher ages largely builds on the assumption that cognitive functioning is modifiable within limits, as is widely acknowledged when it comes to physical functioning. The modifiability of cognitive functioning is not only observable in the improvement of cognitive abilities as a result of training efforts (Baltes et al., 1999) but also in improved cognitive functioning over historical time – the so-called Flynn Effect – both in the general population (Pietschnig & Voracek, 2015) and in older adults (Brailean et al., 2018; Hessel, Kinge, Skirbekk, & Staudinger, 2018; Karlsson, Thorvaldsson, Skoog, Gudmundsson, & Johansson,

2015). Besides the longer exposure to education of later born cohorts (Brailean et al., 2018; Karlsson et al., 2015), changes in health-related and work-related conditions are also considered important explanatory factors of the Flynn Effect (Baltes et al., 1999). Acknowledging the relevance of medical, biological and behavioral factors, the sociological perspective taken in this dissertation stresses the role of experiences made throughout the life course for cognitive functioning.

1.3.1 The life course perspective

The life course perspective acknowledges the individual ability to make choices and engage in actions, but it emphasizes that the surrounding social circumstances represent opportunities and constraints that make certain behaviors and experiences more or less likely than others and thereby affect the chances of achieving certain outcomes at later stages (Elder, 1994; Elder, Johnson, & Crosnoe, 2003; Hendricks, 2012). In other words, this means that "commonalities among and differences between people are rooted in life experiences and that those experiences are grounded in social circumstances" (Hendricks, 2012, p. 227). The social circumstances experienced by individuals are specific to historical time and place, and they are linked to other people in their social network (Elder, 1994; Elder et al., 2003; Hendricks, 2012). As one of the foundations of sociological reasoning, this idea has been expressed in much classical work like Max Weber's distinction of life chances *(Lebenschancen)* and life conduct *(Lebensführung)* (Abel & Cockerham, 1993), Émile Durkheim's concept of social facts as "any way of acting, whether fixed or not, capable of exerting over the individual an external constraint" (1982, p. 59), or Anthony Giddens' (1984) duality of agency and structure (also see Cockerham, 2005 and Hagestad & Dannefer, 2001). Empirically, the importance of these so-called opportunity structures for health-related outcomes manifests impressively in the negative association between socioeconomic status and old age mortality that has consistently been demonstrated across a wide range of countries (Eikemo, Huisman, Bambra, & Kunst, 2008; Huisman et al.,

2004; Huisman, Read, Towriss, Deeg, & Grundy, 2013). In the next paragraph, I present theories about the experiences and behaviors that are relevant when it comes to cognitive functioning in old age.

1.3.2 Linking the life course perspective to cognitive functioning

The Revised Model of the Scaffolding Theory of Aging and Cognition (STAC-r; Reuter-Lorenz & Park, 2014) is conceptualized as a life course model of cognitive functioning (see *Figure 1.1*). Important foundations of the model are that the level of and change in cognitive functioning are closely related to brain structure and brain function (arrow 1), with the latter two being subject to neural changes and maladaptive brain activity that are in turn related to biological aging processes (arrow 2). Besides age-related changes to the brain, life course experiences play an important role for cognitive functioning in two ways. Firstly, life course experiences can exert both beneficial and compromising influences *directly on the brain* (arrow 3). On the beneficial side, engagement in intellectual and social activities, education, and cardiovascular and physical fitness are examples of experiences and behaviors that enhance the brain's structure and function (so-called neural resource enrichment). On the compromising side, experiences like stress, depression, and behaviors increasing vascular risk like smoking, obesity and diabetes can exert negative influences on the brain (so-called neural depletion). Secondly, life course experiences can influence level and decline of cognitive functioning *by affecting (i.e. moderating) how strongly changes to the brain affect its output* via so-called compensatory

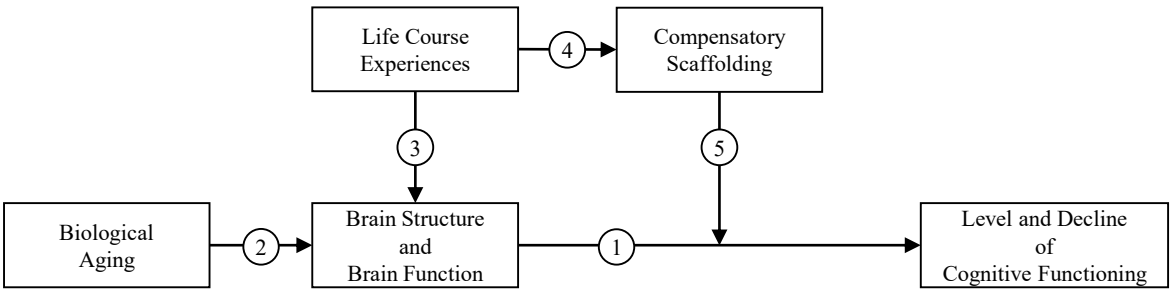

Figure 1.1: The Revised Model of the Scaffolding Theory of Aging and Cognition. Adapted from Reuter-Lorenz and Park (2014) and modified.

scaffolding (arrows 4 and 5).[3] Compensatory scaffolding entails for example the use of alternative brain regions and networks to counteract effects of changes in brain structure and function.

As the Revised Model of the Scaffolding Theory of Aging and Cognition is a rather comprehensive model, its implications regarding successful cognitive aging show commonalties with other theories of cognitive functioning. Especially the idea that activities involving cognitive functioning (e.g. education or intellectual activities/cognitive stimulation) are beneficial to maintain cognitive functioning at higher ages is part of concepts like the Environmental Complexity Hypothesis (Schooler, 1984) or the Use It or Lose It Hypothesis (Hultsch, Hertzog, Small, & Dixon, 1999), with the importance of challenges and novelty of experiences – for example at the workplace or in spare time activities – being emphasized more recently (Oltmanns et al., 2017; Park et al., 2014). Links exist also with the concept of cognitive reserve, which proposes that a reserve is built up by life course experiences including education or occupation and that a large reserve can reduce the impact of changes of the brain on cognitive functioning by allowing more efficient task processing (Stern, 2002). While cognitive reserve is concerned with modifying the relationship between brain and cognitive functioning (arrow 5), the concept of cognitive plasticity emphasizes that the functional supply provided by the brain can be improved within biological constraints given prolonged exposure to cognitive demands (arrow 3). These changes are dependent on cognitive demands that exceed the current functional supply provided by the brain (e.g. enduring challenging activities or environmental demands) and should eventually allow for better cognitive functioning (Hertzog, Kramer, Wilson, & Lindenberger, 2009; Lövdén, Bäckman, Lindenberger, Schaefer, & Schmiedek,

[3] In their graphical representation of the model, Reuter-Lorenz and Park (2014) depict compensatory scaffolding as a mediator variable between brain characteristics and cognitive functioning, presumably because more compensatory scaffolding is necessary to maintain cognitive functioning if the brain shows age-related changes. Since they further suggest that „scaffolding [...] moderates the effects of deleterious brain influences on cognitive performance" (p. 356), I considered the representation of compensatory scaffolding as a moderating variable for the association between brain characteristics and cognitive functioning more informative.

2010). On the side of compromising experiences, neuropsychological research suggests that stress is detrimental for the brain because it may result in dysregulation of the hypothalamic-pituitary-adrenal axis which might lead to impairment of cognitive functioning (Leng et al., 2013; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; McEwen & Sapolsky, 1995; Shields, Sazma, McCullough, & Yonelinas, 2017).

Empirically, disentangling the specific mechanisms at work is difficult with observational data. However, the importance of such life course experiences in modifying cognitive functioning is exemplified in research showing improved cognitive functioning – especially memory – in persons who participated in cognitive training interventions compared to their counterparts in the control groups (Kelly et al., 2014; Martin, Clare, Altgassen, Cameron, & Zehnder, 2011), cognitive improvements in persons who engaged in more general mental stimulation, for example through piano instruction, acting classes or computer courses (Kelly et al., 2014; Martin et al., 2011), and in research demonstrating the positive effects of physical activity – both aerobic exercise and strength training – on cognitive functioning (Northey, Cherbuin, Pumpa, Smee, & Rattray, 2018).

Theoretical links between the cognitively relevant individual-level experiences and behaviors discussed in this paragraph and their social determinants (or the social circumstances) represented by more general living conditions and life experiences (education), other people in the social network (spousal loss), and the place where one lives (neighborhood) are established in Chapters 2 to 4, which are briefly summarized in the next paragraph.

1.4 Summarizing the three studies

Each of the Chapters 2 to 4 contains a self-contained study of one of the potential social determinants of old age cognitive functioning. The studies have either been published in scientific journals or are currently prepared for submission to a scientific journal. *Table 1.1* gives an overview of main aspects and the current status of the studies. While each of them

Table 1.1: Overview of the studies included in this dissertation

	Study 1 (Chapter 2)	**Study 2 (Chapter 3)**	**Study 3 (Chapter 4)**
Title	Trajectories of Cognitive Decline in Old Age: Does Education Predict Maintenance of Cognitive Abilities?	Spousal Loss and Change in Cognitive Functioning: An Examination of Temporal Patterns and Gender Differences	Cognitive Functioning Among Dutch Older Adults: Do Neighborhood Socioeconomic Status and Urbanity Matter?
Research Question(s)	Does education predict the development of cognitive functioning at higher ages?	Is losing the spouse at higher ages associated with changes in cognitive functioning? Does the effect differ by gender?	Are neighborhood socioeconomic status and urbanity associated with the level and decline of older adults' cognitive functioning?
Dependent Variables	Global cognitive functioning Processing speed Memory	Global cognitive functioning Processing speed Memory Reasoning	Global cognitive functioning Processing speed Memory Reasoning
Core Independent Variables	Education	Spousal loss	Neighborhood socioeconomic status, neighborhood urbanity
Data	LASA waves B to I (1992/3 to 2015/6)	LASA waves B to H (1992/3 to 2011/2)	LASA waves B to D (1992/3 to 2001/2); Neighborhood information from Statistics Netherlands
Statistical Units	Timepoints nested within persons	Timepoints nested within persons	Timepoints nested within persons nested within neighborhoods
Statistical Method	Growth curve models (linear mixed models framework) combined with accelerated longitudinal design	Within-person fixed-effects	Multilevel analysis combined with growth curve modeling (linear mixed models framework)
Current status	In preparation for journal submission	Published in Journals of Gerontology: Social Sciences	Published in Social Science & Medicine

Note: LASA = Longitudinal Aging Study Amsterdam;
In Study 3, different wording was used for the dependent variables: General cognitive functioning for global cognitive functioning and problem solving for reasoning.

addresses a different research question, all studies build on empirical data from the Longitudinal Aging Study Amsterdam (Hoogendijk et al., 2016; Huisman et al., 2011) for their empirical analyses. The project is an ongoing longitudinal study of the cognitive, social, emotional and physical functioning of older adults in the Netherlands, which started in 1992/3. At the first wave, a sample of 3,107 adults born between 1908 and 1937 was randomly selected from municipal registries, and additional cohorts were recruited after 10 and 20 years. Follow-ups were conducted every three to four years and each data collection involved face-to-face main interviews, drop-off questionnaires and additional medical interviews. Due to the inclusion of tests of global cognitive functioning/cognitive status, processing speed, logical reasoning and episodic memory, the data provide a great potential for longitudinal analyses of older adults' cognitive functioning. The availability of information about education and partner status, and the linkage to neighborhood data make the Longitudinal Aging Study Amsterdam a particularly suitable database to answer the research questions addressed in this dissertation.

Chapter 2, *Trajectories of Cognitive Decline in Old Age: Does Education Predict Maintenance of Cognitive Abilities,* examines whether educational attainment is related to the maintenance and decline of cognitive abilities relative to the starting level at a given age. I argue that more educated people are more likely to be exposed to life circumstances that put high demands on their cognitive abilities than their less educated counterparts, not only in the work context but possibly also during spare time activities. According to the concept of cognitive plasticity, sustained high demands should result in higher cognitive functioning via adaptations of the brain. Similarly, education itself and the resulting occupational trajectories are expected to form a cognitive reserve that pays off in longer maintenance of especially the memory domain in the face of brain damage.

While previous research unequivocally reported that education is positively associated with cross-sectional assessments of cognitive functioning, findings regarding their maintenance and decline are mixed. Although it is known that decline in many fluid abilities accelerates as people

age, this has rarely been considered in previous studies. In Chapter 2, I therefore examine whether educational differences in cognitive decline can be identified if differences in the initial rate of decline and its acceleration that jointly define the shape or course of cognitive decline are taken into account.

For the empirical analyses, a subsample of 1,182 respondents born between 1916 and 1927 is selected from the Longitudinal Aging Study Amsterdam in order to avoid generational differences in both education and cognitive functioning. Data were observed between 1992/3 and 2015/6 and are organized in an accelerated longitudinal design which allows to observe higher ages within shorter follow-up, reducing problems of panel attrition. For global cognitive functioning, processing speed and memory, the average level at age 65, initial rate of decline, and its acceleration are determined for different educational groups by the estimation of latent growth curve models in a linear mixed models framework.

The results corroborate previous findings that more education is associated with higher levels of cognitive functioning at age 65, specifically in the domains of global cognitive functioning and processing speed. Empirical support for educational differences in decline is weak even considering the potential for different shapes of decline: Tentative evidence of educational differences in decline is only found in the memory domain for women, where highly educated women seem to maintain their functioning into higher ages than their less educated counterparts but lose it faster once decline started.

In Chapter 3, *Spousal Loss and Change in Cognitive Functioning: An Examination of Temporal Patterns and Gender Differences,* I investigate whether spousal loss accelerates cognitive decline beyond the decline that can be expected due to aging. This might be the case because an important source of cognitive stimulation is lost and associated stress and depressive symptoms might further affect cognitive functioning. As men seem to experience stronger negative health-related effects after spousal loss, the chapter also examines whether the effects of spousal loss on cognitive decline vary by gender. Previous research on the topic brought

about mixed findings. This might be because a focus has often been on widowhood as a state of being, rather than on losing the spouse as an event that might have consequences in the short and intermediate run which might level off over time. The study is among the first to describe the co-evolution of spousal loss and the changes in cognitive functioning that occur in the years following the loss of the spouse.

The topic is examined analyzing a sample of 1,269 initially married, cognitively unimpaired older adults aged 65 and older from the Longitudinal Aging Study Amsterdam who have been observed over up to 20 years (1992/3 to 2011/2). Within person-fixed effects models allow to investigate whether losing the spouse is associated with immediate or subsequent decline in global cognitive functioning, processing speed, logical reasoning and episodic memory when statistically controlling for age-related decline and time-constant characteristics like education. The analyses suggest that reasoning abilities in women decline in the second year after spousal loss, but not before or after this time. Spousal loss is not associated to changes in the other cognitive domains among women, and no association between spousal loss and cognitive change is observed among men. The absence of strong evidence of associations between spousal loss and cognitive functioning in a dynamic longitudinal perspective challenges the common assumption that spousal loss accelerates cognitive decline. Furthermore, the findings imply that the consequences of spousal loss on cognitive functioning are not stronger for men than for women.

Chapter 4, *Cognitive Functioning Among Dutch Older Adults: Do Neighborhood Socioeconomic Status and Urbanity Matter?,* builds on the assumption that residential neighborhoods are among the most important contexts in which people spent their lives, especially at higher ages when other contexts like the working environment become less relevant. The socioeconomic status of the neighborhood and its urbanity are major structural characteristics with links to opportunity structures that are relevant for cognitive functioning. Specifically, neighborhoods with a higher (vs. lower) socioeconomic status might provide their

older inhabitants with more or better opportunities to engage in physical, social and cognitively stimulating activities. These activities are considered important determinants of cognitive functioning in different approaches, including the Cognitive Enrichment Hypothesis, the Use It or Lose It Hypothesis, the Environmental Complexity Hypothesis and the Revised Model of the Scaffolding Theory of Aging and Cognition. Similarly, the amount of information to be processed by persons in more urban neighborhoods is arguably larger, for example when moving in traffic or making shopping- and leisure-related decisions, and mentally stimulating offers like museums and theatres are more easily accessible. While previous research brought about mixed findings regarding the cross-sectional association between neighborhood socioeconomic status and cognitive functioning, less is known about the association of neighborhood socioeconomic status with cognitive decline and the role of neighborhood urbanity on both cross-sectional levels and longitudinal decline of cognitive functioning. The study examines these associations combining neighborhood information from Statistics Netherlands with individual information from 985 persons aged 65 to 88 years who participated in the Longitudinal Aging Study Amsterdam. Differences in cross-sectional levels of cognitive functioning and in longitudinal decline over six years are estimated using latent growth curve models in a multilevel framework. Additional to level 1 representing time points and level 2 where timepoints are clustered in persons, a third level is added to account for the clustering of respondents in neighborhoods.

The results provide some evidence that levels of cognitive functioning are higher in neighborhoods with a higher socioeconomic status, specifically for processing speed, reasoning, and, by trend, for memory, but not for global cognitive functioning. Also, average levels of all functions are higher in more urban neighborhoods, except for processing speed which is relatively stable at low and intermediate urbanity and decreases when urbanity is high. When statistically adjusting for a respondent's individual socioeconomic status, the effects of neighborhood urbanity decrease but are generally still observable. In contrast, level differences

by neighborhood socioeconomic status are largely explained by the respondents' individual socioeconomic status. This suggests that neighborhood socioeconomic status does not causally affect levels of cognitive functioning but rather that individuals with a similar socioeconomic status and cognitive functioning tend to concentrate in – or self-select into – neighborhoods with a corresponding socioeconomic status. There is no evidence of an association between neighborhood characteristics and cognitive decline during the analyzed period of six years.

1.5 Conclusion

Against the background of population aging, individual and societal concerns about aging successfully are widespread. According to the concept of Successful Aging, this pertains also to the maintenance of cognitive abilities into higher ages and the prevention of cognitive decline as one grows older. This dissertation examines whether social factors of typically long-lasting or intense nature are determinants of cognitive functioning and/or the decline of cognitive status and multiple fluid cognitive functions in old age. Specifically, I look at the role of education, spousal loss, and the residential neighborhood and argue that they provide opportunity structures for cognitively relevant experiences and behaviors through links with cognitive stimulation, physical and social activity, but also with detrimental experiences like stress and depressive symptoms.

All in all, it appears that the examined determinants relate differently to cross-sectional level differences in cognitive functioning than to longitudinal change in cognitive functioning over time. Level differences are examined for education as well as neighborhood socioeconomic status and urbanity.[4] Education makes a clear and positive difference in cognitive levels at age 65, while neighborhood urbanity showed weak but mostly positive and independent associations with cognitive levels at age 65. However, effects of neighborhood socioeconomic

[4] Note that the association between spousal loss and cognitive functioning was not examined cross-sectionally in Chapter 3.

status were explained by individual education, income, and employment status.

In contrast, evidence of an association with cognitive decline is only found between high education and the memory domain, with slower decline of memory functioning at younger old age but subsequent faster decline in more (vs. less) educated women. Evidence of associations with decline in other outcomes is weak, as well as associations between cognitive decline and other determinants – i.e. neighborhood characteristics or spousal loss – are scarce.

From a theoretical perspective, the associations between levels of cognitive functioning and education and neighborhood urbanity, respectively, are in agreement with a life course perspective on cognitive functioning as proposed by the Revised Model of the Scaffolding Theory of Aging and Cognition (STAC-r; Reuter-Lorenz & Park, 2014). Specifically, the effects of education show that experiences made early in life have a long reach and are still relevant for cognitive functioning at higher ages. The importance of education is additionally backed up by research suggesting that the effect of education is likely to go beyond the association between childhood cognitive abilities and later-life cognitive functioning being mirrored in educational attainment (Ritchie et al., 2016). In addition, the effects of neighborhood urbanity indicate that experiences from midlife and later life are relevant for cognitive functioning in their own right.

The theoretical implications of the longitudinal results are less conclusive. The weak evidence of associations between the determinants under consideration and cognitive decline was not to be expected from the Revised Model of the Scaffolding Theory of Aging and Cognition, which implicitly suggests that level and decline of cognitive functioning equally depend on relevant determinants. Instead, the findings demonstrate considerable robustness of cognitive functioning to major stressful experiences that come with changes in daily living conditions – like the loss of the spouse – or to enduring exposure to living environments like neighborhoods. This allows for multiple implications:

Firstly, taking the findings at face value, they would suggest that beneficial life experiences help building up a cognitive reserve that is used up at a similar rate for individuals during the aging process, as is suggested by a passive model of cognitive reserve (Lenehan, Summers, Saunders, Summers, & Vickers, 2015). This conclusion is only seemingly in contrast to findings showing interindividual heterogeneity in cognitive decline (Mella, Fagot, Renaud, Kliegel, & Ribaupierre, 2018; Wilson et al., 2002) considering that average rates of cognitive decline are at the focus.

Secondly, it might also be the case that respondents with low cognitive functioning or overly fast decline drop out of the sample due to the healthy participant effect. This would result in both attenuation of observed decline and underestimation of differences in decline between different groups. As a consequence, conclusions regarding the importance of examined determinants for cognitive decline have to be made with caution, as the rate of decline might indeed be associated with certain determinants but variation in decline is not adequately represented in the data.

By emphasizing the sociological perspective, this dissertation provides an important perspective to the field of cognitive aging, which typically gains more attention in developmental psychology, neuropsychology, and gerontology. Combining theoretical perspectives from these disciplines can inform the development of interesting hypothesis, which highlight that self-determination in taking actions to age successfully in general and with regard to cognitive functioning is limited (Stowe & Cooney, 2015). This becomes most obvious in findings suggesting that the place of residence – specifically its urbanity – might play a role for old age cognitive functioning. Additionally, the longitudinal perspective highlights that cross-sectional findings cannot simply be transferred into conclusions about determinants of decline or maintenance of cognitive functioning over time.

Although this dissertation was based on a rich and high-quality longitudinal dataset from the Netherlands, the Longitudinal Aging Study Amsterdam, there are limitations to the conclusions being derived from it. Firstly, selective panel attrition due to low cognitive functioning or fast decline is a common problem and most likely also affects the results of the analyses. When calculating growth models in Chapters 2 and 4, I followed the suggestion by Baraldi and Enders (2010) to handle missing data by analyzing all available data using maximum likelihood estimation. Future research might benefit from further improvements in data quality – for example through high efforts to survey and conduct cognitive tests with all respondents including those with low or declining cognitive functioning – to allow for more firm conclusions about determinants of cognitive decline. Also, applying more advanced weighting strategies might be instrumental in approaching problems of selective panel attrition (Weuve et al., 2012). Secondly, some important concepts like the opportunities for physical activity or mental stimulation in the neighborhood could only be proxied through neighborhood socioeconomic status and urbanity. The availability of more direct measures of such opportunities would be an asset in developing more direct tests of associated hypotheses. Thirdly, the data used in this dissertation are observational and provide little information about the mechanisms behind the reported associations. Fourthly, even though this dissertation is concerned with cognitive functioning in the general older population and therefore with age-related decline, it cannot be precluded that findings are influenced by some respondents who showed low or declining cognitive functioning due to pathology (Deary et al., 2009). To attenuate this issue in the absence of data on dementia diagnosis, I exclude those with very low cognitive functioning in Chapters 3 and 4. Finally, the data from the Longitudinal Aging Study Amsterdam only allow conclusions about the Netherlands. Despite the plausible assumption that the determinants and mechanisms referred to in this dissertation are rather universal, their impact might depend on the differences in living conditions of more or less educated persons, widowed and married persons, or persons in different neighborhoods.

Future research might push the field forward by combining and weighting more equally the structural perspective and the individual perspective. This might be because the impact of socio-structural and contextual factors depends on individual characteristics that determine, for example, the degree to which a situation is perceived as stressful or cognitively challenging. If that was the case, different situations might play out differently for different persons, and this might be an explanation why some of the examined associations in this dissertation are weak or absent. I partly tried to integrate such a mixed approach by examining whether the effect of spousal loss differs by gender, but measuring potential moderators more directly might allow for new insights. Potential candidates for future research into moderators of structural effects might include personality characteristics like neuroticism or the perceived support of the social network as factors influencing how stressful an event like spousal loss is perceived. Another approach to link structural and individual determinants is to examine how social determinants moderate effects of variants of the APOE gene (Cook & Fletcher, 2015).

From a practical point of view, this dissertation is in line with the idea that maintaining cognitive functioning at higher ages is a life time endeavor that depends on experiences made at different stages of the life course. Specifically, the results support the notion that investments into education at younger ages pay off in form of better cognitive functioning in old age. Also, living environments in midlife and old age seem to play a role in old age cognitive functioning. Surprisingly, it appears that severe events like the loss of the spouse are not generally associated with cognitive decline, thus the current findings do not call for specific interventions with the aim of supporting the cognitive functioning of older widows and widowers. The same holds for the examined neighborhood characteristics, since neighborhoods explained only little variation in cognitive functioning.

In general, it does not only seem that cognitive functioning is a major resource for engagement with life, but also that engagement with life benefits cognitive functioning. Encouraging and assisting especially people in structurally disadvantaged positions – but also others – to engage

in physical activity (Sofi et al., 2011), cognitively challenging activities (Kelly et al., 2017), and fostering integration into a social network (Kelly et al., 2017) might however be beneficial for their cognitive functioning and have additional benefits for their health and well-being. Besides targeting such efforts at the individual and group level, they might also include measures at the community level like the design of urban public spaces (World Health Organization, 2017). Especially broader approaches have the potential to benefit not only those persons that have already reached old age, but also those that will approach old age in the nearer or more distant future.

1.6 Status of the studies and contribution of co-authors

Chapter 2: ***Trajectories of Cognitive Decline in Old Age: Does Education Predict Maintenance of Cognitive Abilities?***, is currently prepared for journal submission.

As the lead author, I developed the research question and theoretical framework, prepared the data for analyses, conducted the analyses and prepared the manuscript. Co-authors Prof. Dr. Martijn Huisman, Vrije Universiteit Amsterdam, Prof. Dr. Hannie Comijs, Vrije Universiteit Amsterdam, and Prof. Dr. Marja Aartsen, Oslo Metropolitan University, commented on the manuscript. Marja Aartsen also assisted in developing the data analytical approach.

Chapter 3: ***Spousal Loss and Change in Cognitive Functioning: An Examination of Temporal Patterns and Gender Differences***, is published (advance access) in *The Journals of Gerontology: Series B*, gby104. doi.org/10.1093/geronb/gby104.

As the lead author, I developed the research question and theoretical framework, prepared the data for analyses, conducted the analyses and prepared the manuscript. Co-authors Prof. Dr. Hannie Comijs, Vrije Universiteit Amsterdam, and Prof. Dr. Marja Aartsen, Oslo Metropolitan University, commented on different versions of the manuscript.

Chapter 4: ***Cognitive Functioning Among Dutch Older Adults: Do Neighborhood Socioeconomic Status and Urbanity Matter?***, is published in *Social Science & Medicine*, 187, 29-38. doi.org/10.1016/j.socscimed.2017.05.052.

As the lead author, I developed the research question and theoretical framework, prepared the data for analyses, conducted the analyses and prepared the manuscript. Co-authors Prof. Dr. Lea Ellwardt, University of Cologne, Prof. Dr. Marja Aartsen, Oslo Metropolitan University, and Prof. Dr. Martijn Huisman, Vrije Universiteit Amsterdam, commented on different versions of the manuscript.

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Chapter 2: Trajectories of Cognitive Decline in Old Age: Does Education Predict Maintenance of Cognitive Abilities?

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Abstract

Objectives: It is a widely held assumption that education is positively related to cognitive functioning in old age. While this seems to be the case for levels of cognitive functioning when entering this life stage, the findings for subsequent cognitive decline are mixed. Considering findings pointing towards an accelerated course of cognitive decline, the current study goes beyond the common assumption that decline is faster for some and slower for others and examines whether the shape of decline trajectories differs between educational groups.

Methods: Education-specific decline trajectories were estimated for global cognitive functioning, processing speed and memory based on data from up to 1,182 respondents aged 65 to 89 (4,194 person-observations) from the Longitudinal Aging Study Amsterdam. Estimation of latent growth curve models allowed to examine educational differences in cognitive levels at age 65, the initial speed of decline and its subsequent acceleration, with the latter two describing the shape of a decline trajectory for each educational group.

Results: The analyses revealed that more education is associated with higher levels of global cognitive functioning and processing speed, but not memory. Tentative evidence for systematic differences in the shape of cognitive decline trajectories was only found among women in the memory domain.

Discussion: The results suggest that educational effects on the maintenance of cognitive functioning in old age operate rather through the cognitive levels when entering old age than through altering the course of subsequent decline.

2.1 Introduction

Over the last decades, both the number of older persons and their population share have grown in many regions of the world and are predicted to grow further (United Nations, 2017). As people age, especially fluid cognitive abilities (e.g. memory, executive functioning, processing speed) have been shown to decline and the decline seems to be faster at higher ages (Lipnicki et al., 2017; Reas et al., 2017; Wilson et al., 2002; Zaninotto, Batty, Allerhand, & Deary, 2018). However, maintaining cognitive functioning is an important precondition to live independently, with implications for quality of life at the individual level and for costs of care at the societal level (Deary et al., 2009; Hertzog, Kramer, Wilson, & Lindenberger, 2009). Although older populations on average typically show patterns of declining cognitive functioning, previous research has identified substantial interindividual heterogeneity, reporting not only patterns of decline but also of stability and even improvement (Mella, Fagot, Renaud, Kliegel, & Ribaupierre, 2018; Wilson et al., 2002). In parts, this heterogeneity can be explained by the concept of cognitive plasticity, according to which individual experiences and conditions can alter the course of cognitive development within biological constraints (Baltes, 1987; Hertzog et al., 2009; Lövdén, Bäckman, Lindenberger, Schaefer, & Schmiedek, 2010).

In this study, we examine whether education is one of the factors that predicts the development of cognitive functioning at higher ages. We argue that education shapes relevant experiences and conditions that individuals make throughout the life course. This might not only concern the complexity experienced during the educational phase but also the complexity experienced in the work environment or maybe even in spare time activities. Schooler (1984) characterized environmental complexity as the diversity of stimuli, the number of decisions required, the amount of information to be considered in these decisions, and the degree to which contingencies are vague and/or seemingly contradictory. Similarly, Lövdén et al. (2010) suggested that according to the cognitive plasticity hypothesis, a prolonged mismatch between

"functional supply" and "environmental demands" should trigger an adaptation of the cognitive functions towards the demands experienced by a person. Empirically, higher cognitive demands at work have indeed been demonstrated to associate with higher cognitive functioning (Fisher et al., 2014; Then et al., 2014), even if early adulthood intelligence was taken into account (Potter, Helms, & Plassman, 2008). From the perspective of cognitive reserve hypothesis, more educated persons build up a larger cognitive reserve that allows for longer maintenance of especially the memory domain in the face of brain damage by allowing more efficient task processing (Stern, 2002). Similar to the complexity argument, it is assumed that cognitive reserve is not merely determined by innate intelligence but also by experiences made throughout the life course, e.g. an individual's education or occupation (Richards & Sacker, 2003; Stern, 2002). Although cognitive reserve has largely been discussed in the context of dementia-related changes to the brain, it has been suggested that the concept also applies to normal cognitive aging in nondemented persons (Barulli & Stern, 2013; Stern, 2002). Notwithstanding a presumably causal effect of childhood cognitive ability on cognitive ability at higher ages, independent effects of education by age 26 and occupation at age 43 on later life cognitive functioning have been demonstrated (Richards & Sacker, 2003).

In line with these theoretical suggestions, previous studies have relatively consistently shown that higher levels of education predict higher levels in a number of cognitive functions in older adults (Alley, Suthers, & Crimmins, 2007; Cadar et al., 2017; Lipnicki et al., 2017; Tucker-Drob, Johnson, & Jones, 2009; Zahodne et al., 2011, but see Reas et al., 2017 for mixed findings). However, it is much less clear whether the positive association between education and levels of cognitive functioning translates into a protective effect against cognitive decline at higher ages. In their review, Anstey and Christensen (2000) concluded that education was associated with slower decline in mental status, crystallized abilities and memory, but not in speed and other fluid abilities. Little empirical support for an association between education and cognitive decline was reported in a more recent review of ten studies looking at multiple

cognitive domains, with the majority of studies finding no association and two studies pointing towards faster or slower decline in more educated persons, respectively (Lenehan, Summers, Saunders, Summers, & Vickers, 2015). The mixed findings might imply that there is no replicable effect of education on cognitive decline, but it could also mean that cognitive decline follows different patterns than commonly assumed. With few exceptions (Alley et al., 2007; Christensen et al., 2001; Muniz-Terrera et al., 2009; van Dijk, van Gerven, van Boxtel, van der Elst, & Jolles, 2008; Wilson et al., 2009; Zahodne, Stern, & Manly, 2015), previous research on educational differences mirrored the implicit assumption that educational differences in cognitive decline might manifest in either faster or slower rates of cognitive decline, irrespective of the age of the older adults. This is represented in empirical analyses that dichotomize cognitive change, examine change between two timepoints, or examine effects of education on linear patterns of decline. However, since average cognitive decline has been shown to follow a pattern of acceleration at higher ages, it might be the case that educational differences in cognitive decline are more complex. For example, decline might occur at a similar rate for different educational groups in younger older adults and educational differences might manifest in differential acceleration of decline at higher ages. Similarly, decline might be initially slower for one educational group but accelerate more strongly compared to others at higher ages. This would suggest that cognitive decline cannot merely differ in the speed of decline; rather, differences could occur in both the initial speed of decline and its acceleration over time, resulting in different shapes of decline of cognitive functioning.

Thus, the current study sets out to examine educational differences in the shapes of decline in global cognitive functioning, processing speed and memory in older adults aged 65 to 89 years. Data are from up to 1,182 individuals from the Longitudinal Aging Study Amsterdam who have been followed for up to 23 years at the individual level.

2.2 Data and methods

2.2.1 Sample

The Longitudinal Aging Study Amsterdam (Hoogendijk et al., 2016; Huisman et al., 2011) is an ongoing longitudinal study of older adults in the Netherlands, starting in 1992/3 with follow-ups every 3 to 4 years thereafter (t_1=1992/3, t_2=1995/6, t_3=1998/9, t_4=2001/2, t_5=2005/6, t_6=2008/9, t_7=2011/2, t_8=2015/6). The initial sample of 3,107 individuals born between 1908 and 1937 was randomly selected from municipal registries, with an oversampling of older men and the oldest age groups. Data were collected by trained interviewers in a face-to-face main interview at the respondent's home, during which the interviewers left a drop-off questionnaire and asked respondents to participate in a subsequent medical interview. For the present study, we selected 1,185 individuals born between 1916 and 1927 to reduce generational heterogeneity in both education and cognitive functioning. Of those, one respondent with missing information on the variable measuring education had to be excluded. Apart from that, only respondents who never provided information on a cognitive measure had to be excluded from the analysis of the respective cognitive domain, resulting in sample sizes of 1,182 respondents for global cognitive functioning, 1,066 for processing speed, and 1,077 for memory, with 4,194 person-observations for global cognitive functioning, 3,557 for processing speed and 3,631 for memory, respectively.

2.2.2 Variables

Cognitive functioning

Three measures of cognitive functioning were examined, including one widespread measure of global cognitive functioning (assessed at the main interview) and two measures of fluid abilities, specifically processing speed and memory (both assessed at the medical interview). Examining fluid abilities is of particular interest because of their higher susceptibility to age-related decline compared to crystallized abilities (Lindenberger & Baltes, 1997).

Global cognitive functioning was assessed with the Mini Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975). The measure was originally developed as a screening tool for cognitive impairment and assesses basic abilities in orientation in time and space, registration, attention, recall, language, and visuospatial abilities. Up to 30 points can be scored, with higher values indicating better cognitive functioning.

Processing speed was measured using the Coding Task, a letter-letter substitution task described by Piccinin and Rabbitt (1999). Respondents were presented with rows of letters, each of which had an empty row below it. For each upper row letter, a key defined a corresponding letter to be added to the lower row. Respondents were asked to verbally name as many lower-row letters that match the letters in the upper row as possible. We analyzed the mean number of letters mentioned during three trials of one minute each.

Memory was tested with a slightly modified version of the 15 Words Test, the Dutch version of the Auditory Verbal Learning Test (Rey, 1964; Saan & Deelman, 1986). In three instead of originally five trials, respondents were verbally presented with the same 15 words and asked to recall as many words as possible. We used the delayed recall score, i.e. the number of words correctly remembered after a distraction period of 20 minutes.

Education

Education was assessed at t_1 and is grouped into *low* (elementary education or less), *medium* (lower/intermediate vocational education or general intermediate/secondary education), and *high* education (higher vocational education, college, or university) (Hoogendijk, van Groenou, Marjolein Broese, van Tilburg, & Deeg, 2008).

Age

Age of the respondent (in years) was calculated from the difference between the first year of a study wave and the birthyear. For the growth models, age was centered to 65 years.

2.2.3 Analytical approach

Data were analyzed combining latent growth curve analysis with an accelerated longitudinal design.

Latent growth curve analysis (Wickrama, Lee, O'Neal Catherine Walker, & Lorenz, 2016) uses measurements from different time points to determine the growth parameters of a variable (in our case intercept, linear slope, and quadratic slope) that describe the temporal development of this variable at the individual level. For a given sample, the mean and variance of each growth parameter can be determined and effects of predictor variables on these growth parameters can be estimated. In the current study, we used a linear mixed models approach to growth curve modeling, with observations at the survey waves (level 1) nested within respondents (level 2). Educational differences in the level of cognitive functioning are represented by dummy variables for educational groups, while educational differences in the initial speed of cognitive decline (linear slope) and its subsequent acceleration (quadratic slope) are investigated by interacting education dummies with the respective growth parameter, i.e. with age and age squared, respectively. Due to our interest in cognitive decline as an aging process, respondents' age was chosen as the time metric for the growth models. Wald χ^2-statistics were calculated to check for statistically significant differences in the cognitive trajectories jointly described by the linear and quadratic slope. Additionally, visual inspection of the trajectories was applied to assess differences in the shape of cognitive decline. Effects with a *p*-value smaller than .050 were considered statistically significant.

With the so-called accelerated longitudinal or cohort-sequential design (Duncan & Duncan, 2011), longitudinal data from different birth cohorts are merged into an overall trajectory such that it covers the age interval from the age of the youngest respondent at the first interview to the age of the oldest respondent at the last interview. One advantage of such a design is that it reduces the issue of panel attrition since higher ages can be observed within shorter follow-up periods, while attrition becomes more severe as follow-up time increases (Duncan & Duncan,

2011). Another advantage is that this design allows studying an age-interval that is longer than the study duration. During the 23 years covered by this study, the youngest cohort born in 1927 could theoretically be observed from about age 65 at t_1 to about age 88 at t_8. For the oldest cohort born in 1916, the study covers ages 76 to 99, and the intervals for the other cohorts vary accordingly. However, since only about 2% of the observations were observed at age 90 or older, these observations were excluded from the analyses because of the presumably highly selected group surviving to these ages and their potential impact on the overall trajectory. The overall trajectory estimated from different birth cohorts is a valid description of the mean trajectory for persons from these cohorts under the assumption that there are no cohort differences. We aimed to reduce the influence of cohort differences in cognitive functioning (Brailean et al., 2018) and its development by limiting the sample to respondents born in a 12-year interval, for which it is reasonable to assume that no major changes in cognitive functioning occurred. Also, systematic increase in educational levels over these birthyears is relatively weak, as shown by significant but small positive correlations for the MMSE sample (Pearson's r=.06, p=.032), but not for the Coding Task or 15 Words Test samples (Pearson's rs=.05, ps≥.112).

Due to centering age to 65 years in all latent growth models, the intercept represents level differences at age 65, while the slope (age) and the quadratic slope (age squared) represent instantaneous change at age 65 and its acceleration, respectively. Random effects for the intercept, age and age squared were estimated with these models. Respondents were included in the analyses if they provided valid information on the cognitive outcome of interest for one or more waves. Missing data on the cognitive outcomes for the remaining waves was handled by using maximum likelihood estimation (Baraldi & Enders, 2010). In additional analyses, we tested if practice effects were present by adding a dummy variable identifying the first wave in which a respondent was tested on a specific cognitive outcome as a predictor and, if so, whether they affected our findings. We also examined whether the smaller likelihood of women (vs.

men) to obtain higher levels of education (about 6% vs. 13% for high education and 37% vs 56% for medium education) goes along with different education specific cognitive trajectories. Because our study is concerned with identifying the total effect of education on cognitive decline, variables like job characteristics, income or health status are considered potential mechanisms linking education and cognitive decline rather than confounders and were therefore not accounted for in the models. All analyses were conducted using Stata 14 (StataCorp, 2015).

2.3 Results

Descriptive statistics for the MMSE sample can be found in *Table 2.1*. Across all person-observations in the MMSE sample, the average age was about 76 years, somewhat less than half of the observations were from male respondents (43%), and the majority of observations were from the low (42%) and medium (49%) education groups. Respondents were followed up for an average duration of 8.2 years. As regards health, about 8.8 depressive symptoms on the CES-D were reported, while at least one functional limitation and at least one chronic disease were present in about 61% and 85% of the observations, respectively. Except for the follow-up

Table 2.1: Descriptive statistics for the analyses of the MMSE.

MMSE	Mean / %	SD	Min	Max	*N/n*
Age	75.8	5.8	65	89	4,194
Male	43.2%	--	0	1	4,194
MMSE	26.7	3.1	2	30	4,194
CES-D	8.8	7.7	0	54	4,119
Functional limitations	60.8%	--	0	1	4,111
Chronic diseases	85.4%	--	0	1	4,174
Follow-up (years)[a]	8.2	6.6	0	23	1,182
Education					
low	41.8%	--	--	--	1,753
medium	48.5%	--	--	--	2,035
high	9.7%	--	--	--	406

Note. Numbers refer to observations, except for [a] referring to respondents. Deviations from 4,194 observations/ 1,182 individuals are due to missing values on variables not included in the latent growth models.
MMSE = Mini Mental State Examination, CES-D = Center for Epidemiological Studies Depression Scale.

time being about half a year shorter, the samples for the Coding Task and 15 Words Test were comparable with regard to the above variables (see *Tables 2.A1* and *2.A2* in the Appendix). In all samples, the comparison of the high and low education groups using *t*-tests and *p*-tests showed that high education is associated with being male, longer follow-up duration, higher scores on the cognitive tests, and better health as indicated by lower numbers of depressive symptoms and lower shares of functional limitations and chronic diseases present.

Models 1 (*Table 2.2*) describe the overall cognitive trajectories by age and age squared while accounting for educational level differences and including random effects for the intercept, age, and age squared. The effects of age indicated that decline at age 65 in MMSE and Coding Task was not yet observable, while scores on the 15 Words Test seemed to increase. The effects of age squared suggest that, on average, scores in all three domains decline more strongly as respondents age. Accounting for the lack of practice when taking a test for the first time revealed a statistically significant decline already at age 65 in both Coding Task and Memory but not MMSE (models not shown). While the lack of practice at the first assessment was substantial for the 15 Words Test (B=-1.30 or 0.45 SD, p<.001), this was not the case for MMSE (B=-0.13 or .04 SD, p=.162) and Coding Task (B=-0.31 or .04 SD, p=.501). Thus, in addition to our main models which do not account for the lack of practice at the first assessment, we present additional analyses demonstrating that our conclusions regarding the 15 Words Test remained unchanged when practice effects were accounted for. The level of functioning increased for all outcomes as the level of education increased, except for the 15 Words Test where the difference between medium and high education groups was not statistically significant. In a next step, educational levels were additionally interacted with age and age squared, yielding a model describing education-specific cognitive trajectories. The intercept and the effects of age and age squared in these models are conditional on the reference group (low education) and represent the level of cognitive functioning at age 65, the rate of cognitive change at age 65, and the

Table 2.2: Latent growth models of cognitive functioning including educational level differences. Unstandardized coefficients with 95% confidence intervals.

	MMSE M1		Coding Task M1		15 Words Test M1	
Constant	26.95***	[26.70,27.21]	21.95***	[21.32,22.59]	4.95***	[4.64,5.26]
Age	0.01	[-0.03,0.06]	-0.06	[-0.13,0.01]	0.10***	[0.06,0.14]
Age squared	-0.01***	[-0.01,-0.01]	-0.02***	[-0.02,-0.01]	-0.01***	[-0.01,-0.01]
Education (Ref. Low)						
Medium	1.02***	[0.76,1.28]	4.72***	[3.97,5.47]	0.47**	[0.16,0.78]
High	1.50***	[1.07,1.94]	7.87***	[6.58,9.16]	0.89***	[0.37,1.42]
Random Components						
Var(Constant)	1.43*		30.64***		4.10***	
Var(Age)	0.04***		0.01***		0.01***	
Var(Age squared)	0.00***		0.00***		0.00***	
Var(Residual)	3.08***		6.29***		3.28***	
AIC	19,359		20,003		16,563	
BIC	19,416		20,059		16,618	
$N_{observations}$	4,194		3,557		3,631	
$N_{respondents}$	1,182		1,066		1,077	

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion

acceleration of the rate of cognitive change, respectively, for the low education group. The dummy variables for medium and high education give the respective difference in the cognitive level at age 65 compared to the low education group. Accordingly, the interactions of medium and high education with age and age squared indicate the difference in the rate of change at age 65 and in the acceleration of the rate of change compared to the low education group. Wald χ^2-statistics were used to test whether the temporal development over time that is jointly described by the terms for age and age squared differ significantly between educational groups. Results are shown in *Table 2.3* and in *Figure 2.1*.

For MMSE, a pattern of accelerated decline in MMSE was observed for the low education group, as jointly represented by the positive but statistically insignificant effects of linear age (*B*=0.05, *p*=.168) and the statistically significant and negative effect of squared age (*B*=-0.01, *p*<.001). Compared to the low education group, decline at age 65 did not differ in the medium and high education groups, as indicated by the interactions of age with medium and high education (*p*s≥.234). However, the positive and statistically significant interaction terms with age squared hinted towards a slower acceleration of decline at higher ages in the medium and high (vs. low) education groups (Age squared x medium education; *B*=.01, *p*=.012; Age squared x high education: *B*=.01, *p*=.033). Wald χ^2-tests revealed that the linear and squared term for age differed neither separately nor jointly between medium and high education groups, such that no differences in the development of MMSE between medium and high education groups can be concluded (all *p*≥.529). As regards level differences at age 65, average scores of respondents in the medium and high (vs. low) education groups were higher (both *p*<.001) but no significant difference was observed between the medium and high education groups (χ_1^2=1.48, *p*=.224). Average Coding Task scores seemed to be relatively stable at age 65 in the low education group, as indicated by the small negative, statistically insignificant term for linear

Table 2.3: Latent growth models of educational differences in cognitive trajectories. Unstandardized coefficients with 95% confidence intervals.

	MMSE M2		Coding Task M2		15 Words Test M2	
Constant	26.86***	[26.53,27.20]	21.75***	[20.99,22.51]	5.19***	[4.79,5.60]
Age	0.05	[-0.02,0.11]	-0.05	[-0.16,0.06]	0.05	[-0.02,0.12]
Age squared	-0.01***	[-0.02,-0.01]	-0.01***	[-0.02,-0.01]	-0.01***	[-0.01,-0.00]
Education (Ref. Low)						
Medium	1.19***	[0.73,1.65]	5.15***	[4.12,6.19]	0.05	[-0.51,0.60]
High	1.64***	[0.90,2.38]	7.97***	[6.25,9.68]	0.54	[-0.36,1.43]
Age x Education (Ref. Low)						
Medium	-0.05	[-0.14,0.03]	-0.02	[-0.17,0.12]	0.09	[-0.00,0.18]
High	-0.06	[-0.20,0.09]	0.02	[-0.21,0.26]	0.07	[-0.08,0.22]
Age squared x Education (Ref. Low)						
Medium	0.01*	[0.00,0.01]	-0.00	[-0.01,0.00]	-0.00	[-0.01,0.00]
High	0.01*	[0.00,0.01]	-0.00	[-0.01,0.01]	-0.00	[-0.01,0.00]
Random Components						
Var(Constant)	1.41*		30.66***		4.10***	
Var(Age)	0.04***		0.01***		0.01***	
Var(Age squared)	0.00***		0.00***		0.00***	
Var(Residual)	3.08***		6.29***		3.28***	
Wald χ^2-Tests (Age and Age squared)						
Low Education	.000		.000		.000	
Low vs. Medium Education	.006		.002		.170	
Low vs. High Education	.012		.401		.629	
Medium vs. High Education	.529		.643		.885	
AIC	19,353		19,999		16,567	
BIC	19,436		20,079		16,647	
$N_{observations}$	4,194		3,557		3,631	
$N_{respondents}$	1,182		1,066		1,077	

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion

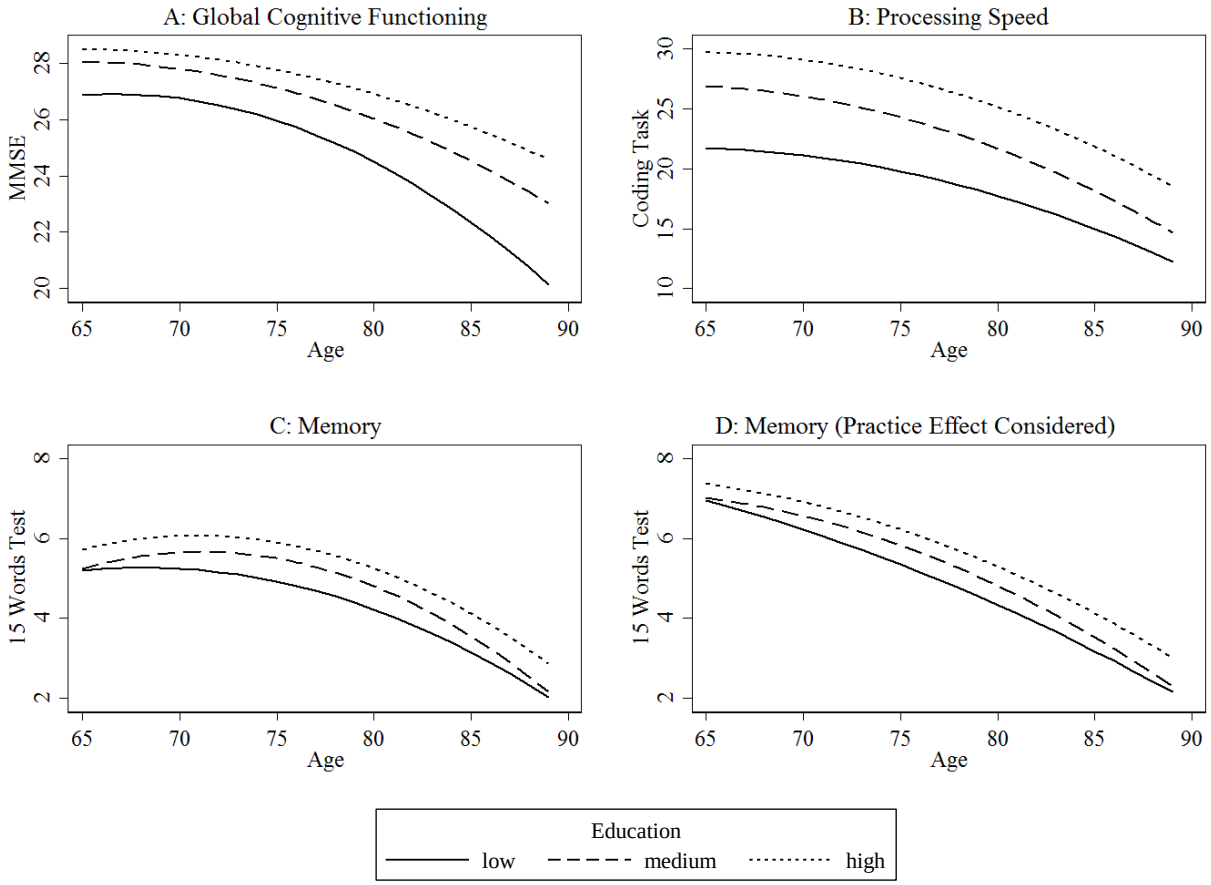

Figure 2.1: Panel A to C show predicted trajectories in three cognitive domains for low, medium, and high education (results from *Table 2.3*). Panel D portraits the hypothetical situation in which the respondents already at the first assessment have the same experience with the 15 Words Test as at the subsequent assessments (see *Table 2.A3* in the Appendix).

age (*B*=-0.05; *p*=.332). At higher ages, decline in Coding Task scores accelerated in this group, as indicated by the negative effect of age squared (*B*=-0.01; *p*<.001). The shape of the trajectories of the medium and high education groups did not seem to differ from the low education group as indicated by non-significant interactions of age and age squared with medium and high education (*p*s≥.265); however, Wald χ^2-statistics testing the joint difference of linear and squared age indicated that the average trajectories for persons with low and medium education were not the same (χ^2_2=12.46, *p*=.002). *Figure 2.1* suggests that the shape of the trajectories for the medium and low education groups are similar, but that decline is faster for the medium education group, which results in a narrowing gap between both educational

groups at higher ages. However, the decline in Coding Task scores does not seem to vary systematically with education since the trajectory for the high education group did not differ from either the low or medium education group according to Wald χ^2-statistics (all $p \geq .401$). In contrast, educational differences in the level at age 65 were evident: As education increased, so did the average Coding Task score (medium education: B=5.15, p<.001; high education: B=7.97, p<.001; difference between medium and high education: χ_1^2=10.57, p=.001).

As regards the 15 Words Test, a tendency towards an initial improvement with subsequent accelerated decline at higher ages in the low education group was implied by the positive but statistically insignificant effect of age (B=0.05, p=.174) and the negative effect of age squared (B=-0.01, p<.001). This is suggestive of a practice effect, which did not affect our conclusions (see below). Compared to the low education group, the medium education group tended to show stronger improvement at younger ages and faster acceleration of decline at higher ages (Age x medium education: B=0.09, p=.060; Age squared x medium education: B =-0.00, p=.076), which did however not substantiate in a significant difference between both trajectories (Wald χ_2^2=3.54, p=.170). The trajectory of the high education group differed from neither the low education group (Age x high education: B=0.07, p=.373; Age squared x high education: B=-0.00, p=.478; for the joint difference of age and age squared in the high vs. low education groups: Wald χ_2^2=0.93, p=.629) nor the medium education group (all $p \geq .678$). *Figure 2.1* supports the idea that the shape of the high education trajectory is more similar to the low than to the medium education trajectory and that there are thus no systematic educational differences in 15 Words Test development. Significant level differences at age 65 were not observed (all $p \geq .239$).

Across all three cognitive tests, model fit indices indicated that considering educational differences in the shape of cognitive trajectories does little to explain differences in cognitive functioning in our sample. While Akaike's Information Criterion decreased slightly from

Models M1 to M2 for MMSE and Coding Task but not 15 Words Test, the Bayesian Information Criterion – which penalizes the inclusion of additional predictor variables more strongly – increased for all outcomes.

Accounting for the lack of practice with the test at the first assessment improved model fit (LR-Test: χ_1^2=175.36, p<.001) but did not change the conclusions regarding educational differences in 15 Words Test trajectories (see *Table 2.A3* in the Appendix and Panel D in *Figure 2.1*). Findings were in line with the idea of a lack of practice when taking the test for the first time, as respondents scored more than one word less when taking the test for the first time compared to assessments of higher order (B=-1.30, p<.001). When the lack of practice was accounted for, the increase of scores at younger ages disappeared and decline was already observable at age 65 in the low education group (B=-0.13, p<.001). Additionally, only a tendency towards acceleration of decline remained in the low education group, as indicated by the effect of age squared (B=-0.00, p=.062). As in the original model, the three trajectories did not differ significantly from each other, as is indicated by both separate and joint comparisons of the coefficients of age and age squared between educational groups (all p≥.134).

Gender differences might have affected all outcomes studied, especially since our descriptive findings showed that women had lower probabilities of achieving medium or high education than men. On the one hand, this could mean that education is not as good a representation of innate abilities for women than it is for men of the generations studied. On the other hand, it might be the case that highly educated women had to achieve outstanding results to achieve these educational degrees and that thus, at the aggregate level, highly educated women are more skilled than their male counterparts. To test for gender differences, we ran gender separate models including practice effects (*Table 2.A4* and *Figure 2.A1* in the Appendix). For MMSE, the divergence of trajectories at higher ages was stronger for women than men and it was only for women but not men that the low, medium and high trajectories differed significantly from

each other. On the contrary, in the domain of processing speed, it was only among men that the trajectory for medium education differed significantly from the low education group. However, only for the 15 Words Test did the education specific trajectories differ significantly by gender, as calculations of interaction terms in a joint model revealed. Specifically, the trajectories for highly educated men and women were significantly different (Wald χ_1^2=5.30, p=.021; models not shown). While the trajectories did not differ in the male sample, the high education trajectory for women started with improvement and declined later, compared to an initially and rather linearly declining average trajectory for women with medium or low education. The relatively small number of highly educated respondents in our sample resulted in small numbers, especially of women but also of men, on which the estimated high education trajectories were based (36 women and 64 men for the 15 Words Test, similar numbers for MMSE and Coding Task). Thus, these gender-specific findings should be treated cautiously.

2.4 Discussion

This study examined whether differences in educational attainment are associated with different shapes of decline trajectories in global cognitive functioning, processing speed, and memory using a sample from the population-based Longitudinal Aging Study Amsterdam. Initial models showed that once practice effects are considered, both processing speed and memory on average decreased already at age 65, while global cognitive functioning was still stable. At higher ages, decline accelerated in all three domains, a finding that is in line with previous research (Lipnicki et al., 2017; Reas et al., 2017; Wilson et al., 2002; Zaninotto et al., 2018). This allows for the opportunity that previous studies focusing on educational differences in linear cognitive decline might have missed educational differences that manifest in differences in the initial rate of decline and/or its acceleration at higher ages. Such a pattern could for example indicate that one educational group maintains a high level of cognitive functioning relative to their starting level but might compensate for this initial advantage by means of faster acceleration later on. To

explore this possibility, education-specific trajectories were estimated to assess differences in the shape of cognitive decline as represented by differences in the initial rate of decline and its acceleration for each of the cognitive domains.

As regards global cognitive functioning, we found that the average decline accelerated more strongly in the low (vs. medium/high) education group among women but not men. The result is in line with a previous finding by Alley et al. (2007), who reported that global cognitive functioning as measured by the Telephone Interview for Cognitive Status declined more rapidly after age 80 in less educated persons. Similarly, Wilson et al. (2009) reported that decline in a composite cognitive score was slower in early years of follow up but became faster later on in the low (vs. average or high) education group, although the trajectories were rather similar in general. An alternative explanation for the observed stronger acceleration of decline in general cognitive functioning among less educated persons might be that a ceiling effect on the MMSE masks the actual extent of decline at higher levels of functioning and does so especially for persons with medium and high education. In line with this reasoning, MMSE-scores of 29 or 30 out of 30 were observed in 35% and 48% of all observations in the medium and high education groups, respectively, while such high scores were only met in 21% of the observations in the low education group. Also, this ceiling effect was stronger among women than among men, with medium educated women scoring 29 or 30 on the MMSE at 41% (men: 30%) and highly educated women at 55% (men: 44%) of the observations. This offers an explanation why a stronger divergence of trajectories between educational groups was observed among women compared to men. We speculate that women of the cohorts studied on average had to demonstrate higher abilities or achievements to obtain high education degrees than their male counterparts, thus indicating higher average functioning. Alternatively, it might be the case that the chances of highly educated women to lead high cognitive load lives compared to less educated women are larger than those of their male counterparts.

For processing speed, neither the initial speed of decline nor its acceleration differed between

the educational groups, suggesting no educational differences in the shapes of processing speed trajectories. However, overall speed of decline seemed to be faster for the medium, but not for the high compared to the low education group, and gender separate analyses showed that this finding was especially true for men. It might be the case that education speeds up the rate of decline in processing speed only up to a certain educational level, after which there is no additional benefit. However, this would require that the trajectories in the medium and high educational groups were similar to each other and differ appreciably from the low education group. This seems rather unlikely because the high education group did not differ significantly from the low education group. Although the smaller sample size in the high (vs. medium) education group might explain differences in statistical significance, the graphical inspection of the trajectory did not support a larger similarity between the high education trajectory and the medium (vs. low) education trajectory. Consequently, we interpret this as a non-systematic finding and conclude that education is neither associated with maintenance nor shape of decline in processing speed.

Memory was the only domain in which significant gender differences were observed. While indication of non-linear decline was weak for men in all of the educational groups as well as for women in the low and medium education groups, women in the high education group initially showed maintenance or even improvement of their memory abilities and started to decline later than their less educated counterparts. In line with this finding, a previous study reported a relatively longer maintenance with a later but steeper decline – as assessed by a composite cognitive measure – in more educated persons, with the qualification that this pattern was limited to persons with less than eight years of schooling (Zahodne et al., 2015). Such a pattern of delayed onset with stronger acceleration of memory decline for more educated persons has been found in persons who develop dementia (Hall et al., 2007) and is in agreement with the active cognitive reserve model according to which individuals with a higher cognitive reserve (as measured e.g. by educational attainment) can tolerate higher levels of brain damage

before showing functional decline (Stern, 2002, 2009). Whereas our data lack information about progression to dementia, Zahodne et al. (2015) could show that the pattern was only present in persons who converted to dementia. Although we analyzed data from a population-based sample rather than a sample of (prospective) dementia patients, we cannot rule out that women who developed dementia were included in the sample and affected the shape of the estimated trajectories. Thus, we are hesitant to interpret this finding as a manifestation of cognitive reserve in non-pathological development of memory. An alternative explanation might be specific abilities and motivations of highly educated women of these cohorts, who might have shown practice effects beyond the first assessment of the memory test controlled in our analyses. The generally weak evidence of differently shaped trajectories of cognitive decline in our study is in accordance with earlier findings that educational differences in cognitive decline do not change as persons become older, a finding that has been reported for a range of cognitive domains (Christensen et al., 2001; Muniz-Terrera et al., 2009; van Dijk et al., 2008). Another interesting finding of our analyses was that a higher level of education is associated with a higher level of global cognitive functioning and processing speed at age 65, but not memory.

Further theoretical implications of our findings relate to the idea of cumulative disadvantages and cognitive plasticity. Regarding cumulative disadvantages, the observation of level differences in global cognitive functioning and processing speed at age 65 could lend support to this concept; however, this is only true to the degree that education causally affects these cognitive functions at higher ages and that education and old age cognitive functioning are not merely a consequence of early life cognitive abilities. Analyses with the Lothian Birth Cohort 1936 however showed that education and processing speed were not associated when controlling for intelligence at age 11 and multiple indicators of socioeconomic status and health, while other cognitive domains were still associated with education (Ritchie et al., 2016). Given this explanation for level differences in processing speed, the potential for systematic

educational effects in the three domains considered in our study remains only for the levels of general cognitive functioning (note the discussion about ceiling effects explaining decline differences) and longer maintenance of memory among women with high (vs. medium or low) education. The weak evidence of educational differences in level or decline of cognitive functioning does not contradict the concept of cognitive plasticity, which essentially supposes that a prolonged discrepancy between cognitive capacity and cognitive demands leads to adjustment in cognitive functioning (Lövdén et al., 2010). Assuming that individuals with a higher educational degree are more likely to experience higher demands in their jobs and possibly also in their spare time activities, positive plasticity should already have occurred before old age and substantiate in level differences in old age. Presumably, remembering a number of items is however a very crucial demand that daily life poses equally on more and less educated persons, such that plasticity might have equalized level differences in the memory domain, if they had existed earlier in life. The concept also includes negative plasticity, i.e. adjustment to lower demands by decreasing cognitive functioning when current capacity exceeds demands. With regard to decline, this means that longer maintenance of cognitive abilities in old age relative to a given starting point would presuppose that a drop in cognitive demands associated with age-related changes like retirement or changes in spare time activities is smaller for more (vs. less) educated persons. It would however also be reasonable to assume that a drop in cognitive demands in old age is even higher for more educated persons when demands in the workplace are lacking, or that change of demands relative to cognitive capacity does not differ by education. Also, the assumption that education is positively associated with complexity in the job or spare time activities is plausible yet untested in our analyses. More refined analyses examining more closely whether the potential for cognitive demands was indeed realized by considering e.g. histories of employment and unemployment over the life course could provide a more specific test of the relationship between education and cognitive functioning at higher ages.

There are limitations to our conclusions. Firstly, panel attrition might have affected our results. We aimed to reduce this issue in general by applying an accelerated longitudinal design that allowed to observe higher ages already at earlier waves of the data collection, where attrition is still less frequent compared to following up single cohorts until higher ages (Duncan & Duncan, 2011). This design builds on the assumption that there are no cohort effects in cognitive levels and decline as well as in the effect of predictor variables on cognitive functioning. Since the sample was restricted to persons born in the relatively short interval between 1916 and 1927, we assume that systematic cohort differences are unlikely. However, the healthy participant effect might nevertheless have affected our results if persons with lower levels of functioning were more likely to drop out of the sample, resulting in underestimation of average initial cognitive decline and its acceleration. This might affect less educated groups more strongly because they are more likely to start from lower levels to decline from, resulting in more severe underestimation of decline for less educated groups because they would reach the threshold faster. Consequently, the current study poses a conservative test of the hypothesis that higher education is associated with a longer maintenance of cognitive functioning. Secondly, although educational effects on cognitive functioning can be assumed to be relatively universal, the context of studies, e.g. the quality of education or the degree of educational inequalities at the societal level could influence the findings. In our study, the disturbances of World War II might have affected access and quality of education especially for some of the younger respondents who aimed for medium or high educational degrees, which could lead to an attenuation of educational effects on cognitive functioning. Thirdly, our analyses do not allow to draw causal conclusions. Beneficial effects of education that were observed in the levels of general cognitive functioning and processing speed are subject to the alternative explanation that inherent intellectual abilities of individuals might affect both their educational attainment and their cognitive functioning at higher ages.

From a practical perspective, our results indicate that highly educated persons on average maintain into their late seventies or even eighties levels of global cognitive functioning and processing speed that are comparable or higher than those that persons with low education show at age 65. Subject to the limitations described above, this difference is mostly due to a higher level of the more educated to decline from, rather than to decline differences. The relative stability of educational differences also implies that less educated persons reach lower levels of cognitive functioning earlier. This supports the notion that education still pays off at higher ages, but it also points out that educational inequalities persist into higher ages. Future research could contribute to a better understanding of this association by tapping into the underlying causes and pathways.

2.5 Conclusion

This study found higher levels in global cognitive functioning and processing speed but not memory for more educated persons in a population-based sample of Dutch older adults. In contrast, even taking the potential for different shapes of cognitive decline into account only revealed little evidence for systematic educational differences in cognitive decline in this sample of presumably predominantly normally aging older adults. This suggests that educational effects on maintenance of cognitive functioning in older adults operate rather through providing higher cognitive levels when entering old age rather than through altering the course of decline in old age. To the degree that educational effects on cognitive levels can be demonstrated to be causal, providing cognitively demanding experiences throughout the life course could be beneficial to increase cognitive functioning at higher ages.

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2.7 Appendix

Table 2.A1: Descriptive statistics for the analyses of the Coding Task.

Coding Task	Mean / %	SD	Min	Max	*N/n*
Age	75.6	5.7	65	89	3,557
Male	44.9%	--	0	1	3,557
Coding Task	23.3	6.8	3.0	43.3	3,557
CES-D	8.5	7.4	0	54	3,535
Functional limitations	59.2%	--	0	1	3,500
Chronic diseases	85.7%	--	0	1	3,552
Follow-up (years)[a]	7.7	6.5	0	23	1,066
Education					
Low	39.7%	--	--	--	1,412
Medium	50.2%	--	--	--	1,787
High	10.1%	--	--	--	358

Note. Numbers refer to observations, except for [a] referring to respondents. Deviations from 3,557 observations/ 1,066 individuals are due to missing values on variables not included in the latent growth models.
CES-D = Center for Epidemiological Studies Depression Scale.

Table 2.A2: Descriptive statistics for the analyses of the 15 Words Test.

15 Words Test	Mean / %	SD	Min	Max	*N/n*
Age	75.7	5.7	65	89	3,631
Male	44.5%	--	0	1	3,631
15 Words Test	5.3	2.9	0	15	3,631
CES-D	8.6	7.5	0	54	3,601
Functional limitations	59.8%	--	0	1	3,571
Chronic diseases	85.8%	--	0	1	3,625
Follow-up (years)[a]	7.8	6.5	0	23	1,077
Education					
Low	40.1%	--	--	--	1,753
Medium	49.8%	--	--	--	2,035
High	10.2%	--	--	--	406

Note. Numbers refer to observations, except for [a] referring to respondents. Deviations from 3,631 observations/ 1,077 individuals are due to missing values on variables not included in the latent growth models.
CES-D = Center for Epidemiological Studies Depression Scale.

Table 2.A3: Latent growth models for the 15 Words Test, accounting for the lack of practice at the first assessment.
Unstandardized coefficients with 95% confidence intervals.

	15 Words Test	
Constant	6.95***	[6.48,7.42]
Age	-0.13***	[-0.20,-0.06]
Age squared	-0.00	[-0.01,0.00]
Education (Ref. Low)		
Medium	0.07	[-0.47,0.61]
High	0.42	[-0.45,1.29]
Age x Education (Ref. Low)		
Medium	0.07	[-0.02,0.16]
High	0.07	[-0.08,0.21]
Age squared x Education (Ref. Low)		
Medium	-0.00	[-0.01,0.00]
High	-0.00	[-0.01,0.00]
Lack of practice		
First assessment dummy	-1.30***	[-1.49,-1.11]
Random Components		
Var(Constant)	3.75***	
Var(Age)	0.01***	
Var(Age squared)	0.00***	
Var(Residual)	3.12***	
Wald χ^2-Tests (Age and Age squared)		
Low Education	.000	
Low vs. Medium Education	.325	
Low vs. High Education	.560	
Medium vs. High Education	.828	
AIC	16,394	
BIC	16,480	
$N_{observations}$	3,631	
$N_{respondents}$	1,077	

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion

Table 2.A4: Latent growth models separately for men and women, controlling for practice effects.
Unstandardized coefficients with 95% confidence intervals.

	MMSE		Coding Task		15 Words Test	
	Men	Women	Men	Women	Men	Women
Constant	26.89***	27.07***	21.21***	22.69***	5.90***	7.59***
	[26.23,27.56]	[26.56,27.58]	[19.66,22.76]	[21.58,23.80]	[5.14,6.65]	[6.99,8.18]
Age	0.01	0.04	-0.14	-0.09	-0.17**	-0.13**
	[-0.12,0.13]	[-0.05,0.12]	[-0.37,0.10]	[-0.24,0.05]	[-0.30,-0.05]	[-0.22,-0.04]
Age squared	-0.01***	-0.01***	-0.01	-0.01***	0.00	-0.00*
	[-0.02,-0.01]	[-0.02,-0.01]	[-0.02,0.00]	[-0.02,-0.01]	[-0.00,0.01]	[-0.01,-0.00]
Education (Ref. Low)						
Medium	1.13**	1.33***	5.54***	5.57***	0.14	0.61
	[0.41,1.86]	[0.70,1.96]	[3.89,7.18]	[4.18,6.97]	[-0.67,0.95]	[-0.12,1.35]
High	1.74***	1.60**	8.91***	7.31***	1.29*	-0.14
	[0.74,2.75]	[0.46,2.75]	[6.59,11.23]	[4.57,10.06]	[0.17,2.41]	[-1.51,1.23]
Age x Education (Ref. Low)						
Medium	-0.04	-0.05	-0.09	0.06	0.14	0.03
	[-0.19,0.11]	[-0.17,0.07]	[-0.35,0.17]	[-0.13,0.24]	[-0.00,0.28]	[-0.09,0.15]
High	-0.07	0.01	-0.01	0.17	-0.01	0.34**
	[-0.27,0.13]	[-0.22,0.23]	[-0.34,0.33]	[-0.22,0.56]	[-0.20,0.17]	[0.10,0.58]
Age squared x Education (Ref. Low)						
Medium	0.00	0.01*	-0.00	-0.01	-0.01*	-0.00
	[-0.00,0.01]	[0.00,0.01]	[-0.02,0.01]	[-0.01,0.00]	[-0.01,-0.00]	[-0.01,0.00]
High	0.01	0.01	-0.00	-0.01	-0.00	-0.01*
	[-0.00,0.01]	[-0.00,0.02]	[-0.02,0.01]	[-0.03,0.01]	[-0.01,0.01]	[-0.02,-0.00]
Lack of practice						
First assessment dummy	-0.08	-0.15	-0.32	-0.28	-1.09***	-1.47***
	[-0.33,0.17]	[-0.39,0.09]	[-0.77,0.14]	[-0.69,0.13]	[-1.34,-0.83]	[-1.74,-1.20]
Random Components						
Var(Constant)	0.91	1.86**	26.55***	31.89***	2.89***	3.47***
Var(Age)	0.05***	0.03***	0.01***	0.01**	0.01***	0.01***
Var(Age squared)	0.00***	0.00***	0.00***	0.00***	0.00	0.00***
Var(Residual)	2.97***	3.14***	6.50***	6.18***	2.72***	3.39***

(continued on next page)

Table 2.A4 (continued)

Wald χ^2-Tests (Age and Age squared)						
Low Education	.000	.000	.000	.000	.000	.000
Low vs. Medium Education	.661	.006	.015	.088	.101	.882
Low vs. High Education	.393	.026	.341	.635	.741	.019
Medium vs. High Education	.734	.442	.647	.718	.199	.040
AIC	8,311	11,050	8,995	10,989	7,032	9,240
BIC	8,389	11,130	9,070	11,067	7,108	9,319
$N_{\text{observations}}$	1,812	2,382	1,598	1,959	1,617	2,014
$N_{\text{respondents}}$	555	627	502	564	505	572

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion

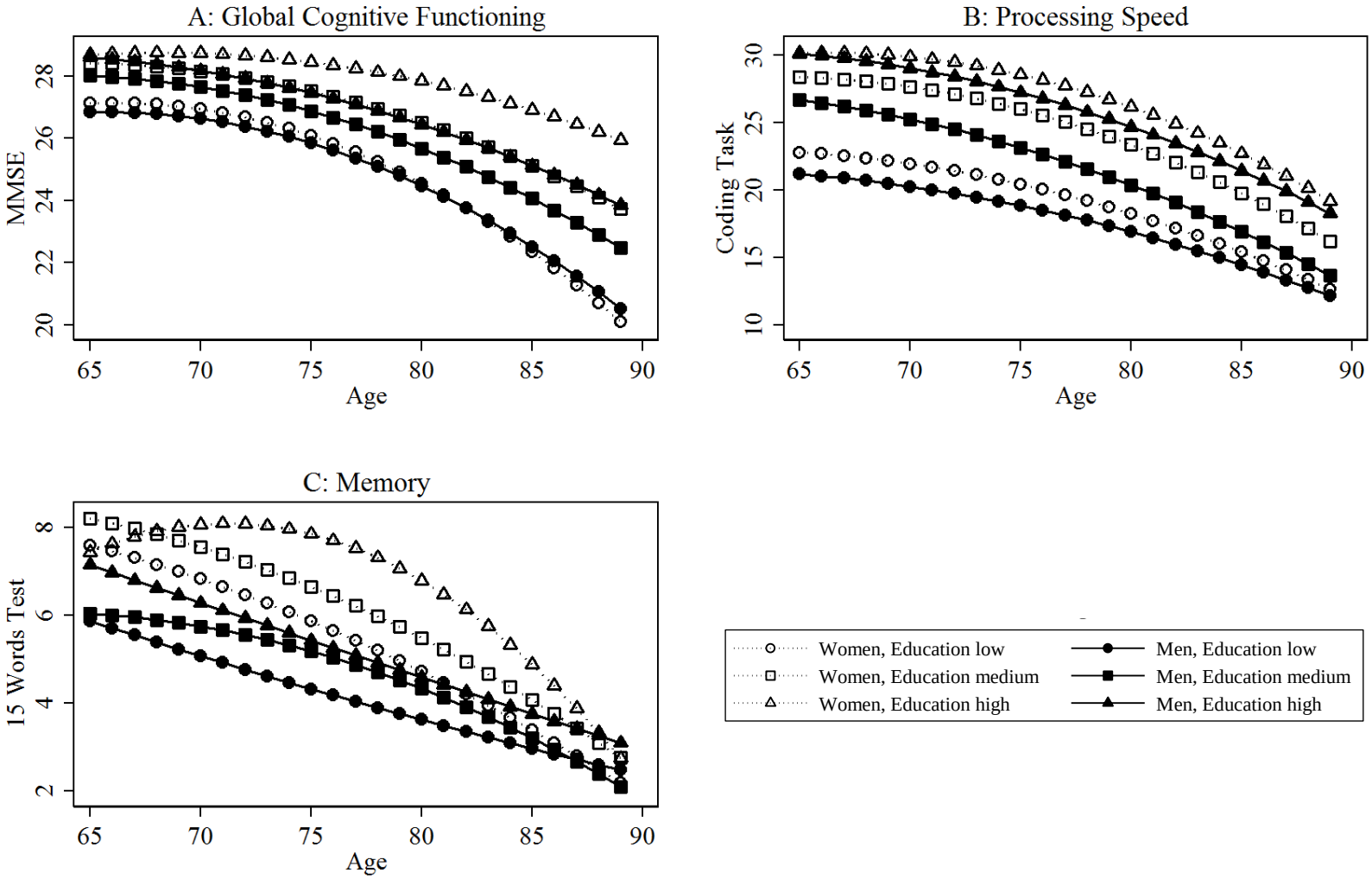

Figure 2.A1: Predicted trajectories for men and women, showing development in three cognitive domains for low, medium, and high education. Graphs show the results from *Table 2.A4* but have been calculated in a joint model interacting all fixed effects involving age and/or education with gender. All graphs show the hypothetical situation in which the respondents already at the first assessment have the same experience with the tests as at the subsequent assessments.

Chapter 3: Spousal Loss and Change in Cognitive Functioning: An Examination of Temporal Patterns and Gender Differences

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Abstract

Objectives: The study investigates whether the disadvantaged position of men in the adverse consequences of widowhood for health and mortality also exists for changes in cognitive health.
Methods: We used data of up to 1,269 men and women aged 65 years and older who participated in the Longitudinal Aging Study Amsterdam in three-yearly assessments between 1992 and 2012 (5,123 person-observations). All were married and without cognitive impairment (MMSE≥24) at baseline and up to 419 lost their spouse. In fixed effects regression models, the effect of spousal loss on change in four domains of cognitive functioning was estimated independently of age-related cognitive change.
Results: For women, a robust temporary decrease was found in the second year after spousal loss in the reasoning domain, but not in global cognitive functioning, processing speed, or memory. No robust effects were found for men.
Discussion: Considering that only one cognitive domain was affected and effects were temporary, cognitive functioning seems rather robust to the experience of spousal loss. Despite men having often been reported to be in a disadvantaged position in other health domains, our analyses indicate no such pattern for cognitive functioning.

3.1 Introduction

Losing the spouse is a stressful but common experience at higher ages. It is associated with depressive symptoms and major depressive disorder (Onrust & Cuijpers, 2006; Vable, Subramanian, Rist, & Glymour, 2015), nutritional risk and weight loss (Stahl & Schulz, 2014), sleep problems (van de Straat & Bracke, 2015), poor immune response (Phillips et al., 2006), and mortality (Moon, Kondo, Glymour, & Subramanian, 2011; Shor et al., 2012). Through increased stress, increased depressive symptoms and changes in the social network, spousal loss may also affect the cognitive functioning of older adults. However, previous research brought about mixed findings (compare Aartsen, van Tilburg, and Smits (2005), Karlamangla et al. (2009), Mousavi-Nasab, Kormi-Nouri, Sundström, and Nilsson (2012), and Vidarsdottir et al. (2014)). One reason might be that these studies often focused on widowhood as a state of being, rather than on the timing of the spousal loss (cf. Aartsen et al., 2005; Vidarsdottir et al., 2014). Since the negative effects of spousal loss on health outcomes tend to attenuate over time (Sasson & Umberson, 2014; Shor et al., 2012), it is important to take the timing of spousal loss into account. Another reason can be that few studies actually observed how cognitive functioning *changes* following spousal loss (see Karlamangla et al. (2009) for an exception). In the present study, we examine changes in cognitive functioning associated with spousal loss and potential gender differences in this association. The presented analyses address the role of time since and time to the loss of the spouse and thus allow conclusions about the recovery from and anticipation of the loss.

3.1.1 Why spousal loss might affect cognitive functioning

Various explanations exist for a potential effect of spousal loss on cognitive functioning. Firstly, losing a spouse is typically considered to be one of the most stressful life events (Rosnick, Small, & Burton, 2010; Vidarsdottir et al., 2014). According to neuropsychological research,

stress is detrimental for the brain because it may result in dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis which might lead to cognitive impairment (Lupien, Maheu, Tu, Fiocco, & Schramek, 2007; McEwen & Sapolsky, 1995), particularly to impaired memory (Shields, Sazma, McCullough, & Yonelinas, 2017), but also to lower MMSE-scores (Leng et al., 2013). Another explanation is that losing a spouse leads to increased levels of depressive symptoms, which in turn lead to lower levels of cognitive functioning (Aartsen et al., 2005). This assumption is supported by empirical findings showing that more depressive symptoms at one time point were associated with faster subsequent decline in processing speed (Comijs, Jonker, Beekman, & Deeg, 2001), global cognitive functioning (Wilson, Mendes de Leon, Bennett, Bienias, & Evans, 2004), and increased risk of developing Mild Cognitive Impairment (Barnes, Alexopoulos, Lopez, Williamson, & Yaffe, 2006). Thirdly, losing a spouse often means the loss of one of the most important social contacts and thus the loss of a vital source of cognitive stimulation, which could further accelerate cognitive decline (van Gelder et al., 2006).

Empirical studies of losing a spouse mainly concentrated on effects on memory. For example, research among 35 to 85 year-olds from Sweden found that episodic but not semantic memory declined faster over a 5-year period in constantly widowed compared to constantly married persons (Mousavi-Nasab et al., 2012). Another investigation drawing on an earlier version of the data we use, but utilizing a less rigorous methodological approach and a smaller widower-sample, reported that older adults aged 60 years and older who became widowed during a 6-year period showed faster decline in memory than those staying married (Aartsen et al., 2005). Opposing our expectations, memory performance was independent of time since spousal loss in this investigation. In an Icelandic study, no effects of spousal loss were found on a number of cognitive functions, except for executive functioning among women (Vidarsdottir et al., 2014). None of these studies examined within person change in marital status and cognitive functioning, i.e. whether cognitive functioning changes as a consequence of losing the spouse.

Rather, they compared married and widowed individuals. Reported effects might thus be confounded even though the authors tried to rule out alternative explanations. We know only one previous study that observed change in *both* marital status *and* cognitive functioning. It showed that loss (vs. no change or gain) of a partner was not associated with change in total cognition score and episodic memory (Karlamangla et al., 2009).

3.1.2 Gender differences in effects of spousal loss on cognitive change

In their work on gender differences in the health risks of widowhood, Stroebe, Stroebe, and Schut (2001) suggested that "men suffer relatively higher consequences of partner loss than do women" (also see Stroebe and Stroebe (1983)). Such effects have been found for some health-related outcomes, e.g. frailty (Trevisan et al., 2016), grip strength (Clouston, Lawlor, & Verdery, 2014), and mortality (Moon et al., 2011). While previous research paid much attention to gender differences in effects of widowhood on subjective well-being, especially depressive symptoms (e.g. Lee & DeMaris, 2007; Lee, DeMaris, Bavin, & Sullivan, 2001; Lee, Willetts, & Seccombe, 1998; Nieboer, Lindenberg, & Ormel, 1999; Schaan, 2013; Umberson, Wortman, & Kessler, 1992), less is known about objective measures of cognitive functioning, specifically cognitive change in older adults.

Various lines of arguments can explain why effects of spousal loss on cognitive change might be stronger for men than for women. One is that the loss of the spouse forces particularly men from older cohorts with predominantly traditional gender roles to take over responsibility for traditionally female typed household tasks that they typically dislike or are unfamiliar with (Leopold & Skopek, 2018; South & Spitze, 1994; Umberson et al., 1992; Utz, Reidy, Carr, Nesse, & Wortman, 2004). This might cause stress that is detrimental to cognitive functioning. In contrast, women might experience less stress since they seem to derive self-confidence and satisfaction from carrying out traditionally male tasks (van den Hoonaard, 2009). Another reason might be men's stronger reliance on their spouses as confidants and for maintenance of

social contacts (Cornwell, Schumm, Laumann, & Graber, 2009; Stroebe & Stroebe, 1983). The resultant change in cognitive stimulation, the loss of access to social support, and the associated increase in stress and depressive symptoms may have consequences for cognitive functioning. Additionally, the generally smaller size of men's (vs. women's) confidant networks (Cornwell et al., 2009) and associated lower availability of social support in the case of spousal loss (Kalmijn, 2012) could be a reason why men suffer stronger consequences than women.

Empirical evidence for a male disadvantage in the consequences of spousal loss on cognitive change is however weak. In their studies, neither Aartsen et al. (2005) nor Mousavi-Nasab et al. (2012) found evidence of a significant gender difference in the effect of widowhood on memory decline. Vidarsdottir et al. (2014) even report that women but not men showed temporarily lower executive functioning in the 2-year interval after their spouse's death compared to the constantly married. No such effect was however found for memory and processing speed. Similarly, another study also reported a negative effect of widowhood for women but not for men (Vable et al., 2015). Interestingly however, episodic memory was lower in the 2-year interval *before* but not in the 2-year interval *after* onset of widowhood (compared to the constantly married), a finding that might be due to stressful caregiving or anticipation of spousal death.

3.1.3 This study

In the current study, we examine whether losing the spouse is associated with negative cognitive change over and above age-related cognitive change and whether there are gender differences in the strength of the effect of losing the spouse on cognitive change. To that end, we study the change in cognitive functioning in older adults who lost their spouse during the study using gender-stratified fixed effects regression models. Our analytical focus is on cognitive change at multiple time points after spousal loss. Additionally, observations before spousal loss allow inferences about pre-loss changes, e.g. due to stressful caregiving or anticipation.

3.2 Data and methods

3.2.1 Sample

The Longitudinal Aging Study Amsterdam (LASA; Hoogendijk et al., 2016; Huisman et al., 2011) is an ongoing longitudinal study among older adults in the Netherlands, initially based on a nationally representative sample. Data collection started in 1992/3 with respondents aged 55 to 85 years, followed up every three to four years thereafter. Respondents were randomly selected from municipal registries, with an oversampling of men and the oldest participants. Trained interviewers visited the respondents at home and conducted face-to-face main interviews, during which respondents were asked to fill in a drop-off questionnaire and to participate in a subsequent medical interview. Besides interviewer training, medical interviewers had to have a relevant professional background.

In the present study, we used data from seven waves (t_1=1992/3, t_2=1995/6, t_3=1998/9, t_4=2001/2, t_5=2005/6, t_6=2008/9, t_7=2011/2). Not least due to the assessment of some cognitive tests in main interviews and others in medical interviews, missing values differed by cognitive domain. Aiming to reduce sample selectivity, we included all observations providing sufficient information on a given outcome in our analyses, rather than using a joint sample with information present on all outcomes, at the cost of having different samples across outcomes. All analytical samples contain respondents who were recruited at t_1, and information on these respondents from follow-up interviews until t_7, i.e. a period of up to nearly 20 years. Respondents' observations were considered from age 65 onwards in order to focus on the age most relevant for age-related cognitive decline. Among these observations, we identified the first observation in which a respondent provided valid information on a specific cognitive outcome and refer to this observation as the person- and outcome-specific baseline. The number of respondents married at baseline differed by outcome (n_{MMSE}=1,766, $n_{\text{Coding Task}}$=1,566, $n_{\text{Raven Matrices}}$=1,667, $n_{\text{15 Words Test}}$=1,593). Respondents with their baseline marital status being *never married*, *divorced* or *registered partnership* were not included in the analyses, the latter

due to lacking information on the date of the partners' death. From the remaining sample, those transitioning from marriage to divorce (up to four respondents, depending on the outcome), those being married or in a registered partnership after widowhood (five respondents), and those providing inconclusive information on their marital status (two respondents) were excluded as well as those showing signs of dementia at baseline (MMSE<24; up to 162 respondents). For technical reasons, observations with missing information on the variable measuring time to spousal loss (four respondents) and respondents providing only one observation of valid data (up to 341 respondents) also had to be excluded. The resulting analytical sample for MMSE consisted of 5,123 person-observations from 1,269 respondents, 419 of whom experienced spousal loss during the observed period (Coding Task: 4,248/1,100/368, Raven Matrices: 4,289/1,189/398, 15 Words Test: 4,319/1,112/376, respectively).

3.2.2 Variables

Outcome variables

Fluid cognitive abilities are more prone to age-related change than crystallized abilities (Lindenberger & Baltes, 1997) and might thus respond more sensitively to spousal loss. Thus, outcome variables comprise a widely used measure of global cognitive functioning and three measures of fluid cognitive abilities.

Global cognitive functioning (main interview) was measured using the Mini Mental State Examination (MMSE), a measure that comprises orientation in time and space, registration, attention, recall, language and visuospatial abilities (Folstein, Folstein, & McHugh, 1975). MMSE scores can range from 0 to 30, with higher values indicating better functioning. Internal consistency is relatively low, representing the multidimensionality of the measure (Tombaugh & McIntyre, 1992).

Processing speed (medical interview) was assessed with a coding task that has been described by Piccinin and Rabbitt (1999). The test contains rows of letters, with each of the rows having

an empty row below it. A key is provided with the test, showing pairs of letters that belong together. Respondents were asked to match as many letters that correspond to the letters in the upper rows as possible by naming the corresponding lower-row letter. We used the mean number of matches over three trials of one minute per assessment (observed range: 3.3 to 44.3), which correlated highly (Cronbach's $\alpha \geq .96$ for each wave).

Reasoning (main interview t_1-t_3, medical interview t_4-t_6, not assessed at t_7) was measured using subsets A and B of the Raven Colored Progressive Matrixes (Raven, 1995), a nonverbal test of abstract reasoning. The test consists of 24 visual patterns that all miss a part of the pattern. From six alternatives printed underneath the patterns, respondents should choose the one that completes the pattern. Correct choices scored one point, thus the maximum score is 24. As intended, the items of both subsets as well as the subsets themselves increased in difficulty (van den Heuvel & Smits, 1994).

Memory (medical interview) was assessed with the delayed recall score of the 15 Words Test, a Dutch version of the Auditory Verbal Learning Test (Rey, 1964; Saan & Deelman, 1986). In three trials, respondents learn 15 words that they should recall after each trial. The delayed recall score is the number of correctly recalled words after a distraction period of 20 minutes following the learning phase. Correct recalls scored one point, thus up to 15 points could be achieved.

Predictor variables

Time to spousal loss indicates the duration between the cognitive assessment (main or medical interview, depending on the outcome variable) and the date of the spouse's death. The date of the spouse's death was obtained from municipality registries, if available, and during the interviews otherwise. Durations were calculated using information on the month and year of these events, with 0 indicating that the cognitive assessment took place in the month in which the spouse deceased. For simplicity, we refer to the 1st year (months 0-11), 2nd year (months

12-23), 3rd year (months 24-35) and 4th and subsequent years (months ≥36) *after* spousal loss and the last year (months -12 to -1), 2nd year (months -13 to -24), 3rd year (months -25 to -36) and 4th and previous years (months ≤-37) *before* spousal loss. The variable is a constant for those who did not lose their spouse during the observed period.

Age at the main or medical interview of each wave (depending on the outcome variable), was measured for both respondents losing and respondents not losing their spouse. To reduce collinearity when estimating effects of squared age, we centered the age variables to their respective mean value of all observations in a sample.

Gender was observed in the main interview at t_1 and was coded 0 for women and 1 for men.

3.2.3 Analytical approach

Change in cognitive functioning associated with spousal loss was analyzed using fixed effects regression for panel data. This method uses the within-person change over time in the predictor variables (e.g. marital status) to predict within-person change in the outcome variable (i.e. cognitive functioning). Consequently, time-constant differences between persons are ruled out as confounding variables. The risk of time-varying confounding is low since changes in e.g. respondents' health conditions or health behaviors might cause changes in their cognitive functioning but are unlikely to be the driving forces behind the spouses' deaths. Time since spousal loss relative to the reference period (i.e. the 4th year and previous years before the spouse's death) was modeled flexibly with multiple dummy variables, allowing to depict different trajectories, including anticipatory effects, effects of spousal loss and recovery thereof. Accounting for age allows to disentangle loss-associated change from age-associated change in the outcomes. Since age was measured for both those losing and those not losing their spouse, the coefficients for age represent general age-related change rather than change of the spousal loss-population only. We split the analyses by gender and tested gender differences in additional joint models for women and men where we interacted gender with both age and time to spousal

loss. Since depressive symptoms constitute a potential pathway from spousal loss to change in cognitive functioning (Barnes et al., 2006; Comijs et al., 2001; Sasson & Umberson, 2014; Vable et al., 2015; Wilson et al., 2004), depressive symptoms are not controlled to allow detection of the total effect of spousal loss on cognitive change. Several sensitivity analyses were carried out. All analyses were conducted using Stata Version 14 (StataCorp, 2015).

3.3 Results

3.3.1 Descriptive results

In the largest sample (MMSE), 55% of observations were from male respondents and 38% from respondents losing their spouse during the observation period, with an average age of about 76 years (*Table 3.1;* see *Tables 3.A1-3.A3* in the Appendix for the other samples). About nine educational years were attained on average, and almost all observations were from respondents indicating Dutch ethnicity. In terms of health, at least one functional limitation was present in about 56% of observations, with an average of two chronic diseases and about eight depressive symptoms (CES-D Scale; Radloff, 1977). There were no substantial differences between the samples on these variables. Average age was higher among observations from male compared to female respondents (except for Coding Task and 15 Words Test) and among those losing their spouse compared to those not losing their spouse. While almost 70% of observations were from male respondents among those not losing the spouse, only about 35% were from male respondents among those losing their spouse. Average cognitive functioning scores were higher among female (vs. male) respondents (reversed pattern for Raven Matrices) and those not losing their spouse (vs. those losing their spouse; except for 15 Words Test).

Table 3.1: Descriptive statistics for analysis of MMSE.

MMSE	Total	Women	Men	*p* for gender diff.
Whole sample				
$N_{observations}$	*5,123*	*2,299*	*2,824*	
MMSE, mean ± SD	27.26 ± 2.60	27.34 ± 2.53	27.19 ± 2.66	.033
Age, mean ± SD	75.98 ± 6.60	75.71 ± 6.58	76.21 ± 6.62	.007
Male, *n* (%)	2,824 (55.1)	--	--	--
Losing spouse, *n* (%)	1,958 (38.2)	1,288 (56.0)	670 (23.7)	<.001
Not losing spouse				
$N_{observations}$	*3,165*	*1,011*	*2,154*	
MMSE, mean ± SD	27.35[a] ± 2.60	27.53[b] ± 2.47	27.26[c] ± 2.66	.006
Age, mean ± SD	74.86[d] ± 6.05	74.04[e] ± 5.75	75.24[f] ± 6.14	<.001
Male, *n* (%)	2,154 (68.1[g])	--	--	
Losing spouse				
$N_{observations}$	*1,958*	*1,288*	*670*	
MMSE, mean ± SD	27.11[a] ± 2.59	27.20[b] ± 2.57	26.96[c] ± 2.64	.054
Age, mean ± SD	77.80[d] ± 7.05	77.01[e] ± 6.89	79.32[f] ± 7.11	<.001
Male, *n* (%)	670 (34.2[g])	--	--	--
Time to spousal loss, *n* (%)				.019
≤ -37 months	688 (35.1)	424 (32.9)	264 (39.4)	
-36 to -25 months	127 (6.5)	83 (6.4)	44 (6.6)	
-24 to -13 months	129 (6.6)	83 (6.4)	46 (6.9)	
-12 to -1 months	101 (5.2)	69 (5.4)	32 (4.8)	
0 to 11 months	131 (6.7)	81 (6.3)	50 (7.5)	
12 to 23 months	126 (6.4)	80 (6.2)	46 (6.9)	
24 to 35 months	107 (5.5)	72 (5.6)	35 (5.2)	
≥ 36 months	549 (28.0)	396 (30.8)	153 (22.8)	

Note. SD = standard deviation; *p*-values were derived from two-sided *t*-tests for mean values, *p*-tests for shares, and a χ^2-test for the distribution of observations by time to spousal loss and gender, respectively.
Identical superscript letters indicate significant differences between those losing spouse and those not losing spouse with $p < .05$.
Observations belong to 1,269 respondents (533 female, 736 male), 419 of whom lost their spouse (270 female, 149 male).

3.3.2 Fixed effects regression models

Results from fixed effects models for women and men are displayed in *Tables 3.2 and 3.3*, respectively, and in *Figure 3.1*. The variable time to spousal loss indicates the difference between the mean cognitive functioning score of the reference period (i.e. the 4th year and previous years before the spouse's death) and the mean score of the period under consideration, accounting for age-related changes. For both men and women, the terms for linear and squared age jointly indicate accelerated age-related decline in MMSE, Coding Task and 15 Words Test, but linear decline of Raven Matrices.

MMSE

With some fluctuation in the time before spousal loss, MMSE-scores among women declined in the second year after the spouse's death ($B_{+2nd\ year}$=-0.68, p=.047) and returned towards the level of the reference period (i.e. the 4th year and previous years before the spouse's death) thereafter. The observed difference between the reference period and the second year after spousal loss corresponds to the age-related cognitive decline occurring during 4.20 years after the mean age of 75.98 years ($B_{age\ linear}$ * age + $B_{age\ squared}$ * age^2 = -0.12 * age + (-0.01) * age^2 = -0.68; solved for age: age=4.20 years, with age=0 equaling the average age in the joint MMSE-sample for both women and men due to centering). The pattern for men was comparable to that of women, but the decrease of 0.52 MMSE-points in the second year after spousal loss (corresponding to 2.77 years of age-related decline after the mean age) failed statistical significance (p=.313). No significant gender differences for loss-associated change in MMSE were found (all $p\geq$.233).

Coding Task

Statistically controlling for age, the Coding Task-scores showed a minor and non-significant decline for women ($B_{-3rd\ year}$=-0.73, p=.076; $B_{-2nd\ year}$=-0.79, p=.122) and an increase for men ($B_{-3rd\ year}$=1.34, p=.012) *before* the loss of the spouse, with a significant gender-difference in change between the reference period and the third year before spousal loss (p=.002). More importantly however, neither men nor women showed a change in Coding Task immediately before the spouse's death or thereafter in comparison to the reference period, but rather tended to show a slight increase in the longer run (women: $B_{+\ 4th\ and\ subs.\ years}$=0.72, p=.106; men: $B_{+\ 4th\ and\ subs.\ years}$=0.84, p=.186).

Table 3.2: Main effects of time to spousal loss and age predicting cognitive functioning in older women.
Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Women	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.12***	[-0.15,-0.10]	.081	-0.37***	[-0.41,-0.33]	**.014**	-0.13***	[-0.17,-0.10]	**.044**	-0.13***	[-0.16,-0.10]	.325
Age^2	-0.01***	[-0.01,-0.00]	.376	-0.01***	[-0.02,-0.01]	.131	-0.00	[-0.01,0.00]	.254	-0.00***	[-0.01,-0.00]	.188
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	-0.14	[-0.68,0.40]	.233	-0.73	[-1.53,0.08]	**.002**	0.10	[-0.52,0.73]	.655	-0.09	[-0.60,0.43]	.783
- 2nd year	-0.37	[-1.00,0.27]	.532	-0.79	[-1.79,0.21]	.071	-0.69	[-1.45,0.07]	.481	-0.12	[-0.75,0.51]	.972
- 1st year	-0.00	[-0.58,0.58]	.573	-0.05	[-0.86,0.75]	.345	-0.06	[-0.81,0.69]	.248	0.09	[-0.55,0.73]	.594
+ 1st year	-0.34	[-0.92,0.23]	.252	0.28	[-0.72,1.28]	.701	-0.24	[-0.91,0.44]	.887	0.08	[-0.57,0.72]	.757
+ 2nd year	-0.68*	[-1.35,-0.01]	.802	0.04	[-0.95,1.02]	.353	-1.54***	[-2.31,-0.78]	**.005**	0.21	[-0.44,0.87]	.827
+ 3rd year	-0.03	[-0.64,0.57]	.669	0.24	[-0.83,1.30]	.613	-0.29	[-1.09,0.52]	.539	-0.32	[-1.22,0.59]	.575
≥ + 4th year	-0.19	[-0.73,0.34]	.764	0.72	[-0.15,1.58]	.868	-0.57	[-1.30,0.16]	.781	0.09	[-0.51,0.68]	.549
Constant	27.70***	[27.50,27.90]	--	25.06***	[24.77,25.34]	--	18.18***	[17.95,18.40]	--	6.64***	[6.45,6.83]	--
$N_{observations}$	2,299			1,847			1,872			1,889		
$N_{individuals}$	533			451			492			456		
AIC	8861.42			8363.48			7941.33			7475.16		
BIC	8913.08			8413.18			7991.15			7525.05		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
Bold letters highlight gender differences with $p < .05$

Table 3.3: Main effects of time to spousal loss and age predicting cognitive functioning in older men.
Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Men	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.16***	[-0.18,-0.13]	.081	-0.44***	[-0.48,-0.40]	**.014**	-0.18***	[-0.21,-0.15]	**.044**	-0.11***	[-0.14,-0.09]	.325
Age^2	-0.01***	[-0.01,-0.00]	.376	-0.01***	[-0.01,-0.00]	.131	-0.00	[-0.00,0.00]	.254	-0.00*	[-0.00,-0.00]	.188
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	0.37	[-0.28,1.02]	.233	1.34*	[0.29,2.40]	**.002**	-0.14	[-1.02,0.74]	.655	0.04	[-0.72,0.80]	.783
- 2nd year	-0.08	[-0.74,0.59]	.532	0.65	[-0.55,1.86]	.071	-0.28	[-1.13,0.56]	.481	-0.10	[-0.71,0.50]	.972
- 1st year	0.26	[-0.46,0.98]	.573	0.66	[-0.59,1.92]	.345	-0.76	[-1.68,0.16]	.248	-0.20	[-1.06,0.66]	.594
+ 1st year	0.22	[-0.56,1.00]	.252	0.62	[-0.80,2.04]	.701	-0.32	[-1.21,0.57]	.887	0.23	[-0.53,1.00]	.757
+ 2nd year	-0.52	[-1.54,0.49]	.802	0.76	[-0.42,1.94]	.353	0.19	[-0.74,1.12]	**.005**	0.11	[-0.55,0.77]	.827
+ 3rd year	-0.27	[-1.15,0.61]	.669	0.65	[-0.56,1.87]	.613	0.17	[-1.05,1.39]	.539	0.05	[-0.86,0.96]	.575
≥ + 4th year	-0.04	[-0.86,0.78]	.764	0.84	[-0.41,2.09]	.868	-0.73	[-1.59,0.13]	.781	-0.18	[-0.84,0.47]	.549
Constant	27.45***	[27.33,27.58]	--	24.32***	[24.16,24.47]	--	18.43***	[18.33,18.54]	--	5.14***	[5.03,5.24]	--
$N_{observations}$	2,824			2,401			2,417			2,430		
$N_{individuals}$	736			649			697			656		
AIC	10982.47			10912.61			9776.80			8985.41		
BIC	11035.99			10964.66			9828.92			9037.57		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
Bold letters highlight gender differences with $p < .05$.

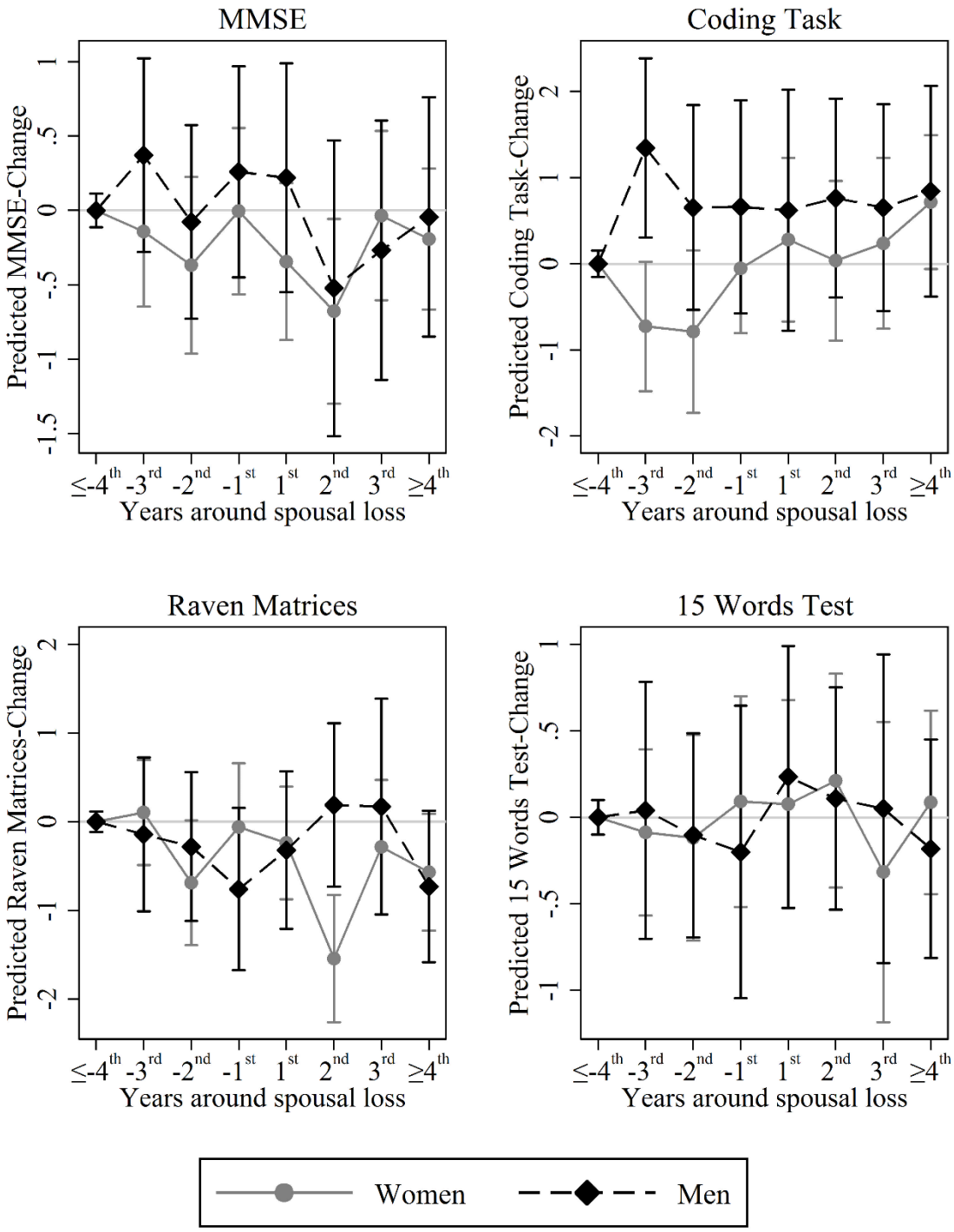

Figure 3.1: Development of four domains of cognitive functioning over time relative to spousal loss, separately for women and men. Scores for the reference period (4th and previous years before spousal loss) were set to 0 for the graphs to show the change relative to the reference period.

Coefficients and 95%-confidence intervals are from joint fixed effects models for women and men, with age and time to spousal loss interacted with gender. The graph was generated holding age constant at the respective sample mean.

Raven Matrices

As concerns the Raven Matrices-scores, there was some fluctuation before and after spousal loss, with the strongest but temporary difference to the reference period in the second year after spousal loss among women ($B_{+2nd\ year}$=-1.54, p<.001), corresponding to 11.85 years of age-related cognitive decline ($B_{+2nd\ year}$ / $B_{age\ linear}$ = -1.54 / -0.13 = 11.85 years; only $B_{age\ linear}$ is considered here since $B_{age\ squared}$=0.00). No such effect was observed among men (p=.005 for gender difference in the second year after spousal loss), but they tended to show weaker losses compared to the reference period – corresponding to about 4 years of age-related decline – in the year before spousal loss ($B_{last\ year\ before}$=-0.76, p=.105) and in the longer run ($B_{+\ 4th\ and\ subs.\ years}$=-0.73, p=.095).

15 Words Test

For both men and women, the 15 Words Test-scores only showed some minor and statistically non-significant fluctuation around the scores of the reference period (all $p \geq .494$).

3.3.3 Sensitivity analyses

Bonferroni adjustment

Responding to α-error inflation in multiple testing, we consulted Bonferroni-adjusted significance tests. Since a single significant coefficient out of three or four coefficients would lead to the rejection of the null-hypothesis of no effect before or after spousal loss, respectively, correction for multiple testing is appropriate (Perneger, 1998). Corrected critical p-values are .05/3=.016 and .05/4=.012, respectively (Bender & Lange, 2001). Given the adjusted p-values, differences in MMSE are no longer significant, but conclusions regarding Processing Speed and Raven Matrices remain unchanged.

Practice effects

Practice effects were controlled by adding to the original models a dummy variable identifying the first observation in which a given cognitive test was completed. A statistically significant practice effect (i.e. lower cognitive scores at the first assessment than thereafter) was only found for the 15 Words Test. The conclusions from this sensitivity analysis were generally in accord with our original results (see *Tables 3.A4* and *3.A5* in the Appendix).

Linear change before and after spousal loss

More long-term trends in cognitive change following spousal loss were examined in fixed effects models with two metric variables indicating years before or after spousal loss, respectively, and being 0 otherwise (range: -19 to 19 years). Except for substantially very weak positive developments on the Coding Task before (women: B=0.08, p=.083; men: B=0.12, p=.034) and after spousal loss (women: B=0.09, p=.070), neither time before nor time after spousal loss was related to cognitive functioning when age was accounted for (see *Tables 3.A6* and *3.A7* in the Appendix). Additional F-tests did not reveal differences between pre- and post-loss slopes, and these differences did not differ by gender (all $p \geq .05$).

ln(31-MMSE)-transformation

To adjust the skewed distribution of MMSE-residuals, a ln(31-MMSE)-transformation was used. The decrease in MMSE in the second year after spousal loss for women failed statistical significance in analyses of the ln(31-MMSE)-transformed variable ($B_{+2\text{nd year}}$=0.13 p=.068; note that higher scores represent lower functioning on the transformed variable; see *Table 3.A8* in the Appendix).

Random effects models

In addition to the original fixed effects models, random effects models were inspected. Hausman tests (Andreß, Golsch, & Schmidt, 2013) indicated endogeneity for MMSE (women

and men) and for Raven Matrices (men), a situation in which fixed effects models are to be preferred over the more efficient but potentially inconsistent random effects models. Random effect models specified analogous to the fixed effects models, but with educational years as an additional time-constant control variable, confirmed the conclusions from the fixed effects models, except that the increase in Coding Task scores in the third year before spousal loss among men was smaller and of borderline significance (B=1.02; p=.05; Tables *3.A9* and *3.A10* in the Appendix).

Attrition analysis

Selective panel attrition directly before or after spousal loss might cause underestimation of the association between spousal loss and cognitive change if the likelihood of attrition in association with spousal loss is larger for those experiencing a larger decline in cognitive functioning than for those experiencing a smaller cognitive decline. It is in the nature of panel attrition that the change in cognitive functioning potentially causing non-participation cannot be observed. However, it is possible to use information measured at wave t, including time to spousal loss, cognitive functioning, and an interaction term of both variables (plus additional control variables) to estimate the likelihood of attrition for other reasons than the respondents' own death at wave t_{+1}. Average marginal effects from logistic regression models indicated that each standard deviation lower in the respective cognitive function in the third and second year before spousal loss was associated with an approximately 10 percentage points larger increase in the likelihood of attrition compared to the fourth and previous years before spousal loss (see *Appendix 3.A1*). Since the analyses concern attrition at the next wave (i.e. about 3 years later), this suggests that the likelihood of attrition in the years directly following spousal loss is indeed larger for those widowed respondents with lower cognitive functioning. However, it cannot be concluded if these are also the ones that experience the largest change in their cognitive functioning after spousal loss.

3.4 Discussion

We investigated whether losing the spouse is associated with a negative cognitive change over and above the effect of age-related cognitive change using gender-stratified models. To that end, fixed effects regression models controlling for age and time-constant confounders were applied to analyze loss-associated change in general cognitive functioning, processing speed, reasoning, and memory. There was little evidence of spousal loss being associated with cognitive change, except that women on average showed a robust temporary decrease in reasoning scores in the second year after spousal loss. No robust effect was observed for men. Evidence that "widowhood effects" might occur in anticipation of the spouse's death (Vable et al., 2015) was weak. For men, an increase in processing speed was observed in the third year before the spouse's death; however, the effect should not be overemphasized because it was relatively small and only of borderline significance in the more efficient random effects model. Across all four cognitive domains, men did not show a more disadvantaged pattern of cognitive change associated with spousal loss, which is in contrast to earlier research on gender differences in other domains of health, but in line with previous findings on cognitive functioning (e.g. Aartsen et al., 2005). Instead, our findings provide support to earlier findings that women's cognitive functioning may be more negatively affected than men's (Vidarsdottir et al., 2014). Comparable to our findings, Vidarsdottir et al. (2014) report the two-year-interval following spousal loss as the critical period. Furthermore, the absence of an effect of change in marital status on memory change in our study is in contrast to reports of faster memory decline among widowed (vs. married) persons (Aartsen et al., 2005; Mousavi-Nasab et al., 2012), a diverging pattern that has previously been found by Karlamangla et al. (2009).

Implications

Implications from our study are based on the overall finding that there were few detrimental effects of spousal loss independently of age-related cognitive decline, and those observed were

temporary and found in women only. Firstly, this might imply that the changes in potential pathways caused by spousal loss, i.e. in stress, depressive symptoms, and cognitive stimulation, might not be severe enough to trigger changes in cognitive functioning. As regards the potential for cognitive stimulation, Kalmijn (2012) found that although women but not men experience increased support from family, friends and neighbors when becoming widowed, neither women nor men experience a *decrease* in support or contact frequency from these groups (Kalmijn, 2012), calling into question both the idea that spousal loss leads to a decrease of cognitive stimulation and does so more for men. Secondly, it might be the case that changes in the potential pathways are not universally related to cognitive change, e.g. stress was not associated with processing speed in a small-scale study among adults of a wide age range (VonDras et al., 2005), and only a selection of cognitive domains was found to be associated with depressive symptoms in another study (Dotson et al., 2008). Yet others found that memory predicted change in depressive symptoms rather than depressive symptoms predicting memory change (Jajodia & Borders, 2011). Thirdly, it might be the case that the suggested pathways actually apply, and the absence or temporariness of effects could be explained by accompanying beneficial processes. The concept of cognitive plasticity (Lövdén, Bäckman, Lindenberger, Schaefer, & Schmiedek, 2010) suggests that a decrease in cognitive functioning following spousal loss may induce a mismatch between environmental demands and supply of cognitive functioning, which can trigger a (re-)adaptation of cognitive functioning to the demands. The complete absence of an effect of spousal loss in other domains suggests that adaptation can either happen quickly or that beneficial consequences counteract the negative consequences of spousal loss. For example, learning to carry out tasks that were previously taken care of by the spouse may foster cognitive functioning, comparably to the beneficial effects of acquiring another language (Bak, Nissan, Allerhand, & Deary, 2014). Similarly, one reason for the absence of a disadvantage for men might be that carrying out disliked household tasks causes new stimulation and only mild stress, which has been suggested to stimulate cognitive

functioning (Comijs, van den Kommer, Minnaar, Penninx, & Deeg, 2011). Fourthly, effect heterogeneity based on other characteristics than gender might explain our findings. For example, spousal loss might be less anticipated, more stressful and more detrimental to cognitive functioning if the deceased partner was younger or in better health. Furthermore, personality characteristics or spousal care activities of the bereaved person might be important factors causing variation in response to spousal loss. To further explore this possibility, future research might want to depart from the examination of gender differences and instead focus on other characteristics of individuals, couples, and circumstances of the spouse's death that can affect the reaction to losing a spouse (Carr, 2004).

Limitations

A first limitation of our study is the relatively small number of observations from men in some periods before and after spousal loss, making it harder to detect statistically significant effects for men or gender differences. However, the substantial patterns suggested that the absence of effects was not due to low power, and a statistically significant gender difference was observed in reasoning nevertheless. Secondly, we report yearly changes in cognitive functioning at the aggregate level even though individuals were surveyed in intervals of about three years. Since a short temporary effect of spousal loss would remain undiscovered if three-year intervals were examined, we preferred to use variation in the date of the spouse's death to be able to report on shorter time intervals, at the cost of not observing each individual in each interval. Although it is plausible that spousal loss triggers more long-term cognitive decline during many years after the loss, this was not supported by sensitivity analyses of linear change during up to 19 years after spousal loss. Thirdly, we exploited all available information by using all observations with valid data, at the cost of analyzing slightly different samples for different outcomes. However, this is unlikely to be the driver behind different findings since these samples did not differ substantially by age, gender, spousal loss, education or health. Fourthly, our findings might

underestimate effects of spousal loss on cognitive decline if the likelihood of panel attrition associated with spousal loss is higher for those showing larger loss-associated cognitive decline. Due to the very nature of panel attrition, cognitive decline resulting from spousal loss could not be examined as a predictor of panel attrition. However, a lower level of cognitive functioning at the previous wave was associated with a higher likelihood of attrition after spousal loss in our attrition analyses. This finding is in line with – but not proof of – the idea that those who experience stronger cognitive decline in association with spousal loss are more likely to drop out of the panel. The resulting potential underestimation of the effect of spousal loss on cognitive decline might be a reason for the reported absence of associations for men and in some cognitive domains for women in our study. However, potential underestimation was not severe enough to mask effects of spousal loss on reasoning for women.

3.5 Conclusion

So far, few studies examined the association between losing the spouse and cognitive change. This population-based study suggests that cognitive functioning is on average hardly affected by spousal loss in the subsequent years. At least for the cognitive domain, this contradicts the notion that becoming widowed accelerates the progression of frailty in older adults. In contrast, older adults' cognitive functioning seems to be rather resilient against this very stressful experience, which might possibly be explained by cognitive plasticity.

3.6 References

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3.7 Appendix

Table 3.A1: Descriptive statistics for analysis of Coding Task.

Coding Task	Total	Women	Men	*p* for gender diff.
Whole sample				
$N_{observations}$	*4,248*	*1,847*	*2,401*	
Coding Task, mean ± SD	24.33 ± 6.72	24.72 ± 6.91	24.02 ± 6.56	<.001
Age, mean ± SD	75.96 ± 6.29	75.78 ± 6.24	76.10 ± 6.33	.092
Male, *n* (%)	2,401 (56.5)	--	--	
Losing spouse, *n* (%)	1,604 (37.8)	1,033 (55.9)	571 (23.8)	<.001
Not losing spouse				
$N_{observations}$	2,644	814	1,830	
Coding Task, mean ± SD	24.76[a] ± 6.60	25.33[b] ± 6.61	24.50[c] ± 6.58	.003
Age, mean ± SD	74.96[d] ± 5.76	74.55[e] ± 5.57	75.14[f] ± 5.83	.016
Male, *n* (%)	1,830 (69.2[g])	--	--	
Losing spouse				
$N_{observations}$	*1,604*	*1,033*	*571*	
Coding Task, mean ± SD	23.62[a] ± 6.87	24.24[b] ± 7.11	22.49[c] ± 6.27	<.001
Age, mean ± SD	77.61[d] ± 6.76	76.74[e] ± 6.56	79.19[f] ± 6.86	<.001
Male, *n* (%)	571 (35.6[g])	--	--	
Time to spousal loss, *n* (%)				.004
≤ -37 months	603 (37.6)	363 (35.1)	240 (42.0)	
-36 to -25 months	104 (6.5)	66 (6.4)	38 (6.7)	
-24 to -13 months	102 (6.4)	63 (6.1)	39 (6.8)	
-12 to -1 months	84 (5.2)	58 (5.6)	26 (4.6)	
0 to 11 months	102 (6.4)	64 (6.2)	38 (6.7)	
12 to 23 months	102 (6.4)	58 (5.6)	44 (7.7)	
24 to 35 months	83 (5.2)	53 (5.1)	30 (5.3)	
≥ 36 months	424 (26.4)	308 (29.8)	116 (20.3)	

Note. SD = standard deviation; *p*-values were derived from two-sided *t*-tests for mean values, *p*-tests for shares, and a χ^2-test for the distribution of observations by time to spousal loss and gender, respectively.
Identical superscript letters indicate significant differences between those losing spouse and those not losing spouse with $p < .05$.
Observations belong to 1,100 respondents (451 female, 649 male), 368 of whom lost their spouse (232 female, 136 male).

Table 3.A2: Descriptive statistics for analysis of Raven Matrices.

Raven Matrices	Total	Women	Men	*p* for gender diff.
Whole sample				
$N_{observations}$	*4,289*	*1,872*	*2,417*	
Raven Matrices, mean ± SD	18.16 ± 3.75	17.94 ± 3.76	18.32 ± 3.73	.001
Age, mean ± SD	75.22 ± 6.29	74.84 ± 6.19	75.51 ± 6.35	<.001
Male, *n* (%)	2,417 (56.4)	--	--	
Losing spouse, *n* (%)	1,639 (38.2)	1,058 (56.5)	581 (24.0)	<.001
Not losing spouse				
$N_{observations}$	*2,650*	*814*	*1,836*	
Raven Matrices, mean ± SD	18.35[a] ± 3.69	17.94 ± 3.66	18.53[c] ± 3.68	<.001
Age, mean ± SD	74.19[d] ± 5.79	73.37[e] ± 5.50	74.55[f] ± 5.88	<.001
Male, *n* (%)	1,836 (69.3[g])	--	--	
Losing spouse				
$N_{observations}$	*1,639*	*1,058*	*581*	
Raven Matrices, mean ± SD	17.84[a] ± 3.83	17.95 ± 3.84	17.65[c] ± 3.80	.133
Age, mean ± SD	76.88[d] ± 6.69	75.97[e] ± 6.45	78.54[f] ± 6.81	<.001
Male, *n* (%)	581 (35.5[g])	--	--	
Time to spousal loss, *n* (%)				.021
≤ -37 months	653 (39.8)	396 (37.4)	257 (44.2)	
-36 to -25 months	116 (7.1)	74 (7.0)	42 (7.2)	
-24 to -13 months	116 (7.1)	71 (6.7)	45 (7.8)	
-12 to -1 months	92 (5.6)	62 (5.9)	30 (5.2)	
0 to 11 months	105 (6.4)	65 (6.1)	40 (6.9)	
12 to 23 months	102 (6.2)	65 (6.1)	37 (6.4)	
24 to 35 months	85 (5.2)	56 (5.3)	29 (5.0)	
≥ 36 months	370 (22.6)	269 (25.4)	101 (17.4)	

Note. SD = standard deviation; *p*-values were derived from two-sided *t*-tests for mean values, *p*-tests for shares, and a χ^2-test for the distribution of observations by time to spousal loss and gender, respectively.
Identical superscript letters indicate significant differences between those losing spouse and those not losing spouse with $p < .05$.
Observations belong to 1,189 respondents (492 female, 697 male), 398 of whom lost their spouse (254 female, 144 male).

Table 3.A3: Descriptive statistics for analysis of 15 Words Test.

15 Words Test	Total	Women	Men	*p* for gender diff.
Whole sample				
$N_{observations}$	*4,319*	*1,889*	*2,430*	
15 Words Test, mean ± SD	5.65 ± 2.97	6.48 ± 3.08	5.00 ± 2.71	<.001
Age, mean ± SD	76.06 ± 6.35	75.86 ± 6.29	76.22 ± 6.39	.066
Male, *n* (%)	2,430 (56.3)	--	--	
Losing spouse, *n* (%)	1,648 (38.2)	1,063 (56.3)	585 (24.1)	<.001
Not losing spouse				
$N_{observations}$	2,671	826	1,845	
15 Words Test, mean ± SD	5.66 ± 2.98	6.81[b] ± 3.23	5.14[c] ± 2.70	<.001
Age, mean ± SD	75.04[d] ± 5.80	74.63[e] ± 5.65	75.22[f] ± 5.86	.016
Male, *n* (%)	1,845 (69.1[g])	--	--	
Losing spouse				
$N_{observations}$	*1,648*	*1,063*	*585*	
15 Words Test, mean ± SD	5.64 ± 2.95	6.23[b] ± 2.93	4.58[c] ± 2.69	<.001
Age, mean ± SD	77.72[d] ± 6.84	76.81[e] ± 6.60	79.37[f] ± 6.96	<.001
Male, *n* (%)	585 (35.5[g])	--	--	
Time to spousal loss, *n* (%)				.003
≤ -37 months	610 (37.0)	370 (34.8)	240 (41.0)	
-36 to -25 months	107 (6.5)	66 (6.2)	41 (7.0)	
-24 to -13 months	106 (6.4)	67 (6.3)	39 (6.7)	
-12 to -1 months	83 (5.0)	57 (5.4)	26 (4.4)	
0 to 11 months	109 (6.6)	67 (6.3)	42 (7.2)	
12 to 23 months	108 (6.6)	61 (5.7)	47 (8.0)	
24 to 35 months	84 (5.1)	54 (5.1)	30 (5.1)	
≥ 36 months	441 (26.8)	321 (30.2)	120 (20.5)	

Note. SD = standard deviation; *p*-values were derived from two-sided *t*-tests for mean values, *p*-tests for shares, and a χ^2-test for the distribution of observations by time to spousal loss and gender, respectively.
Identical superscript letters indicate significant differences between those losing spouse and those not losing spouse with $p < .05$.
Observations belong to 1,112 respondents (456 female, 656 male), 376 of whom lost their spouse (237 female, 139 male).

Table 3.A4: Main effects of time to spousal loss and age predicting cognitive functioning in older women, accounting for practice effects. Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Women	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.12***	[-0.15,-0.09]	.121	-0.37***	[-0.42,-0.32]	**.013**	-0.13***	[-0.17,-0.09]	.054	-0.16***	[-0.19,-0.13]	.692
Age^2	-0.01***	[-0.01,-0.00]	.343	-0.01***	[-0.02,-0.01]	.136	-0.00	[-0.01,0.00]	.240	-0.00**	[-0.01,-0.00]	.260
First assessment	0.15	[-0.13,0.44]	--	0.03	[-0.43,0.48]	--	-0.00	[-0.41,0.41]	--	-1.02***	[-1.37,-0.67]	--
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	-0.13	[-0.67,0.41]	.229	-0.72	[-1.53,0.09]	**.002**	0.10	[-0.52,0.73]	.652	-0.18	[-0.71,0.35]	.778
- 2nd year	-0.37	[-1.01,0.26]	.509	-0.79	[-1.79,0.21]	.073	-0.69	[-1.45,0.07]	.474	-0.14	[-0.74,0.47]	.982
- 1st year	0.01	[-0.57,0.60]	.653	-0.05	[-0.86,0.76]	.334	-0.06	[-0.81,0.69]	.230	-0.05	[-0.69,0.58]	.782
+ 1st year	-0.30	[-0.88,0.27]	.272	0.29	[-0.73,1.31]	.694	-0.24	[-0.94,0.46]	.851	-0.18	[-0.84,0.48]	.610
+ 2nd year	-0.64	[-1.31,0.03]	.847	0.04	[-0.96,1.04]	.342	-1.54***	[-2.33,-0.76]	**.006**	-0.04	[-0.69,0.61]	.989
+ 3rd year	0.01	[-0.61,0.62]	.634	0.24	[-0.85,1.34]	.609	-0.29	[-1.10,0.53]	.560	-0.61	[-1.51,0.30]	.533
≥ + 4th year	-0.17	[-0.70,0.37]	.832	0.72	[-0.16,1.60]	.854	-0.57	[-1.31,0.17]	.747	-0.08	[-0.67,0.50]	.708
Constant	27.67***	[27.47,27.88]	--	25.05***	[24.75,25.36]	--	18.18***	[17.93,18.42]	--	6.82***	[6.62,7.02]	--
$N_{observations}$	2,299			1,847			1,872			1,889		
$N_{individuals}$	533			451			492			456		
AIC	8862.07			8365.47			7943.33			7430.68		
BIC	8919.47			8420.68			7998.68			7486.11		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where variables for age and time to spousal loss were interacted with gender.
Bold letters highlight gender differences with $p < .05$.

Table 3.A5: Main effects of time to spousal loss and age predicting cognitive functioning in older men, accounting for practice effects. Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Men	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.14***	[-0.17,-0.11]	.121	-0.45***	[-0.50,-0.41]	**.013**	-0.16***	[-0.20,-0.13]	.054	-0.15***	[-0.18,-0.13]	.692
Age^2	-0.01***	[-0.01,-0.00]	.343	-0.01***	[-0.01,-0.00]	.136	-0.00	[-0.00,0.00]	.240	-0.00*	[-0.00,-0.00]	.260
First assessment	0.41***	[0.17,0.65]	--	-0.28	[-0.64,0.09]	--	0.29	[-0.02,0.60]	--	-1.02***	[-1.26,-0.77]	--
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	0.41	[-0.24,1.05]	.229	1.32*	[0.26,2.39]	**.002**	-0.12	[-1.00,0.76]	.652	-0.05	[-0.80,0.70]	.778
- 2nd year	-0.07	[-0.72,0.59]	.509	0.64	[-0.57,1.85]	.073	-0.29	[-1.13,0.56]	.474	-0.15	[-0.73,0.43]	.982
- 1st year	0.24	[-0.49,0.96]	.653	0.66	[-0.60,1.92]	.334	-0.79	[-1.71,0.13]	.230	-0.20	[-1.05,0.65]	.782
+ 1st year	0.29	[-0.49,1.08]	.272	0.57	[-0.85,1.99]	.694	-0.29	[-1.18,0.61]	.851	0.08	[-0.68,0.84]	.610
+ 2nd year	-0.47	[-1.48,0.54]	.847	0.72	[-0.46,1.90]	.342	0.22	[-0.71,1.15]	**.006**	-0.04	[-0.68,0.59]	.989
+ 3rd year	-0.20	[-1.08,0.69]	.634	0.58	[-0.63,1.80]	.609	0.21	[-1.02,1.44]	.560	-0.19	[-1.14,0.76]	.533
≥ + 4th year	-0.03	[-0.85,0.79]	.832	0.82	[-0.42,2.07]	.854	-0.74	[-1.60,0.12]	.747	-0.25	[-0.90,0.40]	.708
Constant	27.38***	[27.24,27.51]	--	24.37***	[24.20,24.54]	--	18.37***	[18.25,18.50]	--	5.33***	[5.22,5.43]	--
$N_{observations}$	2,824			2,401			2,417			2,430		
$N_{individuals}$	736			649			697			656		
AIC	10970.52			10911.81			9773.70			8894.44		
BIC	11029.98			10969.64			9831.60			8952.39		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where variables for age and time to spousal loss were interacted with gender.
Bold letters highlight gender differences with $p < .05$.

Table 3.A6: Main effects of time before and after spousal loss measured in years (piecewise regression) and age predicting cognitive functioning in older women. Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Women	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.14***	[-0.17,-0.10]	.259	-0.40***	[-0.45,-0.34]	.092	-0.15***	[-0.20,-0.11]	.320	-0.13***	[-0.17,-0.10]	.529
Age^2	-0.01***	[-0.01,-0.00]	.304	-0.01***	[-0.02,-0.01]	.138	-0.00	[-0.01,0.00]	.201	-0.00***	[-0.01,-0.00]	.306
Time to spousal loss												
Before loss	-0.02	[-0.08,0.03]	.125	0.08	[-0.01,0.16]	.567	-0.02	[-0.08,0.05]	.833	0.02	[-0.04,0.08]	.840
After loss	0.04	[-0.02,0.11]	.262	0.09	[-0.01,0.19]	.661	0.02	[-0.06,0.09]	.305	-0.01	[-0.07,0.05]	.944
Constant	18.07	[-20.62,56.76]	--	-48.73	[-112.27,14.81]	--	18.36	[-32.87,69.59]	--	0.96	[-38.62,40.55]	--
$N_{observations}$	2,299			1,847			1,872			1,889		
$N_{individuals}$	533			451			492			456		
AIC	8852.73			8366.63			7953.31			7467.17		
BIC	8875.69			8388.72			7975.45			7489.35		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
Bold letters highlight gender differences with $p < .05$.

Table 3.A7: Main effects of time before and after spousal loss measured in years (piecewise regression) and age predicting cognitive functioning in older men. Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

Men	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.16***	[-0.19,-0.14]	.259	-0.46***	[-0.50,-0.41]	.092	-0.18***	[-0.21,-0.15]	.320	-0.12***	[-0.14,-0.09]	.529
Age^2	-0.01***	[-0.01,-0.00]	.304	-0.01***	[-0.01,-0.01]	.138	-0.00	[-0.00,0.00]	.201	-0.00**	[-0.01,-0.00]	.306
Time to spousal loss												
Before loss	0.05	[-0.03,0.12]	.125	0.12*	[0.01,0.22]	.567	-0.01	[-0.07,0.06]	.833	0.03	[-0.03,0.09]	.840
After loss	-0.02	[-0.13,0.08]	.262	0.05	[-0.09,0.20]	.661	-0.06	[-0.17,0.06]	.305	-0.01	[-0.08,0.07]	.944
Constant	10.58	[-84.91,106.08]	--	-104.03	[-246.70,38.63]	--	66.43	[-36.48,169.35]	--	-14.14	[-84.74,56.45]	--
$N_{observations}$	2,824			2,401			2,417			2,430		
$N_{individuals}$	736			649			697			656		
AIC	10974.42			10900.04			9775.99			8976.09		
BIC	10998.20			10923.17			9799.15			8999.28		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
Bold letters highlight gender differences with $p < .05$.

Table 3.A8: Main effects of time to spousal loss and age predicting ln(31-MMSE)-transformed global cognitive functioning scores in older women and men.
Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from fixed effects regression models.

ln(31-MMSE)	***Women***		***Men***		
	B	95%-CI	*B*	95%-CI	*p* for gender diff.
Age	0.03***	[0.02,0.03]	0.03***	[0.03,0.04]	.184
Age^2	0.00***	[0.00,0.00]	0.00**	[0.00,0.00]	.297
Time to spousal loss (ref. ≤ - 4th year)					
- 3rd year	0.01	[-0.11,0.13]	-0.02	[-0.17,0.13]	.732
- 2nd year	0.05	[-0.09,0.20]	-0.01	[-0.18,0.16]	.575
- 1st year	0.02	[-0.12,0.17]	-0.05	[-0.24,0.15]	.571
+ 1st year	0.07	[-0.07,0.21]	-0.02	[-0.20,0.15]	.408
+ 2nd year	0.13	[-0.01,0.28]	0.10	[-0.10,0.30]	.787
+ 3rd year	-0.00	[-0.15,0.15]	0.09	[-0.08,0.25]	.439
≥ + 4th year	0.07	[-0.06,0.19]	0.04	[-0.13,0.21]	.819
Constant	1.03***	[0.99,1.07]	1.10***	[1.07,1.12]	--
$N_{observations}$	2,299		2,824		
$N_{individuals}$	533		736		
AIC	2459.40		2711.25		
BIC	2511.07		2764.77		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
Bold letters highlight gender differences with $p < .05$.
Note that higher values on the ln(31-MMSE)-variable indicate lower global cognitive functioning due to the transformation.

Table 3.A9: Main effects of time to spousal loss, age, and educational years predicting cognitive functioning in older women.
Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from random effects regression models.

Women	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.11***	[-0.14,-0.09]	.146	-0.38***	[-0.42,-0.33]	**.046**	-0.14***	[-0.17,-0.11]	**.020**	-0.14***	[-0.16,-0.11]	.218
Age^2	-0.01***	[-0.01,-0.00]	.211	-0.01***	[-0.02,-0.01]	.067	-0.00	[-0.01,0.00]	.102	-0.00***	[-0.01,-0.00]	.200
Education (yrs)	0.23***	[0.18,0.27]	**.008**	0.89***	[0.70,1.08]	.072	0.47***	[0.39,0.55]	**.019**	0.17***	[0.08,0.25]	.744
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	-0.15	[-0.63,0.33]	.792	-0.70	[-1.49,0.09]	**.008**	0.10	[-0.50,0.70]	.683	-0.11	[-0.61,0.39]	.860
- 2nd year	-0.43	[-1.00,0.13]	.364	-0.84	[-1.84,0.16]	.060	-0.61	[-1.34,0.12]	.668	-0.21	[-0.78,0.37]	.615
- 1st year	0.00	[-0.48,0.49]	.683	-0.04	[-0.83,0.74]	.500	-0.03	[-0.72,0.66]	.390	0.02	[-0.61,0.64]	.442
+ 1st year	-0.38	[-0.91,0.14]	.776	0.32	[-0.66,1.31]	.941	-0.22	[-0.85,0.42]	.849	0.11	[-0.47,0.70]	.875
+ 2nd year	-0.71*	[-1.31,-0.11]	.864	0.14	[-0.82,1.09]	.464	-1.39***	[-2.12,-0.66]	**.011**	0.25	[-0.37,0.86]	.876
+ 3rd year	-0.04	[-0.56,0.49]	.386	0.21	[-0.83,1.25]	.750	-0.30	[-1.03,0.43]	.333	-0.30	[-1.14,0.54]	.669
≥ + 4th year	-0.25	[-0.65,0.16]	.702	0.78	[-0.04,1.60]	.686	-0.39	[-1.02,0.25]	.722	0.11	[-0.40,0.61]	.364
No loss	-0.42**	[-0.69,-0.14]	.595	-0.48	[-1.56,0.60]	.294	-0.94***	[-1.47,-0.40]	**.031**	0.08	[-0.40,0.56]	.577
Constant	25.88***	[25.41,26.34]	--	17.36***	[15.63,19.09]	--	14.51***	[13.73,15.30]	--	5.05***	[4.31,5.79]	--
$N_{observations}$	2,299			1,847			1,872			1,889		
$N_{individuals}$	533			451			492			456		
σ_u	1.39			5.40			2.54			2.09		
σ_e	1.89			2.67			2.35			2.01		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
The joint model additionally included the main effect of gender.
Bold letters highlight gender differences with $p < .05$

Table 3.A10: Main effects of time to spousal loss, age, and educational years predicting cognitive functioning in older men.
Unstandardized coefficients, 95%-confidence intervals, and *p*-values for gender differences from random effects regression models.

Men	***MMSE***			***Coding Task***			***Raven Matrices***			***15 Words Test***		
	B	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.	*B*	95%-CI	*p* for gender diff.
Age	-0.14***	[-0.15,-0.12]	.146	-0.43***	[-0.47,-0.39]	**.046**	-0.18***	[-0.21,-0.16]	**.020**	-0.11***	[-0.13,-0.10]	.218
Age^2	-0.00**	[-0.01,-0.00]	.211	-0.01***	[-0.01,-0.00]	.067	0.00	[-0.00,0.00]	.102	-0.00*	[-0.00,-0.00]	.200
Education (yrs)	0.14***	[0.10,0.18]	**.008**	0.68***	[0.56,0.81]	.072	0.36***	[0.30,0.41]	**.019**	0.15***	[0.10,0.20]	.744
Time to spousal loss (ref. ≤ - 4th year)												
- 3rd year	-0.04	[-0.63,0.55]	.792	1.02	[-0.00,2.05]	**.008**	-0.11	[-0.94,0.71]	.683	-0.04	[-0.72,0.65]	.860
- 2nd year	-0.06	[-0.65,0.53]	.364	0.63	[-0.54,1.79]	.060	-0.37	[-1.18,0.44]	.668	-0.00	[-0.55,0.54]	.615
- 1st year	0.17	[-0.45,0.79]	.683	0.44	[-0.77,1.64]	.500	-0.56	[-1.51,0.39]	.390	-0.40	[-1.21,0.42]	.442
+ 1st year	-0.24	[-0.98,0.50]	.776	0.24	[-1.15,1.63]	.941	-0.32	[-1.16,0.53]	.849	0.04	[-0.67,0.74]	.875
+ 2nd year	-0.61	[-1.58,0.36]	.864	0.70	[-0.43,1.83]	.464	0.11	[-0.79,1.01]	**.011**	0.18	[-0.44,0.80]	.876
+ 3rd year	-0.46	[-1.26,0.35]	.386	0.45	[-0.70,1.59]	.750	0.37	[-0.77,1.50]	.333	-0.04	[-0.88,0.79]	.669
≥ + 4th year	-0.40	[-1.10,0.30]	.702	0.46	[-0.72,1.65]	.686	-0.58	[-1.33,0.16]	.722	-0.26	[-0.84,0.33]	.364
No loss	-0.52***	[-0.82,-0.23]	.595	0.32	[-0.71,1.35]	.294	-0.06	[-0.65,0.52]	**.031**	-0.10	[-0.54,0.34]	.577
Constant	26.29***	[25.79,26.78]	--	16.74***	[15.20,18.28]	--	14.65***	[13.85,15.44]	--	3.55***	[2.95,4.15]	--
$N_{observations}$	2,824			2,401			2,417			2,430		
$N_{individuals}$	736			649			697			656		
σ_u	1.54			5.04			2.53			1.76		
σ_e	1.97			2.75			2.17			1.80		

Note. * $p < .05$, ** $p < .01$, *** $p < .001$.
AIC = Akaike's Information Criterion, *BIC* = Bayesian Information Criterion.
p-values for gender differences were obtained from a joint model for women and men where all variables were interacted with gender.
The joint model additionally included the main effect of gender.
Bold letters highlight gender differences with $p < .05$.

Appendix 3.1: Attrition analysis

Attrition due to other reasons than death was analyzed using logistic regression models.[1] The independent variables age, time to spousal loss, cognitive functioning, gender and education were used to predict attrition at the next wave (i.e. about 3 years later). The average marginal effects from these models indicated that aging by 1 year on average increases the likelihood of attrition for other reasons than death by 1 percentage point. Furthermore, in the second year after spousal loss, the likelihood of non-death attrition was increased by about 10 percentage points compared to the reference period (i.e. the fourth and previous years before the loss of their spouse; (see *Table 3.A11*)), indicating a higher probability of panel attrition at the next wave about 3 years later.

Adding interaction terms of time to spousal loss and the respective cognitive outcome variable to the models allowed further insights. Specifically, the marginal effects of the respective cognitive variable on the likelihood of attrition for each period before/after spousal loss (reference category: fourth and previous years before spousal loss) are depicted in *Figure 3.A1*. The negative marginal effects of the cognitive functions in the third and second year before spousal loss on the probability of non-death attrition (except for MMSE) suggest that higher cognitive functioning reduces the likelihood of attrition about 3 years later, i.e. in the period directly following the loss of the spouse. The change in the likelihood of non-death attrition predicted for the third year (compared to the reference period in the fourth and previous years) before spousal loss is about 1 to 3 percentage points smaller for each 1 unit increase in the respective cognitive function. This corresponds to a 7 to 12 percentage points smaller likelihood of attrition for each standard deviation higher on the respective cognitive measure (additional

[1] Respondents' own death was only available as a reason for attrition for the time after but not for the time before the spouse's death. This made it impossible to estimate differences in the likelihood of attrition due to respondents' death before and after the spouse's death. The current attrition analyses excluded respondents that ever drop out of the sample due to their own death.

calculations).[2] Since the attrition variable is a measure of non-participation at the next interview, i.e. about 3 years later, this suggests that the likelihood of participating in the study in the first and second year after spousal loss is especially reduced for those widowed persons with low cognitive functioning.

[2] These calculations assumed average age and education, fixing gender at female; effects for gender fixed at male are the same or slightly smaller.

Table 3.A11: Main effects of age, time to spousal loss, cognitive functioning, gender and educational years on panel attrition for reasons other than death. Average marginal effects and 95%-confidence intervals from logistic regression models.

	MMSE		***Coding Task***		***Raven Matrices***		***15 Words Test***	
	B	95%-CI	*B*	95%-CI	*B*	95%-CI	*B*	95%-CI
Age	0.01***	[0.01,0.01]	0.01***	[0.01,0.01]	0.01***	[0.01,0.01]	0.01***	[0.01,0.01]
Time to spousal loss (ref. ≤ - 4th year)								
- 3rd year	0.07*	[0.00,0.15]	0.07	[-0.02,0.16]	0.07	[-0.00,0.14]	0.04	[-0.05,0.12]
- 2nd year	-0.03	[-0.09,0.03]	-0.01	[-0.08,0.06]	0.00	[-0.06,0.06]	0.02	[-0.05,0.10]
- 1st year	0.03	[-0.05,0.10]	0.02	[-0.06,0.11]	0.01	[-0.07,0.08]	0.00	[-0.08,0.09]
+ 1st year	-0.01	[-0.07,0.04]	0.07	[-0.02,0.17]	0.08	[-0.01,0.16]	0.06	[-0.03,0.16]
+ 2nd year	0.09*	[0.00,0.17]	0.12*	[0.02,0.22]	0.08	[-0.00,0.17]	0.11*	[0.02,0.21]
+ 3rd year	0.06	[-0.03,0.14]	0.04	[-0.04,0.13]	0.04	[-0.05,0.12]	0.05	[-0.04,0.15]
≥ + 4th year	0.02	[-0.03,0.06]	0.02	[-0.03,0.06]	0.01	[-0.03,0.05]	0.02	[-0.03,0.07]
No loss	0.07***	[0.04,0.10]	0.08***	[0.04,0.11]	0.09***	[0.06,0.12]	0.06**	[0.02,0.10]
MMSE	-0.02***	[-0.03,-0.02]	--	--	--	--	--	--
Coding Task	--	--	-0.01***	[-0.01,-0.01]	--	--	--	--
Raven Matrices	--	--	--	--	-0.01***	[-0.02,-0.01]	--	--
15 Words Test	--	--	--	--	--	--	-0.02***	[-0.02,-0.01]
Male	-0.02	[-0.04,0.01]	-0.03*	[-0.06,-0.00]	-0.02	[-0.05,0.00]	-0.03	[-0.06,0.00]
Education (yrs)	-0.00	[-0.01,0.00]	0.00	[-0.00,0.01]	-0.00	[-0.00,0.00]	-0.00	[-0.01,0.00]
$N_{observations}$	2,951		2,689		2,703		2,670	
$N_{individuals}$	1,269		1,100		1,189		1,112	
McFadden Pseudo R^2	0.13		0.11		0.11		0.10	

Note: * $p < .05$, ** $p < .01$, *** $p < .001$.

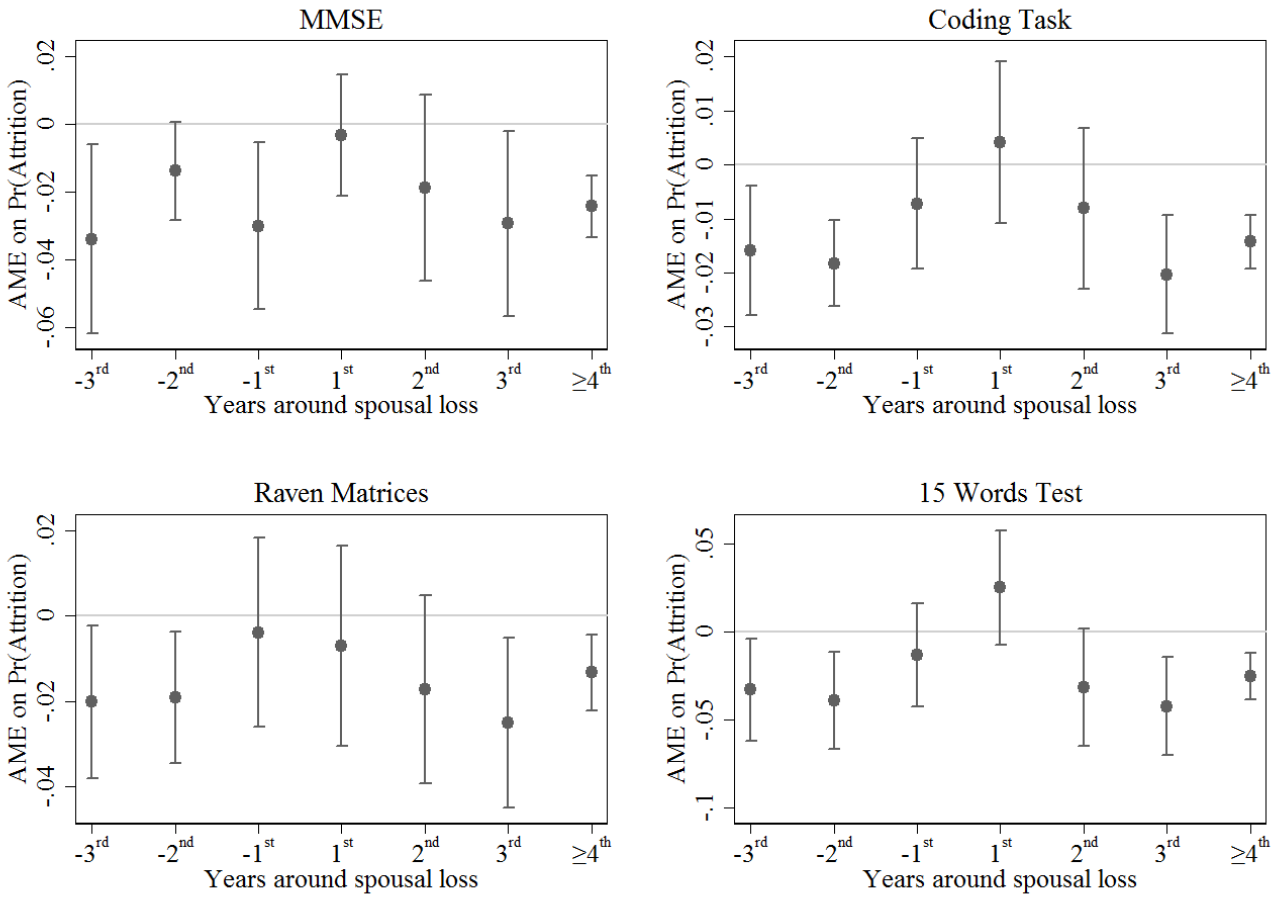

Figure 3.A1: Average marginal effects of the respective cognitive function on the probability of attrition at the next wave for other reasons than the respondents' own death compared to the fourth and previous years before spousal loss, for different periods before and after spousal loss. Estimates are from models presented in *Table 3.A11* with additional interaction terms for the respective cognitive function and time to spousal loss included.

Chapter 4: Cognitive Functioning Among Dutch Older Adults: Do Neighborhood Socioeconomic Status and Urbanity Matter?

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Abstract

Positive associations of neighborhood socioeconomic characteristics and older adults' cognitive functioning have been demonstrated in previous studies, but overall results have been mixed and evidence from European countries and particularly the Netherlands is scarce. We investigated the effects of socioeconomic status (SES) and urbanity of neighborhoods on four domains of cognitive functioning in a sample of 985 Dutch older adults aged 65–88 years from the Longitudinal Aging Study Amsterdam. Besides cross-sectional level differences in general cognitive functioning, processing speed, problem solving and memory, we examined cognitive decline over a period of six years. Growth models in a multilevel framework are used to simultaneously assess levels and decline of cognitive functioning. In models not adjusting for individual SES, we found some evidence of higher levels of cognitive functioning in neighborhoods with a higher SES. In the same models, urbanity generally showed positive or inversely U-shaped associations with levels of cognitive functioning. Overall, effects of neighborhood urbanity remained significant when adjusting for individual SES. In contrast, level differences by neighborhood SES were largely explained by the respondents' individual SES. This suggests that neighborhood SES does not influence levels of cognitive functioning beyond the fact that individuals with a similar SES tend to self-select into neighborhoods with a corresponding SES. No evidence of systematically faster decline in neighborhoods with lower SES or lower degrees of urbanity was found. The findings suggest that neighborhood SES has

no independent effect on older adults cognitive functioning in the Netherlands. Furthermore, the study reveals that neighborhood urbanity should be considered a determinant of cognitive functioning. This finding is in line with theoretical approaches that assume beneficial effects of exposure to complex environments on cognitive functioning. We encourage further investigations into the effect of urbanity in other contexts before drawing firm conclusions.

4.1 Introduction

Recent years have seen a strong interest in the effects of neighborhood environments – typically understood as relatively small areas surrounding a person's place of residence as defined by administrative, geographical or subjective boundaries – on health. A sizeable amount of findings indicating better health (as assessed in terms of self-rated health, depression, cardiovascular and cardiometabolic risk factors, and mortality) in socioeconomically better off neighborhoods is challenged by many studies reporting null-findings (Julien, Richard, Gauvin, & Kestens, 2012; Kim, 2008; Leal & Chaix, 2011; Mair, Diez Roux, & Galea, 2008; Pickett & Pearl, 2001; Richardson, Westley, Gariepy, Austin, & Nandi, 2015; Riva, Gauvin, & Barnett, 2007). Especially for older adults, maintaining cognitive functioning is an important health outcome, influencing their quality of life, and costs of care (Hertzog, Kramer, Wilson, & Lindenberger, 2009). It has been argued that the neighborhood context is especially meaningful for older adults because of their higher vulnerability and because they presumably spend more time in their neighborhoods than younger people, especially after retirement (e.g. Robert & Li, 2001).

A recent review concludes that the majority of studies report that older adults in neighborhoods with a higher socioeconomic status or lower levels of deprivation show better cognitive functioning (Wu, Prina, & Brayne, 2015). However, while many cross-sectional studies examined effects of neighborhood socioeconomic status (NSES) on the *levels* of cognitive functioning, very few studies examined cognitive *decline* over time (but see Boardman, Barnes, Wilson, Evans, & Mendes de Leon, 2012; Sheffield & Peek, 2009; Zeki Al Hazzouri et al., 2011). We thus aim to examine effects of NSES not only on levels but also on decline of cognitive functioning. To get a broader and more detailed picture, we investigate four different aspects of cognitive functioning, particularly general cognitive functioning, processing speed, problem solving, and memory, instead of using a general measure only. We expect all four

domains of cognitive functioning to be associated with NSES. While the different domains may vary in their susceptibility to neighborhood characteristics, we do not explicitly theorize on domain-specific variations in this study. Instead, we include the different domains in our empirical analysis to facilitate a broader test where findings can be cross-validated across domains. Reliance on multiple outcomes rather than a sole measure seems especially important in light of the rather poor discriminatory power of the Mini-Mental State Examination in well-functioning individuals.

We further examine the effect of another key neighborhood characteristic, i.e. the effect of neighborhood urbanity, on older adults' cognitive functioning. Like NSES, neighborhood urbanity may affect access to opportunity structures that benefit cognitive functioning. Given the potential contribution of neighborhood urbanity to the understanding of interindividual differences in cognitive functioning, the scarcity of previous research on this issue signifies the need for our study.

We start by outlining theoretical considerations and empirical findings that propose effects of NSES and urbanity on older adults' cognitive functioning. Subsequently, we use growth models in a multilevel framework to examine the effect of both neighborhood characteristics on levels of and decline in cognitive functioning in a sample of 985 older adults from the Longitudinal Aging Study Amsterdam (LASA; Aartsen & Huisman, 2016). At the beginning of our study in 1995/96, the respondents were aged 65 to 88 years and did not show cognitive impairment. They were followed up for up to six years.

4.1.1 Theory

Associations of NSES and neighborhood urbanity with cognitive functioning could base on two different mechanisms. Firstly, the neighborhood context may have a causal influence on the cognitive functioning of its older inhabitants by affecting opportunity structures that influence behaviors associated with cognitive functioning. For example, neighborhoods with a higher (vs.

lower) NSES may provide their older inhabitants with more and higher quality resources that encourage engagement in physical activities (e.g. parks, gyms, sidewalks of good quality), social activities (e.g. attractive shopping areas, social clubs, neighborhood organizations), and cognitively stimulating activities (e.g. bookstores, libraries) (Wight et al., 2006; also see Sheffield & Peek, 2009 and Wu et al., 2015). This assumption is supported by theoretical approaches like the Cognitive Enrichment Hypothesis arguing that within age-related biological constraints, "behaviors of an individual (including cognitive activity, social engagement, exercise, and other behaviors) have a meaningful positive impact on the level of effective cognitive functioning in old age" (Hertzog et al., 2009). Other theories state more explicitly that cognitive decline may similarly be affected by cognitive, physical, and social activities (e.g. Use It or Lose It Hypothesis (Hultsch, Hertzog, Small, & Dixon, 1999), Environmental Complexity Hypothesis (Schooler, 1984), Revised Scaffolding Theory of Aging and Cognition (Reuter-Lorenz & Park, 2014)).

Besides differential access to opportunities, neighborhoods may influence their inhabitants' motivation to engage in cognitively enhancing activities: Older adults' neighbors in better off neighborhoods are more likely to be well educated and occupationally successful and might stimulate upward comparison. This may in turn motivate older adults' engagement in activities that enhance cognitive functioning (Sisco & Marsiske, 2012).

In view of neighborhood urbanity, we assume that more urban neighborhoods represent more complex environments, as understood by the higher diversity of stimuli and the requirement to make decisions in which a larger amount of information needs to be considered and processed (see Schooler, 1984). For example, moving in busy traffic, not getting sidetracked by distractions along the way, and choosing from a larger number of options when it comes to shopping and leisure time activities characterize complex urban environments, offering cognitive stimulation (Cassarino & Setti, 2015). In line with this, Crowe et al. (2008) assumed that a greater life-space (i.e. the spatial range within which people move regularly) with its

"greater diversity of experiences and greater demands in terms of decision making" represents a component of environmental complexity. They found that older adults who used a greater life-space showed a weaker decline in cognitive functioning over a 4-year period, controlling for baseline cognition and the effect of physical function. Furthermore, urban neighborhoods supposedly offer a high density of mentally stimulating offers like museums and theatres. Also by means of dense public transportation systems, access to a variety of offers should be facilitated for older adults (St. John, Seary, Menec, & Tyas, 2016; also see Wu et al., 2015). We thus expect higher levels of and slower decline in cognitive functioning for older adults residing in neighborhoods with a higher NSES (hypothesis H1a/H1b) or a higher degree of urbanity (H2a/H2b), respectively.

Secondly, better cognitive functioning in neighborhoods with a higher NSES or higher urbanity can be caused by the selection of individuals based on their individual socioeconomic status (SES) into specific neighborhoods. E.g., individual education and income have been shown to be related to cognitive functioning in cross-sectional studies (Opdebeeck, Martyr, & Clare, 2016; Zhang et al., 2015) (longitudinal findings have been more mixed though, see Anstey & Christensen, 2000; Valenzuela & Sachdev, 2006). Thus, individuals with higher SES and associated better cognitive functioning more likely live in neighborhoods with a higher (vs. lower) NSES or a higher (vs. lower) degree of urbanity. In such case, cognitive differences are not necessarily caused by the neighborhood context but by the mingling of certain individuals in the respective neighborhoods. We thus test the alternative explanation that statistical effects of NSES and urbanity dissolve once individual socioeconomic status is considered (H3).

4.1.2 Findings on neighborhood socioeconomic status

Cross-sectional empirical findings on the effect of NSES in older populations are mixed. On the one hand, studies found NSES (defined here as (proxy-)measures of at least one dimension of socioeconomic status, i.e. education, occupation, and/or income) to be positively associated

with the level of cognitive functioning (Clarke et al., 2012; Lang et al., 2008; Lee, Glass, James, Bandeen-Roche, & Schwartz, 2011; Rosso et al., 2016; Shih et al., 2011; Sisco & Marsiske, 2012; Wight et al., 2006; Zeki Al Hazzouri et al., 2011) or negatively associated with cognitive impairment (Basta, Matthews, Chatfield, & Brayne, 2008; Wee et al., 2012). Partly, studies found that the effect of NSES on cognitive functioning depended on individual demographic (Lang et al., 2008), socioeconomic (Aneshensel, Ko, Chodosh, & Wight, 2011; Basta et al., 2008; Deeg & Thomése, 2005; Wight et al., 2006) or genetic (Lee et al., 2011) factors. On the other hand, a range of studies found no or attenuated effects of measures of NSES when adjusting for individual SES (Clarke et al., 2012; Lee et al., 2011; Rosso et al., 2016; Sheffield & Peek, 2009; Sisco & Marsiske, 2012; Wee et al., 2012; Wight et al., 2006). The lack of associations between measures of NSES and cognitive functioning or impairment in models accounting for individual SES is more in line with the selection mechanism than with the causal explanation. Yet, not all findings can undoubtedly be attributed to the former, partly because other factors (e.g. health) where controlled simultaneously.

The available evidence on effects of NSES on cognitive decline is equally mixed. Some studies find associations of higher NSES with reduced rates of cognitive decline, at least for some of the investigated indicators of NSES (Sheffield & Peek, 2009) or cognitive functioning. In one study, these effects showed in white but not black people (Rosso et al., 2016). Taken together, this provides at least some evidence for a causal effect. The NSES-measures in two other studies were however not related to change in cognitive functioning over time when individual SES was controlled (partly simultaneously with additional factors), questioning the existence of a causal effect and supporting the possibility of a selection mechanism (Boardman et al., 2012; Zeki Al Hazzouri et al., 2011).

4.1.3 Findings on urbanity

To the best of our knowledge, little is known about effects of urbanity on normal cognitive functioning. Findings from related fields however give reason to expect that higher neighborhood urbanity might be beneficial for cognitive functioning. E.g., a recent study using brain imaging measures found that urbanity as measured by dwelling density is cross-sectionally associated with better brain health (see online supplements in Cerin et al., 2017). Conclusions from research looking into dementia and cognitive impairment are however mixed. Either no association (Klich-Rączka et al., 2014; St. John et al., 2016) or a negative association is reported between urbanity (partly measured at larger scales than neighborhoods) and risks of dementia and cognitive impairment after adjustment for at least age and education (Arslantas et al., 2009; Gavrila et al., 2009; also see the review by Russ, Batty, Hearnshaw, Fenton, & Starr, 2012). Given the potentially stimulating effect of urban neighborhoods, the lack of empirical studies on its effects on normal cognitive functioning illustrates the need for further investigations of this issue.

4.2 Data and methods

4.2.1 Sample

We used data from the Longitudinal Aging Study Amsterdam (LASA), an ongoing study on the cognitive, social, emotional and physical functioning of older adults in the Netherlands (Aartsen & Huisman, 2016). The first wave of data was collected in 1992/3, with follow-ups approximately every three years. The population-based sample was selected from eleven municipalities within three culturally distinct regions of the country and aimed to represent urban as well as rural inhabitants within each of these regions. Ethics approval was obtained from the medical ethics committee of the VU University Medical Center (IRB numbers: 92/138 and 2002/141). In the present study, we analyzed data from 985 older adults who were aged 65

to 88 years at the start of our study period, which was only in the second wave in 1995/6 (t_1) due to availability of neighborhood data. Respondents were followed up for the two subsequent waves in 1998/9 (t_2) and 2001/2 (t_3), i.e. for up to approximately six years, with reduced sample sizes caused by missing values (see *Table 4.1*)

Table 4.1: Descriptive statistics for cognitive functioning, individual controls and neighborhood characteristics.

Variable	$n_{respondents}$	Mean or %	SD	Range
Cognition				
MMSE (t_1)	985	27.69	1.63	24–30
MMSE (t_2)	833	27.24	2.45	10–30
MMSE (t_3)	645	26.79	3.04	5–30
Coding Task (t_1)	985	24.15	6.68	7.00–42.67
Coding Task (t_2)	777	23.75	6.61	3.00–40.00
Coding Task (t_3)	570	23.40	6.80	3.33–40.67
RCPM (t_1)	985	17.99	3.76	4–24
RCPM (t_2)	787	17.49	3.93	4–24
RCPM (t_3)	575	17.51	3.87	4–24
15WT (t_1)	985	6.13	2.89	0–15
15WT (t_2)	773	5.56	2.94	0–15
15WT (t_3)	579	5.89	3.22	0–14
Individual controls				
Male (Ref. female)	985	48.93	*n.a.*	*n.a.*
Age (t_1)	985	74.75	6.30	64.76–88.33
Education	985	9.20	3.36	5–18
Income (1000 Euro/month; t_1)	985	0.97	0.43	0.34–2.61
Employed (Ref. not employed; t_1)	985	4.37	*n.a.*	*n.a.*
Neighborhood variables (t_1)				
NSES 1st quartile	985	14.72	*n.a.*	6,534€–7,7737€[a]
NSES 2nd quartile	985	34.82	*n.a.*	7,760€–8,395€[a]
NSES 3rd quartile	985	25.38	*n.a.*	8,440€–9,348€[a]
NSES 4th quartile	985	25.08	*n.a.*	9,393€–12,229€[a]
Neighborhood urbanity	985	2.03	1.46	0–4[a]

Note. [a] Range of the original variable within the respective category; SD = standard deviation; MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

4.2.2 Measures

Cognition

Firstly, the Mini Mental State Examination (MMSE) was a general measure of cognition, including orientation in time and space, registration, attention, recall, language and visuospatial abilities (Folstein, Folstein, & McHugh, 1975). The maximum score is 30, with higher values indicating better cognitive functioning (see *Table 4.1* for descriptive statistics of all variables). Secondly, processing speed was assessed with the Coding Task (Piccinin & Rabbitt, 1999). Respondents were presented with a key of two rows of letters in which a letter from the upper row and the lower row belong together. The test consisted of an upper row containing letters and an empty lower row. Within three trials of one minute, respondents had to match as many letters as possible to the upper row by orally naming the corresponding letter from the key. We analyzed the mean number of matches made by the respondents in up to three trials per wave. Thirdly, problem solving was measured with Raven Colored Progressive Matrices (RCPM; Raven, 1995). In this non-verbal visual test of abstract reasoning, respondents were presented with 24 patterns, in each of which one part was missing. Respondents were asked to pick the pattern that correctly fits into the incomplete pattern from six alternatives.

Fourthly, episodic memory was assessed with the 15 words test (15WT), a Dutch version of the Auditory Verbal Learning Test (Rey, 1964; Saan & Deelman, 1986). Respondents were asked to learn 15 words and recall them immediately during the learning phase, which was repeated three times. We analyzed the delayed recall score, i.e. the number of words correctly recalled by the respondent after a distraction period of 20 minutes.

Individual level control variables

We adjusted for individual sex and age to account for potential differences in cognitive functioning as well as for demographic differences in the composition of neighborhoods. Individual education, income and employment status were used to examine whether differences in cognitive functioning by NSES and urbanity remain after accounting for selection based on individual SES. Education was measured in years typically needed to achieve a certain educational level. Our income measure is based on the categorical report of monthly net household income of the respondent and, if applicable, its partner. Following a procedure suggested by Broese van Groenou, Deeg, and Penninx (2003), we generated a continuous measure of net monthly household income by replacing income categories with the median income of each category. For reasons of comparability between respondents with and without a co-residing partner, we divided net monthly household income by 1.5 if the respondent co-resided with a partner, as suggested by the modified OECD-scale (Hagenaars, Vos, & Zaidi, 1994). We controlled for employment status because employment can be an additional source of cognitive stimulation and the likelihood of being employed may differ by NSES and neighborhood urbanity. Our measure assessed whether the respondent was currently in paid work for at least one hour per week. We did not control for individual health because it might be a mechanism linking neighborhood characteristics and cognitive functioning, hiding existing effects of NSES and urbanity. However, we present models including health indicators in a robustness check.

All cognitive outcomes were assessed at t_1, t_2, and t_3, while age, income and employment status were assessed at baseline t_1 in 1995/6. Information on sex and education were assessed at the first collection of LASA data in 1992/3. We treat the latter two variables as if they were measured at t_1 because they rarely change in older adults. Since we tested for quadratic effects of continuous variables, age, education and income were centered to their respective sample means to reduce multicollinearity.

Neighborhood socioeconomic status and urbanity

Neighborhood information for baseline t_1 in 1995 stem from Statistics Netherlands. A neighborhood is represented by a so-called *wijk*, which is an area in a community that consists of one or more adjoining homogenous sub-areas, which are again delineated by historical or built characteristics (Statistics Netherlands, n.d.). At baseline, the respondents in our analytical sample lived in 63 different neighborhoods, corresponding to an average number of 15.63 respondents per neighborhood (*SD*=15.27, *min*=1, *max*=62).

NSES was operationalized as the average net income per inhabitant in the neighborhood in the previous year, i.e. 1994, provided that they had income the whole year (Statistics Netherlands, 2016). In our analyses, we used quartiles of average neighborhood income, calculated on the basis of 63 neighborhoods in which the respondents constituting our sample resided.

To obtain a measure of neighborhood urbanity, the number of addresses within a radius of one kilometer around an address was determined. The average of this measure of address density over all addresses in a neighborhood constitutes our indicator of neighborhood urbanity (reported as the number of addresses/km^2). This measure of neighborhood urbanity was developed to measure human activity in an area and thus includes residential addresses as well as addresses of shops, workplaces etc. (den Dulk, van de Stadt, & Vliegen, 1992). Statistics Netherlands differentiates between areas that are not urbanized (<500 addresses/km^2), little urbanized (500 to <1000 addresses/km^2), somewhat urbanized (1000 to <1500 addresses/km^2), highly urbanized (1500 to <2500 addresses/km^2), and very highly urbanized (>2500 addresses/km^2). We used a continuous measure ranging from 0 (not urbanized) to 4 (highly urbanized), mean-centered at the neighborhood level.

4.2.3 Analytical approach

We restricted the sample to respondents with valid information on all independent variables and the baseline assessment of all four cognitive functioning measures. Out of 1,367

respondents with complete cognitive baseline information, we excluded 135 respondents with cognitive impairment (MMSE-score ≤ 23, Tangalos et al., 1996), 191 respondents who moved since 1992 (i.e. during approximately three years before the baseline measurement t_1 in 1995/6), one respondent with missing neighborhood information, and 55 respondents with missing information on independent variables. Respondents with missing values on cognitive functioning measures at t_2 and/or t_3 were retained in the sample and maximum likelihood estimation was applied to deal with missing data (Baraldi & Enders, 2010). Information on at least one cognitive functioning measure was provided by 833 and 646 respondents at t_2 and t_3, respectively. Those providing information on cognitive functioning at t_3 were more likely to be female, were younger on average and had higher average scores on cognitive measures at t_1. For each domain of cognitive functioning, we estimated growth curve models in a multilevel framework, which allowed us to assess simultaneously the level of cognitive functioning at baseline and the rate of decline during the subsequent six years. In a multilevel approach to growth curves, typically a hierarchical two-level data structure is assumed: Assessments of the same respondent at different time points (level 1) are nested within the respective respondent (level 2). We added a third level for neighborhoods to account for the clustering of respondents living in the same neighborhood at t_1. The temporal dimension of decline is represented by the survey waves at t_1, t_2, and t_3, which were recoded to 0, 3, and 6 years, respectively, so cognitive decline is reported as the yearly rate of change. The effects of individual and neighborhood variables on the levels of cognitive functioning are then indicated by the conditional effects of the respective variables, while their effects on cognitive decline are represented by interaction terms of the respective variable with time. We used the xtmixed-command in Stata version 14.0 and tested for random slopes of time at the individual level and for random slopes of time and individual-level independent variables at the neighborhood level. When testing the joint significance of or differences between fixed effects, we applied Wald χ^2-tests.

4.3 Results

Neighborhood effects from the multilevel linear regressions predicting baseline levels and growth rates over six years for all cognitive outcomes are shown in *Table 4.2* (see *Table 4.A1* in the Appendix for full models). Models M1 were adjusted for sex and age to examine whether NSES and urbanity were associated with cognitive functioning when taking into account differences in neighborhood composition by these demographic variables. Models M2 additionally account for individual SES, i.e. respondents' education, income at baseline and employment status at baseline. This was done to see if potential neighborhood effects in models M1 mainly reflect self-selection of individuals rather than a neighborhood effect. Self-selection describes a situation in which apparent neighborhood effects are explained by the gathering of individuals with certain socioeconomic and cognitive characteristics in certain neighborhoods and thus largely reflect an association at the individual level. For example, individuals with a higher individual SES would have higher cognitive levels and slower rates of decline and would simultaneously be more likely to live in neighborhoods with higher NSES or urbanity (and vice versa). The estimation of curvilinear effects for education (Coding Task) and income (MMSE, Coding Task) was implied by the data.

4.3.1 Levels at baseline

We observed a pattern of increasing levels of cognitive functioning across NSES-quartiles for all outcomes (Models M1, *Table 4.2*). The difference between the first and fourth quartile was statistically significant for Coding Task ($B_{Q4\text{-}Q1}$=2.89, $p\leq$.001) and RCPM ($B_{Q4\text{-}Q1}$=0.97, p=.013), but not MMSE ($B_{Q4\text{-}Q1}$=0.30, p=.126) and 15WT ($B_{Q4\text{-}Q1}$=0.55, p=.066). For Coding Task, also the third and second NSES-quartile had higher average levels than the first ($B_{Q3\text{-}Q1}$=2.11, p=.003; $B_{Q2\text{-}Q1}$=1.32, p=.040), and the level of the fourth was also higher than that of the second ($B_{Q4\text{-}Q2}$=1.57, p=.005). For RCPM, also the third NSES-quartile differed significantly from the first ($B_{Q3\text{-}Q1}$=0.88, p=.026). When accounting for selection into

Table 4.2: Multilevel linear regression models predicting baseline levels (t_1) and growth rates (t_1– t_3) for four cognitive functioning outcomes among 985 respondents aged 65 and older at t_1. Independent variables refer to t_1. Unstandardized regression coefficients (*p*-values in parentheses).

	MMSE		Coding Task		RCPM		15WT	
	M1	M2	M1	M2	M1[a]	M2	M1	M2[b]
Fixed Effects								
Baseline	27.58***	27.81***	22.98***	25.21***	16.89***	17.69***	6.37***	6.66***
Intercept	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
NSES (Ref. 1st quartile)								
2nd quartile (t_1)	0.03	-0.04	1.32*	0.71	0.60	0.28	0.43	0.30
	(0.865)	(0.854)	(0.040)	(0.257)	(0.113)	(0.438)	(0.134)	(0.318)
3rd quartile (t_1)	0.26	0.08	2.11**	0.92	0.88*	0.26	0.50	0.26
	(0.192)	(0.703)	(0.003)	(0.180)	(0.026)	(0.495)	(0.101)	(0.410)
4th quartile (t_1)	0.30	0.05	2.89***	1.16	0.97*	0.10	0.55	0.28
	(0.126)	(0.802)	(0.000)	(0.077)	(0.013)	(0.789)	(0.066)	(0.373)
Neighborhood urbanity	0.15***	0.10*	0.69***	0.35*	0.17*	0.03	0.18**	0.14*
	(0.001)	(0.024)	(0.000)	(0.013)	(0.042)	(0.736)	(0.004)	(0.036)
Neighborhood urbanity^2	*n.a.*	*n.a.*	-0.33*	-0.28*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.011)	(0.032)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Growth rate (linear)								
Time (years)	-0.23***	-0.18**	-0.25**	-0.28**	-0.26***	-0.25***	-0.34*	-0.36*
	(0.000)	(0.003)	(0.003)	(0.001)	(0.000)	(0.000)	(0.014)	(0.010)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years	0.01	-0.01	-0.03	-0.02	0.06	0.06	-0.15	-0.14
	(0.913)	(0.888)	(0.680)	(0.770)	(0.361)	(0.404)	(0.337)	(0.386)
3rd quartile (t_1) × years	0.03	0.01	0.02	0.04	0.12	0.12	-0.23	-0.19
	(0.641)	(0.852)	(0.854)	(0.668)	(0.091)	(0.093)	(0.157)	(0.235)
4th quartile (t_1) × years	-0.03	-0.06	-0.02	0.01	0.10	0.10	-0.06	-0.06
	(0.691)	(0.371)	(0.850)	(0.937)	(0.152)	(0.164)	(0.705)	(0.738)
Neighborhood urbanity × years	-0.01	-0.02	-0.03	-0.02	0.01	0.01	-0.04	-0.03
	(0.355)	(0.154)	(0.116)	(0.193)	(0.441)	(0.524)	(0.308)	(0.346)
Neighborhood urbanity^2 × years	*n.a.*	*n.a.*	-0.01	-0.01	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.536)	(0.544)	*n.a.*	*n.a.*	*n.a.*	*n.a.*

(continued on next page)

Table 4.2 continued

Growth rate (quadratic)								
Time^2 (years^2)	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.04	0.04
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.097)	(0.069)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.02	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.508)	(0.584)
3rd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.03	0.02
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.342)	(0.476)
4th quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.609)	(0.647)
Neighborhood urbanity × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.00	0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.517)	(0.570)
Random Variance Components								
Neighborhood level								
Intercept	0.04***	0.06***	0.33	0.53	0.08	0.10*	0.08*	0.12
Respondent level								
Intercept	0.96	0.81	30.00***	24.09***	7.59***	6.06***	4.26***	3.81***
Slope (years)	0.18***	0.17***	0.18***	0.18***	*n.a.*	*n.a.*	0.04***	0.04***
Residual	2.08***	2.08***	4.76***	4.75***	5.63***	5.60***	2.57***	2.57***
$N_{observations}$	2,463	2,463	2,332	2,332	2,347	2,347	2,337	2,337
$N_{respondents}$	985	985	985	985	985	985	985	985
$N_{neighborhoods}$	63	63	63	63	63	63	63	63
Wald χ^2-tests for time trends								
p_{NSES}	0.80	0.61	0.92	0.86	0.34	0.34	0.36	0.40
$p_{Urbanity}$	0.35	0.15	0.22	0.34	0.44	0.52	0.38	0.40

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Models M1 control for age and sex and their effects on time. Models M2 additionally control for education, income and employment status. If implied by the data, curvilinear effects of continuous control variables were estimated. Details are shown in *Table 4.A1* in the Appendix.

[a] To achieve model convergence, a restricted maximum likelihood instead of a maximum likelihood algorithm had to be used.

[b] Model included a random slope for income at the neighborhood level: Slope(income)=1.14, p=.814.

MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

neighborhoods by individual SES in models M2, no significant differences between NSES-quartiles remained (all $p \geq .077$).

Degree of urbanity was positively associated with levels of MMSE (B_{URB}=0.15, p=.001), RCPM (B_{URB}=0.17, p=.042), and 15WT (B_{URB}=0.18, p=.004). For Coding Task, an inversely U-shaped association was found ($B_{URB\ linear}$=0.69, $p \leq .001$; $B_{URB\ squared}$=-0.33, p=.011; Wald $\chi^2(2)$=27.19, $p \leq .001$). Accounting for individual SES in models M2, the coefficients were somewhat reduced but the linear association with MMSE and 15WT and the inversely U-shaped association with Coding Task remained statistically significant (all $p \leq .036$).

4.3.2 Decline over time

In models accounting for sex and age (and their effects on cognitive change over time), we found cognitive decline in all four outcomes (MMSE: B_{Years}=-0.23, Coding Task: B_{Years}=-0.28, RCPM: B_{Years}=-0.18, all p<.001; 15WT: B_{Years}=-0.47, $B_{Years\ squared}$=0.05; Wald $\chi^2(2)$=70.61, p<.001, corresponding to a change of -0.96 points after three years and -1.02 points after six years, obtained by replacing Years = 3 and Years = 6 in Δ15WT = $B_{Years\ linear}$ * Years + $B_{Years\ squared}$ * Years^2 = -0.47 * Time + 0.05 * Time^2, respectively; models not shown) for female respondents of average age. A curvilinear change for 15WT was estimated since descriptive analyses hinted at a non-linear development of 15WT scores over time. Causes might be the re-use of the same set of words at t_1 and t_3, practice effects and measurement inequivalence between t_2 vs. t_1 and t_3.

Findings on differences in cognitive decline by neighborhood characteristics hardly differed between partly and fully adjusted models. None of the models showed a pattern of systematically slower decline of cognitive functioning in higher NSES-quartiles and change did not differ significantly between quartiles (all $p \geq .091$; *Table 4.2* and additional analyses, considering also the curvilinear trend for 15WT). Similarly, rates of decline did not significantly

depend on urbanity (all $p \geq .116$, considering also the curvilinear effect of urbanity on Coding Task) (see *Figure 4.1* and *Figure 4.2*).

4.3.3 Random components and intraclass correlation coefficients (ICCs)

Two things should be noted when evaluating random components. Firstly, the variation of cognitive functioning at the neighborhood level was relatively small in random intercept models where only sex, age, and the linear decline (plus quadratic term of time for 15WT) and the effects of sex and age on decline were accounted for (ICC_{MMSE}=.02, $ICC_{Coding\ Task}$=.06, ICC_{RCPM}=.02, ICC_{15WT}=.02; models not shown). This implies that after taking into account these predictors, a much larger part of variation in cognitive functioning is located at the individual compared to the neighborhood level. Secondly, adding random slopes for time at the neighborhood level did not significantly improve these models, indicating that variation in cognitive decline between neighborhoods was rather small.

4.3.4 Robustness checks

We ran several checks to evaluate the robustness of our findings. Firstly, we added health indicators (number of functional limitations, number of chronic diseases, number of depressive symptoms (CES-D Scale; Radloff, 1977)). The results are reported in *Table 4.A2* in the Appendix. Due to missing values on the added variables, this sample consists of 974 individuals from 62 neighborhoods. Overall, these analyses support our conclusions from the original models. The most notable deviations were that the level-difference in MMSE between the second and fourth NSES-quartile and the effect of urbanity on linear change in Coding Task became statistically significant in the partly adjusted model M1.

Secondly, we checked whether findings have been affected by respondents who moved between t_1 and t_3. We re-ran our analyses with a subsample including only respondents of the initial

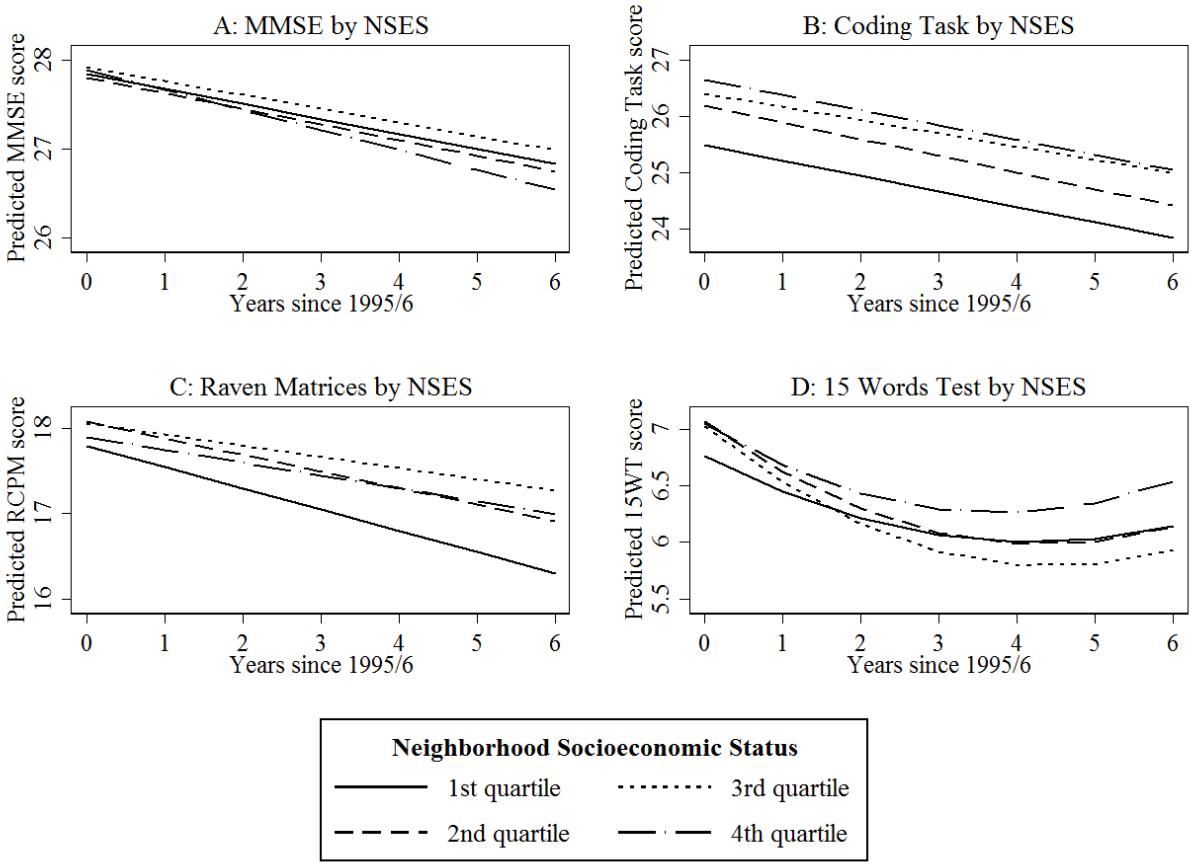

Figure 4.1: Predicted growth curves by neighborhood socioeconomic status for all four cognitive outcomes in the observed period t_1 to t_3. Graphs depict results from models M2 (see *Table 4.2*) for women of average age, education and income who were not employed at t_1.

sample who did not move between t_1 and t_3 (n=506), excluding 137 movers (64 movers between t1 and t2, 63 movers between t_2 and t_3, 9 respondents moved between both periods) and 343 respondents with missing information on at least one of the moving periods. The results of this robustness-check (see *Table 4.A3* in the Appendix) are generally in accord with the results from the initial sample. A notable difference was that the robustness check showed significantly higher levels of MMSE in the fourth (vs. second) quartile and higher levels of 15WT for the second, third and fourth (vs. first) quartile in partly adjusted models M1, which were however no longer significant in the fully adjusted models M2. Furthermore, for MMSE, Coding Task and 15WT, the level difference by urbanity was not statistically significant in the fully adjusted models M2 of the robustness check. However, for Coding Task and 15WT, the coefficients were of similar size, so their non-significance can be attributed to the smaller sample size. In

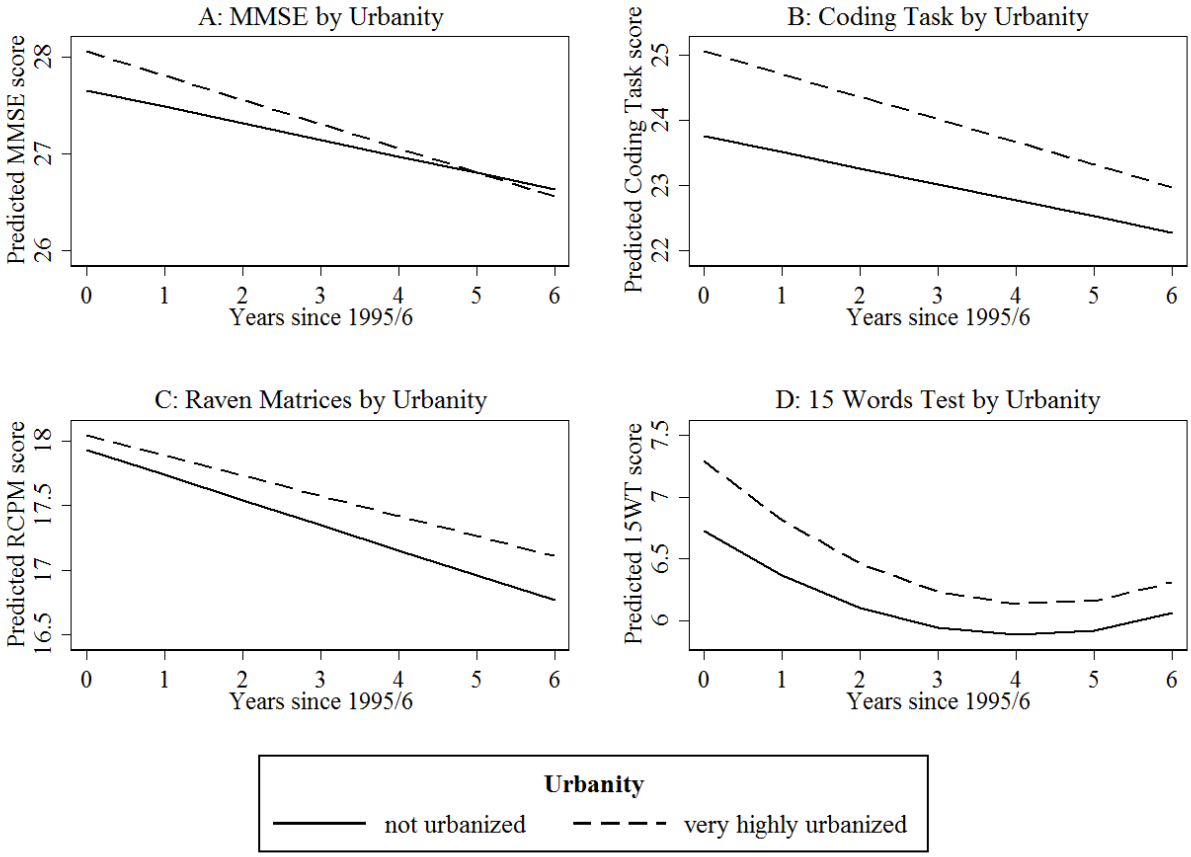

Figure 4.2: Predicted growth curves for the most extreme values of neighborhood urbanity for all four cognitive outcomes in the observed period t_1 to t_3. Graphs depict results from models M2 (see *Table 4.2*) for women of average age, education and income who were not employed at t_1.

this sample, decline of MMSE was significantly slower in the second and third (vs. first), but not in the fourth NSES-quartile, and the U-shaped trend of 15WT differed significantly between the third vs. fourth quartile of NSES, indicating faster initial decline in the third quartile. Nevertheless, these differences did overall not imply systematically slower decline in neighborhoods with a higher NSES. Instead, this robustness check showed again that differences in cognitive functioning by NSES can be attributed to selection on individual SES and that level differences by urbanity are relatively robust to selection on individual SES. Both neighborhood characteristics hardly systematically affected decline in cognitive functions.

Thirdly, due to the negative skewness of MMSE-scores, we additionally re-ran the original models with a log(31-MMSE)-transformation to achieve a more normal distribution of model residuals. Overall, the conclusions from these models support the original conclusions (higher

levels in more urban neighborhoods in both partly and fully adjusted models, no significant differences in levels by NSES, and no differences in decline by neither NSES nor urbanity).

Fourthly, respondents might improve on cognitive tests due to practice effects when their cognitive functioning is assessed repeatedly. Similar to a procedure described by Vivot et al. (2016), we added a dummy variable to identify the first observation in our study. Practice effects would then be represented by a negative deviation at t_1 from the general pattern of cognitive decline. We found evidence of a practice effect for MMSE but not for Coding Task, RCPM, and 15WT. Accounting for practice effects did not change our substantial conclusions (see *Table 4.A4* in the Appendix).

4.4 Discussion

Our study is among the first to examine neighborhood effects on levels of and decline in cognitive functioning among Dutch older adults. We used official statistics on neighborhoods to avoid same source bias and employed a longitudinal design to allow for the study of cognitive decline. According to H1a, we expected higher levels of cognitive functioning in neighborhoods with a higher NSES. We found partial support for H1a, indicating higher levels of processing speed and problem solving, but not general cognitive functioning and memory, in neighborhoods with higher NSES. No support was found for H1b, predicting cognitive decline to be slower in neighborhoods with higher NSES. For all cognitive outcomes, levels were positively associated with urbanity, supporting H2a. However, the inversely U-shaped association for processing speed showed that this association might be reversed at high levels of urbanity. The lack of significant effects of urbanity on decline lead us to reject H2b, predicting slower cognitive decline in urban neighborhoods. The selection mechanism (H3) found partial support because controlling for individual SES largely reduced the associations that we previously found between NSES and levels of processing speed and problem solving,

respectively (note that NSES was not associated with cognitive decline and levels of general cognitive functioning and memory). However, observed effects of urbanity on levels were overall relatively robust to controlling for individual SES (except for problem solving; note that no effects of urbanity on decline were found), giving little additional support for H3.

The findings show that if neighborhood differences in levels by NSES are observable, these can be explained by the selection of individuals into neighborhoods based on their individual SES. The lack of systematic differences in cognitive decline as a function of NSES does not support the idea that more opportunities for stimulating activities in the neighborhood slow down decline. An explanation for the findings on NSES may be that access to supposedly relevant opportunity structures was not worse in neighborhoods with lower NSES (Pearce, Witten, Hiscock, & Blakely, 2007). Alternative explanations might be that average income in the neighborhood is a rather indirect measure of neighborhood characteristics that are assumed to be relevant (e.g. parks, conditions of sidewalks, shopping areas, or libraries) or that these concepts are unrelated to cognitive functioning (Clarke et al., 2012). The findings imply that individuals with lower SES who might only afford living in neighborhoods with lower NSES are not additionally disadvantaged by the NSES of their neighborhood when it comes to cognitive functioning. Nevertheless, it should be noted that levels of processing speed and problem solving were higher in neighborhoods with higher NSES in partly adjusted models, indicating that older adults with lower cognitive functioning gather in neighborhoods with a lower NSES.

We found higher levels of cognitive functioning in more urban neighborhoods, findings that were overall robust to our tests of the selection mechanism, with the exception of problem solving. Thus, results for levels of cognitive functioning by urbanity largely comply with the assumed stimulating effect of urban neighborhoods. However, the inversely U-shaped association for processing speed shows that very high urbanity may be less beneficial, e.g. because the environment is too stressful or too challenging and discourages older adults from

using opportunity structures. The finding of level differences by urbanity is in line with the idea that access to stimulating resources may well differ between more and less urban neighborhoods in the Dutch context, while it may differ less by NSES. The lack of an effect of urbanity on decline implies that level differences by urbanity are not due to urbanity differences in decline in old age, but might rather arise already earlier in life.

Besides the merits of our study, there are some limitations. Firstly, neighborhood effects earlier in the life-course might well have effects on cognitive functioning in older adults. Our findings may also be biased by respondents moving during the observed period. We addressed both issues by restricting the sample to people who did not move within three years before the start of our study and by conducting a robustness-check with a non-mover subsample.

Secondly, the ICCs and random components from models accounting only for sex, age, and time showed little variation in cognitive functioning at the neighborhood level, also – although not directly comparable – in comparison to studies on cognitive functioning in older populations from the American context, which report neighborhood level ICCs ranging from .19 to .29 in intercept only models (Aneshensel et al., 2011; Clarke et al., 2012; Wight et al., 2006). This implies that investigating determinants at the individual level, which might include differential use of opportunity structures in the neighborhood as a function of individual characteristics, seems more promising to understand the determinants of cognitive functioning in our sample of Dutch older adults.

Thirdly, further research is necessary before generalizing our findings. This refers to different aspects: (1) Although the data were collected from a population-based sample to depict a good representation of older adults in the Netherlands, the findings of our study cannot be generalized to the population of older adults in the Netherlands. To do so, using sample weights or census data would be necessary. (2) Neighborhood effects can be expected to depend on country characteristics (e.g. strength of differences between neighborhoods and buffering effects of welfare states) and thus our findings can also not be generalized to other country contexts. Also,

the Netherlands are a highly urbanized country, yielding little variation in the degree of urbanity. Thus, neighborhood urbanity might be more important in understanding interindividual differences in cognitive functioning in countries with more variation in urbanity. (3) Although neighborhood characteristics can be assumed to change slowly, it is conceivable that characteristics of some neighborhoods may have changed since our data had been collected. Yet, we believe that the theoretical mechanisms linking NSES and urbanity with cognitive functioning are as valid today as they were in the 1990s and early 2000s. Consequently, we assume that one would find similar associations with more recent data. To empirically assess the transferability of our conclusions to more recent times, replication with newer data would be desirable.

4.5 Conclusion

We conclude that in the Dutch context, individual SES is more relevant than NSES to understand level differences in cognitive functioning. Thus, interventions should target individuals based on their individual risk profile instead of their residence in a neighborhood with certain NSES. Simultaneously, it should be highlighted that change in cognitive functioning overall was not systematically associated with individual SES or NSES. The study also reveals that neighborhood urbanity should be considered as a determinant of levels of cognitive functioning. This is in line with theoretical approaches that assume beneficial effects of exposure to complex environments on cognitive functioning. However, since rates of decline in our sample of older adults did not differ by urbanity, we assume that urbanity differences in cognitive functioning arise already earlier in life. Future research might follow up on our findings and test if urbanity makes a bigger difference in contexts that are less homogenously urbanized than the Netherlands.

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4.7 Appendix

Table 4.A1: Full multilevel linear regression models predicting baseline levels (t_1) and growth rates (t_1– t_3) for four cognitive functioning outcomes among 985 respondents aged 65 and older at t_1. Independent variables refer to t_1. Unstandardized regression coefficients (*p*-values in parentheses).

	MMSE		Coding Task		RCPM		15WT	
	M1	M2	M1	M2	M1[a]	M2	M1	M2[b]
Fixed Effects								
Baseline								
Intercept	27.58***	27.81***	22.98***	25.21***	16.89***	17.69***	6.37***	6.66***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
NSES (Ref. 1st quartile)								
2nd quartile (t_1)	0.03	-0.04	1.32*	0.71	0.60	0.28	0.43	0.30
	(0.865)	(0.854)	(0.040)	(0.257)	(0.113)	(0.438)	(0.134)	(0.318)
3rd quartile (t_1)	0.26	0.08	2.11**	0.92	0.88*	0.26	0.50	0.26
	(0.192)	(0.703)	(0.003)	(0.180)	(0.026)	(0.495)	(0.101)	(0.410)
4th quartile (t_1)	0.30	0.05	2.89***	1.16	0.97*	0.10	0.55	0.28
	(0.126)	(0.802)	(0.000)	(0.077)	(0.013)	(0.789)	(0.066)	(0.373)
Neighborhood urbanity	0.15***	0.10*	0.69***	0.35*	0.17*	0.03	0.18**	0.14*
	(0.001)	(0.024)	(0.000)	(0.013)	(0.042)	(0.736)	(0.004)	(0.036)
Neighborhood urbanity^2	*n.a.*	*n.a.*	-0.33*	-0.28*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.011)	(0.032)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female)	-0.02	-0.21	0.32	-1.06**	0.72**	0.08	-1.35***	-1.60***
	(0.855)	(0.050)	(0.397)	(0.003)	(0.001)	(0.723)	(0.000)	(0.000)
Age	-0.07***	-0.06***	-0.44***	-0.40***	-0.19***	-0.18***	-0.16***	-0.16***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Education		0.09***		0.60***		0.28***		0.08**
		(0.000)		(0.000)		(0.000)		(0.005)
Education^2		*n.a.*		-0.04***		*n.a.*		*n.a.*
		n.a.		(0.001)		*n.a.*		*n.a.*
Income		0.29		3.72***		1.17***		0.67*
		(0.131)		(0.000)		(0.000)		(0.020)
Income^2		-0.02		-1.33*		*n.a.*		*n.a.*
		(0.915)		(0.042)		*n.a.*		*n.a.*
Employed (Ref. not employed)		0.16		0.91		-0.03		0.19
		(0.541)		(0.289)		(0.954)		(0.637)

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Table 4.A1 continued

Growth rate (linear)								
Time (years)	-0.23***	-0.18**	-0.25**	-0.28**	-0.26***	-0.25***	-0.34*	-0.36*
	(0.000)	(0.003)	(0.003)	(0.001)	(0.000)	(0.000)	(0.014)	(0.010)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years	0.01	-0.01	-0.03	-0.02	0.06	0.06	-0.15	-0.14
	(0.913)	(0.888)	(0.680)	(0.770)	(0.361)	(0.404)	(0.337)	(0.386)
3rd quartile (t_1) × years	0.03	0.01	0.02	0.04	0.12	0.12	-0.23	-0.19
	(0.641)	(0.852)	(0.854)	(0.668)	(0.091)	(0.093)	(0.157)	(0.235)
4th quartile (t_1) × years	-0.03	-0.06	-0.02	0.01	0.10	0.10	-0.06	-0.06
	(0.691)	(0.371)	(0.850)	(0.937)	(0.152)	(0.164)	(0.705)	(0.738)
Neighborhood urbanity × years	-0.01	-0.02	-0.03	-0.02	0.01	0.01	-0.04	-0.03
	(0.355)	(0.154)	(0.116)	(0.193)	(0.441)	(0.524)	(0.308)	(0.346)
Neighborhood urbanity^2 × years	*n.a.*	*n.a.*	-0.01	-0.01	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.536)	(0.544)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female) × years	0.02	0.00	-0.20***	-0.18**	0.00	0.01	0.09	0.09
	(0.606)	(0.974)	(0.000)	(0.001)	(0.938)	(0.842)	(0.342)	(0.366)
Age × years	-0.02***	-0.02***	-0.02***	-0.02***	-0.01	-0.01	0.00	0.00
	(0.000)	(0.000)	(0.000)	(0.000)	(0.079)	(0.081)	(0.910)	(0.901)
Education × years		0.00		-0.01		-0.00		-0.02
		(0.657)		(0.277)		(0.810)		(0.367)
Education^2 × years		*n.a.*		-0.00		*n.a.*		*n.a.*
		n.a.		(0.870)		*n.a.*		*n.a.*
Income × years		0.17*		-0.01		0.05		0.12
		(0.018)		(0.879)		(0.385)		(0.372)
Income^2 × years		-0.15*		0.09		*n.a.*		*n.a.*
		(0.048)		(0.370)		*n.a.*		*n.a.*
Employed (Ref. not employed) × years		0.03		-0.05		-0.16		0.26
		(0.736)		(0.711)		(0.141)		(0.300)
Growth rate (quadratic)								
Time^2 (years^2)	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.04	0.04
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.097)	(0.069)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.02	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.508)	(0.584)
3rd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.03	0.02
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.342)	(0.476)
4th quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.609)	(0.647)

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Table 4.A1 continued

Neighborhood urbanity × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.00	0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.517)	(0.570)
Male (Ref. female) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.01	-0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.512)	(0.563)
Age × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.00	-0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.288)	(0.280)
Education × years^2		*n.a.*		*n.a.*		*n.a.*		0.00
		n.a.		*n.a.*		*n.a.*		(0.481)
Income × years^2		*n.a.*		*n.a.*		*n.a.*		-0.01
		n.a.		*n.a.*		*n.a.*		(0.559)
Employed (Ref. not employed) × years^2		*n.a.*		*n.a.*		*n.a.*		-0.06
		n.a.		*n.a.*		*n.a.*		(0.182)
Random Variance Components								
Neighborhood level								
Intercept	0.04***	0.06***	0.33	0.53	0.08	0.10*	0.08*	0.12
Respondent level								
Intercept	0.96	0.81	30.00***	24.09***	7.59***	6.06***	4.26***	3.81***
Slope (years)	0.18***	0.17***	0.18***	0.18***	*n.a.*	*n.a.*	0.04***	0.04***
Residual	2.08***	2.08***	4.76***	4.75***	5.63***	5.60***	2.57***	2.57***
$N_{observations}$	2,463	2,463	2,332	2,332	2,347	2,347	2,337	2,337
$N_{respondents}$	985	985	985	985	985	985	985	985
$N_{neighborhoods}$	63	63	63	63	63	63	63	63
Wald χ^2-tests for time trends								
p_{NSES}	0.80	0.61	0.92	0.86	0.34	0.34	0.36	0.40
$p_{Urbanity}$	0.35	0.15	0.22	0.34	0.44	0.52	0.38	0.40

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[a] To achieve model convergence, a restricted maximum likelihood instead of maximum likelihood algorithm had to be used.

[b] Model included a random slope for income at the neighborhood level: Slope(income)=1.14, p=.814.

MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

Table 4.A2: Unstandardized coefficients from multilevel linear regression models of baseline levels (t_1) and growth rates (t_1– t_3) of four cognitive functioning outcomes on neighborhood characteristics among respondents aged 65 and older, additionally controlling for health variables (n=974, p-values in parentheses).

	MMSE		Coding Task		RCPM		15WT	
	M1	M2	M1	M2	M1[a]	M2	M1	M2[b]
Fixed Effects								
Baseline								
Intercept	27.67***	27.88***	23.43***	25.55***	17.01***	17.78***	6.56***	6.83***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
NSES (Ref. 1st quartile)								
2nd quartile (t_1)	-0.01	-0.09	1.05	0.36	0.65	0.30	0.36	0.23
	(0.954)	(0.648)	(0.116)	(0.579)	(0.096)	(0.430)	(0.245)	(0.484)
3rd quartile (t_1)	0.11	-0.03	1.89**	0.94	0.65	0.16	0.28	0.11
	(0.571)	(0.889)	(0.007)	(0.163)	(0.102)	(0.682)	(0.385)	(0.745)
4th quartile (t_1)	0.31	0.07	2.66***	0.97	0.95*	0.09	0.52	0.25
	(0.114)	(0.734)	(0.000)	(0.137)	(0.016)	(0.811)	(0.099)	(0.442)
Neighborhood urbanity	0.14***	0.10*	0.72***	0.41**	0.16	0.03	0.20**	0.16*
	(0.001)	(0.023)	(0.000)	(0.005)	(0.061)	(0.717)	(0.005)	(0.029)
Neighborhood urbanity^2	*n.a.*	*n.a.*	-0.34**	-0.28*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.009)	(0.027)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female)	-0.10	-0.28*	-0.14	-1.43***	0.56*	-0.05	-1.49***	-1.73***
	(0.391)	(0.014)	(0.723)	(0.000)	(0.018)	(0.841)	(0.000)	(0.000)
Age	-0.05***	-0.05***	-0.38***	-0.36***	-0.17***	-0.17***	-0.14***	-0.15***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Functional limitations	-0.15***	-0.13***	-0.44**	-0.32*	-0.22**	-0.15	-0.26***	-0.22***
	(0.000)	(0.001)	(0.001)	(0.012)	(0.008)	(0.051)	(0.000)	(0.000)
No. chronic diseases	0.09	0.09	-0.09	-0.08	0.03	0.02	0.16*	0.16*
	(0.053)	(0.062)	(0.594)	(0.592)	(0.760)	(0.850)	(0.025)	(0.028)
Depressive symptoms (CES-D)	-0.01	-0.01	-0.06*	-0.06*	-0.00	0.00	-0.01	-0.01
	(0.454)	(0.449)	(0.029)	(0.021)	(0.993)	(0.983)	(0.439)	(0.327)
Education		0.09***		0.60***		0.28***		0.08**
		(0.000)		(0.000)		(0.000)		(0.005)
Education^2		*n.a.*		-0.04***		*n.a.*		*n.a.*
		n.a.		(0.001)		*n.a.*		*n.a.*
Income		0.24		3.50***		1.13***		0.60*
		(0.221)		(0.000)		(0.000)		(0.031)
Income^2		-0.02		-1.30*		*n.a.*		*n.a.*
		(0.904)		(0.045)		*n.a.*		*n.a.*
Employed (Ref. not employed)		0.19		1.04		0.01		0.24
		(0.454)		(0.220)		(0.980)		(0.549)

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Table 4.A2 continued

Growth rate (linear)								
Time (years)	-0.23***	-0.18**	-0.24**	-0.28**	-0.26***	-0.26***	-0.39**	-0.41**
	(0.000)	(0.003)	(0.003)	(0.001)	(0.000)	(0.000)	(0.005)	(0.004)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years	0.02	0.00	-0.03	-0.02	0.04	0.03	-0.10	-0.09
	(0.783)	(0.959)	(0.713)	(0.838)	(0.591)	(0.619)	(0.533)	(0.583)
3rd quartile (t_1) × years	0.03	0.01	0.01	0.03	0.13	0.14	-0.25	-0.22
	(0.663)	(0.836)	(0.904)	(0.718)	(0.057)	(0.054)	(0.123)	(0.173)
4th quartile (t_1) × years	-0.02	-0.05	-0.02	0.01	0.12	0.12	-0.05	-0.04
	(0.729)	(0.434)	(0.780)	(0.945)	(0.085)	(0.079)	(0.772)	(0.796)
Neighborhood urbanity × years	-0.01	-0.02	-0.04*	-0.03	0.01	0.01	-0.04	-0.04
	(0.497)	(0.252)	(0.042)	(0.082)	(0.591)	(0.674)	(0.236)	(0.246)
Neighborhood urbanity^2 × years	*n.a.*	*n.a.*	-0.01	-0.01	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.512)	(0.494)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female) × years	-0.00	-0.02	-0.21***	-0.19**	-0.01	0.00	0.16	0.16
	(0.979)	(0.705)	(0.000)	(0.001)	(0.824)	(0.925)	(0.119)	(0.143)
Age × years	-0.02***	-0.02***	-0.02***	-0.02***	-0.01	-0.01	-0.00	-0.00
	(0.000)	(0.000)	(0.000)	(0.000)	(0.076)	(0.062)	(0.847)	(0.840)
Functional limitations × years	-0.00	-0.00	-0.03	-0.03	0.00	0.01	0.01	0.01
	(0.755)	(0.887)	(0.119)	(0.134)	(0.865)	(0.687)	(0.729)	(0.709)
No. chronic diseases × years	-0.01	-0.01	-0.00	-0.00	-0.01	-0.01	-0.04	-0.04
	(0.525)	(0.565)	(0.974)	(0.949)	(0.680)	(0.790)	(0.371)	(0.401)
Depressive symptoms (CES-D) × years	-0.00	-0.00	0.00	0.00	-0.00	-0.00	0.02*	0.02*
	(0.393)	(0.418)	(0.264)	(0.252)	(0.336)	(0.426)	(0.023)	(0.022)
Education × years		0.00		-0.01		-0.00		-0.01
		(0.766)		(0.218)		(0.681)		(0.508)
Education^2 × years		*n.a.*		-0.00		*n.a.*		*n.a.*
		n.a.		(0.859)		*n.a.*		*n.a.*
Income × years		0.18*		-0.02		0.05		0.11
		(0.015)		(0.862)		(0.404)		(0.419)
Income^2 × years		-0.15*		0.09		*n.a.*		*n.a.*
		(0.042)		(0.374)		*n.a.*		*n.a.*
Employed (Ref. not employed) × years		0.04		-0.04		-0.16		0.23
		(0.707)		(0.768)		(0.136)		(0.350)

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Table 4.A2 continued

Growth rate (quadratic)								
Time^2 (years^2)	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.05*	0.05*
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.045)	(0.036)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.734)	(0.799)
3rd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.03	0.03
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.253)	(0.344)
4th quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.689)	(0.714)
Neighborhood urbanity × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.00	0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.414)	(0.432)
Male (Ref. female) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.02	-0.02
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.188)	(0.243)
Age × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.00	-0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.455)	(0.450)
Functional limitations × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.00	-0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.741)	(0.738)
No. chronic diseases × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.293)	(0.307)
Depressive symptoms (CES-D) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.00*	-0.00*
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.014)	(0.015)
Education × years^2		*n.a.*		*n.a.*		*n.a.*		0.00
		n.a.		*n.a.*		*n.a.*		(0.666)
Income × years^2		*n.a.*		*n.a.*		*n.a.*		-0.01
		n.a.		*n.a.*		*n.a.*		(0.648)
Employed (Ref. not employed) × years^2		*n.a.*		*n.a.*		*n.a.*		-0.05
		n.a.		*n.a.*		*n.a.*		(0.231)

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Table 4.A2 continued

Random Variance Components								
Neighborhood level								
Intercept	0.05***	0.07***	0.43	0.56	0.10	0.11*	0.13**	0.17**
Respondent level								
Intercept	0.91	0.77	29.05***	23.48***	7.49***	5.98***	4.11***	3.72***
Slope (years)	0.18***	0.17***	0.18***	0.18***	*n.a.*	*n.a.*	0.04***	0.04***
Residual	2.08***	2.08***	4.76***	4.75***	5.64***	5.60***	2.56***	2.56***
$N_{observations}$	2,440	2,440	2,309	2,309	2,324	2,324	2,314	2,314
$N_{respondents}$	974	974	974	974	974	974	974	974
$N_{neighborhoods}$	62	62	62	62	62	62	62	62
Wald χ^2-tests for time trends								
p_{NSES}	0.81	0.65	0.94	0.93	0.13	0.11	0.38	0.43
$p_{Urbanity}$	0.50	0.25	0.10	0.17	0.59	0.67	0.33	0.33

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[a] To ensure comparability with the original model, a restricted maximum likelihood algorithm was used instead of a maximum likelihood algorithm.

[b] Model included a random slope for income at the neighborhood level: Slope(income)=0.98, p=.969.

MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

Table 4.A3: Unstandardized coefficients from multilevel linear regression models of baseline levels (t_1) and growth rates (t_1– t_3) of four cognitive functioning outcomes on neighborhood characteristics among respondents aged 65 and older that are known to remain living in their neighborhood (n=506, p-values in parentheses).

	MMSE		Coding Task		RCPM		15WT	
	M1	M2	M1	M2	M1[a]	M2	M1	M2[b]
Fixed Effects								
Baseline								
Intercept	27.87***	28.19***	23.63***	26.05***	17.00***	17.90***	6.42***	6.77***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
NSES (Ref. 1st quartile)								
2nd quartile (t_1)	-0.10	-0.23	1.11	0.19	0.70	0.28	0.87*	0.62
	(0.641)	(0.277)	(0.163)	(0.807)	(0.165)	(0.552)	(0.024)	(0.124)
3rd quartile (t_1)	0.11	-0.04	1.80*	0.73	1.20*	0.65	0.95*	0.73
	(0.611)	(0.842)	(0.037)	(0.377)	(0.023)	(0.198)	(0.018)	(0.085)
4th quartile (t_1)	0.33	0.04	2.00*	0.14	1.08*	0.13	1.01*	0.69
	(0.135)	(0.845)	(0.016)	(0.865)	(0.040)	(0.794)	(0.012)	(0.106)
Neighborhood urbanity	0.13**	0.06	0.77***	0.29	0.26*	0.04	0.22*	0.14
	(0.005)	(0.181)	(0.000)	(0.102)	(0.018)	(0.724)	(0.011)	(0.132)
Neighborhood urbanity^2	*n.a.*	*n.a.*	-0.25	-0.21	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.116)	(0.177)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female)	0.02	-0.22	0.52	-0.96	1.06***	0.27	-1.45***	-1.70***
	(0.902)	(0.114)	(0.311)	(0.057)	(0.001)	(0.367)	(0.000)	(0.000)
Age	-0.04**	-0.03**	-0.37***	-0.33***	-0.16***	-0.15***	-0.11***	-0.11***
	(0.001)	(0.006)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Education		0.10***		0.62***		0.32***		0.09*
		(0.000)		(0.000)		(0.000)		(0.034)
Education^2		*n.a.*		-0.03		*n.a.*		*n.a.*
		n.a.		(0.088)		*n.a.*		*n.a.*
Income		0.43		3.76***		1.16**		0.71
		(0.082)		(0.000)		(0.002)		(0.066)
Income^2		-0.30		-1.97*		*n.a.*		*n.a.*
		(0.218)		(0.025)		*n.a.*		*n.a.*
Employed (Ref. not employed)		0.36		0.41		0.17		0.29
		(0.283)		(0.729)		(0.811)		(0.616)

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Table 4.A3 continued

Growth rate (linear)								
Time (years)	-0.26***	-0.23***	-0.22*	-0.27*	-0.30***	-0.30***	-0.48**	-0.51**
	(0.000)	(0.000)	(0.025)	(0.014)	(0.000)	(0.000)	(0.007)	(0.005)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years	0.13*	0.12	-0.00	0.02	0.10	0.09	-0.13	-0.11
	(0.048)	(0.065)	(0.991)	(0.865)	(0.224)	(0.249)	(0.516)	(0.577)
3rd quartile (t_1) × years	0.13*	0.13*	0.02	0.05	0.14	0.13	-0.25	-0.21
	(0.048)	(0.048)	(0.877)	(0.664)	(0.087)	(0.117)	(0.221)	(0.315)
4th quartile (t_1) × years	0.05	0.04	0.05	0.10	0.13	0.14	0.12	0.13
	(0.420)	(0.533)	(0.586)	(0.353)	(0.131)	(0.111)	(0.561)	(0.539)
Neighborhood urbanity × years	-0.00	-0.01	-0.02	-0.01	0.00	0.01	-0.04	-0.04
	(0.735)	(0.608)	(0.401)	(0.706)	(0.870)	(0.764)	(0.404)	(0.434)
Neighborhood urbanity^2 × years	*n.a.*	*n.a.*	-0.01	-0.01	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.555)	(0.611)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female) × years	-0.03	-0.03	-0.19**	-0.14*	-0.00	0.02	0.18	0.17
	(0.491)	(0.558)	(0.003)	(0.029)	(0.975)	(0.706)	(0.169)	(0.212)
Age × years	-0.01***	-0.01**	-0.01*	-0.01*	-0.01*	-0.01*	-0.01	-0.00
	(0.000)	(0.002)	(0.041)	(0.034)	(0.049)	(0.035)	(0.623)	(0.683)
Education × years		-0.01		-0.02		-0.01		-0.01
		(0.279)		(0.067)		(0.501)		(0.577)
Education^2 × years		*n.a.*		-0.00		*n.a.*		*n.a.*
		n.a.		(0.936)		*n.a.*		*n.a.*
Income × years		0.15		0.04		0.03		0.10
		(0.051)		(0.737)		(0.673)		(0.570)
Income^2 × years		-0.12		0.00		*n.a.*		*n.a.*
		(0.102)		(0.983)		*n.a.*		*n.a.*
Employed (Ref. not employed) × years		0.07		-0.03		-0.26*		0.42
		(0.492)		(0.838)		(0.036)		(0.192)
Growth rate (quadratic)								
Time^2 (years^2)	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.06*	0.06*
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.037)	(0.029)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.01	0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.680)	(0.755)
3rd quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.03	0.02
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.344)	(0.472)
4th quartile (t_1) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.01	-0.01
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.732)	(0.717)

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Table 4.A3 continued

Neighborhood urbanity × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	0.00	0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.536)	(0.554)
Male (Ref. female) × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.03	-0.02
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.221)	(0.307)
Age × years^2	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	-0.00	-0.00
	n.a.	*n.a.*	*n.a.*	*n.a.*	*n.a.*	*n.a.*	(0.843)	(0.760)
Education × years^2		*n.a.*		*n.a.*		*n.a.*		0.00
		n.a.		*n.a.*		*n.a.*		(0.732)
Income × years^2		*n.a.*		*n.a.*		*n.a.*		-0.01
		n.a.		*n.a.*		*n.a.*		(0.715)
Employed (Ref. not employed) × years^2		*n.a.*		*n.a.*		*n.a.*		-0.09
		n.a.		*n.a.*		*n.a.*		(0.095)
Random Variance Components								
Neighborhood level								
Intercept	*n.a.*[c]	0.00	0.00***	0.15	0.13	0.14	0.07	0.15*
Respondent level								
Intercept	0.79	0.64*	28.82***	23.56***	6.99***	5.37***	4.15***	3.58***
Slope (years)	0.10***	0.10***	0.17***	0.17***	*n.a.*	*n.a.*	0.04***	0.04***
Residual	1.92***	1.92***	4.85***	4.82***	5.63***	5.56***	2.68***	2.67***
$N_{observations}$	1,516	1,516	1,445	1,445	1,455	1,455	1,449	1,449
$N_{respondents}$	506	506	506	506	506	506	506	506
$N_{neighborhoods}$	59	59	59	59	59	59	59	59
Wald χ^2-tests for time trends								
p_{NSES}	0.12	0.12	0.91	0.75	0.35	0.38	0.24	0.27
$p_{Urbanity}$	0.73	0.61	0.57	0.80	0.87	0.76	0.61	0.66

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[a] To ensure comparability with the original model, a restricted maximum likelihood algorithm was used instead of a maximum likelihood algorithm.

[b] Model included a random slope for income at the neighborhood level: Slope(income)=1.84, p=.305.

[c] The models for this robustness check were specified identically to the original models. For MMSE model M1 however, the random variance components for the intercept at the neighborhood level had to be omitted to achieve model convergence.

MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

Table 4.A4: Multilevel linear regression models predicting baseline (t_1) levels and growth rates (t_1–t_3) for four cognitive functioning outcomes among 985 respondents aged 65 and older at t_1, accounting for practice effects by adding a dummy variable for t_1. Independent variables refer to t_1.
Unstandardized regression coefficients (*p*-values in parentheses).

	MMSE		Coding Task		RCPM		15WT	
	M1	M2	M1	M2	M1[a]	M2	M1	M2[b]
Fixed Effects								
Baseline								
Intercept	27.84***	28.07***	23.22***	25.45***	16.57***	17.37***	5.50***	5.78***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
NSES (Ref. 1st quartile)								
2nd quartile (t_1)	0.03	-0.03	1.32*	0.71	0.59	0.28	0.39	0.27
	(0.854)	(0.866)	(0.039)	(0.255)	(0.113)	(0.440)	(0.162)	(0.362)
3rd quartile (t_1)	0.26	0.08	2.11**	0.92	0.88*	0.26	0.44	0.22
	(0.193)	(0.702)	(0.003)	(0.180)	(0.026)	(0.493)	(0.138)	(0.485)
4th quartile (t_1)	0.30	0.05	2.89***	1.16	0.98*	0.11	0.52	0.25
	(0.127)	(0.804)	(0.000)	(0.076)	(0.013)	(0.782)	(0.077)	(0.419)
Neighborhood Urbanity	0.15***	0.10*	0.69***	0.35*	0.17*	0.03	0.18**	0.13*
	(0.001)	(0.024)	(0.000)	(0.013)	(0.043)	(0.747)	(0.005)	(0.044)
Neighborhood Urbanity^2	*n.a.*	*n.a.*	-0.33*	-0.28*	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.011)	(0.032)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female)	-0.01	-0.21	0.32	-1.05**	0.72**	0.08	-1.33***	-1.58***
	(0.894)	(0.055)	(0.392)	(0.003)	(0.002)	(0.735)	(0.000)	(0.000)
Age	-0.06***	-0.06***	-0.43***	-0.40***	-0.19***	-0.18***	-0.16***	-0.16***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Education		0.09***		0.60***		0.28***		0.08**
		(0.000)		(0.000)		(0.000)		(0.007)
Education^2		*n.a.*		-0.04***		*n.a.*		*n.a.*
		n.a.		(0.001)		*n.a.*		*n.a.*
Income		0.29		3.71***		1.18***		0.70*
		(0.140)		(0.000)		(0.000)		(0.014)
Income^2		-0.02		-1.33*		*n.a.*		*n.a.*
		(0.929)		(0.042)		*n.a.*		*n.a.*
Employed (Ref. not employed)		0.16		0.91		-0.03		0.31
		(0.530)		(0.288)		(0.948)		(0.433)

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Table 4.A4 continued

Growth rate (linear)								
Time (years)	-0.28***	-0.23***	-0.30***	-0.33***	-0.20**	-0.19**	0.04	0.04
	(0.000)	(0.000)	(0.001)	(0.001)	(0.005)	(0.009)	(0.442)	(0.456)
NSES (Ref. 1st quartile)								
2nd quartile (t_1) × years	0.00	-0.01	-0.04	-0.03	0.06	0.06	-0.05	-0.05
	(0.947)	(0.852)	(0.662)	(0.751)	(0.347)	(0.389)	(0.323)	(0.313)
3rd quartile (t_1) × years	0.03	0.01	0.01	0.04	0.12	0.12	-0.08	-0.08
	(0.671)	(0.887)	(0.876)	(0.688)	(0.086)	(0.087)	(0.126)	(0.133)
4th quartile (t_1) × years	-0.03	-0.06	-0.02	0.01	0.10	0.10	0.02	0.02
	(0.665)	(0.351)	(0.834)	(0.953)	(0.144)	(0.155)	(0.731)	(0.746)
Neighborhood Urbanity × years	-0.01	-0.02	-0.03	-0.02	0.01	0.01	-0.01	-0.01
	(0.344)	(0.148)	(0.110)	(0.184)	(0.419)	(0.500)	(0.227)	(0.226)
Neighborhood Urbanity^2 × years	*n.a.*	*n.a.*	-0.01	-0.01	*n.a.*	*n.a.*	*n.a.*	*n.a.*
	n.a.	*n.a.*	(0.523)	(0.532)	*n.a.*	*n.a.*	*n.a.*	*n.a.*
Male (Ref. female) × years	0.02	-0.00	-0.20***	-0.18**	0.00	0.01	0.03	0.04
	(0.654)	(0.976)	(0.000)	(0.001)	(0.920)	(0.827)	(0.317)	(0.274)
Age × years	-0.02***	-0.02***	-0.02***	-0.02***	-0.01	-0.01	-0.01*	-0.01**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.108)	(0.112)	(0.010)	(0.008)
Education × years		0.00		-0.01		-0.00		-0.00
		(0.669)		(0.274)		(0.821)		(0.493)
Education^2 × years		*n.a.*		-0.00		*n.a.*		*n.a.*
		n.a.		(0.866)		*n.a.*		*n.a.*
Income × years		0.17*		-0.01		0.05		0.04
		(0.016)		(0.883)		(0.397)		(0.311)
Income^2 × years		-0.15*		0.09		*n.a.*		*n.a.*
		(0.049)		(0.368)		*n.a.*		*n.a.*
Employed (Ref. not employed) × years		0.03		-0.05		-0.16		-0.06
		(0.760)		(0.701)		(0.143)		(0.481)
Practice Effect								
Dummy t_1	-0.30*	-0.30*	-0.27	-0.27	0.36	0.37	0.90***	0.89***
	(0.021)	(0.020)	(0.180)	(0.180)	(0.095)	(0.087)	(0.000)	(0.000)

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Table 4.A4 continued

Random Variance Components								
Neighborhood level								
Intercept	0.04***	0.06***	0.33	0.53	0.08	0.10*	0.07*	0.12**
Respondent level								
Intercept	0.96	0.81	30.01***	24.09***	7.59***	6.06***	4.25***	3.80***
Slope (years)	0.18***	0.17***	0.18***	0.18***	*n.a.*	*n.a.*	0.04***	0.04***
Residual	2.06***	2.06***	4.73***	4.73***	5.62***	5.59***	2.58***	2.59***
$N_{observations}$	2,463	2,463	2,332	2,332	2,347	2,347	2,337	2,337
$N_{respondents}$	985	985	985	985	985	985	985	985
$N_{neighborhoods}$	63	63	63	63	63	63	63	63
Wald χ^2-tests for time trends								
p_{NSES}	0.80	0.61	0.91	0.86	0.33	0.32	0.13	0.14
$p_{Urbanity}$	0.34	0.15	0.21	0.32	0.42	0.50	0.23	0.23

Note. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

[a] To ensure comparability with the original model, a restricted maximum likelihood algorithm was used instead of a maximum likelihood algorithm.

[b] Model included a random slope for income: Slope(income)=1.14, p=.811.

MMSE = Mini Mental State Examination; RCPM = Raven Colored Progressive Matrices; 15WT = 15 Words Test; NSES = neighborhood socioeconomic status; Ref. = reference category; n.a. = not applicable.

For this robustness check, we added a dummy variable to identify the first observation in our study. Practice effects would then be represented by a negative deviation at t_1 from the general pattern of cognitive decline. For 15WT however, simultaneous estimation of coefficients for years, years squared and the dummy variable for t_1 to assess practice effects was not possible. Thus, the models for 15WT consist of the linear term for years and the dummy variable for t_1.