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The mix matters: Complex personal networks relate to higher cognitive functioning in old age



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ABSTRACT

Stronger engagement of older adults in social activities and greater embeddedness in networks is often argued to buffer cognitive decline and lower risks of dementia. One of the explanations is that interaction with other people trains the brain, thereby enhancing cognitive functioning. However, research on the relationship between personal networks and cognitive functioning is not yet conclusive. While previous studies have focused on the size of personal networks as a proxy of cognitive stimulation, little attention has been paid to the complexity of the personal network. Adults embedded in a broad range of network relationships (i.e., various relationship types) are likely to be exposed to a wider range of stimuli than adults embedded in a homogeneous network including similar relationship types. We expect that higher numbers of personal relationship types rather than a higher number of similar contacts relate to higher levels of cognitive functioning and slower cognitive decline. Data are from the Longitudinal Aging Study Amsterdam (LASA) and include 2959 Dutch participants aged 54 to 85 at baseline in 1992 and six follow-ups covering a time span of twenty years. Cognitive functioning is assessed with the Mini-Mental State Examination (MMSE), and for network complexity we use the Social Network Index. We test our expectations using fixed-effects regression models. The results reveal that a reduction in network complexity is associated with a reduction in cognitive functioning, which is neither explained by size of the network nor by presence of specific relationship types. However, enhanced complexity has only a marginal buffering effect on decline in cognitive functioning. We conclude that network characteristics and cognitive functioning are intertwined and that their association is mostly cross-sectional in nature.

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1. Introduction

Scholars in gerontology hypothesize that older adults who are more socially embedded show less decline in cognitive functioning and decreased risk of dementia than those who are less socially embedded (Cacioppo et al., 2009; Ellwardt et al., 2013; Ertel et al., 2008; Fratiglioni et al., 2004; Wang et al., 2002; Wilson et al., 2007). One of the arguments is that integration into rich personal networks stimulates cognition and enhances neural plasticity in aging, which in turn preserves cognitive abilities (Hultsch et al., 1999; Katzman, 1995). Yet, empirical evidence of the association between large personal networks and cognitive functioning is not

conclusive. While some studies show positive relations (Barnes et al., 2004; Bassuk et al., 1999; Crooks et al., 2008; Ertel et al., 2008; Holtzman et al., 2004), others show only a moderating relation (Bennett et al., 2006; Hughes et al., 2008) or no relation (Helmer et al., 1999; Krueger et al., 2009; Seeman et al., 2001; Wilson et al., 2007). Other studies have demonstrated reversed causation (Aartsen et al., 2002, 2004; Hultsch et al., 1999), suggesting that changes in networks and reduction in number of relationships likely follow from progressing cognitive impairments. We argue that personal networks and cognitive functioning are closely intertwined, and that more conclusive insights may be reached by tapping into the underlying mechanisms.

One issue not sufficiently discussed is whether the size of the personal network is the best available indicator of social embeddedness. Importantly, little attention has been paid to the complexity of the personal network. Adults embedded in a broad range of network relationships (i.e., various relationship types) are likely to

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be exposed to a wider range of social activities and more complex stimuli than adults embedded in a homogeneous network including similar relationship types. Results from epidemiologic studies suggest that complexity in personal networks may be one of the drivers of preventing a broad range of negative health outcomes (Cohen and Janicki-Deverts, 2009)—including upper respiratory illness (Cohen et al., 1997), disability (Escobar-Bravo et al., 2012), blood pressure (Troxel et al., 2010), heart disease (Barefoot et al., 2005), stress (Haines and Hurlbert, 1992), and mortality (Berkman and Syme, 1979; House et al., 1988; Litwin and Shiovitz-Ezra, 2006). Likewise, complex leisure activities and mentally demanding jobs have been shown to increase intellectual stimulation in late adulthood and to relate to higher cognitive functioning (Bosma et al., 2003; Schooler and Mulatu, 2001). Given this overwhelming evidence, the absence of research into network complexity and cognition seems striking.

We opt for a framework more closely resembling the formerly suggested process of preserved cognitive abilities, which predicts an increased intellectual stimulation through a *variety* of social relationships. Personal networks in old age are heterogeneous (Aartsen et al., 2004; Fiori et al., 2007; Glass et al., 1997; Litwin and Shiovitz-Ezra, 2006; Van Tilburg, 1998) and differ considerably in their network complexity, defined as the number of different social relationship types (Cohen et al., 1997). While it has been emphasized that relationship types have differential impacts on cognitive decline (Giles et al., 2012), the importance of *mixing* diverse types has remained unaddressed. In the present study we investigate: How can variations in cognitive functioning and age-related decline thereof be understood from differences in network complexity? We expect that greater complexity in networks relates to higher cognitive functioning and reduced cognitive decline.

A main reason for the former inconsistencies and the neglect of personal network composites is the scarcity of appropriate data. Studies thus often rely on global measures like friend counts and social support (Amieva et al., 2010; Ellwardt et al., 2013; Holtzman et al., 2004), or use marital status and organizational membership as proxies of social integration (Fratiglioni et al., 2000; Helmer et al., 1999; Wang et al., 2002). The present study is based on a longitudinal dataset with a comprehensive delineation of the personal network (Van Tilburg, 1998), allowing for analyses on network complexity.

2. Theory and evidence

2.1. Why network size matters (less)

Since Berkman and Syme's influential article in 1979 (Berkman and Syme, 1979), social integration into personal networks has been widely recognized as a powerful promoter of physical and mental health. Proposed benefits of networks include the provision of social support, social influence, social engagement and access to resources and material goods (Berkman et al., 2000). Larger networks presumably offer more of these benefits. However, because the underlying mechanisms likely co-occur and are difficult to empirically disentangle, in many studies it is hardly certain which mechanism is at play. In a similar vein, being embedded in large personal networks is believed to enhance cognitive functioning in older adults (Fratiglioni et al., 2004; Holtzman et al., 2004), as it was shown to increase social activities (James et al., 2011; Krueger et al., 2009; Wang et al., 2002; Zunzunegui et al., 2003), reduce stress (Dickinson et al., 2011; Wilson et al., 2011), and provide intellectual stimulation (Hultsch et al., 1999). But again, size of the network is only indirectly argued to relate to cognitive outcomes and multiple explanations of the underlying mechanism are possible.

We argue that a model solely relying on the size of the personal network is underspecified. On the one hand, the formerly proposed mechanisms do not necessarily rest on network size. Large networks can be ineffective if contacts are not useful or ambivalent (Uchino et al., 2004). On the other hand, in later life personal networks typically shrink and change in their composition (Broese van Groenou et al., 2013; Carstensen, 1993; Wrzus et al., 2013). Transitions often result from changing needs that come with life events (e.g., widowhood; Antonucci et al., 2001; Guiaux et al., 2007; Zettel and Rook, 2004), physical disability and cognitive impairments (Aartsen et al., 2004; Green et al., 2008), so that a reversed mechanism is likely observed at the same time. This underspecification is partly responsible for the lack of substantiated evidence on the relationship between social integration and cognition in older adults. Research inquiring this relationship therefore needs to capture the multidimensional facets of the personal network, most importantly its composition.

2.2. Why network complexity matters (more)

A widely overlooked but consequential network composite is complexity, defined as the total number of distinct social roles—like being relative, friend, neighbor, former colleague—in which a person has regular contact with (Cohen et al., 1997). Although this composite bases on size of the personal network (larger networks allow greater complexity), it embraces more on the aspects of aging networks. On the one hand, from previous research we know that personal networks of older adults are differentiated (Fiori et al., 2007; Litwin and Shiovitz-Ezra, 2006; Van Tilburg, 1998), e.g., women retain friendships longer than men (Stevens and Van Tilburg, 2011). On the other hand, the composition of the network was shown to change in late life, e.g., in large networks friendship relations are replaced by family and neighbor relations (Aartsen et al., 2004; Carstensen, 1993).

An analysis incorporating also structural aspects of the personal network permits a more rigorous investigation of the *use-it-or-lose-it hypothesis* (Bassuk et al., 1999; Hultsch et al., 1999). It states that not using and challenging the brain may lead to atrophy in cognitive capacities, whereas the deliberate practice of cognitive skills can preserve, manifest and enhance performance. Increased task difficulty may even result in recruitment of additional brain areas, so that an individual with more reserve might be able to call on a larger array of alternate networks and thereby delay the time it takes to show impairments (Stern, 2002). Based on this hypothesis, improved cognitive functioning is not only achieved through greater number of relationships, but primarily through the complexity that the set of these relationships brings about.

In a similar vein, the *environmental complexity hypothesis* (Schooler, 1987) expects that complex environments—characterized by diverse stimuli, socializing, coordination of multiple decision making processes and sometimes contradictory contingencies—promote brain activation due to their cognitively challenging nature. Three classes of biological mechanisms have been theorized: enriched environments imply reduction in neuronal loss, provision of compensatory strength in case of neuropathologic damage and modification of Alzheimer's disease (Kempermann et al., 2002; Valenzuela et al., 2012). Together these mechanisms should lead to a greater number of healthy neurons in brain regions most affected by Alzheimer's disease, better adaptation to brain atrophy and memory dysfunction, and less Alzheimer's disease pathology at death.

In support of the latter hypothesis, biological studies have shown protective effects of complex environments on memory, neurogenesis and neural function in humans (Valenzuela et al., 2012). Experimental research in neurology has demonstrated

enhanced cognitive control abilities and boosted task performance in older adults who participated in effortful video gaming and multitasking training as compared to non-participating older adults (Anguera et al., 2013). Similarly, carrying out mentally demanding jobs and pursuing complex leisure activities have been found to reduce the risk of cognitive impairments (Bosma et al., 2003; Schooler and Mulatu, 2001; Schooler et al., 1999).

Based on these insights, we expect that integration into complex networks increases the likelihood of being stimulated, and thus the preservation of cognitive functioning (Hultsch et al., 1999). Such networks more likely expose individuals to rich, cognitively challenging environments than less complex networks. Some relationship types will generate more intellectual engagement than others, and the intellectual value attributed to each of the relationship types will vary across individuals. A homogeneous network consisting of a small number of relationship types captures only certain social domains (e.g., only family), which may result in intense interaction though limited cognitive stimulation. In contrast, a network with multiple relationship types covers different social domains and life spheres (e.g., family, neighborhood, organizations), thereby providing older adults with a broader range of social activities and cognitive input (e.g., taking care of grandchildren, volunteering). A trade-off effect of complex networks is that they require coordinating and switching between a variety of social roles, interactions and contexts. This trains the ability to process, memorize and recall information about relatively unconnected people and events. In networks of homogeneous contacts, the same social context may be shared among many contacts, which further adds to the chances of intense but redundant stimulation.

In addition to these direct influences, complex networks may contribute to cognitive functioning via other important mechanisms (Berkman et al., 2000). Greater variety in relationship types potentially carries more access to and greater diversity in resources of social support. A diverse choice of resources better allows the individual to select optimal support when a needy situation occurs.

We are aware of only one recent study that explicitly addresses how different types of networks relate to memory. In that research, Giles et al. (2012) classified personal networks of older adults into four network types (children network, relatives' network, friends' network, and confidants' network) and subsequently modeled memory decline for each type separately. Having a network of friends was most protective against memory loss, suggesting a differential impact of social roles on cognition. Yet, this classification technique does not permit conclusions about the overall complexity in personal networks.

Based on our discussion, we hypothesize: *Greater complexity in the personal network is associated with higher levels of cognitive functioning and slower decline in cognitive functioning. These effects are independent of size of the personal network.*

3. Data and methods

3.1. Respondents

Data were derived from the Longitudinal Aging Study Amsterdam (LASA), an ongoing longitudinal, multidisciplinary research project focused on physical, cognitive, social and emotional functioning of the aging population (Huisman et al., 2011). This program employed a stratified random sample of men and women born between 1908 and 1937. The oldest participants, particularly the men, were over-represented in the sample. The sample was taken from population registers of 11 municipalities, varying in religion and urbanization. The LASA sample was initially recruited for the Living Arrangements and Social Networks of Older Adults (LSN)

research program (Knipscheer et al., 1995). Of the 6107 eligible individuals in the LSN sample, 2302 (38%) were unwilling to participate due to lack of interest or time; another 734 had died or were too ill or cognitively impaired to be interviewed. A total of 3107 LSN sample respondents took part in the first LASA observation (1992–1993). Follow-ups were conducted in 1995–1996 ($N = 2545$), 1998–1999 ($N = 2076$), 2001–2002 ($N = 1691$), 2005–2006 ($N = 1257$), 2008–2009 ($N = 835$) and 2011–2012 ($N = 764$). For each follow-up, on average 81% of the respondents were re-interviewed, 12% had died, 2% were too ill or too cognitively impaired to be interviewed, 5% refused to be re-interviewed, and less than 1% could not be contacted due to a residential relocation to another country or an unknown destination. We missed network data for 147 respondents due to their incapacity to undergo a full interview at any observation, and one respondent who did not answer questions on memory.

On average, 3.5 observations on networks and memory were available for each respondent ($N = 2959$). The pooled data set included 10,376 observations. Reasons for missing data at follow-up were premature termination of an interview or item non-response (<1%), use of an abridged version of the questionnaire at a specific observation (5%), or a telephone interview for respondents who were too frail to be interviewed with the full questionnaire (8%).

The age of the 1433 male and 1526 female respondents varied between 54 and 100 years ($M = 73.9$, $SD = 8.5$) at the time of the observation. Respondents were followed for a maximum of 19.8 years ($M = 6.2$; $SD = 5.8$). From observation to observation, we had an increasingly selective sample composition. Respondents for whom no follow-up data is available when compared with respondents with follow-up data were more often male (odds ratio, $OR = 1.18$, $CI = 1.07, 1.30$; Wald = 11.4, $p < .001$), and they were older ($OR = 1.07$, $CI = 1.06, 1.07$; Wald = 404.9, $p < .001$), were lower on memory (MMSE-score; $OR = 0.85$, $CI = 0.83, 0.87$; Wald = 311.7, $p < .001$) and had a smaller network ($OR = 0.99$, $CI = 0.98, 0.99$; Wald = 17.0, $p < .001$) in the previous observation.

3.2. Measures

3.2.1. Cognitive functioning

The Mini-Mental State Examination (MMSE) is a global indicator of cognitive functioning. This index is mainly used as a screening instrument for cognitive decline and dementia. It involves indications of memory, orientation, registration, attention, language, and construction. Scale scores range from 0 to 30. Higher scores on the MMSE indicate better cognitive performance. The traditional cut-point is <24, but a cut-point of <23 is also used as indicative for dementia (Cullen et al., 2005). Altogether, the MMSE is judged to assess the severity of cognitive impairment and cognitive changes satisfactorily (Tombaugh and McIntyre, 1992).

3.2.2. Personal network

In each observation, a domain-specific approach for network delineation was employed that encompasses the following classification of personal relationships: household members, children and their partners, other family members, neighbors, contacts through work and school, members of associations, and other non-kin relationships. For each of the seven domains the following question was asked: "Name the people you have frequent contact with and who are also important to you" (Van Tilburg, 1998). The criteria of importance was left to the interpretation of the respondent and only persons older than age 18 could be considered. Next, for each generated name, it is asked what type of relationship the respondent had with this person, and how often the

respondent had contact with this person. The identification method was similar across observations.

Network size included the total count of all the members in the personal network, including the partner, if there was one.

Network complexity was measured with Cohen's Social Network Index (Cohen et al., 1997). This is the number of social roles in which a respondent has regular contact with at least one person. Regular contact is defined as contacting a person at least once every two weeks. Less regular contacts are not considered in this index. Next, these regular contacts are classified into 13 different social roles: spouse, child, child-in-law, sibling, sibling-in-law, parent, relative, close friend, acquaintance, neighbor, (former) colleague, voluntary organization, other group member. Respondents receive one point for every role that is covered by their contacts. This means that per role only one point is given, even if multiple relationships fit in. A higher sum score indicates a greater complexity in the personal network. Note that the Cohen classification was slightly modified to better represent typical roles of older adults in the Netherlands (i.e., there are hardly contacts from school, and there is a clear distinction between acquaintances and close friends). Because the maximum category of 11 social roles contained very few observations ($N = 5$), the categories of 10 and 11 social roles were merged prior to the analyses.

3.2.3. Control variables

The analyses controlled for time since baseline (0–20 years), age at baseline, gender (1 = female), educational level, living with a partner (1 = yes), and physical functioning. These variables were assessed at all seven time points of observations. For an easier interpretation of the regression coefficients, the time variable was rescaled into decades ($\text{time}_{\text{decades}} = \text{time}_{\text{years}}/10$) beforehand in the analyses. To measure physical functioning, respondents were asked six questions about activities of daily living (Katz et al., 1963), such as "Can you walk up and down stairs". Possible answers included (1) not at all, (2) only with help, (3) with a great deal of difficulty, (4) with some difficulty, and (5) without difficulty. Scale reliability was 0.83, and a sum score was obtained. Because the resulting scale was negatively skewed, a log-transformed version was used. High scores indicated good functional capacities. Time, network complexity and network size were centered prior to the analyses.

3.3. Analytical approach

The data consisted of repeated measurements, that is the respondents' characteristics had been observed at multiple time points. This means a total of 10,376 observations were nested in 2959 respondents. Nested data is typically modeled with random-effects regression models that include a random intercept for each subject, thereby allowing subjects to vary in their level of cognitive functioning. These *random-intercept models*, which were estimated with the maximum likelihood procedure, compared network complexity and cognitive functioning between subjects.

Importantly, based on these longitudinal data it was also possible to assess whether variations in a respondent's network complexity were related to variations in the same respondent's level of cognitive functioning. A powerful tool to model such within-subject variability is the *fixed-effects model* (Rabe-Hesketh and Skrondal, 2012, p. 228). The interpretation of this model, which is often referred to as the differencing model, is that changes in the time-varying covariate (i.e., network complexity) between time $t - 1$ and t may affect changes in the time-varying outcome (i.e., cognitive functioning) over the same time period $t - 1$ to t . A major advantage is that the fixed-effects model rules out unobserved heterogeneity between subjects when this heterogeneity is constant over time, and thereby eliminates subject-level

confounding. This implied that controlling for time-invariant covariates like gender, age and educational level was neither necessary nor possible in the fixed-effects models.

Furthermore, we were interested in longitudinal effects of network complexity on *decline* in cognitive functioning over time (i.e., with increasing age). First, to assess whether network complexity had the potential to buffer age-related decline in cognitive functioning, we tested an interaction effect between network complexity and time since baseline. The same was done for network size. Second, we carried out additional analyses employing lagged covariates. Specifically, the outcome cognitive functioning at a time point t was regressed on network complexity at a time point $t - 1$ prior to that. The analyses controlled for network complexity at time point t , and previous level of cognitive functioning at a prior time point $t - 1$. This model has the same differencing interpretation as the original fixed-effects model, except that changes in the covariate between time $t - 1$ and t (an earlier period) may affect changes in the outcome between time t and $t + 1$ (a later period). The latter analyses only included respondents who participated in at least two subsequent survey rounds ($N = 2201$).

4. Results

4.1. Descriptive results

The median number of observations per respondent was 3 (IQR, 2–6), and the median number of follow-up years since baseline was 9 (IQR, 3–16). At baseline, the median MMSE was 28 (IQR, 26–29). Many older adults in our data experienced slight cognitive decline and few adults experienced severe decline: Of 300 respondents who started with an MMSE of 30 points and who had at least one follow-up interview, 173 respondents experienced a decline of 3 points or greater (52.4%), while 25 respondents (7.6%) experienced a decline of 8 points or greater. There were 17 respondents (5.2%) whose scores remained stable at 30 for all available data points. Respondents had an average of 4.5 social roles ($SD = 1.8$) in their personal networks. The most frequently mentioned contacts by respondents included children (82%), the spouse (67%), children-in-law (62%), neighbors (56%) and siblings (40%) at baseline. Table 1 presents the descriptive statistics and the correlations between all variables.

4.2. Hypothesis test

We hypothesized that individuals with more complex networks show higher cognitive functioning and slower cognitive decline than their counterparts with less complex networks. Table 2 presents the estimates of the random-intercept regression models, which compare network complexity and cognitive functioning between individuals. Individuals with more complex networks scored higher on the MMSE index than individuals with less complex networks. Furthermore, the interactions of the network variables with time were of substantive interest: Model 6 shows that older adults experienced lower cognitive functioning as time had passed, and that this time effect was weaker for older adults embedded in more complex networks than for those embedded in less complex networks.

A Hausman specification test revealed that the coefficients generated by the random-intercept models differed systematically when a fixed-effects approach was applied instead ($\chi^2 = 355.70$, $p < .001$, using Model 4). We therefore proceed with the more consistent estimates from the fixed-effects models, which compare changes within individuals.

Table 3 summarizes the results from the fixed-effects regression models. Likelihood-ratio tests revealed that each addition of

Table 1Descriptive statistics and correlations between the variables across all waves (pooled sample, $N_{\text{observations}} = 10,376$).

	M	SD	Time	Age	Gender	Education	Partner	Physical functioning	Cognitive functioning	Network complexity
Time	3.58	6.05								
Age at baseline	67.69	8.27	−0.31***							
Gender (1 = female)	53.86%	–	0.04***	0.01						
Education	3.60	1.99	0.07***	−0.13***	−0.22***					
Partner	0.63	0.48	−0.08***	−0.29***	−0.35***	0.14***				
Physical functioning ^a	2.22	0.98	0.09***	−0.37***	−0.18***	0.18***	0.29**			
Cognitive functioning	27.08	2.80	0.01	−0.33***	−0.01	0.26***	0.16***	0.27***		
Network complexity	4.59	1.85	0.03**	−0.28***	0.03**	0.02	0.29***	0.19***	0.21***	
Network size	15.16	8.75	0.06***	−0.21***	−0.01	0.12***	0.22***	0.17***	0.20***	0.61***

^a Log-transformed. * $p < .05$, ** $p < .01$, *** $p < .001$.

the variables carried an improvement in model fit (χ^2 between 8.66 and 70.34, $p < .003$). Estimates for the control variables in Model 1 suggest that cognitive functioning declined as age increased (i.e., time elapsed since baseline) and physical functioning decreased.

Model 2 demonstrates a small positive association of differences in the size of the personal network with differences in cognitive functioning. In support of our expectation, older adults reporting declines in the variety of social roles in their network also exhibited declines over time in their cognitive functioning (Model 3). This effect was independent of changes in the size of the personal network (Model 4). Note that controlling for network size attenuated the effect estimate for network complexity, suggesting that the influence of changes in network complexity was still sensitive to changing network size. Fig. 1 illustrates the predictive margins of cognitive decline by changes in number of social roles in the personal network.

The positive and significant interaction of network complexity with time (Model 6) suggested that age-related decline in cognitive functioning was dampened as number of social roles remained high or became higher. Table A1 in the Online Supplemental Material presents the predictive margins for number of years since baseline (i.e., time) and the different values of network complexity based on the final model including all variables (Model 7). It shows that respondents with greatest network complexity had both highest MMSE scores and smallest differences in MMSE scores during the course of the follow-up study. For example, respondents with two roles were predicted to experience a drop of more than two score points (27.4–25.3), whereas their counterparts with eight roles would experience a drop of less than one point (27.8–27.0). Fig. 2 illustrates the interaction finding: There was slightly faster decline in cognitive functioning for older whose personal networks became less complex over time.

4.3. Additional analyses

4.3.1. Lagged covariates

To further investigate the longitudinal effects of network complexity on cognitive decline, we performed additional analyses including time lags between the variables of interest. Specifically, the fixed-effects regression models used cognitive functioning at a time point t as the outcome variable and network complexity at a prior time point $t - 1$ as the predictor variable. Model 3 in Table 4 shows a small positive effect of differences in network complexity after $t - 1$ on differences in cognitive functioning after t , further supporting the notion that an increased number of social roles potentially buffered cognitive decline. However, changes in network complexity after $t - 1$ had a weaker association with changes in cognitive functioning than had changes in network complexity after t , implying that the cross-sectional association was stronger than the longitudinal association.

4.3.2. Non-linearity

Next, we carried out tests of non-linearity. Based on the theory, we assumed that complex networks provided stimulation and would therefore be associated with high levels of cognitive functioning. It could be argued, however, that once a certain level of complexity is achieved, additional increases in complexity may yield fewer differences in stimulation. To account for the possibility of a curvilinear relationship, we added squared effects to the final model (Model 7). There was a small and negative parameter estimate ($B = -0.02$, $CI = -0.03, -0.01$) indicating that the effect of increased network complexity on cognitive decline slightly extenuated towards large increases in the network. No such effect was found for network size.

4.3.3. Separate roles

We wanted to rule out that presence of specific relationship types in the network was responsible for the positive association between changes in network complexity and cognitive functioning. We therefore reran the final model including the separate social roles. This resulted in a series of 13 models, with every model testing one of the 13 social roles. Only changes in three roles were associated with changes in cognitive functioning: keeping parents ($B = -0.38$, $CI = -0.59, -0.16$) and colleagues ($B = -0.17$, $CI = -0.34, -0.01$) related to less cognitive decline, while losing neighbors ($B = 0.13$, $CI = 0.01, 0.24$) related to more cognitive decline. Still, regardless of the roles' effects, the significant positive effect of network complexity and its interaction with time continued to exist unmodified across all models.

4.3.4. Predictor variable

We examined the robustness of our results against alternative operationalizations of network complexity. Network complexity was measured as the number of social roles in which a respondent had *bi-weekly* contact with at least one person. Two alternative operationalizations of network complexity were tested, of which both deviated from the original measure only with respect to the regularity of a contact: The first operationalization solely included very regular contacts in the index—the number of social roles in which a respondent had *weekly* contact with at least one person. The second operationalization included all contacts in the index—the number of social roles in which a respondent had *any* contact with at least one person. Overall, the results did not substantially differ from the original findings, so that our findings appeared to be relatively robust against alternative definitions of network complexity. These results are presented in the Online Supplemental Material.

4.3.5. Outcome variable

Finally, the MMSE index was a global measure of cognitive functioning indicative of demented diseases. We were interested whether our findings would also hold for more specific outcomes of

Table 2
Random-intercept regression models on cognitive functioning (MMSE).^a

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)
Controls							
Time (in decades) ^b	−0.658*** (−0.734, −0.582)	−0.655*** (−0.730, −0.580)	−0.636*** (−0.711, −0.560)	−0.641*** (−0.716, −0.566)	−0.648*** (−0.724, −0.573)	−0.650*** (−0.725, −0.574)	−0.651*** (−0.726, −0.576)
Age at baseline	−0.109*** (−0.119, −0.098)	−0.105*** (−0.115, −0.094)	−0.101*** (−0.112, −0.091)	−0.101*** (−0.112, −0.091)	−0.102*** (−0.112, −0.091)	−0.102*** (−0.113, −0.092)	−0.102*** (−0.113, −0.092)
Gender (1 = female)	0.368*** (0.193, 0.543)	0.332*** (0.158, 0.505)	0.309*** (0.135, 0.482)	0.304*** (0.130, 0.477)	0.303*** (0.130, 0.477)	0.306*** (0.132, 0.479)	0.305*** (0.132, 0.479)
Educational level	0.362*** (0.319, 0.405)	0.348*** (0.305, 0.390)	0.364*** (0.321, 0.407)	0.354*** (0.312, 0.397)	0.356*** (0.313, 0.399)	0.355*** (0.312, 0.397)	0.355*** (0.313, 0.398)
Partner	0.166* (0.031, 0.301)	0.083 (−0.052, 0.218)	0.037 (−0.099, 0.173)	0.025 (−0.111, 0.161)	0.027 (−0.109, 0.163)	0.032 (−0.104, 0.168)	0.031 (−0.105, 0.167)
Physical functioning	0.272*** (0.210, 0.334)	0.257*** (0.195, 0.318)	0.257*** (0.196, 0.319)	0.252*** (0.190, 0.314)	0.248*** (0.187, 0.310)	0.247*** (0.185, 0.309)	0.247*** (0.185, 0.308)
Predictors							
Network size		0.032*** (0.026, 0.038)		0.021*** (0.013, 0.028)	0.020*** (0.013, 0.027)	0.021*** (0.013, 0.028)	0.020*** (0.013, 0.028)
Network complexity			0.159*** (0.130, 0.189)	0.110*** (0.075, 0.144)	0.111*** (0.077, 0.145)	0.108*** (0.074, 0.142)	0.109*** (0.075, 0.143)
Network size × time ^b					0.012** (0.005, 0.019)		0.005 (−0.004, 0.014)
Network complexity × time ^b						0.072*** (0.035, 0.108)	0.057* (0.012, 0.102)
<i>Intercept</i>	32.062*** (31.211, 32.914)	31.946*** (31.102, 32.790)	31.718*** (30.873, 32.564)	31.753*** (30.909, 32.597)	31.788*** (30.942, 32.633)	31.829*** (30.983, 32.676)	31.828*** (30.982, 32.675)
<i>N</i> _{observations}	10,376	10,376	10,376	10,376	10,376	10,376	10,376
<i>N</i> _{individuals}	2959	2959	2959	2959	2959	2959	2959
<i>Var</i> _{observations}	1.97	1.94	1.94	1.93	1.94	1.94	1.94
<i>Var</i> _{individuals}	1.84	1.83	1.83	1.83	1.83	1.82	1.82
Log likelihood	−23,224.51	−23,173.21	−23,169.37	−23,153.72	−23,148.77	−23,146.23	−23,145.68

^a Unstandardized coefficients (B) and 95% confidence intervals (CI).

^b Time was measured in years since baseline, divided by 10. * $p < .05$, ** $p < .01$, *** $p < .001$.

cognitive functioning, such as information processing speed and immediate recall and learning. Two sets of fixed-effects regression models were estimated: one set on information processing speed as the outcome, measured with the Coding Task, and another set on recall and learning as the outcome, measured with the 15 Words Test. Note that both outcome measures were not assessed among all respondents, so that the models used 2659 individuals and a total of 8271 observations. A reduction in network complexity was related to a deterioration of the performance in both the Coding Task and the 15 Words Test, and there was a small and significant interaction effect with time since baseline, suggesting slightly slower decline in older adults who retained complex networks. Altogether these results resembled the findings from the analyses based on the MMSE index. The description of the measures and the corresponding results tables and figures are presented in the [Online Supplemental Material](#).

5. Discussion

Building on the *use-it-or-lose-it hypothesis* (Hultsch et al., 1999) and on the *environmental complexity hypothesis* (Schooler, 1987), we suggested that greater network complexity (i.e., having a variety of social relationships or roles in the personal network) provides older

adults with enhanced intellectual engagement (Giles et al., 2012; Stephens et al., 2011), thereby stimulating neural activity and maintaining cognitive capabilities in the long run. This expectation was tested with unique data from the Longitudinal Aging Study Amsterdam (LASA), covering information on the personal network and cognitive functioning over a period of twenty years.

In our investigation, older adults reporting greater number of relationship types in their network were characterized by higher cognitive capacities—both in global and in specific terms—than their counterparts reporting fewer types. Importantly, a reduction in network complexity was related to a decline in cognitive functioning. These findings were neither explained by size of the personal network nor by presence of specific relationship types in the network, which supports our notion that complexity may carry additional benefits based on the mechanism of activated brain reserves. There was also indication for a buffering effect of network complexity on cognitive change over time. This effect, however, was not substantial as differences in decline appeared to be rather marginal across different levels of network complexity. We conclude that network complexity and cognitive functioning are intertwined and that their association is mostly cross-sectional in nature. More research is needed to assess the extent to that characteristics of the personal network can preserve cognitive

Table 3
Fixed-effects regression models on cognitive functioning (MMSE).^a

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)	B (CI)
Controls							
Time (in decades) ^b	-0.793*** (-0.874, -0.712)	-0.782*** (-0.863, -0.701)	-0.767*** (-0.848, -0.686)	-0.767*** (-0.848, -0.686)	-0.788*** (-0.869, -0.707)	-0.791*** (-0.872, -0.710)	-0.795*** (-0.877, -0.714)
Partner	0.155 (-0.017, 0.326)	0.115 (-0.057, 0.286)	0.085 (-0.087, 0.257)	0.078 (-0.093, 0.250)	0.076 (-0.095, 0.248)	0.080 (-0.091, 0.252)	0.079 (-0.093, 0.250)
Physical functioning	0.160*** (0.085, 0.235)	0.153*** (0.078, 0.228)	0.152*** (0.077, 0.227)	0.150*** (0.075, 0.225)	0.138*** (0.063, 0.213)	0.136*** (0.061, 0.211)	0.133*** (0.058, 0.208)
Predictors							
Network size		0.025*** (0.017, 0.032)		0.016*** (0.008, 0.024)	0.014** (0.005, 0.022)	0.015*** (0.007, 0.024)	0.014*** (0.006, 0.022)
Network complexity			0.124*** (0.090, 0.158)	0.089*** (0.051, 0.128)	0.090*** (0.051, 0.128)	0.077*** (0.039, 0.116)	0.081*** (0.043, 0.120)
Network size × time ^b					0.022*** (0.014, 0.030)		0.012* (0.003, 0.022)
Network complexity × time ^b						0.117*** (0.079, 0.155)	0.082*** (0.035, 0.129)
Intercept	26.630*** (26.432, 26.828)	26.670*** (26.473, 26.868)	26.691*** (26.494, 26.889)	26.700*** (26.503, 26.898)	26.722*** (26.525, 26.920)	26.726*** (26.529, 26.923)	26.731*** (26.533, 26.928)
Nobservations	10,376	10,376	10,376	10,376	10,376	10,376	10,376
Nindividuals	2959	2959	2959	2959	2959	2959	2959
Varobservations	1.80	1.80	1.80	1.80	1.79	1.79	1.79
Varindividuals	2.87	2.83	2.83	2.82	2.84	2.84	2.84
Log likelihood	-19,103.14	-19,071.98	-19,067.97	-19,057.49	-19,035.70	-19,031.77	-19,027.44

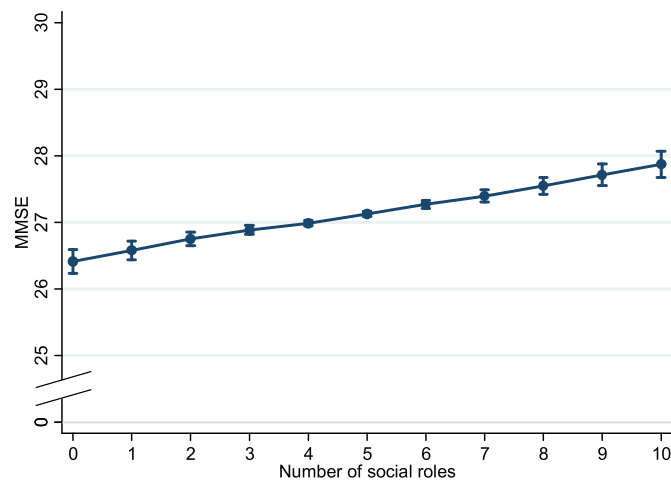
^a Unstandardized coefficients (B) and 95% confidence intervals (CI).
^b Time was measured in years since baseline, divided by 10. **p* < .05, ***p* < .01, ****p* < .001.

capabilities and postpone the onset of normal or pathological cognitive decline in older age.

Still, the findings presented emphasize that complex networks with diversified social relationships may contribute to enriched environments. These environments are cognitively demanding, they require handling and switching between multiple contexts, and facilitate training of brain activities vital to neural plasticity

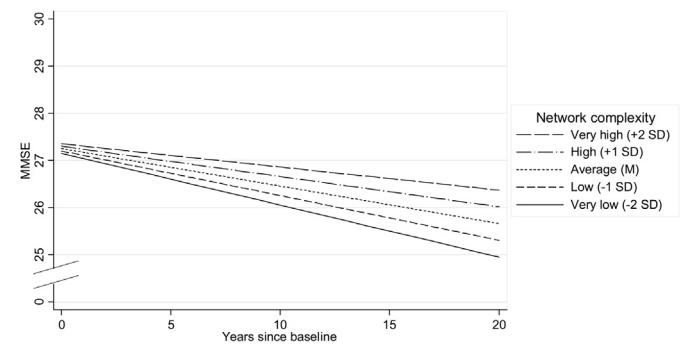
(Schooler, 1987; Valenzuela et al., 2012). It seems that benefits are not only obtained by the integration into large personal networks, but by the integration into certain network structures.

A cautious note on reversed causation should be added. Personal networks often change and shrink in later life (Broese van Groenou et al., 2013; Wrzus et al., 2013). Previous research has shown that such decrease in size of personal networks likely follows from deterioration in cognitive and physical functioning (Aartsen et al., 2002, 2004). We argue that changes in network characteristics and cognitive functioning are interlocked and hence reinforce one



Note. ^a Estimates base on the full fixed-effects model (Model 7).

Fig. 1. Predicted margins of changes in cognitive functioning (MMSE) by changes in number of social roles.



Note. ^a Respondents were grouped into those with average level of network complexity (mean), very high (+2 standard deviation), high (+1 standard deviation), low (-1 standard deviation) and very low (-2 standard deviation) level of network complexity.

Fig. 2. Changes in cognitive functioning (MMSE) over time, by different levels of network complexity.

Table 4
Fixed-effects regression models on cognitive functioning including lagged variables (at $t - 1$).^a

	Model 1	Model 2	Model 3	Model 4
	B (CI)	B (CI)	B (CI)	B (CI)
Controls				
Cognitive functioning at $t - 1$	−0.069*** (−0.102, −0.036)	−0.074*** (−0.107, −0.041)	−0.072*** (−0.105, −0.039)	−0.074*** (−0.107, −0.041)
Time (in decades) ^b	−0.844*** (−0.958, −0.729)	−0.827*** (−0.941, −0.712)	−0.804*** (−0.918, −0.689)	−0.805*** (−0.920, −0.691)
Partner	0.245* (0.031, 0.459)	0.191 (−0.023, 0.405)	0.169 (−0.045, 0.384)	0.152 (−0.062, 0.367)
Physical functioning	0.186*** (0.092, 0.280)	0.182*** (0.088, 0.275)	0.183*** (0.090, 0.277)	0.181*** (0.087, 0.275)
Predictors				
Network size		0.021*** (0.013, 0.030)		0.013** (0.003, 0.023)
Network size at $t - 1$		0.017*** (0.009, 0.026)		0.015** (0.005, 0.024)
Network complexity			0.119*** (0.077, 0.160)	0.090*** (0.044, 0.136)
Network complexity at $t - 1$			0.055** (0.014, 0.096)	0.023 (−0.023, 0.069)
Intercept	28.693*** (27.746, 29.639)	28.846*** (27.901, 29.791)	28.801*** (27.857, 29.745)	28.878*** (27.934, 29.822)
$N_{\text{observations}}$	7186	7186	7186	7186
$N_{\text{individuals}}$	2201	2201	2201	2201
$Var_{\text{observations}}$	1.76	1.75	1.75	1.75
$Var_{\text{individuals}}$	2.98	2.94	2.93	2.92
Log likelihood	−12,932.86	−12,906.95	−12,906.56	−12,896.09

^a Unstandardized coefficients (B) and 95% confidence intervals (CI).

^b Time was measured in years since baseline, divided by 10. * $p < .05$, ** $p < .01$, *** $p < .001$.

another simultaneously. Yet, because these changes may root in different explanations—e.g., deficient stimulation versus impaired health—reversed causation is unlikely to outweigh the added value of complexity in personal networks on cognitive functioning altogether. Shrinking networks, if diverse, still yield sources of intellectual engagement and stabilize individuals at risk of cognitive impairment.

Besides its contributions, the present research permits limited interpretations. First, the association between changes in network complexity and cognitive functioning over time was relatively weak, which suggests that the mechanism of environmental enrichment may not have been captured fully in our study. The data contained no information on the actual level of stimulation and activation by the (diverse) contacts in a network. Because of this it is desirable to tap into the causal effect of network complexity on cognition. Second, while the variety of social roles is potentially a better proxy for a complex environment than size of the network, it is still limited. Depending on the context, it is possible to receive more cognitive stimulation from one social role rather than multiple roles. Not all relationship types may be equally intellectually engaging and enriching, and their impact on cognitive functioning is likely differential. Friendship networks, for instance, most profoundly prevented cognitive decline in previous research (Giles et al., 2012). Finally, more research is needed into other dimensions of complexity. This could feature the diversity of individual attributes—such as age—in the personal network; for example, a network mixing young and old people might generate more stimulation than a network containing old people only.

Based on this study, preventive strategies may aim at restoring and expanding complexity in networks of older adults who are exposed to elevated risks of cognitive impairment and have relatively few social roles, and at stabilizing role diversity in the remaining older adults. Personal networks have been an established component of the research agenda on cognition and other health outcomes. But relatively little tribute has been paid to the composites of personal networks. We demonstrated that network complexity deserves to be part of this agenda.

Appendix A. Supplementary data

Supplementary data related to this article can be found at <http://dx.doi.org/10.1016/j.socscimed.2014.05.007>.

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