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A longitudinal study of the impact of social network size and loneliness on cognitive performance in depressed older adults

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ABSTRACT

Objectives: To examine the association of social network size and loneliness with cognitive performance and -decline in depressed older adults.

Method: A sample of 378 older adults [70.7 (7.4) years] with a *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* diagnosis of current depressive disorder were recruited from primary care and specialized mental health care. Cognitive performance was assessed at baseline and 2 years follow-up with the Stroop colored-word test, a modified version of the Auditory Verbal Learning Task and the Digit Span subtest from the Wechsler Adult Intelligence Scale, encompassing four cognitive domains; processing speed, interference control, memory, and working memory. Social network size was assessed with the Close Person Inventory and loneliness with the de Jong Gierveld Loneliness Scale at baseline.

Results: After adjusting for baseline working memory performance, loneliness was associated with impaired working memory after 2 years [B = −0.08 (−0.17 to 0.00)]. This association was no longer significant after adjusting for age, sex, education level, physical activity, alcohol use and depressive symptom severity [B = −0.07 (−0.16 to 0.03)]. A backward elimination procedure revealed education level to be the only covariable to explain this association. Loneliness was not associated with impairments or decline in other cognitive domains. Social network size was not associated with cognitive impairments or decline.

Conclusion: Social network size and loneliness do not predict cognitive decline in depressed older adults.

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Depression; social network size; loneliness; working memory; processing speed; interference control; verbal memory

Introduction

Depression in older adults is associated with faster development of cognitive impairments (van den Kommer et al., 2013; Gallagher, Kiss, Lanctot, & Herrmann, 2016). Cognitive impairments are associated with increased risk of disabilities, impaired functioning in instrumental activities (Dodge et al., 2005), dementia (Petersen et al., 2009), and nursing home admission (Nuutinen, Leskelä, Suojalehto, Tirronen, & Komssi, 2017). Current pharmacological treatments for cognitive impairments are not effective and might even be harmful in some persons (Raschetti, Albanese, Vanacore, & Maggini, 2007). Because depressed older adults are at increased risk of developing cognitive decline it is important to identify modifiable risk factors for cognitive decline in depressed older adults. Two potentially modifiable risk factors for cognitive decline are the size of the social network and loneliness.

Social network size and cognitive decline

Small social network size has been found to predict adverse health outcomes, including Type 2 diabetes (Brinkhues et al., 2017), coronary heart disease (Leigh-Hunt et al., 2017), stroke, and mortality (Holt-Lunstad, Smith, & Layton, 2010; Becofsky et al., 2015). By performing two recent systematic reviews and meta-analyses we found that small social network size was also associated with an increased risk of cognitive decline (Kuiper et al., 2016), and the onset of dementia (Kuiper et al., 2015) in the general population.

Several pathways may explain the association of a small social network with cognitive decline in the general population. First, the 'stress-buffering' hypothesis suggests that the social network may work as a buffer against stress (Cohen & Wills, 1985). Stress induces an increased release of glucocorticoids. A cumulative exposure to glucocorticoids in turn results in neurodegeneration of the hippocampus (Sapolsky, Krey, & McEwen, 1986). Indeed, psychological distress has been found to be associated with cognitive decline and development of Alzheimer's Disease (Wilson et al., 2003). Another pathway in which stress is thought to induce cognitive impairments is through decreased connectivity and plasticity of the prefrontal cortex, resulting in impairments in working memory specifically (McEwen & Morrison, 2013). Second, the

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'cognitive-reserve' theory suggests that stimulating environments lead to neuronal changes that keep the brain cognitively intact (Cohen & Wills, 1985; Van Praag, Kempermann, & Gage, 2000). A smaller social network may therefore result in more cognitive decline through a less stimulating environment. Third, the 'main-effect' hypothesis suggests that the social network stimulates positive health behaviors (such as non-smoking and physical exercise), and increases effective use of available health institutions (Kawachi & Berkman, 2001).

Loneliness and cognitive decline

In older adults, the size of the social network might become less important than the emotional perception of support from their social contacts. With increasing age, the social network size decreases while the number of close contacts stays the same (English & Carstensen, 2014), and meaningful emotional exchanges with close contacts become more relevant than interaction with more distant contacts (Carstensen, 1992). A concept that is closely related to (the absence of) meaningful emotional exchanges with close contacts is loneliness.

Loneliness is a psychological condition that encompasses a deep sense of emptiness, worthlessness and a feeling of insecurity (Cacioppo, Cacioppo, & Boomsma, 2014). In the general population, loneliness is associated with several adverse health outcomes including cardiovascular disease, stroke (Valtorta, Kanaan, Gilbody, Ronzi, & Hanratty, 2016), cognitive decline (Kuiper et al., 2016), and dementia (Kuiper et al., 2015). One study found loneliness to mediate the association between poor social support and cognitive decline in older adults (Ellwardt, Aartsen, Deeg, & Steverink, 2013). Loneliness might impact cognitive decline through exaggerated response to stress (Brown, Gallagher, & Creaven, 2017), leading to neurodegeneration of the hippocampus (Sapolsky et al., 1986) or decreased connectivity within the prefrontal cortex (McEwen & Morrison, 2013). Alternatively, a recent study found increased cortical amyloid burden in cognitively healthy older adults with increased loneliness, suggesting that loneliness as a first symptom of preclinical Alzheimer's Disease (Donovan et al., 2016).

Because loneliness and depression are strongly interrelated in older adults (Golden et al., 2009), depressed older adults might be particularly vulnerable for the poor health outcomes associated with loneliness. Supportive of this, loneliness in depressed older adults is associated with increased mortality rates (Stek et al., 2005; Holwerda et al., 2016), and in another study loneliness was associated with poor cognitive performance only in individuals with depressive symptoms (Lam, Yu, & Lee, 2017). Thus, in depressed older adults loneliness might be an important risk factor for cognitive decline.

Aims of the present study

To our knowledge, no study evaluated the impact of loneliness on cognitive decline in depressed older adults, and only two studies examining the impact of social network size on cognitive decline among depressed older patients found no relation (Dickinson, Potter, Hybels, McQuoid, & Steffens, 2011; Riddle, McQuoid, Potter, Steffens, & Taylor, 2015). These negative findings are somewhat disappointing, and need replication. In the present study, we examined the association of social network size and loneliness with cognitive performance and 2-year cognitive decline in a well-defined sample of 378 depressed older adults.

Methods

Study design

The present study used baseline and two-year follow-up data from the Netherlands Study of Depression in Older persons (NESDO). Briefly, NESDO was designed to examine the course and the consequences of depressive disorders in older adults (≥60 years). Detailed description of the methods of NESDO can be found elsewhere (Comijs et al., 2011).

Inclusion criteria for NESDO were being 60 years and older and having a primary diagnosis of major or minor depression or dysthymia within the last 6 months according to the criteria of *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (DSM-IV) which was determined with the Composite International Diagnostic Interview (CIDI) (Wittchen, 1994). Participants were excluded from the study if they had a primary diagnosis of or were suspected for dementia or primary severe psychiatric disorder other than depression according to the clinician, had a Mini Mental State Examination-score under 18 (out of 30 points), or if they had insufficient command of the Dutch language. Before participation, all participants signed for informed consent. The study protocol was approved by the Ethical Review Board of the VU University Medical Center, the Leiden University Medical Center, University Medical Center Groningen and the Radboud University Medical Center in Nijmegen.

Procedures

At baseline (between 2007 and 2010) and at 2 years follow-up (between 2009 and 2012) trained research assistants gathered demographic, psychosocial, biological, cognitive and mental health parameters with interviews, questionnaires, and physiological examinations. Participants not able to come to the site were interviewed at their homes. When necessary, the assessment was spread over two appointments.

Cognitive measures

Three neuropsychological tests were performed: a modified version (10 instead of 15 words) of the Auditory Verbal Learning Test (Rey, 1964; Van der Elst, van Boxtel, van Breukelen, & Jolles, 2005), the subtest Digit Span (both forward and backward) from the Wechsler Adult Intelligence Scale (WAIS) (Wechsler, 1958), and the Stroop Colour-word test (Stroop, 1935).

During the Stroop Colour Word test, participants were first shown a card with the names of four colours printed in black ink. They were asked to read this card as fast as possible (Stroop task I). Second, participants were shown a card with patches in four types of colours, and were asked

to name the colours of the patches as fast as possible (Stroop task II). Third, participants were shown a card with names of colours printed in an incongruent colour, and they had to name the colour as fast as possible (Stroop task III).

During the WAIS Digit Span, participants had to repeat a series of digits recited by the research assistant. With every correct series a number was added. The Digit Span Forward score comprised the longest number of digits correctly repeated. The Digit Span Backward score comprised the longest number of digits correctly repeated in reverse order.

During the Modified Auditory Verbal Learning test, participants had to recall as many words as possible from a list of 10 common nouns recited by the research assistant. This was repeated 5 times. After a delay of approximately 15 minutes participants were asked to recall the words again. The total score comprised the total number of recalled words during the five rounds. The delayed recall score comprised the total number of recalled words during the delayed round.

Based on a previous principal component analysis on these three neuropsychological test results, four cognitive domain scores were created: *processing speed, interference control, verbal memory,* and *working memory* (Korten et al., 2014). *Processing speed* comprised the total number of seconds to complete Stroop I and II. This variable was transformed by taking the multiplicative inverse (i.e. 1/x) to make it normally distributed. *Interference control*, which is a component of executive function, comprised the interference score from the Stroop test, which was computed with the formula: (tIII − .5 * (tI + tII))/(.5 * (tI + tII)) * 100% (Klein, Ponds, Houx, & Jolles, 1997). This variable was transformed by taking the natural logarithm (after adding a constant (50)) to make it normally distributed and multiplied by −1 so higher scores represent better scores. *Verbal memory* comprised the total number of correct words on the 5 tasks and delayed recall task of the modified Auditory Verbal Learning test. *Working memory* comprised the total number of correct items of the Forward and Backward scores of the WAIS Digit Span. For all four domains, higher scores represent better scores. For both time-points the four cognitive domains were calculated in the same way (including transformations).

Social network size and loneliness

Social network size was assessed with the first question of the Close Person Inventory (Stansfeld & Marmot, 1992). Participants were asked to indicate how many family members, friends and good acquaintances, over the age of 18, they had regular and important contact with, disregarding roommates. Responses to this question were classified as 1 (0 to 1 contacts), 2 (2 to 5 contacts), 3 (6 to 10 contacts), 4 (11 to 15 contacts), 5 (16 to 20 contacts) or 6 (more than 20 contacts). These categories were based on the original Close Person Inventory, and thus social network size was entered as a categorical variable in the analyses.

Loneliness was measured with the De Jong Gierveld loneliness scale, which is shown to be a valid and reliable instrument (De Jong Gierveld & Kamphuis, 1985). The questionnaire consists of eleven items that have to be answered with yes or no, resulting in a sum score (range 0–11) with higher scores indicative of a more severe level of loneliness.

Covariables

In addition to age and sex, we included years of education, alcohol use, physical activity, depressive symptom severity at baseline and depressive symptom severity at 2 years follow-up as covariables. Low education level, heavy alcohol use and physical inactivity were previously found to be associated with higher risk of poor social support (Kirchner et al., 2007; Cohen-Mansfield, Hazan, Lerman, & Shalom, 2016; Böhm, Mielke, da Cruz, Ramires, & Wehrmeister, 2016) as well as faster cognitive decline (Stern, 2002; Sabia et al., 2014; Guure, Ibrahim, Adam, & Said, 2017). Within depressed persons depressive symptom severity is associated with loneliness (Holvast et al., 2015) and with worse cognitive functioning (Sheline et al., 2006; McClintock, Husain, Greer, & Cullum, 2010; Korten et al., 2014). Furthermore, loneliness has been found to precede changes in depressive symptoms (Cacioppo, Hawkley, & Thisted, 2010) and changes in depressive symptoms have been found to covary with changes in cognitive functioning (Douglas & Porter, 2009). Therefore, depressive symptom severity at baseline and 2 years follow-up were also included as covariables.

At baseline, demographic data were collected on age, sex, and years of education. Alcohol use was classified as no drinking, moderate alcohol use and problematic alcohol use. Problematic alcohol use was defined as taking $\geq$5 units on a typical drinking day irrespective of the frequency of drinking, or as $\geq$3 units on a typical drinking day for $\geq$4 days a week. Physical activity was classified into low, moderate and high physical activity according to the International Physical Activities Questionnaire (IPAQ) (Craig et al., 2003). Finally, at baseline and at 2 years follow-up depressive symptom severity was measured with the 30-item Inventory of Depressive Symptoms (IDS) (Rush, Gullion, Basco, Jarrett, & Trivedi, 1996), a valid and reliable self-report instrument with higher scores indicating more severe levels of depressive symptoms.

Statistical analysis

First, participants at follow-up were compared to dropouts with independent *t*-tests for normally distributed variables, Mann-Whitney *U* tests for not normally distributed variables, and Chi Square tests for categorical variables.

Next, multiple imputation was applied, using the fully conditional specification approach (Rubin, 1987). Since 27% of cases had at least one missing value, 27 datasets were created with 100 iterations for each dataset (White, Royston, & Wood, 2011). As using the outcomes for imputation of missing predictor values gives more reliable results (Moons, Donders, Stijnen, & Harrell, 2006), the imputation model included all variables that were used in the analyses, including the outcomes. Missing values on the outcomes themselves were not imputed, because this introduces noise to the estimates (Von Hippel, 2007; White et al., 2011). Therefore, analyses were performed in the subset of cases with complete data on the cognitive outcomes.

Table 1. Descriptive statistics at baseline.

	Total sample ($n = 378$)	Available for follow-up ($n = 265$)	Dropped out at follow-up ($n = 113$)	p-value
Demographics:				
Age, mean (SD); n	70.7 (7.4); 378	70.2 (7.3); 265	72.0 (7.6); 113	.025[a]
Sex, n (%) female	250 (66.1); 378	170 (64.2); 265	80 (70.8); 113	.21[b]
Education in years, mean (SD); n	10.4 (3.4); 378	10.7 (3.4); 265	9.8 (3.4); 113	.014[a]
Depression characteristics:				
IDS score, mean (SD); n	30.1 (13.0); 373	29.3 (12.7); 262	32.1 (13.6); 111	.053[a]
Lifestyle characteristics:				
Alcohol use, n (%)				
No drinking	150 (40.4)	97 (37.3)	53 (47.7)	.108[b]
Moderate alcohol use	188 (50.7)	141 (54.2)	47 (42.3)	
Problematic alcohol use	33 (8.9)	22 (8.5)	11 (9.9)	
Physical activity, n (%)				
Low physical activity	98 (31.1)	54 (24.3)	44 (47.3)	<.001[b]
Moderate physical activity	119 (37.8)	91 (41.0)	28 (30.1)	
High physical activity	98 (31.1)	77 (34.7)	21 (22.6)	
Social support:				
Social network size				.577[b]
0–1 contacts	53 (14.2)	38 (14.4)	15 (13.6)	
2–5 contacts	172 (46.1)	120 (45.6)	52 (47.3)	
6–10 contacts	97 (26.0)	67 (25.5)	30 (27.3)	
11–15 contacts	34 (9.1)	24 (9.1)	10 (9.1)	
16–20 contacts	6 (1.6)	5 (1.9)	1 (0.9)	
>20 contacts	11 (2.9)	9 (3.4)	2 (1.8)	
Loneliness (total score), median (IQR); n	7 (4–10); 351	7 (3–10); 253	8 (4–10); 98	.343[c]
Cognitive performance:				
Processing speed, median (IQR); n	45 (40–52); 369	44 (39–50); 262	48 (42–57); 107	<.001[c]
Interference control, median (IQR); n	121 (92–161); 362	119 (90–154); 256	127 (96–176); 106	.292[c]
Verbal memory, mean (SD); n	37.2 (8.9); 375	38.4 (8.7); 263	34.3 (8.7); 112	<.001[a]
Working memory, mean (SD)	13.2 (3.2); 367	13.5 (3.2); 257	12.4 (2.9); 110	.002[a]

Notes: SD, standard deviation; IDS, Inventory of Depressive Symptomatology; IQR, interquartile range.
[a] With independent samples *t*-test.
[b] With chi-square.
[c]With Mann Whitney *U* test.

Next, linear regression analyses were performed on the imputed datasets, pooling the results from the different datasets using Rubin's rules (Rubin, 1987). First, to evaluate the potential role of each covariable in the examined associations, we examined the univariable association of each covariable with performance in the four cognitive domains at baseline and at 2 years follow-up. Next, we examined the relation of social network size or loneliness (independent variable) with cognitive performance in each of the four cognitive domains (dependent variable) at baseline, and with 2-year cognitive decline in each of the four cognitive domains. Loneliness was entered as a continuous variable, and social network size as a categorical variable with the response category of more than 20 contacts as reference category. Cognitive decline over 2 years was determined by defining cognitive performance at 2 years follow-up as dependent variable while adjusting for cognitive performance in the same domain at baseline. Separate models were performed for social network size and loneliness. In the fully adjusted models, adjustments were made for age, sex, education level, alcohol use, physical activity, and depressive symptom severity at baseline because these variables might act as confounders in the associations of social network size and loneliness with cognitive outcomes. All models with cognitive performance at 2 years follow-up as dependent variable also included baseline cognitive performance, and depressive symptom severity at follow-up as covariables.

Finally, for those models in which social network size or loneliness was significantly associated with cognitive performance or -decline univariably, but not anymore after adjustment for covariables, we examined which covariable(s) explained the association using the backward elimination procedure. For this purpose, we started with the full model. Subsequent models were examined, eliminating the independent variable with the highest p-value in the previous model, until a final model was achieved retaining all independent variables with $p < .10$ or $p < .05$.

IBM SPSS statistics software version 22 was used for the statistical analysis. Significance levels were set at $p < 0.05$ and all tests were two-tailed.

Results

The sample consisted of 378 depressed older adults, of whom 250 (66%) were female and 99% had the Dutch nationality. The mean age was 70.7 years (SD: 7.4; range 60–90). Of the 378 participants at baseline, 285 (75.4%) participated in the two-year follow-up of which cognitive testing was available in 265 (93.0%) participants. Compared to those lost to follow-up, those still available for follow-up were younger and had more years of education at baseline. They also had less severe depressive symptoms, performed more in moderate or high physical activity, and had better baseline cognitive performance (except for interference control) (see Table 1). The Spearman correlation between social network size and loneliness was −.33 ($p < .001$).

Older age was significantly associated with worse baseline and follow-up performance in processing speed, interference control and verbal memory. Lower education level was associated with worse cognitive performance in all domains at baseline and follow-up. Alcohol use was associated with worse performance in verbal memory at baseline, and worse working memory at baseline and follow-up. Low physical activity was associated with worse performance in verbal memory and working memory at baseline and follow-up. Depressive symptom severity at baseline was associated with worse performance in verbal memory and working memory at baseline only (see Table 2).

Table 2. Results of linear regression analysis showing the univariable association of each covariable with cognitive performance at baseline and follow-up.[a]

	Transformed processing speed[b,c]	Transformed interference control[b]	Verbal memory	Working memory
	Baseline B (95% CI)			
Age, per year	−0.14 (−0.21 to −0.09)***	−0.02 (−0.02 to −0.01)*	−0.34 (−0.45 to −0.22)***	−0.03 (−0.07 to 0.02)
Sex, female	−0.85 (−1.81 to 0.11)	−0.05 (−0.16 to 0.07)	1.74 (−0.16 to 3.63)	0.04 (−0.65 to 0.72)
Education, per year	0.32 (0.19 to 0.45)***	0.02 (0.00 to 0.04)*	0.58 (0.32 to 0.83)***	0.31 (0.22 to 0.39)***
Alcohol				
Moderate vs low	0.45 (−0.50 to 1.41)	0.03 (−0.09 to 0.14)	1.02 (−0.88 to 2.92)	1.12 (0.45 to 1.79)**
Problematic vs low	1.49 (−0.19 to 3.17)	0.12 (−0.08 to 0.32)	3.96 (0.64 to 7.28)*	2.91 (1.75 to 4.08)***
Physical activity				
Moderate vs high	−0.58 (−1.71 to 0.55)	0.11 (−0.03 to 0.25)	−1.86 (−4.16 to 0.44)	0.21 (−0.66 to 1.08)
Low vs high	−2.80 (−3.97 to −1.62)***	−0.02 (−0.17 to 0.13)	−4.65 (−7.05 to −2.25)***	−1.66 (−2.53 to −0.79)***
Inventory of Depressive Symptomatology total score, per point increase	−0.05 (−0.09 to −0.02)	0.00 (−0.01 to 0.00)	−0.08 (−0.15 to −0.01)*	−0.04 (−0.07 to −0.02)**
	Follow-up: B (95% CI)			
Age, per year	−0.19 (−0.25 to −0.12)***	−0.02 (−0.02 to −0.01)***	−0.52 (−0.66 to −0.38***	−0.02 (−0.07 to 0.03)
Sex, female	−0.83 (−1.87 to 0.21)	0.01 (−0.09 to 0.11)	1.19 (−1.15 to 3.54)	−0.45 (−1.23 to 0.33)
Education, per year	0.27 (0.13 to 0.41)***	0.02 (0.01 to 0.03)**	0.70 (0.38 to 1.01)***	0.27 (0.17 to 0.37)***
Alcohol				
Moderate vs low	0.52 (−0.56 to 1.60)	0.07 (−0.03 to 0.17)	0.59 (−1.81 to 2.98)	1.15 (0.36 to 1.95)**
Problematic vs low	0.54 (−1.38 to 2.46)	−0.01 (−0.19 to 0.17)	3.72 (−0.55 to 7.99)	2.39 (1.00 to 3.78)**
Physical activity				
Moderate vs high	−0.63 (−1.89 to 0.63)	−0.05 (−0.16 to 0.06)	−0.26 (−2.98 to 2.46)	0.23 (−0.70 to 1.16)
Low vs high	−1.93 (−3.36 to −0.49)**	−0.03 (−0.16 to 0.10)	−5.17 (−8.19 to −2.15)**	−1.06 (−2.17 to −0.01)*
Inventory of Depressive Symptomatology total score at baseline, per point increase	−0.01 (−0.05 to 0.03)	0.00 (0.00 to 0.00)	0.04 (−0.05 to 0.13)	−0.03 (−0.06 to 0.00)
Inventory of Depressive Symptomatology total score at 2 years follow-up, per point increase	−0.03 (−0.07 to 0.01)	0.00 (−0.01 to 0.00)	−0.02 (−0.11 to 0.07)	−0.02 (−0.05 to 0.01)

Notes: CI, confidence interval.
[a] Results were obtained by using the imputed data.
[b] The transformed variables of processing speed and interference control were used.
[c] Results of the Processing Speed variable were multiplied by a constant of 1000 to make the presentation of the results more informative.

Neither social network size nor loneliness was significantly associated with baseline measures of any of the cognitive domains (all $p > 0.05$; see Table 3 and Table 4). Loneliness was significantly associated with 2-year decline in working memory (B = −0.08; 95% CI: −0.17 to 0.00; p = .049; see Table 4), but not anymore after adjustments for age, sex, education level, alcohol use, physical activity and depressive symptom severity (B = −0.07; 95% CI: −0.16 to 0.03; p = .162; see Table 4). Apart from this, loneliness and social network size were not significantly associated with 2-year cognitive decline (Table 3 and Table 4).

Next, we examined which covariable explained the association between loneliness and 2-year decline in working memory. The backward elimination procedure retaining all independent variables with $p < .10$ retained baseline working memory (b = 0.61; 95% CI = 0.52 to 0.70: $p < .001$), education level (b = 0.08; 95% CI = 0.00 to 0.17; p = .049) and loneliness (b = −0.08; 95% CI = −0.16 to 0.01; p = .066). When retaining all independent variables with $p < .05$ baseline working memory (b = 0.61; 95% CI = 0.52 to 0.70; $p < .001$) and education level (b = 0.09; 95% CI = 0.01 to 0.17; p = .035) were retained.

Discussion

This study evaluated the potential impact of social network size and loneliness on cognitive performance and 2-year cognitive decline in a well-representative sample of 378 clinically depressed older adults. While two previous studies evaluated the potential impact of social network size on cognitive decline in depressed older adults (Dickinson et al., 2011; Riddle et al., 2015), this was the first study in depressed older adults to evaluate the potential impact of loneliness on cognitive decline. After adjustment for demographics, lifestyle and depressive symptom severity, loneliness and social network size were not associated with cognitive performance and 2-year cognitive decline.

Loneliness and cognitive performance after 2 years

We found a univariable association between loneliness and 2-year cognitive decline in working memory, but this was not present anymore in the multivariable analysis. A lower education level appeared to confound the association between loneliness with 2-year decline in working memory. This suggests against the hypothesis that loneliness impacts decline in working memory through exaggerated responses to stress and reduced connectivity of the prefrontal cortex (McEwen & Morrison, 2013; Brown et al., 2017) in depressed older adults. The present finding does suggest however that lower educated depressed older adults have an increased risk of being lonely as well as declining faster in working memory performance. Indeed, previous studies found lower education level to be associated with loneliness (Cohen-Mansfield et al., 2016). Low education level is often accompanied by lower income, which has been found to limit the social and activity opportunities, and reduce self-esteem and self-efficacy (Cohen-Mansfield et al., 2016). In addition to its association with loneliness, low education-level is also associated with worse working memory performance as well as lower prefrontal cortex volumes (Leonard, Mackey, Finn, & Gabrieli, 2015). Thus, low education-level may result in loneliness through reduced social opportunities, as well as in reduced working memory performance through an affected

Table 3. Linear regression models showing cross-sectional and longitudinal associations between social network size and cognitive performance.[a]

	Processing speed[b] Unstandardized B (95% CI)	Interference control Unstandardized B (95% CI)	Verbal memory Unstandardized B (95% CI)	Working memory Unstandardized B (95% CI)
Cross-sectional associations (n = 378)				
Univariate				
0–1 contacts	−0.72 (−3.61 to 2.17)	0.01 (−0.33 to 0.35)	−1.98 (−7.72 to 3.76)	−0.85 (−2.91 to 1.22)
2–5 contacts	0.16 (−2.55 to 2.86)	0.09 (−0.23 to 0.41)	0.39 (−5.00 to 5.77)	0.31 (−1.64 to 2.25)
6–10 contacts	−0.05 (−2.82 to 2.73)	−0.07 (−0.39 to 0.26)	0.65 (−4.86 to 6.16)	−0.15 (−2.14 to 1.83)
11–15 contacts	−0.08 (−3.10 to 2.94)	−0.17 (−0.53 to 0.18)	−1.07 (−7.11 to 4.98)	−0.50 (−2.66 to 1.67)
16–20 contacts	−1.92 (−6.32 to 2.48)	−0.14 (−0.65 to 0.38)	−2.21 (−11.00 to 6.59)	1.67 (−1.53 to 4.86)
>20 contacts	Reference category	Reference category	Reference category	Reference category
Adjusted model[c]				
0–1 contacts	0.01 (−2.75 to 2.76)	0.00 (−0.34 to 0.33)	−1.61 (−7.00 to 3.79)	−0.35 (−2.26 to 1.57)
2–5 contacts	0.52 (−2.04 to 3.08)	0.07 (−0.24 to 0.38)	−0.09 (−5.13 to 4.95)	0.05 (−1.75 to 1.85)
6–10 contacts	0.56 (−2.07 to 3.18)	−0.07 (−0.39 to 0.25)	0.44 (−4.73 to 5.61)	−0.21 (−2.05 to 1.63)
11–15 contacts	0.65 (−2.20 to 3.49)	−0.17 (−0.51 to 0.18)	−1.21 (−6.84 to 4.43)	−0.67 (−2.67 to 1.34)
16–20 contacts	−0.65 (−4.80 to 3.50)	−0.09 (−0.60 to 0.41)	−1.07 (−9.28 to 7.14)	1.63 (−1.34 to 4.60)
>20 contacts	Reference category	Reference category	Reference category	Reference category
Longitudinal associations (n = 265):				
Univariate[d]				
0–1 contacts	0.48 (−1.58 to 2.54)	0.00 (−0.25 to 0.24)	−0.09 (−5.13 to 4.95)	−0.47 (−2.12 to 1.18)
2–5 contacts	−0.01 (−1.94 to 1.91)	0.04 (−0.19 to 0.27)	−0.59 (−5.28 to 4.11)	0.33 (−1.22 to 1.88)
6–10 contacts	0.16 (−1.81 to 2.14)	0.05 (−0.19 to 0.28)	2.04 (−6.86 to 2.79)	−0.08 (−1.68 to 1.51)
11–15 contacts	−0.36 (−2.53 to 1.81)	0.06 (−0.20 to 0.32)	−2.45 (−7.79 to 2.90)	−0.45 (−2.21 to 1.31)
16–20 contacts	0.38 (−2.72 to 3.48)	−0.17 (−0.54 to 0.20)	−3.07 (−10.64 to 4.49)	0.84 (−1.67 to 3.35)
>20 contacts	Reference category	Reference category	Reference category	Reference category
Adjusted model[e]				
0–1 contacts	0.06 (−1.97 to 2.09)	−0.03 (−0.28 to 0.22)	−1.25 (−6.27 to 3.77)	−0.34 (−2.03 to 1.35)
2–5 contacts	−0.19 (−2.09 to 1.70)	0.01 (−0.22 to 0.24)	−1.26 (−5.94 to 3.42)	0.40 (−1.18 to 1.98)
6–10 contacts	−0.03 (−1.97 to 1.92)	0.03 (−0.21 to 0.26)	−2.63 (−7.44 to 2.18)	0.00 (−1.63 to 1.63)
11–15 contacts	−0.25 (−2.38 to 1.87)	0.06 (−0.20 to 0.32)	−2.09 (−7.38 to 3.20)	−0.36 (−2.14 to 1.42)
16–20 contacts	0.43 (−2.63 to 3.48)	−0.16 (−0.53 to 0.21)	−2.82 (−10.37 to 4.73)	1.16 (−1.40 to 3.71)
>20 contacts	Reference category	Reference category	Reference category	Reference category

Notes: CI, confidence interval.
[a]Results were obtained by using the transformed and imputed data.
[b] Results of the Processing Speed variable were multiplied by a constant of 1000 to make the presentation of the results more informative.
[c] Adjusted for age, sex, years of education, alcohol use, physical activity, and depressive symptom severity at baseline.
[d]Adjusted for baseline cognitive performance only.
[e] Adjusted for age, sex, years of education, alcohol use, physical activity, depressive symptom severity at baseline, depressive symptom severity at 2 years follow-up, and baseline cognitive performance.

Table 4. Linear regression models showing cross-sectional and longitudinal associations between loneliness and cognitive performance.[a]

	Processing speed[b] Unstandardized B (95% CI)	Interference control Unstandardized B (95% CI)	Verbal memory Unstandardized B (95% CI)	Working memory Unstandardized B (95% CI)
Cross-sectional associations (n = 378)				
Univariate	−0.11 (−0.24 to 0.03)	0.00 (−0.01 to 0.01)	−0.17 (−0.44 to 0.09)	−0.04 (−0.13 to 0.06)
Adjusted model[c]	−0.03 (−0.16 to 0.10)	0.00 (−0.01 to 0.02)	−0.03 (−0.29 to 0.23)	0.04 (−0.05 to 0.14)
Longitudinal associations (n = 265)				
Univariate[d]	−0.01 (−0.11 to 0.09)	0.00 (−0.01 to 0.01)	−0.03 (−0.28 to 0.23)	−0.08 (−0.17 to 0.00)*
Adjusted model[e]	−0.02 (−0.13 to 0.09)	0.00 (−0.01 to 0.02)	−0.06 (−0.33 to 0.22)	−0.07 (−0.16 to 0.03)

*$p < 0.05$; CI = confidence interval.
[a]Results were obtained by using the transformed and imputed data.
[b] Results of the Processing Speed variable were multiplied by a constant of 1000 to make the presentation of the results more informative.
[c] Adjusted for age, sex, years of education, alcohol use, physical activity, and depressive symptom severity at baseline.
[d]adjusted for baseline cognitive performance only.
[e] Adjusted for age, sex, years of education, alcohol use, physical activity, depressive symptom severity at baseline, depressive symptom severity at 2 years follow-up and baseline cognitive performance.

prefrontal cortex, perhaps due to increases in stress (McEwen & Morrison, 2013; Brown et al., 2017).

The absence of an association between loneliness and cognitive performance and -decline in depressed older adults might be because loneliness has no predictive value anymore over the strong impact of depression itself on cognitive performance and -decline. In line with this potential explanation, depressive symptom severity (which is another variable that is often found to be associated with cognitive decline in general population samples (e.g. Van den Kommer et al., 2013; Bunce, Batterham, Christensen, & Mackinnon, 2014; Gallagher et al., 2016), was also not associated with cognitive decline in the present sample. Depression may exert its impact on cognitive decline through many different pathways that compete with loneliness, including many psychological, physiological, and behavioral pathways that may differ between persons. Therefore, interventions aiming to postpone cognitive decline in depressed older adults might be more beneficial if they target the full array of potential factors between depression and cognitive decline as well as the depression itself. This means that for lonely depressed older adults interventions targeting the loneliness might be beneficial, particularly if they also improve the depression.

Social network size and cognitive performance after 2 years

We found no association of social network size with cognitive performance after 2 years in our sample of depressed

older adults. This is in contrast to what is often found in general population samples (Shankar, Hamer, McMunn, & Steptoe, 2013; Kuiper et al., 2016), yet consistent with two previous studies in depressed older adults (Dickinson et al., 2011; Riddle et al., 2015). Riddle et al., evaluated the association of social network size, frequency of social interactions, instrumental social support and emotional perception of social support with conversion to cognitive impairment or dementia in 299 depressed older adults. Lower levels of or a change in these social measures over the first year did not predict conversion to cognitive impairment or dementia in 299 depressed older adults (Riddle et al., 2015). In the study of Dickinson et al., social support did not predict cognitive decline in 112 depressed older adults (Dickinson et al., 2011). However, a decrease in frequency of social interaction was associated with a subsequent decrease in global cognitive performance and Digit Span Forward scores specifically (Dickinson et al., 2011). Therefore, frequency of social interaction might be more important in predicting cognitive decline in depressed older adults than social network size.

An alternative explanation why social network size was not associated with cognitive performance in the present study is that our measure of social network size did not specify the social network composition. A relatively higher proportion of friends might protect against cognitive impairments, whereas a relatively higher proportion of family contacts might be associated with impairments in cognitive performance (Aartsen, van Tilburg, Smits, & Knipscheer, 2004). Finally, as with loneliness, also social network size might lose its predictive value on cognitive performance- and decline in depressed older adults over the strong impact of the depression itself on cognitive performance and -decline. Thus, interventions aiming the social network size might be effective in reducing cognitive decline in depressed older adults, as long as they also improve the depression.

Strengths and limitations

Strengths of the present study are the large sample size of clinically depressed older adults, structured psychiatric diagnostic interviews, assessment of cognitive performance across four different domains, and adjustment for all relevant covariables including depressive symptom severity. Moreover, to our knowledge, this was the first study evaluating the potential impact of loneliness on cognitive decline in depressed older adults. There are also some considerations to take into account when interpreting the results of the present study. First, the dropout rate was higher among patients with the worst cognitive performance which might represented accelerated cognitive decline among the dropouts. Therefore, it remains unclear to what extent the findings are generalizable to depressed older adults with worse cognitive performance. Second, depressed persons have biased emotional processing (Gotlib & Joormann, 2010), which may have resulted in a more negative rating of social network size and loneliness. This may have obscured any relationship between our social measures and cognitive decline. Third, our social network size measure did not distinguish between friends and family, while a relatively higher proportion of friends might protect against cognitive impairments, whereas a relatively higher proportion of family contacts might be associated with impairments in cognitive performance (Aartsen et al., 2004). Furthermore, we did not assess frequency of social interactions, a measure that was found to be associated with cognitive decline in depressed older adults previously (Dickinson et al., 2011).

Implications and general conclusion

Results from the present study suggest that after adjustment for demographics, lifestyle and depressive symptom severity social network size and loneliness are not associated with cognitive performance and 2-year cognitive decline in depressed older adults. The absence of an association of loneliness and social network size with cognitive performance and -decline in depressed older adults might be caused by the fact that their impact is overruled by the strong impact of depression itself on cognitive performance and decline. Depression may exert its impact on cognitive decline through many different pathways that compete with loneliness and social network size, including many psychological, physiological, and behavioral pathways that may differ between persons. Therefore, interventions aiming to postpone cognitive decline in depressed older adults might be more beneficial if they target the full array of potential factors between depression and cognitive decline as well as the depression itself. Thus, interventions aiming loneliness and the social network size might be effective in reducing cognitive decline in depressed older adults, as long as they also improve the depression.

Disclosure statement

No potential conflict of interest was reported by the authors.

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