RESEARCH ARTICLE

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Change in stress and social support as predictors of cognitive decline in older adults with and without depression

Whitney J. Dickinson, Guy G. Potter, Celia F. Hybels, Douglas R. McQuoid and David C. Steffens

Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Durham, NC, USA
Correspondence to: D. C. Steffens, E-mail: steff001@mc.duke.edu

Objective: The relationship between stress, social support, and cognition in geriatric depression is complex. In this study, we sought to examine whether an increase in stressful life events or a decrease in social support would lead to subsequent cognitive decline among older adults with and without depression.

Methods: The sample consisted of 112 depressed and 101 non-depressed older adults who enrolled in the Neurocognitive Outcomes of Depression in the Elderly (NCODE) study. Participants were assessed clinically, agreed to interviews focusing on stressful life events and social support, and underwent a battery of neuropsychological tests annually. Our global measure of cognition was the Consortium to Establish a Registry in Alzheimer's disease Total Score (CERAD TS).

Results: We found that a decline in the total number of stressors was associated with a subsequent improvement on CERAD TS. In terms of social support, decreased social interaction, and instrumental social support predicted decline in cognitive performance. These relationships were significant even after controlling for depression status, age, education, and sex.

Conclusions: These findings extend prior research on the importance of social factors in aging and depression which have largely focused on mood-related outcomes. Future confirmatory studies are needed. In addition, biological and other studies should be conducted to further our understanding of the relationship between stress, social support and cognition in older adults with and without depression.

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Cognitive impairment in late-life depression is a risk factor for multiple adverse outcomes in physical health, functional independence, and mortality (Mehta *et al.*, 2003). Depression in late life may be a prodromal symptom of Alzheimer's disease and a risk factor for dementia more broadly (Kessing and Nilsson, 2003), while cognitive impairment during depression is also associated with persistent deficits (Lee *et al.*, 2007) and dementia (Alexopoulos *et al.*, 1993). Based on community samples, the prevalence of cognitive impairment and comorbid depression doubles at 5-year intervals after the age of 70, and is estimated to be present in 25% of individuals aged 85 and older (Arve *et al.*, 1999). These rates make it important to understand the factors that contribute to acute and persistent cognitive dysfunction in depressed older adults.

While studies of depression have examined neurobiological correlates of cognitive dysfunction in depression, less attention has been devoted to the role of psychosocial factors. The association between greater life stress and higher rates of depression has been well established (Kessler, 1997; Mazure *et al.*, 2002), and higher perceived stress is associated with incomplete remission of depression (Hybels *et al.*, 2006). Higher levels of perceived stress and stressful

life events also have been associated with greater age-related differences on objective measures of episodic memory and executive functions (VonDras *et al.*, 2005). Evidence supports the idea that stress-related coping requires a certain amount of cognitive attention and therefore the presence of major stress reduces the amount of attention one can devote to other information processing tasks (Kahneman, 1973; Stawski *et al.*, 2006), which would be exacerbated in later life (Smith, 2003), and presumably more so when stress results in a depressive episode. It has also been posed that stress-related hypercortisolemia during depression may have a progressive toxic effect on the hippocampus and associated cognitive functions, which has some support among non-depressed, but is more mixed in studies of late-life depression (Lupien *et al.*, 1999; Steffens and Potter, 2007).

Social support (both perceived and actual) is another psychosocial factor that may contribute to cognitive dysfunction in late-life depression. Low levels of perceived social support are associated with longitudinal depression severity (Steffens *et al.*, 2005). Instrumental social support appears to provide a buffer against functional decline in late-life depression (Hays *et al.*, 2001), but the role in cognitive decline is not well studied in this condition. There is evidence in non-depressed elderly, however, that low satisfaction with social support is associated with 5-year decline in episodic memory (Hughes *et al.*, 1988) and that perceived social isolation (i.e., loneliness) is associated with cognitive decline over a 10-year period (Tilvis *et al.*, 2004). Thus, poor social support, or the perception of poor support, may also contribute to cognitive decline among depressed older adults as well.

The purpose of this study was to examine the effects of stress and social support on cognition in depressed and non-depressed older adults. We expected that a change in stress from baseline to 1-year (and first year of treatment for patients) would predict a change in cognitive performance in follow-up assessments across both the depressed and non-depressed population. In addition, we hypothesized that changes in the level of social support during the first year of the study would affect changes in cognitive ability the following year for the whole sample. We hypothesized an increase in stress or a decrease in social support would predict a decrease in cognitive ability over the following year. We hypothesized that these associations would be significant after controlling for age, sex, years of education, and depression status (depressed/non-depressed) at baseline. A secondary objective was to explore which aspects of cognition might be sensitive to the influence of social factors. Overall, the potential clinical importance of this study is that finding specific associations between stress and social support with cognition could highlight potentially modifiable intervention targets to treat depression and prevent cognitive decline.

Methods

Study sample

Data were obtained from the National Institutes of Health-supported Neurocognitive Outcomes of Depression in the Elderly (NCODE) study at Duke University, a longitudinal naturalistic study of depression in older adults. Depressed participants who met criteria for a current episode of unipolar major depression and were age 60 and older were enrolled in the study. Participants were referred to the study from the Duke inpatient and outpatient psychiatry services and from the Duke General Internal Medicine Clinic. Exclusion criteria included presence of another major psychiatric illness such as schizophrenia, schizoaffective disorder, bipolar disorder, and lifetime alcohol or substance dependence. Patients with psychotic depression were included, as were those with co-morbid anxiety disorders, as long as major depression was deemed by the study psychiatrist to be the primary psychiatric disorder. Patients were excluded if they had dementia or suspected dementia at baseline based on information available to the assigned geriatric psychiatrist, who examined the subject, reviewed medical records, and conferred with referring physicians for all patients. Other neurological conditions that could affect cognitive function were also excluded, including Parkinson's disease, multiple sclerosis, and seizure disorder. Participants in the parent study from which these date were collected were also excluded if there were contraindications to brain MRI.

Non-depressed older adults were recruited from the Center for Aging Subject Registry at Duke University, which includes more than 1900 community-dwelling elders in the Durham, Chapel Hill, and Raleigh (North Carolina) area who expressed a willingness to participate in the Duke Center for Aging Research. Eligible comparison participants must have had a non-focal neurological examination, no self-report of neurologic (including dementia) or depressive illness, no contraindication to brain MRI, and no evidence of a lifetime depression diagnosis based on the Diagnostic Interview Schedule portion of the Duke Depression Evaluation Schedule (DDES) (Robins *et al.*, 1981).

Patients are evaluated at least every 3 months, while comparison participants are screened annually for current and past depression. After complete description of the study to the subjects, written informed consent was obtained. The study was approved by the Institutional Review Board at Duke University Medical Center.

Data collection. Baseline demographic characteristics obtained from participants included age, sex, and years of education completed. A trained interviewer administered the DDES to all patients and comparison participants at baseline and annual follow-ups. The DDES assesses depression using the NIMH Diagnostic Interview Schedule (Robins *et al*., 1981), and also includes assessments of overall cognitive status, physical health, and the four measures that compose the Duke Social Support Index (DSSI): instrumental social support, social interactions, subjective social support, and non-family social network (Landerman *et al*., 1989).

Clinical evaluation and follow-up. At baseline, a geriatric psychiatrist interviewed each depressed subject and completed standardized clinical assessments, including the 17-item Hamilton Rating Scale for Depression (Hamilton, 1960), the Montgomery Asberg Depression Rating Scale (Montgomery and Asberg, 1979), and the Clinical Global Impression Scale (Guy, 1976). The NCODE study operates in a naturalistic treatment milieu using treatment guidelines established by the Duke Affective Disorders Program (Steffens *et al*., 2002). Treatment modalities available included antidepressant medications, electroconvulsive therapy, and individual and group cognitive-behavioral psychotherapy. Treatment was monitored to ensure that clinical guidelines were followed appropriately. Patients were evaluated when clinically indicated and at least every 3 months while they were in the study. The protocol recommends that patients receive continuation treatment for at least 1–2 years (some indefinitely) once they achieve remission. Each patient is thus ensured to receive the most appropriate care we are able to provide. For this particular analysis focusing on cognitive outcomes, we note the small frequency of subjects receiving treatment with tricyclic antidepressants (3.85%) or ECT (1.54%).

Measures of stress and social support

Overall stress and negative stressful events were assessed during the baseline interview and follow-up using the Life Events Scale developed at Duke University (Hughes *et al*., 1988), which asks 20 event-related items focused on changes in respondents' health, family and living situations, work, and finances. The negative life event measure used here is the sum of the number of events that respondents reported as having negative effect on their lives the previous year. The total number of stressors includes both positive and negative events. We created two variables to indicate change in number of total stressors and change in negative stressors between baseline and year 1. Each component was used as a continuous variable in these analyses, with higher totals indicating more stress. Change was assessed by subtracting the year 1 scores from the baseline scores. Therefore, positive change scores indicated a reduction in stress, whereas negative scores indicated an increase in stress.

Social networks and social support were assessed with items from the DSSI (Landerman *et al*., 1989), which has four subscales:

Subjective social support. Assessed by 10 items (e.g., 'When you are talking with your family and friends, do you feel you are being listened to?'); response options were *hardly ever* (coded as 1), *some of the time* (2), and *most of the time* (3).

Instrumental social support. Assessed by twelve items (e.g., 'Do your family and friends ever help out when you are sick?'); response options were yes (1) and no (0).

Social network size. Assessed by four items (e.g., 'Other than persons living with you, how many of your brothers and sisters live within one hour's travel of your home?').

Social interaction. Assessed by four items (e.g., 'Other than at work, how many times during the past week did you spend some time with someone who does not live with you, that is, you went to see them or they came to visit you, or you went out together?'); response options were *not at all*, *once*, *twice*, *three times*, *four times*, *five times*, *six times*, and *seven times or more*, coded from 0 to 7.

Summary scores were computed for each domain by summing all relevant items, with higher numbers corresponding to larger networks and more social support. We created one change score for each of the four domains to reflect changes in social support between baseline and year 1. The change scores were created by subtracting the year 1 total from the baseline total. Negative change scores indicated an increase in social support, while positive scores indicated a decrease.

Cognitive assessment

A neuropsychological test battery was administered to depressed participants at baseline while still symptomatic and then annually to patients, regardless of depression status. Non-depressed participants received annual assessments as well. The battery consisted of the Consortium to Establish a Registry in Alzheimer's Disease (CERAD) neuropsychological battery (Morris *et al*., 1989); which includes (i) the mini mental state examination (MMSE) (Folstein *et al*., 1975); (ii) language tasks consisting of category fluency (Animal Naming and object naming); (iii) constructional praxis copy of four geometric designs; and (iv) verbal learning and memory consisting of immediate recall of the learning trials of a 10-item word list, delayed recall of the list, and recognition/discrimination of target words from non-target foils. The CERAD battery was supplemented by other common neuropsychological measures used in clinical practice for assessing (i) immediate and delayed verbal memory (Logical Memory subtest of the Wechsler Memory Scale—Revised (Wechsler, 1987)), (ii) attentional/executive functions (Trail Making Test Parts A (TMT-A) and B (TMT-B) (Reitan, 1992), Symbol Digit Modalities Test (SDMT) (Smith, 1982), Digit Span Forward and Backward from the Wechsler Adult Intelligence Scale—Revised (Wechsler, 1981), and a separate ascending Digit Span task modeled after the Digit Ordering Test (Hoppe *et al*., 2000)). A few additional tests in the battery did not pertain to our scientific questions and were not used in the current analysis. The CERAD Total Score (TS) (Chandler *et al*., 2005), was used as an index of global neurocognitive functioning of the sample at baseline and at follow-up assessments. This score is the sum of the individual CERAD neuropsychological tests, excluding the MMSE.

Statistical analysis

Initial comparisons were done using *t*-tests to explore differences between means for age, education, social support measures, stressful life events summary measures and neuropsychological measures between patients and comparison participants. Chi-square tests were used to compare differences in frequencies of sex and race between groups.

Pearson's correlation coefficients were calculated to test for significant correlations between changes in stressful life events (total and negative separately) and social support measures over the first year and changes in cognition over the second year. Linear regression models were fit for the measures that had a significant correlation ($p < 0.05$) in the initial analyses. Sex, age, education, and participants' diagnostic status (depressed or comparison participant) were included in the models as covariates along with the first year cognition measure to create residualized change models. Significance levels were set at $p < 0.05$. All tests were two-tailed.

Results

The characteristics of the depressed ($n = 112$) and non-depressed ($n = 101$) groups at baseline are described in Table 1. There were statistically significant differences between depressed and non-depressed participants with respect to age, sex, and education, but not race. Depressed individuals performed significantly worse than non-depressed at baseline on all cognitive measures. Depressed individuals reported significantly more negative stressful events and total stressful events than non-depressed individuals. Depressed individuals reported significantly lower subjective social support, lower instrumental social support, and less social interaction relative to non-depressed individuals, but there was no difference in social network size.

As an initial step to define the longitudinal models, we examined the correlations between changes in stress and social support between baseline and year 1 and change in cognitive scores between years 1 and 2. A decline in the total number of stressors was associated with an improvement on CERAD TS ($r = -0.16$, $p = 0.021$). There were four measures for which a change in social support was associated with a change in cognitive performance. A decrease social interaction was associated decline on CERAD TS ($r = 0.17$, $p = 0.013$) and Digit Span Forward ($r = 0.14$, $p = 0.041$). Decrease in instrumental social support was associated with decline on Ascending Digit Span ($r = 0.18$, $p = 0.014$) and SDMT ($r = 0.16$, $p = 0.027$).

The significant bivariate correlations between changes in stress/social interaction measures and cognitive performance formed the basis of our linear regression models, in which we sought to control for other possible explanatory variables: depression status (patient or comparison participant), age, education level, and sex (see Table 2). The first linear model regressed change in total number of stressors from baseline to year 1 on change in CERAD TS from years 1 to 2 while controlling for depression status (patient or comparison participant), age, education, and sex (see Table 2). An increase in the number of stressors from

Table 1 Baseline demographic and clinical characteristics of sample ($n = 213$)

Sample characteristic	Patients $n = 112$	Comparison participants $n = 101$	Total $n = 213$	Statistics t, df, p-value
Age	68.69 (6.34)	70.46 (5.72)	69.53 (6.11)	2.13, 211, 0.035
Sex (female) % (n)	56.25 (63)	72.28 (73)	63.85 (136)	$\chi^2 = 5.91$, 1, 0.015
Race (Caucasian) % (n)	88.39 (99)	88.12 (89)	88.26 (188)	$\chi^2 = 0.004$, 1, 0.951
Education	14.21 (2.51)	15.42 (1.76)	14.78(2.26)	4.07, 199, 0.0001
Social support				
Subjective	23.15 (3.89)	27.25 (1.16)	25.12 (3.56)	10.47, 127, <0.0001
Instrumental	9.30 (1.86)	10.72 (0.85)	9.98 (1.63)	7.18,154, <0.0001
Network	2.01 (1.99)	1.50 (1.83)	1.76 (1.93)	−1.94, 208, 0.053
Interaction	6.31 (2.60)	8.01 (2.34)	7.13 (2.62)	4.96, 208, <0.0001
Stress measures				
Total stressors	2.50 (1.72)	1.33 (1.23)	1.94 (1.61)	−5.78, 201, <0.0001
Negative stressors	1.60 (1.52)	0.42 (0.65)	1.04 (1.33)	−7.50, 154, <0.0001
Neuro-psych measures				
CERAD TS	73.29 (11.15)	83.17 (7.82)	77.97 (10.88)	7.55, 199, <0.0001
MMSE total	27.80 (2.36)	28.89 (1.25)	28.31(1.99)	4.30, 172, <0.0001
Digit span F	8.48 (2.57)	9.21 (2.34)	8.82 (2.48)	2.01, 182, 0.046
Digit span A	8.12 (2.74)	9.75 (1.99)	8.86 (2.55)	4.67, 178, <0.0001
Digits span B	6.85 (2.44)	7.83 (2.44)	7.30 (2.49)	2.72, 182, 0.007
Logical memory	24.11 (7.99)	29.68 (6.61)	26.74 (7.86)	5.50, 210, <0.0001
Logical memory II	20.08 (9.28)	26.07 (7.71)	22.92 (9.06)	5.07, 209, <0.0001
SDMT	36.36 (11.47)	44.38 (9.67)	40.19 (11.36)	5.47, 209, <0.0001
Trails A time	50.09 (34.86)	36.08 (11.97)	43.45 (27.44)	139, −4.00, 0.0001
Trails B time	130.30 (77.52)	85.46 (35.71)	108.42 (64.71)	−5.39, 149, 0.0001

CERAD total, Consortium to Establish a Registry in Alzheimer's Disease; Digit Span F (forward), A (ascending), B (backward); SDMT, Symbol-Digit Modalities Test.

baseline to year 1 was found to be negatively associated with a change in the CERAD TS from years 1 and 2 ($t = -2.85$, df = 206, $p = 0.0048$). Depression status at baseline also emerged as the other statistically significant predictor of decline in cognitive ability independent of changes in stress, but there were no significant effects of age, education, or sex on changes in cognition measured by CERAD TS.

As shown in Table 2, results from linear regression models also showed consistent patterns of decreased social interaction and instrumental social support predicting decline in cognitive performance while controlling for depression status, age, education, and sex. A decrease in social interaction from baseline to year 1 was associated with worse performance on both the CERAD TS ($t = 2.66$, df = 201, $p = 0.0084$) and Digit Span Forward ($t = 2.08$, df = 195, $p = 0.0385$) from years 1 to 2. In addition, decrease in instrumental social support from baseline to year 1 was associated with decline from years 1 to 2 on Ascending Digit Span ($t = 2.76$, df = 183, $p = 0.0064$) and SDMT ($t = 2.14$, df = 187, $p = 0.0333$). In analyzing effects independent of changes in instrumental support, there was a significant effect of education on changes in the Ascending Digit Span score and of depression status and age on SDMT. Subjective social support and social network size did not appear to be associated with any changes in cognition.

In post-hoc analyses, we explored the combined effect of changes in stressors and in social interactions in a similar multivariate model and found that increase in number of stressors ($t = -3.10$, $p = 0.0022$) and decrease in number of social interactions ($t = 2.80$, $p = 0.0057$) continued to be significant independent predictors of decrease in CERAD TS. An additional model with the interaction of change in stressors-by-change in social interactions found the interaction term to be non-significant ($p = 0.6906$). Thus, change in social support did not appear to modify the effect of change in stressors on cognitive change.

To determine if the effects of stress or social support on cognition varied by depression status (patient vs. non-depressed comparison), we ran the five models with the appropriate interaction term included. The interaction term was significant ($p < 0.05$) in two of the models. For Model 2, change in social interaction predicting change in CERAD TS, the effect of change in social interaction differed between the patients and non-depressed comparison group. As social interaction decreased by 1, CERAD TS decreased by 0.72 points for the patients ($p = 0.0005$). A change in social interaction did not predict change in CERAD TS for the non-depressed comparison group ($p = 0.7116$). For Model 4, change in social interaction predicting change in Digit Span Forward also varied by group. Change in social interaction did not predict change

Table 2 Linear regression models of change in stress and social support baseline (BL) to year 1 predicting change in cognitive scores years 1 to 2 ($n = 213$)

	b	SE	*t*	*p*
Model 1: change in number of stressors predicting change in CERAD TS				
Age	0.111	0.073	1.52	0.1296
Sex (male)	0.497	0.886	0.56	0.5756
Education	−0.349	0.207	−1.69	0.0926
Depression Status (patients)	2.244	0.979	2.29	0.0229
Year 1 CERAD TS	0.254	0.046	5.51	<0.0001
Change in number of stressors BL to year 1	−0.647	0.227	−2.85	0.0048
Model 2: change in social interaction predicting change in CERAD TS				
Age	0.110	0.073	1.50	0.1339
Sex (male)	0.462	0.894	0.52	0.6059
Education	−0.384	0.210	−1.83	0.0693
Depression Status (patients)	1.765	0.964	1.83	0.0686
Year 1 CERAD TS	0.270	0.047	5.73	<0.0001
Change in social interaction BL to year 1	0.449	0.169	2.66	0.0084
Model 3: change in instrumental social support predicting change in digit span ascending				
Age	0.027	0.024	1.09	0.2788
Sex (male)	0.119	0.300	0.40	0.6911
Education	−0.231	0.073	−3.16	0.0018
Depression Status (patients)	0.219	0.318	0.69	0.4918
Year 1 Digits A Score	0.492	0.059	8.31	<0.0001
Change in instrumental social support BL to year 1	0.284	0.103	2.76	0.0064
Model 4: change in social interaction predicting change in digit span forward				
Age	0.005	0.017	0.28	0.7825
Sex (male)	−0.162	0.222	−0.73	0.4655
Education	−0.053	0.050	−1.04	0.2987
Depression Status (patients)	0.136	0.223	0.61	0.5431
Yr1 Digits F Score	0.242	0.043	5.69	<0.0001
Change in social interaction BL to year 1	0.088	0.042	2.08	0.0385
Model 5: change in instrumental social support predicting change in symbol-digit modalities test				
Age	0.233	0.068	3.42	0.0008
Sex (male)	−1.456	0.794	−1.83	0.0682
Education	0.079	0.192	0.41	0.6829
Depression Status (patients)	2.760	0.875	3.15	0.0019
Year 1 SDMT Score	0.132	0.040	3.28	0.0013
Change in instrumental social support BL to year 1	0.578	0.270	2.14	0.0333

Note: depressed = 1 and non-depressed = 0.

in Digit Span Forward for the non-depressed group ($p = 0.4957$). As social interaction decreased by 1 unit, Digits Span Forward decreased by 0.16 points for the patient group ($p = 0.0025$). While these findings suggest some topics for future work, they must be interpreted with caution as we tested interactions in five models, and the results are susceptible to Type I errors. We decided therefore to use a more stringent significance level for these interaction terms ($p < 0.01$), and did not include the terms in our final models. As a final step in model interpretation, we ran our five models also controlling for our social variables (e.g., total number of stressors, subjective social support, instrumental social support, social network size, and social interaction) at the year 2 level, but the results were essentially unchanged from the models presented here.

Discussion

The current study found that increased stress and decreased social support over a 1-year-period are associated with worse cognitive performance during the following year. The effects of stress were most consistent on a measure of global cognitive function. This study builds on the existing literature relating stress to cognitive decline by looking at both depressed and non-depressed participants in relation to cognitive decline. While depression status at baseline was associated with decline in cognitive ability, having a non-depressed comparison group in the study allowed us to see that changes in stress have an adverse effect on cognition in healthy older adults as well. By assessing these participants over a 2-year time period, we were able to see how psychosocial changes in late adulthood affect cognition

over time. Since the comorbidity of cognitive impairment and late-life depression are strong risk factors for dementia (Steffens and Potter, 2008), knowing the effects of stress in relation to depression and cognition will hopefully promote interventions to for these conditions in late adulthood or even earlier.

In terms of social support, this study found decline in social interaction and instrumental social support to be associated with cognitive decline in the following year. Reported decreases in social interaction over 1 year predicted the cognitive decline over the next year. Decreased instrumental support showed similar predictive effects of cognitive decline, but more in the area of verbal working memory and processing speed/executive functions than in global cognitive function. While decreased social interaction has been found to be associated with cognitive decline in several previous studies (Bassuk *et al*., 1999; Seeman *et al*., 2001; Holtzman *et al*., 2004), cognitive decline as a result of decreased instrumental support highlights an under-recognized aspect of the relationship between environmental factors and cognitive performance. These findings were all significant despite controlling for age, sex, education, and depression status.

Our results are consistent with much of the literature on stress and social support related to cognition in the elderly. While depression has consistently proven to be a risk factor for cognitive impairment (Stuck *et al*., 1999; Potter and Steffens, 2007; Steffens and Potter, 2008), this study has added support to the claim that there are other salient environmental factors that contribute to cognitive decline in the elderly. Stress has been found to have an impact not only in cognition for day-to-day memory tasks (VonDras *et al*., 2005; Sliwinski *et al*., 2006), but it has also been found to affect neurobiological structures as well (McEwen and Sapolsky, 1995; McEwen, 2000; Kim and Diamond, 2002). Stress and stress hormones have been found to impair hippocampus-dependent forms of memory in both humans and animals (Kirschbaum *et al*., 1996; McEwen, 2000). The hippocampus is strongly related in memory recall and since the CERAD total is largely representative of episodic memory (i.e., 50% of total points), the results of this study provide some support for arguments that stress is toxic to the hippocampus, or given that the CERAD TS is sensitive to MCI and AD (Chandler *et al*., 2005), there is some indication that increased stress may produce cognitive decline similar to what is seen in these conditions. We do note that stress was not associated with memory decline as assessment by WMS-R Logical Memory, which suggests that different memory processes may be affected differentially by stress.

In addition to stress, positive levels of social interaction and instrumental social support have been found to be protective factors for cognition in various studies (Hays *et al*., 2001; Seeman *et al*., 2001), which provides reasonable support for why a loss of these support systems would be predictive of cognitive decline, as was found in this study. Ultimately, learning how to manage stress and keep social support at stable levels could help maintain healthy cognitive function in late adulthood, especially if depression can also be simultaneously treated.

This study does have limitations to note. While we cannot rule out the possibility of a Type I error associated with multiple tests, these findings can lead to more narrowly defined studies of social factors and changes in cognition. Recent stress or problems with social support could have had more impact on how a patient or comparison participant reported their current state or changed one's performance on the follow-up cognitive tests. In fact, we did run tests to see if stress or social support within the year that the cognitive follow-ups were administered affected the outcome, but we found there to be no meaningful change in the results. When studying older adults, it is important to take into account the levels of attrition, but in this study we only used participants in the analyses with at least two years of available data. Doing so, however, cut down on the number of participants available for the analyses, but we find the effect sizes to still be clinically significant despite this limitation. Although this study did control for depression, the treatment that was given to the depressed patients, and not the non-depressed older adults, may have somehow affected the reporting of stressful events or changes in social support.

Even though we had follow-up tests that monitored the level of depression, we did not control for changes in severity of depression over the course of the study, which may have affected the cognitive scores and which could be addressed in a future study. Potential confounding between changes in stress or social support and change in depression severity might have explained the findings in part. That is, adverse changes in stress and social support may have been mediated by their effect on depression symptomatology which might have directly affected the cognitive outcomes. We also note, however, that patients had been in the study and under treatment at least a year before we assessed cognitive change and that their depression was relatively stable. These low and stables levels of depression reduce the likelihood that our results were affected by additional depression improvement over the same interval as cognitive change was assessed. While we did not examine this likelihood specifically,

Key points

- Along with biological factors, social factors have been shown to play a role in late-life depression and cognitive decline.
- Social factors salient in research in late-life depression include stressful life events and social support.
- Improvement in stress was found to positively influence cognition.
- Worsening social support negatively influenced cognition.

our study's specific contribution to the current literature is its focus on two social factors, stress and social support, over several years in the context of depressed and non-depressed patients. For future studies, the neurobiological symptoms of stress and changes in social support should be explored in relation to both depression symptomatology and their ability to predict neurocognitive decline.

Conflict of interest

None of the authors have any conflicts of interest to report.

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