Impact of Education on Memory Deficits in Subclinical Depression

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Accepted 29 May 2015

Abstract

Elevated depressive symptoms are associated with cognitive deficits, while higher education protects against cognitive decline. This study was conducted to test if education level moderates the relationship between depressive symptoms and cognitive function. Seventy-three healthy, dementia-free adults aged 18–81 completed neuropsychological tests, as well as depression and anxiety questionnaires. Controlling for age, sex, and state anxiety, we found a significant interaction of depressive symptoms and education for immediate and delayed verbal memory, such that those with a higher education level performed well regardless of depressive symptomatology, whereas those with lower education and high depressive symptoms had worse performance. No effects were found for executive functioning or processing speed. Results suggest that education protects against verbal memory deficits in individuals with elevated depressive symptoms. Further research on cognitive reserve in depression-related cognitive deficits and decline is needed to understand the mechanisms behind this phenomenon.

Keywords: Depressive symptoms; Cognition; Memory; Cognitive reserve; Educational achievement

Introduction

Approximately two thirds of depressed patients experience cognitive deficits (Afridi, Hina, Qureshi, & Hussain, 2011; Rock, Roiser, Riedel, & Blackwell, 2014). These deficits occur in multiple domains including executive function, episodic memory, visuospatial memory, attention, and processing speed (McDermott & Ebmeier, 2009; Rock et al., 2014). Additionally, structural and functional brain abnormalities in frontotemporal regions that subserve these cognitive functions have been noted in individuals who suffer from depression (Dotson et al., 2014; Naismith, Norrie, Mowszowski, & Hickie, 2012). Cognitive deficits are present not only in major depressive disorder, but also in individuals who exhibit “minor” or subclinical levels of depression (Dotson, Resnick, & Zonderman, 2008; Dotson et al., 2014; Elderkin-Thompson et al., 2003). Despite multiple findings of cognitive deficits, there is variability in the pattern and severity of depression-related cognitive difficulties across individuals (Baune et al., 2010; McDermott & Ebmeier, 2009). This variability may be due in part to differences in the severity of depressive symptoms across studies (McDermott & Ebmeier, 2009). Variability may also result from the heterogeneous nature of depression, as it has been suggested that specific symptom dimensions of depression may be associated with unique cognitive impairments (Baune, Suslow, Arolt, & Berger, 2007; Elderkin-Thompson et al., 2003; McDermott & Ebmeier, 2009). Additionally, variability in the relationship between depressive symptoms and cognitive functioning may be affected by individual differences in cognitive reserve.

Cognitive reserve is a term used to describe the brain’s ability to maintain adequate function despite neuropathology (Booth et al., 2013; Stern, 2002). Various proxies for cognitive reserve are used in the literature, most commonly vocabulary ability, leisure time activity, occupational attainment, and education (Baune et al., 2007). There is some debate over the extent to which education level accurately captures an individual’s cognitive reserve (Reed et al., 2010; Zahodne et al., 2013), but studies have demonstrated a high correlation between education and alternative measures of reserve such as vocabulary knowledge.
Research examining the role of cognitive reserve in the relationship between depressive symptoms and cognitive dysfunction is limited. One study found that lower education and higher depressive symptoms were associated with poorer performance on a brief mental status questionnaire in older, community-dwelling men (Wight, Anshensel, & Seeman, 2002). Another study found a similar interaction between education and depressive symptoms on task switching, verbal memory, and processing speed tasks in a group of depressed older adults (Avila et al., 2009). In contrast, a third study found no significant interactions between education level and depressive performance in predicting cognitive performance on tasks of processing speed, visuospatial functioning, executive functioning, language, or memory in older adults (Bhalla et al., 2005). Finally, a recent study found the reverse pattern of what would be expected, as older adults with higher levels of education showed more cognitive deficits at higher depressive symptoms than those with lower education (O’Shea et al., 2015).

Differing findings between studies may be partially explained by varying levels of education and depression severity; however, with such limited research on the subject, the effects of education on cognitive deficits in depression are still unclear. Additionally, previous studies have exclusively examined this relationship in older adults. It is, therefore, unknown whether the potential protective effects of education on cognitive deficits in depression are seen in younger adults or if it is unique to older individuals, who may be at greater risk for cognitive decline due to brain changes associated with aging (Fjell, & Walhovd, 2010). This study was, therefore, conducted to further explore the impact of education level and depressive symptoms on executive functioning, processing speed, and verbal memory in adults who ranged in age from 18 to 81 years. These domains were selected based on previous research indicating deficits in these areas in depressed individuals (McDermott, & Ebmeier, 2009; Rock et al., 2014). It was hypothesized that higher depressive symptoms and lower education levels would be associated with worse cognitive performance. Moreover, we predicted that higher education levels would be associated with less impact of depressive symptoms on cognitive functioning.

Methods

Participants

The sample included 73 community-dwelling adults between the ages of 18 and 81 years who were recruited from the University of Florida and the surrounding community via the Claude D. Pepper Registry for Research, local flyer and newspaper advertisements, and public service announcements. Inclusion criteria included right-handedness, native English speaking, normal or corrected-to-normal vision, an education of at least 9 years, and a score of >30 on the Telephone Interview for Cognitive Status (TICS; Brandt, Spencer, & Folstein, 1988). The TICS has a score range of 0–41, with higher scores reflecting better cognitive functioning. The cutoff of 30 has been shown to have a sensitivity of 94% as well as 100% specificity for differentiating between cognitively intact and demented individuals (Brandt et al., 1988). Participants were given the mood disorders, psychotic screeners, substance use disorders, and anxiety disorder modules of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-IV; First, Spitzer, Gibbon, & Williams, 2002) to assess the presence of major psychopathology, other than unipolar depression or anxiety disorders, that would be an exclusionary criteria. Individuals with an anxiety disorder were not excluded from the study due to the high comorbidity of anxiety and depressive symptoms (Fava et al., 2000; Kvaal, McDougall, Brayne, Matthews, & Dewey, 2008). Instead, anxiety symptoms were controlled for in all analyses. Two participants met criteria for major depression, six for an anxiety disorder, and four for comorbid major depression and anxiety disorder based on SCID-IV administration. Other exclusionary criteria included a history of major medical or neurological illness, head trauma, learning disorders, current antiepileptic or antipsychotic use, and language comprehension difficulties. All participants gave written and verbal consent to participate in the study. All procedures were in compliance with the Declaration of Helsinki, and the protocol was approved by the University of Florida Health Science Center Institutional Review Board. Sample demographic data are presented in Table 1.

Measures

Symptoms of depression were assessed using the Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977), a 20-item self-report questionnaire measuring the frequency and severity of depressive symptoms experienced in the past week that has been validated for use in both young and older community-dwelling adults (Haringsma, Engels, Beekman, & Spinhoven, 2004; Radloff, 1991). Each item on the CES-D is scored from 0 to 3, resulting in a potential total score range of 0–60, with higher scores signifying greater severity of depressive symptoms. State anxiety was assessed using the state form of the State-Trait Anxiety Inventory (STAI-S; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983).
Participants were administered a brief neuropsychological battery that focused on measures of executive functioning, processing speed, and verbal memory. The current study used the following measures: total immediate recall and delayed recall of the Hopkins Verbal Learning Test-Revised (HVLT-R; Brandt, & Benedict, 2001), completion time on the Trail Making Test parts A (TMT-A) and B (TMT-B; Reitan, 1992), and the Stroop Color–Word interference score (Golden, 1978). The Stroop inference score was calculated based on the traditional \( CW^2 \) formula, where \( CW \) corresponds to the color–word score and \( CW' \) is the predicted color-word score, calculated based on the word (\( W \)) and color (\( C \)) subtest scores \( \left( \frac{W \times C}{W + C} \right) \).

Statistical Analysis

Of the 73 subjects in the study, 1 had missing data for both TMT-A and TMT-B and 2 were missing data for the Stroop interference task. Additionally, three outliers (>3 SDs above the mean) were removed from the TMT-B analysis, resulting in sample sizes ranging from 69 to 73 for each test. Data were analyzed using linear regression models in SPSS version 21.0 (IBM Corp., Armonk, NY). Separate models were conducted for each of the aforementioned cognitive measures, with years of education and CES-D scores as independent variables and with age, sex, and STAI-S scores as covariates (see Table 1). All variables besides sex were continuous measures in the models. TMT-A and TMT-B scores were transformed via natural log and inverse transformations, respectively, to account for significant positive skew. Raw scores were used for Stroop interference, HVLT-R total recall, and HVLT-R delayed recall. Statistical significance was set at \( \alpha \leq 0.05 \).

Results

Results of the regression analyses are summarized in Table 2 and shown graphically in Fig. 1. Significant CES-D \( \times \) education effects were observed for HVLT-R total recall \( [\beta = 0.27, t(66) = 2.59, p = .01] \) and HVLT-R delayed recall \( [\beta = 0.28, t(66) = 2.53, p = .01] \). For both tests, higher depressive symptom severity was associated with worse performance in individuals with lower levels of education, but performance was preserved at higher depressive symptom severity in individuals with higher levels of education. This trend was seen in both younger and older adults when results were graphed separately. Additionally,
a similar pattern was observed when individuals with CES-D scores $>2$ $SD$s above the mean were removed, suggesting that the results, while influenced by more severe depression scores, are not solely explained by them. There were no main effects of CES-D or education on any measure and no significant CES-D $\times$ education interactions for Stroop interference, TMT-A, or TMT-B.

**Discussion**

Results from this study suggest that education level interacts with depressive symptoms to affect verbal memory, such that individuals with higher education levels may not show deficits despite elevated depressive symptoms, whereas those with lower education levels perform more poorly at increasingly elevated levels of depressive symptoms. This finding is consistent with previous work which found that education level serves as a protective factor against deficits in verbal memory in late-life depression (Avila et al., 2009) and against deficits in general cognitive status in older adults with elevated subclinical depressive symptoms (Wight et al., 2002). The current study extends previous findings to a sample that ranged from young adults to older adults, demonstrating that the effect is not limited to late life. Results are also similar to previous research on education’s impact on cognition in multiple disease states, including Hepatitis C, Alzheimer’s disease, and Parkinson’s disease (Bieliauskas et al., 2007; Cohen et al., 2007; Stern, 2012).

Our results are not consistent with a recently published study which found that higher depressive symptoms were associated with memory, executive functioning, and language deficits in individuals with higher education, but not in those with lower education (O’Shea et al., 2015). These former findings suggest that those with higher education may lose the protective nature of education at increased depressive symptoms, in contrast to our finding that those with higher education are able to maintain performance at elevated depressive symptoms. This discrepancy may be due in part to differences between studies in the range of depressive symptoms. While both studies examined subclinical depressive symptoms, the previous study used the CES-D short form and scores ranged from 0 to 9, while we used the full 20-item CES-D and scores ranged from 0 to 45. Perhaps the greater variability in scores in our sample allowed us to detect relationships across a wider range of severity.

The verbal memory results are consistent with the cognitive reserve hypothesis, which postulates that those with higher levels of cognitive reserve can maintain cognitive performance despite neuropathology because they are able to utilize alternative cognitive strategies and neurological pathways to compensate for the effects of the disease (Stern, 2012). It is unclear whether higher education resulted in participants in our study using more effective strategies to remember words or if they were simply better able to manage the demands of the test compared with those with lower education. The effective implementation of strategies may also explain why we found a protective effect of education on memory but not on executive functions or processing speed, because
mnemonic strategies are frequently taught in educational settings. Results of previous research examining a potentially protective effect of education on depression-related dysfunction in executive and speed abilities have been mixed, with one study finding significant protective effects (Avila et al., 2009), another noting no significant interactions (Bhalla et al., 2005), and a third study finding higher education associated with more decline (O’Shea et al., 2015). Although reasons for the discrepant findings are unclear, differences may be partially explained by differences in educational attainment between samples. Education moderated the relationships of depression with executive functioning and processing speed in a study that compared individuals with 1–4 years of education with those with 5 or more years (Avila et al., 2009) and in a study where the average level of education was relatively low (9.7 years; O’Shea et al., 2015), but not in the two studies, including the present study, that included samples with overall higher education levels (Bhalla et al., 2005). In the current study, education level ranged from 10 to 20 years, and in the study by Bhalla and colleagues (2005), the mean age of even their “low education” group was 11.3 years. Perhaps the protective effect of education on executive functioning and processing speed is apparent after just a few years of schooling, but plateaus or shows smaller gains as more years of education are attained. In contrast, educational attainment may continue to benefit verbal memory after more years of schooling. Alternatively, deficits in processing speed may be difficult to compensate for compared with verbal memory tasks, and therefore, increased education may not significantly assist in the retention of this ability. The specific tasks used to measure executive functioning and processing speed may have also affected the results, because alternative measures may have been more sensitive to the effects of depressive symptoms, education, or both.

Results from the current study suggest that individuals with elevated depressive symptoms and low education may be more susceptible to verbal memory deficits. Depression-related memory deficits are associated with functional disability, particularly in older adults. For example, impairments in everyday problem-solving ability in depressed older adults were mediated by memory and reasoning deficits in the Advanced Cognitive Training for Independent and Vital Elderly study (Yen, Rebok, Gallo, Jones, & Tennstedt, 2012). In another study (Gallo et al., 2003), memory and problem-solving ability mediated the relationship between depression symptoms and everyday problem-solving and observed tasks of daily living. In conjunction with these findings, our results indirectly suggest that individuals with low education and elevated depressive symptoms may be at increased risk for functional decline. Future research should directly test this possible relationship.

This study sample was well educated overall and had mostly subclinical levels of depression. Research examining more severely depressed individuals, those with significantly lower education, and additional aspects of executive functioning may provide more insight into the protective nature of education on cognitive deficits in depression. The potential impact of age on the interactive effect of education and depressive symptoms on cognitive performance could not be assessed given the limited sample size, which precluded analyzing a three-way interaction between age, education, and CES-D scores. Our significant results across a wide age range suggest that the protective effect of education may not be unique to older adults, and therefore, future research would benefit from examining this potential three-way interaction. Additionally, although education is commonly used as a proxy for cognitive reserve, it may not fully capture all of the variance associated with cognitive reserve (Reed et al., 2010). Reed and colleagues (2010) suggested that cognitive reserve should be directly measured by examining the difference between expected and actual performance given brain pathology. In other words, the variance in cognitive performance that cannot be attributed to measured brain pathology could be identified as the variance due to cognitive reserve. Further research examining cognitive reserve’s influence on cognitive deficits in depression by using either additional proxy measure for reserve, such as vocabulary knowledge, leisure activities, or occupational attainment or attempting to implement Reed’s operational definition for cognitive reserve, would be beneficial to further understand the extent to which reserve protects against cognitive deficits in depression (Reed et al., 2010; Zahodne et al., 2013).

Overall, results suggest that memory deficits related to depressive symptoms may be attenuated by higher levels of education. This phenomenon may occur across the lifespan, not only in older adults who are more vulnerable to cognitive deficits. These results emphasize the importance of accounting for lower education levels during clinical evaluation, as less-educated individuals may be particularly vulnerable to memory deficits, even at subclinical depression levels. Gaining a better understanding of the neuropsychological deficits seen in subclinical depression and how education level affects these deficits will help to further clarify the role cognitive reserve plays in the neuropsychological functioning of individuals suffering from depressive disorders.

Funding

This work was supported by an Age-Related Memory Loss award from the McKnight Brain Research Foundation (VMD). VMD is partially supported by the University of Florida Claude D. Pepper Center, which is funded by the National Institute on Aging (P30 AG028740-01). SMS is supported by a grant from the National Institute of Aging (T32AG020499-11).
Conflict of Interest

None declared.

Acknowledgement

We thank Dr. Christopher Sozda for assistance with data collection.

References


