Auditory Memory Decrement: Without Dissimulation, among Patients with Major Depressive Disorder

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Abstract

Questions have been raised about whether poor performance on memory tasks by individuals with major depressive disorder (MDD) might be the result of poor or variable effort or disease-related disruption of neural circuits supporting memory functions. The present study examined performance on a measure of task engagement and on an auditory memory task among 45 patients with MDD (M age = 47.82, SD = 19.55) relative to 32 healthy controls (HC; M age = 51.03, SD = 22.09). One-hundred percent of HC and MDD volunteers performed above the threshold for adequate effort on a formal measure of task engagement. The MDD subjects performed significantly more poorly than the HC subjects on an auditory learning and memory test. The present results suggest that auditory memory difficulties do occur among those with MDD and that decrements in performance in this group may be related to factors other than lack of effort.

Keywords: Depression; Malingering/symptom validity testing; Learning and memory

Introduction

Major depressive disorder (MDD) has been associated with poor neuropsychological functioning, including lowered performance on tasks that measure executive functions (e.g., Langenecker et al., 2005), attention (Comblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989; Porter, Gallagher, Thompson, & Young, 2003), and memory (Austin, Ross, Murray, & O’Carroll, 1992; Brown, Scott, Bench, & Dolan, 1994; Burt, Zembar, & Niederehe, 1995; Elliott, Sahakian, McKay, & Herrod, 1996), among other domains (King & Caine, 1996). Euthymic individuals with a history of MDD continue to exhibit cognitive deficits in attention and executive functioning domains during remission of symptoms (i.e., Paelecke-Habermann, Pohl, & Leplow, 2005; Weiland-Fiedler et al., 2004). Cognitive functioning has also been shown to be worse in MDD individuals who experience consecutive episodes, suggesting a sensitization process (Basso & Bornstein, 1999; Fossati et al. 2004), although it may also be an index of severity and reduced cognitive reserve.

With specific regard to memory, there are reports of decreased performance on measures of initial and delayed recall and delayed recognition by individuals with MDD (Austin et al., 1992; Basso & Bornstein, 1999; Burt et al., 1995). For example, a meta-analysis by Burt and colleagues (1995), which predominantly included studies conducted before formal effort tests were more widely in use, demonstrated moderate effect sizes on memory tests among those with MDD. Also, some prospective studies suggest that depression may be associated with a subsequent increased risk of general cognitive decline (Brodaty et al., 2003; Dufoil et al., 1996). Alternatively, other studies of individuals with depressive symptoms, including those who were determined to meet MDD criteria, suggest that individuals with MDD or depressive symptoms may not demonstrate significant deficits in implicit memory and episodic recall (Elderkin-Thompson, Moody,
Knowlton, Helleman, & Kumar, 2011), psychomotor speed (Hermens, Naismith, Redoblado Hodge, Scott, & Hickie, 2010), or memory (Langenecker et al., 2005). The apparent discrepancies across the literature of whether depression results in memory difficulties, and in which domains these difficulties might be expected, are further fueled by variability in ascertainment samples and methods used to determine the presence of depression (Langenecker, Lee, & Bieliauskas, 2009).

Inconsistencies in the presence, extent, and foci of cognitive difficulties in depression have been further confounded by variable reports of poor effort in those with MDD and/or significant depressive symptoms. For example, poor performance on tasks of memory, attention, and executive functioning among individuals with MDD has been attributed to poor effort in executing a given task, particularly in tasks with greater levels of difficulty or effortful requirements (Rohling, Green, Allen, & Iverson, 2002; Sachs-Ericsson, Joiner, & Blazer, 2008). The effort hypothesis posits that individuals with MDD demonstrate difficulty with effectively allocating effort when challenged by a more demanding cognitive task (Hasher & Zacks, 1979). It has also been proposed that the impairment in sustained effortful tasks is related to a generalized state of low motivation in depressed individuals (Cohen, 1982). Some early studies of effort and motivation in mood disorders suggested that MDD patients often perform automatic memory tasks (e.g., recognition) within normal limits, but perform poorly on tasks that are more challenging, and hence effortful (e.g., recall tasks; Cohen, 1982; Rohling & Scogin, 1993; Roy-Byrne, 1986; Weingartner, 1981; Zakzanis, Leach, & Kaplan, 1998).

Complicating matters and potentially obscuring these reports, research with groups of individuals undergoing forensic workers compensation neuropsychological evaluations demonstrated that those who exhibited failed performance on measures of dissimulation also had significantly higher symptoms of depression or clinical diagnosis of psychological dysfunction (Gervais, Rohling, Green, & Ford, 2004; Rohling et al., 2002; Sumanti, Boone, Savodnik, & Gorsuch, 2006). In contrast, more recent studies of individuals with depressive symptomatology or diagnosis of MDD have challenged the notion that lowered performances on cognitive measures among patients with MDD reflect poor effort. Using the Test of Memory Malingering (TOMM; Tombaugh, 1996) for instance, Ashendorf, Constantinou, and McCaffrey (2004) did not observe a significant relationship between failure of the TOMM and the presence of depressive symptoms in a community-dwelling sample of individuals without MDD. Low levels of task disengagement were also reported in another study of depressed inpatients (Rees, Tombaugh, & Boulay, 2001), where the vast majority of inpatients diagnosed with MDD scored above the traditional cut-off for the TOMM. Finally, a study by Egeland and colleagues (2005) reported that research volunteers with recurrent, Structured Clinical Interview for DSM (SCID)-verified MDD did not exhibit higher levels of effort test failure relative to the matched control group and that memory retrieval and executive functioning deficits were present.

It is possible that conflicting reports of memory deficits being present and effort measures being passed or failed by MDD individuals is related to the samples from which study volunteers are drawn, methods by which depression is classified, presence of comorbid condition(s), possibility of primary and secondary gain, sample size, or other methodological differences. Thus, there is the possibility that significant relationships between performance on measures of dissimulation and depressive symptomatology reflect sampling bias related to medico-legal contexts. It appears that there is a greater likelihood that the presence of depressive symptoms in a neuropsychology clinic setting, where primary and secondary gain may be present, results in a much different sample than of those with MDD who are volunteers or are being seen in non-neuropsychology clinic settings. It may be that the presence and opportunity for gain, and not diagnosis, is the operational driving mechanism in these cases, as failure rates on formal measures of effort are high across TBI, pain, and psychiatric diagnoses in neuropsychology clinic settings (Gervais et al., 2004; Greve, Ord, Curtis, Bianchini, & Brennan, 2008; Slick, Hopp, Strauss, & Spellacy, 1996).

Apart from the mixed and apparently conflicting reports of cognitive dysfunction in MDD, increasing evidence is emerging about the neurobiological underpinnings of cognitive decrements that have been reported. Findings of memory decrements among individuals with MDD are supported by evidence of hippocampal atrophy among individuals with MDD (Bremner et al., 2000; van der Flier et al., 2004). Further, Sheline, Wang, Gado, Csernansky, and Vannier (1996) found that the degree of volume loss correlated with months of untreated illness, and others have hypothesized that glucocorticoid toxicity contributes to volume loss in the hippocampus and subsequent memory problems (Bremner & Narayan, 1998; Sapolsky, 1985). One recent meta-analysis of hippocampal volume in MDD reported that children and middle-aged or older adults with more than one episode and greater than 2 years of illness exhibited less hippocampal volume when compared with controls (McKinnon, Yucel, Nazarov, & MacQueen, 2009), while an older meta-analysis reported average volume loss of 8% on the left and 10% on the right across studies (Videbech & Ravnikilde, 2004).

There is also ample evidence to conclude that cognitive dysfunction among individuals with MDD might serve as a reflection of neural abnormalities within frontal-limbic circuits, including amygdala, hippocampus, subgenual and rostral anterior cingulate gyrus, and dorsolateral prefrontal cortex (Drevets & Raichle, 1992; Grimm et al., 2008; Hamilton, Siemer, & Gotlib, 2008; Hastings, Parsey, Oquendo, Arango, & Mann, 2004; Langenecker et al., 2007; Neumeister et al., 2006;
Sheline, Sanghavi, Mintun, & Gado, 1999; van Eijndhoven et al., 2009). These circuits are critically important in the performance of learning and memory tasks (Bremner & Narayan, 1998; Johnson, Schmitz, Asthana, Gluck, & Myers, 2008; Schacter, Savage, Alpert, Rauch, & Albert, 1996; Squire, 1992). Structural and functional disruption to neural circuitry has also been related to performance decrements on cognitive measures among individuals with MDD (Sheline et al., 1999) and as negatively predictive of response to antidepressant medication (i.e., s-citalopram) in MDD (Langenecker et al., 2007).

The current study was designed to test the proposition that memory difficulties are present in moderate to severe MDD (verified with SCID; First, Spitzer, & Gibbon, 1995) and that these decrements in learning and memory are not the result of sub-optimal effort. A critical question is whether previously mixed reports of memory difficulties in MDD are related to the sample collected, whether the sample was actually diagnosed with MDD or not, whether a comorbid diagnosis or rule-out diagnosis was present, whether primary or secondary gain was present, whether formal effort measures were used, the specific cognitive tests used, and sample size. This appears to be a critical gap in the literature. For the current study, we tested whether SCID verified MDD is associated with a higher than the normal level of dissimulation, and/or whether dissimulation sufficiently explains poor memory performance among individuals with MDD. We hypothesized that MDD and healthy control (HC) subjects would perform equivalently on a measure of dissimulation, but that MDD subjects would perform more poorly than the HC subjects on an auditory serial learning and memory task. By using a more homogeneous, paid volunteer sample with SCID-verified MDD, we attempted to address the question of whether MDD, absent many of the previously reported confounding features, could be related to intact effort and impaired memory ability.

Methods

Participants

Seventy-seven subjects participated in the study, including 45 with MDD and 32 HC, after matching the two groups for age and education level. Three of the MDD individuals had previously undergone ECT more than 7 months prior to their study participation, which was considered sufficient interim time for inclusion (see Semkovska & McLoughlin, 2010). Subjects were recruited from outpatient clinics, participant databases, and community advertisement. The HC and MDD participants were all community dwelling volunteers, recruited from the same community using the same advertisements and reimbursed at the same rate. MDD participants were also made aware of study opportunities in outpatient psychiatry and primary care clinics through use of flyers and bulletins.

Each subject was evaluated with the SCID (First et al., 1995) and the 17-item Hamilton Depression Rating Scale (HDRS; Hamilton, 1967). Participants were considered depressed if the SCID interview supported a current diagnosis. Exclusion criteria included a history or diagnosis of any traumatic brain injury, loss of consciousness of >3 min, dementia, history of stroke, current substance abuse, untreated diabetes, medical instability (e.g., acute, terminal, or worsening major medical condition), currently undergoing major medical treatment such as chemotherapy or radiation, or inability to speak English fluently. Individuals with MDD could not have a history of bipolar disorder or psychosis. Thirty-two individuals with MDD were receiving treatment with an antidepressant medication during study participation and 10 individuals with MDD had not been treated with antidepressant medication. We did not have medication data for three participants with MDD. Eighteen of 45 individuals with MDD had a comorbid anxiety disorder. For the total sample, there were no significant differences between the HC and MDD groups in years of education, t(75) = 1.05, p = .23; sex, χ²(77) = 1.44, p = .23; or age, t(75) = 0.67, p = .50. We also obtained estimated IQ scores, as measured by the Shipley Institute of Living Scale (Shipley, 1940), for 27 HC and 30 MDD, a measure added after the study was underway to address the relative specificity of learning and memory difficulties in MDD. The two groups were equivalent on this measure, t(55) = 0.36, p = .72, and they were also equivalent in education, suggesting no general cognitive differences between these groups. Please refer to Table 1 for further description of the sample.

Table 1. Participant demographics

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Sex (M/F)</th>
<th>Age (M [SD])</th>
<th>Age Range</th>
<th>Education (M [SD])</th>
<th>HDRS (M [SD])</th>
<th>Prev. ECT</th>
<th>Est. IQ-SILS (M [SD])</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC</td>
<td>32</td>
<td>15/17</td>
<td>51.03 (22.09)</td>
<td>19–80</td>
<td>16.09 (1.91)</td>
<td>0.81 (1.03)</td>
<td>0</td>
<td>106.9 (22.0)</td>
</tr>
<tr>
<td>MDD</td>
<td>45</td>
<td>25/30</td>
<td>47.82 (19.55)</td>
<td>18–79</td>
<td>15.49 (2.84)</td>
<td>21.12 (5.41)</td>
<td>3</td>
<td>108.7 (15.4)</td>
</tr>
</tbody>
</table>

Notes: HC = healthy control; MDD = major depressive disorder; HDRS = Hamilton Depression Rating Scale-17 item; ECT = electroconvulsive therapy; SILS = Shipley Institute of Living Scale.

*a n = 27 HC and n = 30 MDD.*
Measures

Test of Memory Malingering. The TOMM (Tombaugh, 1996) was administered to all subjects prior to completion of the California Verbal Learning Test, 2nd Edition (CVLT-II; below). The TOMM is a formal measure of dissimulation and is used to help identify insufficient effort on memory tasks. Subjects are shown 50 images and then given 50 forced-choice questions with feedback. During the forced choice phase, two images are displayed, one image previously seen and the other a novel image. Subjects are then shown the same 50 images and given a new set of forced-choice questions, with each of the initially viewed items matched to a different novel image. Feedback is given for performance on TOMM 1 and 2 trials. A low score (<.45) on the second trial suggests that the subject may have put forth poor effort.

California Verbal Learning Test, 2nd Edition. The CVLT-II (Delis, Kramer, Kaplan, & Ober, 2000) was also administered to all subjects. In this test, subjects are read a list of 16 words five consecutive times. Following each trial, subjects are asked to recall as many words as possible. The words in the list belong to four semantic categories, but are read to the subject in a standard, uncategorized order. After the five learning trials, the examiner reads aloud a distracter list that the subject is asked to recall immediately. The subject is then asked to recall the original list without any prompts (short delay free recall, SDFR). After this free recall phase, subjects are asked to recall words from each semantic category using the category descriptor as a prompt (short-delay cued recall, SDCR). After a 20–30-min delay, the subject is asked to recall the original list without (long-delay free recall, LDFR) and with (long-delay cued recall, LDCR) category prompts. In addition, a yes/no recognition trial is administered, which includes words from the target list, distracter list, and words that were not on either list but may be semantically related to the target list (long-delay recognition discriminability). Ten minutes later, the subject is administered a forced choice task (not included as part of main analyses in this study due to ceiling effects in performance).

There were four distinct orders in which the tests were administered. TOMM was always administered first. Initial trials of the CVLT-II were administered after the TOMM. A visual learning and memory test in development (Langenecker et al., 2005) was then administered in the delay for some individuals (n = 58), whereas others completed tests of visual spatial attention (n = 19). Fine motor tests, measures of executive functioning, and questionnaires were also used to fill the delay period. Test administration order used in the delay period for the CVLT-II did not influence the results; it was not a significant covariate, F(10, 63) = 1.53, p = .15.

Statistical Analyses

Forty-five individuals with MDD (M age = 47.82, SD = 19.55) and 32 HC (M age = 51.03, SD = 22.09) completed the TOMM and CVLT-II. For the TOMM Trial 2, we conducted an analysis of covariance, with MDD status entered as the independent variable and age and education included as covariates. To examine CVLT-II performance between groups, a multivariate analysis of covariance was performed, with MDD status entered as the independent variable and age and education included as covariates. The 10 dependent variables were z-scores from the five learning trials, SDFR and SDCR trials, LDFR and LDCR trials, and long-delay recognition. Scores are age-corrected using the CVLT-II sample of 1,087 U.S. adults (Delis et al., 2000). The age-corrected z/t scores were only somewhat successful in removing the impact of age in our sample. For example, a correlation of SDFR raw with age was r = -0.68, p < .001 (r = -.71 for MDD only), whereas the correlation of SDFR z-scores with age was r = -0.42, p < .001 (r = -.51 for MDD only). Age was used as a covariate in analyses, and z/t scored were the dependent variables to minimize the impact of age within the model, as that was beyond the purview of this study.

Results

Consistent with hypothesis 1, there was no significant difference in TOMM performance between the HC (M trial 2 = 49.83, SD = 0.79) and the MDD groups (M trial 2 = 49.70, SD = 0.80), F(1, 73) = 0.50, p = .48. No participants scored <.45 on Trial 2. Thus, all participants demonstrated normal TOMM performance, overall, as described in the TOMM manual (Tombaugh, 1996). There was no significant interaction between TOMM scores and age, F(1, 73) = 1.81, p = .18. Consistent with hypothesis two, the MDD group performed significantly worse overall than did the HC group on the CVLT-II, F(10, 64) = 2.68, p < .01. The HC group performed significantly better than that did the MDD group on: Trial 2, F(1, 73) = 13.55, p < .001; Trial 3, F(1, 73) = 8.50, p < .01; Trial 4, F(1, 73) = 10.34, p < .01; Trial 5, F(1, 73) = 7.06, p < .01; SDFR, F(1, 73) = 4.89, p < .05; LDCR, F(1, 73) = 5.43, p < .05; and long-delay recognition, F(1, 73) = 7.41, p < .01. Differences in the remaining non-significant post hoc tests were in the same direction (HC > MDD). Results are displayed in Fig. 1 using both standardized (Fig. 1a) and raw scores (Fig. 1b). In a post hoc analysis using the higher TOMM 2 cutoff suggested by Greve and colleagues (2008), results were significant, and nearly identical, after using a
more stringent threshold for TOMM 2 performance (raw score of 49 or more; 30 HC and 43 MDD), $F(10, 60) = 2.19, p < .05$. Age was a significant covariate in the model across all but the recognition trial. The effects of group were independent of age effects. While forced choice recognition performance was not included in the MANCOVA due to severe lack of range, we report results for informational purposes. All but two individuals (both in the MDD group) achieved a score of 15 or 16/16 on forced choice recognition. These two subjects achieved a score of 14/16. Results did not change when these subjects were excluded from analyses.

To explore whether depression severity (as measured by the HDRS) was correlated with performance on the CVLT-II in the MDD sample, we conducted partial correlation analyses with CVLT-II scores and HDRS scores, covarying for age and education. Depression severity was not significantly related to performance on any of the CVLT-II trials (all $p > .05$).

**Discussion**

This is one of the first studies to include a well-powered sample of individuals with MDD who are evaluated with both formal dissimulation and memory measures. Both HC and subjects diagnosed with MDD were considered to be putting...
forth good effort, as demonstrated by equivalent scores on the TOMM, and no scores were below the standard exclusion criteria in either group on the second trial. Even after using a more stringent threshold as demonstrated by Greve and colleagues (2008) in a clinical, workers compensation sample, the results were still significant. Differential performance between HC and MDD individuals on the CVLT-II also supports the hypothesis that individuals with MDD may have learning and/or memory deficits. While individuals with MDD performed equivalently to non-depressed HC during the first learning trial, they failed to match the HC learning curve for subsequent learning trials, with relative impairment in most subsequent memory trials. This pattern of performance suggests that consolidation and organization of memory may be deficient in MDD. The same pattern was present for short-delay free recall, LDCR, and long-delay recognition tasks, relative to HC, suggesting poor encoding overall relative to non-depressed individuals. These results support previous studies of poor retrieval and encoding processes among individuals with MDD (for review of these findings see Burt et al., 1995; Ingram & Reed, 1986).

These results are important for both clinical and social reasons. While the presence of primary and secondary gain may skew the validity of neuropsychological scores within medico-legal contexts, findings suggest that clinically depressed individuals who volunteer for research studies (with payment for participation not being attached to performance) do not exhibit a lack of motivation, as measured by formal effort testing, yet still demonstrate auditory learning and memory impairment on one measure. For clinical evaluations where primary and secondary gain are not present, clinicians should explore the hypotheses that memory and other cognitive difficulties in MDD may be of genetic, endocrinological, metabolic, and/or neurodegenerative significance. Second, there is an emerging area of research focused on neuropsychological techniques assisting in treatment outcome prediction (Goldman, Axelrod, Tandon, & Ribeiro, 1993; Lantz & Sterman, 1992; Xu, Wang, Liu, Zhang, & Yan, 1998). For example, in clinical contexts, having greater confidence that observed cognitive decrements are not related to poor effort and have neurological underpinnings may suggest a poorer treatment prognosis (Kampf-Sherf et al., 2004; Taylor et al., 2006). Use of effort and cognitive tests in those with MDD can be a legitimate and potentially productive tool to understand the probability of treatment response.

Third, impaired behavioral and cognitive performance by those with MDD is often explained by the lay person (and some clinicians) as resulting from lethargy, apathy, and diminished ability to automatically engage complex neural circuits when challenged. This diagnosis-related stigma attached to many mood disorders is now being challenged by findings suggesting neurobiological underpinnings of MDD and mixed evidence of reversibility in cognitive deficits (Basavaraju & Phillips, 1989; Langenecker, Lee, & Bieliauskas, 2009; Perri, Carlesimo, Serra, & Caltagirone, 2009; Racine, Lawton, Hett, & Josephson, 2008). The results presented here help to demonstrate that poor effort cannot uniformly explain auditory memory performance in those with MDD and that other hypotheses should be entertained when a primary diagnosis of MDD is present.

One possible critique of studies that use dissimulation measures is the limited temporal validity and breadth of specificity of the measures. We administered the TOMM before the memory measure in the current protocol. Dissimulation measures that are administered early in a neuropsychological battery may not gauge decreasing effort as the evaluation proceeds. Future studies in line with this research could counterbalance the order of memory tasks and dissimulation measure, or use an additional measure of dissimulation to sandwich the memory tasks. However, as 45 individual volunteers with MDD were recruited from multiple settings (e.g., outpatient psychiatric clinic, community advertisement) and all passed this formal measure of dissimulation, it is less likely that poor effort could account for the difficulties in memory performance. Also, the TOMM was administered prior to the CVLT-II for all participants. It is possible that proactive interference from the TOMM influenced performance on the CVLT-II, although interference from a visual recognition memory paradigm followed by an auditory learning paradigm would be expected to be quite minimal. Further, one study of 44 patients with MDD found no evidence of proactive interference in a verbal learning and memory test (Porter et al., 2003). Another potential limitation is that only one effort and one memory measure are reported. We cannot comment on the breadth of difficulties, nor the stability of intact effort in this sample. Furthermore, there is significant debate within the literature about whether performance on the TOMM may be insensitive to poor effort, with some studies showing reduced sensitivity (Gervais et al., 2004; Green, 2007; Greve et al., 2008), and others showing equivalent sensitivity relative to other measures (Greiffenstein, Greve, Bianchini, & Baker, 2008). To guard against this interpretation, we use the standard cutoff of 45 on Trial 2, plus a more sensitive cutoff of 49 as suggested by Greve and colleagues (2008), without significant differences observed between groups in either analysis. Finally, participants were not assessed for the presence of PTSD, which has known cognitive sequelae, including memory deficits. While performance on the CVLT-II did not differ among participants with and without other comorbid anxiety disorders, we cannot say whether the presence or absence of PTSD, specifically, influenced results. Future studies should consider whether PTSD that is comorbid with MDD impacts findings of effort and cognition.

This study re-evaluated the veracity of the argument that poor memory performance by depressed individuals is due to poor effort. With the presented results suggesting that there is more to the story than lack of effort, future studies should consider the influence of severity of current depressive symptoms, number of episodes, length of illness, medications, and similar features
related to treatment course and chronicity on effort and memory performance in depressed individuals. In particular, this study and a growing body of literature suggest that context of ascertainment, homogeneity of sample including the method of diagnosis, and other critical features reported here should be considered prior to attributing low effort to MDD.

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Conflict of Interest

None declared.

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References


