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Effects of depression on sensory/motor vs. central processing in visual mental imagery

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Visual mental imagery is often affected by those experiencing a major depressive disorder (MDD), and is commonly used in cognitive behavioural therapy to treat these patients. However, the nature of imagery in this population has not been studied in depth. Moreover, it remains unclear what aspects of cognitive processing are responsible for psychomotor retardation observed in some patients with MDD. Control participants and participants who were experiencing MDD performed a mental image generation task, a mental image rotation task, and a task that required them to identify objects seen from canonical vs. noncanonical viewpoints. In all three tasks, participants with MDD performed “central processing” (which leads to the decisions required by the task) as well as control participants, but were slower in sensory/motor processing. These results suggest that the psychomotor retardation observed in patients with depression may result from an encoding or motor output deficit rather than a cognitive deficit.
Most individuals with major depressive disorder (MDD) are notoriously slow and unresponsive; they are said to have “psychomotor retardation” (e.g., Barkley & Tryon, 1995; Cornell, Suarez, & Berent, 1984; Dantchev & Widlocher, 1998). This characteristic could arise for one of three reasons. First, the MDD participants could be cognitively impaired, leading them to think slowly and with difficulty. Second, they could have difficulty encoding perceptual stimuli or making responses, but not be cognitively impaired. And third, they could be both cognitively impaired and have difficulty encoding information and making responses. We administered three tasks in an effort to distinguish among these alternatives. The processing required by the tasks can be divided into two types. On the one hand, some processes are required at input and output, to encode the stimuli and produce the responses. We refer to these operations as “sensory/motor processing”. On the other hand, processing is required to use the stimulus to perform the task itself, which requires operating on the stimulus and making a decision. We refer to these operations as “central processing”.

Many cognitive studies in patients with depression have documented that these individuals are impaired in various cognitive processes. These processes include, but are not limited to, executive function (e.g., Austin et al., 1992), memory (e.g., Austin, et al., 1992; Stromgren, 1977), and attention (e.g., Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989). Implicit in their interpretations of these results, researchers seem to assume that central processing is affected by depression. We have chosen cognitive tasks where central processing can be distinguished from other components of task processing. Our tasks are designed so that we can manipulate the difficulty of central processing without altering the difficulty of sensory/motor processing. For example, in one of our tasks, the hand rotation task, we vary the degree to which a picture of a hand must be mentally rotated (which requires central processing) without changing either the complexity of the stimulus or the nature of the responses (which require sensory/motor processing). Thus, these tasks can be used to discover whether major depression slows central processing, sensory/motor processing, or both. If depression impairs central processing, we would expect patients with depression to perform increasingly poorly, relative to control participants, when increasing amounts of central processing are necessary. A problem in sensory/motor processing, on the other hand, would not lead patients to perform increasingly poorly (relative to control participants) when more central processing is necessary.

Two of our tasks require visual mental imagery. This was appropriate because individuals diagnosed with major depression sometimes report changes in their imagery. For example, they sometimes report that stimuli evoke disturbing images more frequently than they did prior to the major depressive episode (Beck, Rush, Shaw, & Emery, 1979). In addition, some
forms of treatment for major depression rely on the ability to form mental images (e.g., cognitive behavioural therapy). For example, “induced images” are used to demonstrate to depressed clients the relationship between their thinking and their feelings (Beck & Weishaar, 1989). These forms of dynamic, narrative, emotional mental imagery draw on basic processes assessed with cognitive mental imagery techniques. In addition, we utilised a third task which does not involve visual mental imagery (Kosslyn, Thompson, & Alpert, 1997). This was to ensure that the effects we observed were not specific to visual mental imagery per se.

The present study is one of only a few that has investigated mental imagery in participants with depression. One other example is the study reported by Cocude, Charlot, and Denis (1997), who measured the amount of time participants needed to create images and the amount of time they could retain images once created. They found that participants with depression had trouble creating mental images, but that, once formed, these individuals could retain images for comparable amounts of time as the control participants. However, for the most part, previous cognitive research on participants with depression has focused on verbal processes, not mental imagery.

Specifically, in the present study we administered an image generation task, which required the participants to solve problems by generating (i.e., creating) an image. We also administered a task in which participants had to imagine pictures of hands rotating, which draws on a different sort of imagery (Shepard & Cooper, 1986). And finally, we asked the participants to identify objects seen from a familiar point of view (that is, a canonical viewpoint) vs. objects depicted from an unusual point of view (that is, a noncanonical viewpoint). Top-down perceptual processing is used when people identify pictures of objects seen from noncanonical points of view; moreover, this task and the image generation task activate about two thirds of the same brain areas (Kosslyn et al., 1997).

EXPERIMENT 1

Images of objects are not always present, nor are they produced instantaneously. Rather, a process of “image generation” is necessary to activate stored information to produce an image representation in short-term memory. To assess image generation, we used a version of a task developed by Kosslyn, Cave, Provost, and von Gierke (1988), and subsequently used in a neuroimaging study by Kosslyn et al. (1993). The task was shortened in order to avoid fatigue in depressed participants. The task required participants to visualize block letters within a 4 × 5 grid. An X mark later appeared in one of the cells in the grid, and the participants were to decide
whether the X would fall on or off the block letter if the letter were present in
the grid. Previous results with this task validated that performance does
indeed reflect one’s ability to generate mental images (Kosslyn et al., 1988,
1993). A perceptual version of the task was also administered to ensure that
the imagery task was in fact tapping the processes used in image generation
and not simply those used to inspect image patterns.

We manipulated the difficulty of central processing by varying two
aspects of the task. First, the letters were simple or complex, based on the
number of constituent segments. Kosslyn et al. (1988) found that more time
was required to generate images of more complex letters, but no more time
was required to inspect images of more complex letters when they were
presented perceptually. Second, we also varied the location of the probe
marks along the letter. Kosslyn et al. (1988) found that participants required
more time to evaluate probe X marks that fell on segments typically printed
later in the sequence of strokes. This finding, which was only present when
the image had to be generated (and not when it was generated before the X
was presented or when a block letter was physically present in the grid),
suggested that the segments are visualised in the order in which they are
typically printed.

By varying these two factors, complexity and probe location, we assessed
the efficacy of central processing without affecting the processing used to
encode the stimuli or to produce the responses. If participants with
depression have impaired central processing, they should have increased
difficulty (relative to controls) when generating images of complex letters
compared to simple ones; similarly, they should have increased difficulty
(relative to controls) when generating images to evaluate probes on later
segments compared to early ones. That is, if we were to graph the
performance against two levels of task difficulty, the slope for the depressed
participants should be steeper than that for the controls and we should see a
statistical interaction of depressive status and task difficulty. In contrast, if
central processing in this task is intact, we should instead find that the
depressed participants and controls have a comparable increase in response
times and error rates as difficulty increases, and no such interaction should
be observed. That is, if impaired responses arise because of the processing
used to encode the stimuli or produce the responses, we would expect overall
increases in response times or errors, but – and this is the crucial point –
increased difficulty should affect the patients and control participants to the
same degree. In other words, we would see a main effect difference in the
intercept, but not an interaction in the slope.

Our studies are cast within the framework developed by Saul Sternberg in
his famous work on “additive factors” analyses (Sternberg, 1966, 1998). The
encoding and response aspects of the tasks remain constant over the
variations of the independent variables. Thus, any changes in performance
that occur when the independent variables are manipulated cannot reflect the encoding and response processes. By definition, the slope – which is plotted against variations in the independent variable – reflects the effects of manipulating the independent variable. The intercept represents the processes that do not vary with changes in the independent variable – namely, encoding and response processes.

Method

Participants

All participants in this experiment, and the others described herein, responded to advertisements placed in various local newspapers (i.e., *Boston Globe, Boston Herald, Cambridge Tab, Harvard Gazette*). All participants were screened by telephone to determine whether they had any characteristics that would exclude them from DSM-IV (1994) criteria of MDD or make them unsuitable control participants. Respondents who met the general criteria of MDD, or who were control participants, were scheduled for a one-to-two hour Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1994) that was administered by either a trained research assistant or a clinical psychologist (PJD). A second trained interviewer listened to approximately one quarter of the SCID interview tapes to confirm the diagnosis of MDD. The rate of agreement between the two interviewers was 100%.

After the SCID interview, the participants were assigned to one of three categories: (1) control participants; (2) participants with MDD; or (3) not qualified for either category. All potential participants were compensated for their time, but only the participants who were assigned to the first two categories were tested further. The experimental session occurred on a different day than the interviews. All experiments, however, were completed on the same day.

The MDD participants with major medical illness (e.g. epilepsy, cancer, diabetes), head injury resulting in unconsciousness for more than 10 minutes, manic episodes, or current substance abuse were excluded from publication. Control participants were also excluded from participating in the experiment for these reasons or if they had past or present Axis I disorders.

All participants were unaware of the purposes and predictions of the experiment at the time of testing. Immediately before the experiment, the participant’s level of depression and anxiety was assessed using the following scales: the Beck Depression Inventory (BDI), which is a self-reported measure used to determine severity of depression (Beck, 1967; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Metcalfe & Goldman, 1965); the Beck Hopelessness Scale (BHS), which quantifies the participant’s expectation of positive and negative future events (Beck & Weissman, 1974); and the
Spielberger State-Trait Anxiety Inventory (STAI-S/STAI-T), two questionnaires that measure both an individual’s current anxiety state (STAI-S) and general anxiety trait (STAI-T; Spielberger, Gorusch, & Luschene, 1970).

Overall, 22 control participants and 22 patients with MDD took part in at least one of the three experiments in this study. Table 1 provides the mean ages of the two groups as well as their scores on the BDI, BHS, STAI-S, and STAI-T. A subset of participants from each pool took part in Experiment 1, a different subset in Experiment 2, and a different subset in Experiment 3. Although there was considerable overlap of participants among experiments, none of the three subsets was exactly the same. In each experiment, the MDD and control groups were balanced for age, education, and gender. The differences in the self-reported assessment scores remained highly significant for all subsets.

Of the 22 MDD participants who took part in these experiments, the mean major depressive episode duration was 16 months ($SD = 17$; range 1–60 months). Of the participants, 86% ($n = 19$) reported a previous depressive disorder and 54% ($n = 12$) also had comorbid dysthymia. Several participants with MDD had comorbid anxiety disorders, such as posttraumatic stress disorder ($n = 2$), obsessive–compulsive disorder ($n = 3$), social phobia ($n = 3$), general anxiety disorder ($n = 1$), and panic disorder ($n = 2$); 59% ($n = 13$) of MDD participants did not have any comorbid anxiety disorders. There were no other comorbid Axis I disorders. Of the MDD participants, 54% ($n = 13$) had sought treatment for their MDD in the past, and 36% ($n = 8$) were currently seeking treatment for their MDD. Six MDD participants (27%) were currently on medication for their depression. In all experiments, there was no difference between the performance of the medicated participants, or those with comorbid anxiety disorders, and that of the other MDD participants, therefore both medicated and unmedicated participants were included.

<table>
<thead>
<tr>
<th>TABLE 1</th>
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<tbody>
<tr>
<td>Description of gender, age, and self-reported depression and anxiety scores for all members of the two groups that participated in this study (standard deviations are in parentheses)</td>
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<tr>
<td>Total number</td>
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<tr>
<td>Age (years)</td>
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<td>Beck Depression Inventory</td>
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<td>Beck Hopelessness Scale</td>
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<tr>
<td>Spielberger State Anxiety Inventory</td>
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<tr>
<td>Spielberger Trait Anxiety Inventory</td>
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MDD, major depressive disorder.

*An unpaired two-tailed t-test was used to compare the data from the two groups.
In Experiment 1, 16 control participants (8 women and 8 men), and 16 participants diagnosed as currently experiencing MDD (10 women, and 6 men) volunteered to take part in the study. The participants were balanced for age (\(M = 38.2, SD = 14.4\) for the control participants; \(M = 37.6, SD = 14.4\) for the depressed participants, \(p = .91\)). Balancing the groups on age is important because age has been shown to affect performance in the tasks we administered (Dror & Kosslyn, 1994). All participants were right handed.

**Materials**

All tasks were administered using the MacLab 2.0.0.d.50 program (Costin, 1988) on a Macintosh LC computer, with a black-and-white monitor. Four upper case, block letters were used as stimuli in this experiment, H, U, S, and J, and an X mark was used as a probe. The stimulus letters were created by filling in cells of the \(4 \times 5\) grid to form segments of block letters. Hence, the end result was an angular block letter with no curves. These letters were classified as simple or complex depending on the number of segments used to depict them: H and U were considered simple letters (three segments each) whereas J and S were considered complex letters (four or more segments). The X mark was created by connecting the diagonal corners of one of the cells in the grid. All letters and the X mark were used during all three phases of the study (acquisition, imagery, and perception).

There were 16 trials in both the imagery and perception portions of the experiment. In the imagery task, each letter served as a stimulus four times in each phase, twice covering the X mark on the grid (true trials) and twice having the X mark fall on a cell that was adjacent to a segment of the letter (false trials). In addition, for half of the trials of each type, the X mark was placed on a cell, or adjacent to a cell, of a segment that was at the beginning of the sequence in which the segments are typically printed (i.e., the left vertical bar of the H; see Kosslyn et al., 1988). These are referred to as early trials. For the other half, the X mark was placed on or adjacent to a segment that is typically printed at the end of the sequence (i.e., the horizontal bar in H). These are referred to as late trials. Two versions of this experiment were prepared, with the order of the stimuli in one being the reverse of order in the other. This was to ensure that the effects of practice or fatigue did not systematically alter responses to items that happened to be at the beginning or end of the trial sequence. Half the participants in each diagnostic group received one version, half the other.

In addition, two versions of perceptual task were prepared. These stimuli consisted of \(4 \times 5\) grids with the upper case letters presented in light grey. The perception task differed from the image generation task only in that the upper-case letter was presented in the grid at the same time as the probe.
Four additional trials were designed for practice; these trials included two complex trials, one early, one late, and two simple trials, one early, one late. Half of the trials required a response of “true,” and half required a response of “false”.

Procedure

The experiment consisted of three phases: (1) the acquisition phase; (2) the imagery phase; and (3) the perception phase.

Acquisition phase. First, participants were asked to learn the shapes of the upper-case letters in grids. They were told to pay close attention because they would later be tested on the letters. Each letter was presented three times, individually, in the centre of the screen. The lower-case script version of the letter was also presented with each block letter, directly below the grid. The participants had as much time as they desired to study the letters. After all the letters were studied, the participants were given a sheet containing empty $4 \times 5$ grids and a pencil. Each participant was presented with a lower-case script letter and asked to draw its corresponding block letter in the grid. If the participants made mistakes, they were corrected and asked to redraw the letter after a brief waiting period. If the participants made more than one mistake, they were asked to review the letters again and to redraw them. No participant continued to make a mistake on the same letter more than twice.

Imagery phase. During the imagery phase of the experiment, participants were asked to decide whether an X would fall on the letter if the letter were physically present in the grid. On each trial, the sequence of events was as follows: An exclamation point was presented in the center of the screen, which served as a fixation point. When ready, the participant pressed the space bar and a blank screen appeared for 500 ms. A lower-case script version of a letter then appeared for 500 ms, followed by another blank screen for 500 ms. The stimulus, an empty $4 \times 5$ grid containing an X mark in one of the cells, as well as the lower-case script letter below the grid, was then presented. Participants made their judgements by pressing either a key labelled “T” (the “b” key on the keyboard) if the X would fall on the letter, or by pressing a key labelled “F” (the “n” on the keyboard) if the X would not fall on the letter. Participants were asked to respond as quickly and accurately as possible. The participants’ response times and responses were recorded for each trial.

Perception phase. For all participants, the perception phase always followed the imagery phase. In the perception phase the participants were simply asked to indicate whether an X fell on or off a visible block letter. The
sequence of events was exactly the same as in the imagery phase except that instead of having to generate an image when the lower-case cue appeared, a light grey version of the block letter actually appeared in the grid. Participants made their judgements by using the same keys as in the imagery phase. Again, the response times and responses were recorded for each trial.

Results

We first computed the mean response times per condition per participant. Data from trials on which participants responded incorrectly were not included in the analysis of response times. In addition, we excluded responses that were outliers, defined as response times greater than 2.5 standard deviations from the mean response time for that condition (imagery or perception) for that participant. No participant had more than 1 outlier. The resulting means from the imagery phase of the experiment were submitted to an analysis of variance. Replicating previous studies, participants required more time to evaluate more complex letters (with means of 2340 ms, $SD = 1168$, vs. 1961 ms, $SD = 894$ for complex and simple letters, respectively), $F(1, 30) = 11.87, p < .002$. Moreover, participants required more time for probes placed on or near the last segment (with means of 2256 ms, $SD = 1134$, vs. 2045 ms, $SD = 963$, for late and early probes, respectively), $F(1, 30) = 4.95, p < .05$.

As expected the participants diagnosed with MDD required more time overall than the control participants (with mean response times of 2468 ms, $SD = 827$, vs. 1803 ms, $SD = 868$, respectively), $F(1, 30) = 4.93, p < .05$. However, and crucially for delineating the affected processes, there was no hint of an interaction between the complexity of the stimuli and the performance of the diagnostic groups, $F(1, 30) < 1, p = .99$. Indeed, visual inspection of Figure 1 reveals that complexity increased response times to the same degree in both groups. Moreover, the same was true for probe location, with $F(1, 30) < 1, p = .87$ for the interaction of that variable and group (see Figure 2).

The analysis of error rates revealed that the two groups performed comparably (with means of 5.9%, $SD = 7.0$, and 6.2%, $SD = 6.8$, for the controls and participants with MDD, respectively), with $F(1, 30) < 1, p = .87$. Although all participants were three times more likely to make mistakes for complex letters than simple ones, $F(1, 30) = 9.41, p < .005$, there was no interaction between difficulty and diagnostic group, $F(1, 30) < 1, p = .45$. Finally, error rates did not increase significantly with decreases in response times, belying a speed-accuracy trade-off.

The perception phase of this experiment revealed that the control participants once again were much faster than the MDD participants.
(with means of 937 ms, $SD = 382$, vs. 1276 ms, $SD = 470$), $F(1, 30) = 5.00$, $p < .05$, which, again, is evidence of slowed performance in the MDD group. In addition, the participants required more time to evaluate complex letters (with means of 1133 ms, $SD = 494$, versus 1080 ms, $SD = 453$), $F(1, 30) = 7.35$, $p = .01$. Thus, the effects of complexity we observed in the imagery phase may reflect, at least in part, image inspection, not image generation. However, there was an increase of 53 ms for the complex stimuli here, compared to 379 ms in the imagery phase – and thus not all of the increase in the imagery phase can be ascribed to inspection processes. However, in the perception condition there was no effect of probe location, $F(1, 30) < 1$, $p = .49$, which implies that the effect observed in the imagery condition does indeed reflect image generation per se. In addition, there were no hints of

![Figure 1](image1.png)

**Figure 1.** Interaction plot of stimulus complexity and response time for each diagnostic group, in the image generation experiment.

![Figure 2](image2.png)

**Figure 2.** Interaction plot of probe location and response time for each diagnostic group, in the image generation experiment.
interactions between complexity and diagnosis, $F(1, 30) < 1$, $p = .77$, nor between probe location and diagnosis, $F(1, 30) < 1$, $p = .60$. Similarly, the errors did not increase significantly with decreased response times. Hence, as before, there was no evidence of a speed-accuracy trade-off.

Discussion

Although we found evidence of slowed responses in participants with MDD, there was no evidence that this decrement in performance arose from impaired central processing. Increasing the difficulty of the task, by varying both complexity and probe location, affected the depressed participants and control participants to the same degree; these manipulations did not lead the patients to take a disproportionately longer time to process the task than did the controls. These findings suggest that depression impairs encoding or response processes. The fact that the same pattern was observed in the imagery and perception conditions when we varied letter complexity is further evidence that central processing was not affected by depression. Indeed, even for the probe location, which clearly reflected imagery processing, increasing task difficulty did not selectively impair the depressed participants.

EXPERIMENT 2

Many uses of mental imagery involve transforming an image in some way. Without question, the best understood image transformation is mental rotation (e.g., Shepard & Cooper, 1986). In these tasks, participants typically see pairs of similar shapes and decide whether they are identical or one is a mirror reversal of the other. The key manipulation is the relative orientations of the stimuli; when one is rotated relative to the other, participants typically report “mentally rotating” the shapes into alignment before comparing them. The time to perform such a task is predicted by the amount of rotation necessary, with more time being required for greater rotation. The rotation task used in this study requires participants to evaluate pictures of pairs of human hands, and was an abridged version that used in a positron emission tomography (PET) study by Kosslyn, DiGirolamo, Thompson, and Alpert (1998).

In this experiment, the slope of the response time graphed over increasing angular disparity between the stimuli reflects the mental rotation process itself. The intercept is a measure of the time required to encode the stimuli and to produce a response. As in Experiment 1, by examining the intercept and slope, and comparing these two values between populations, we have a good measure of the performance of the central processes (slope) and
sensory/motor processes (intercept). If, as in Experiment 1, central processing is preserved in depression, the slope of the data from the control and depressed participants should be comparable and no statistical interaction should be observed. Instead, one would expect to see simple main effects of depressive status and of angular disparity. However, if central processing is impaired, the slopes should be significantly different and a statistical interaction should be evident.

Method

Participants

The candidates for this experiment were chosen from the pool of participants described earlier. Most individuals (78%) had participated in Experiment 1. There were 32 participants: 16 controls (9 women, 7 men) and 16 participants diagnosed with current MDD (8 women, 8 men). Once again, the two groups were balanced for age (M = 30.3, SD = 13.8 for the control participants; M = 39.6, SD = 14.7 for the depressed participants, p = 0.41). All participants were right-handed.

Materials

The stimuli were the 2-dimensional line drawings of hands used by Kosslyn et al. (1998). The hand stimuli were created by orienting the figures in 20° increments, from 0° to 180°. The plane of rotation intersected through the middle of the hand (through all the fingers – parallel to a flat hand). Eight sets of hand stimuli were created: a palm-up version and back-up version of four different finger configurations. Images of each of these configurations were produced in all 10 orientations of the rotation. In addition, there was a right-handed and left-handed version of each configuration.

In the rotation condition, pairs of stimuli were presented; the stimulus on the left side was always a left hand, while the stimulus on the right side of the screen varied. This arrangement was designed to avoid interference that could have occurred if the hands appeared in opposite locations (Kosslyn et al., 1998). Also, the left hand on the screen was always upright, whereas the one on the right could appear at any angle in the stimulus set. Half of the items presented on the right side of the screen were left hands, and the other half were right hands.

The stimuli were pseudo-randomly ordered so that no more than three of the same type of stimulus (i.e., left or right hands) on the right side were presented in a row. In addition, the same angle of rotation could not appear twice before all other angles had been presented, and could not appear three times until all others had occurred twice, and so on. Each of the four hand configurations appeared once before being repeated, and each appeared
twice before any other appeared three times. Our experiment included 80 trials, and we created two versions of this experiment, where the order of the stimuli of one version was the reverse of the other one. This was done in order to remove effects of fatigue or practice, which could have differentially affected different types of stimuli. As before, half of the participants in each group received one version, half the other.

A practice session was also created to familiarise the participants with the stimuli and procedure. This set included 9 trials, familiarising the participant with an example of all non-upright orientations.

**Procedure**

Participants were first presented with the instructions for the task and asked to paraphrase them to show that they understood. Only after the participant understood the instructions did the practice trials start. Each trial began with a fixation point (an exclamation mark), which appeared for 500 ms; after this, one of the stimulus pairs appeared. The participants were told to decide whether both hands were left hands or one was a left hand and one was right hand. They were asked respond as quickly and accurately as possible by pressing a key – “S” when the hands were the same (the number “2” on the keyboard) and “D” when the hands were different (the number “0”). Immediately after their response, a fixation point appeared and the sequence started over.

**Results**

To analyse these data, we plotted the response time for each trial against its corresponding angle of rotation. For trials in which the participant responded in error, the response time was deleted. In addition, if a response time to a particular stimulus was more than 2.5 standard deviations from the mean response time, it was not included in the analysis. No participant had more than 2 such outliers. For each individual participant we computed the slope and intercept. The intercept gave us an estimate of the amount of time necessary for sensory/motor processes, and the slope gave us an estimate of the rotation time per se, which relies on central processing.

Again supporting the delayed response time associated with depression, the intercepts of the two diagnostic populations were different (2541 ms, $SD = 1048$ vs. 1790 ms, $SD = 842$, for the depressed and control groups, respectively), $F(1, 30) = 4.98$, $p < .05$. In contrast, as shown in Figure 3, the slopes from the two groups were not different (6.3 ms/degree, $SD = 3.8$, vs. 4.7 ms/degree, $SD = 4.7$, for the depressed and control groups, respectively), $F(1, 30) = 1$, $p = .33$. The aggregate data replicated previous experiments, in
that the more rotation required, the more time it took participants to respond.

The error rates for the two groups were comparable (mean for the depressed, 11.9%, $SD = 11.35$; mean for controls, 6.7%, $SD = 5.98$), $F(1, 30) = 2.66$, $p = .11$. Moreover, error rates did not increase when response times decreased, and hence a speed-accuracy trade-off cannot account for our results.

**Discussion**

Similar to Experiment 1, the hand rotation task replicated previously published studies by showing that the hands that needed more mental rotation required more time to evaluate. The slopes and intercepts were comparable to those from previous studies (Kosslyn et al., 1998), further bolstering the validity of these results.

The findings support the hypothesis that people with MDD perform central processing normally in the image rotation task, and their overall slowed responses are a result of impaired sensory or motor processing. This experiment clearly shows the dissociation between the intercept and slope; the former is affected by MDD, but the latter is not. These results are consistent with those from Experiment 1.
EXPERIMENT 3

The third task we administered also involves top-down processing, but not mental imagery. In this task the participants were asked whether a spoken name is appropriate for a pictured object. In one condition, the objects were pictured from familiar, “canonical” viewpoints, whereas in another condition the objects were pictured from unusual, “noncanonical” viewpoints. Previous studies have suggested that top-down processing is used to identify the object in the noncanonical condition (Kosslyn et al., 1994). Thus, using the logic of the previous two experiments, by comparing the canonical vs. noncanonical manipulation we can assess central processing.

Method

Participants

The candidates for this experiment were drawn from the same pool described earlier. Of the participants in this task, 69% also took part in Experiment 1, and 62% of the participants also took part in Experiment 2. There were 30 participants: 15 controls (10 women, and 5 men) and 15 participants with MDD (10 women and 5 men). The two groups were balanced for age ($M_{C}=30$ years, $SD_{C}=11.7$, and $M_{D}=38.7$, $SD_{D}=13.6$ for the depressed participants). All participants were right-handed.

Materials

Four versions of 27 pictures of common objects were created. Two versions of each object were a canonical depiction; the other two were noncanonical depictions. The stimuli were presented in the centre of the screen. The words corresponding to each object were recorded on the Macintosh computer using SoundEdit 1.0. The words were the “entry-level” name of the picture (Jolicoeur, Gluck, & Kosslyn, 1984) or names of similarly shaped objects (with two distractor names per object). An additional set of stimuli was created for practice trials; four objects were chosen for this part of the experiment.

The 27 objects were split into three groups of nine. (For a more detailed list of objects and the experimental organisation see Kosslyn et al., 1994.) All objects were used to create canonical and noncanonical conditions for the experiment. All participants received both the canonical and noncanonical conditions; half in each group received the canonical followed by the noncanonical, and vice versa for the other half.

Within each version of the experiment, each object appeared twice, once as a “yes” trial (where the word did in fact name the object in the picture),
and once as a “no” trial (where the word did not name the object). The stimuli were pseudo-randomly ordered to ensure that no three consecutive responses were the same and that the same object was not presented in three consecutive images. There were 36 trials in each condition; thus each participant received a total of 72 trials, half of which were canonical and the other half noncanonical.

Procedure

Participants were first presented with the instructions for the experiment and asked to paraphrase them to show they understood. Only after the participants understood the instructions did the practice trials start. Each trial began with the computer’s showing a line drawing of an object and, immediately after the presentation, playing a prerecorded word (i.e., either the name of the object or a distractor). The participants were asked to decide whether the word correctly named the picture on the screen; they were to respond by pressing a key labelled “S” when the stimuli were the same (the number “2” on the keyboard) or a key labelled “D” if the word and picture were different (the number “zero”). Participants were asked to respond as quickly and accurately as possible: 500 ms after their response, a new trial began.

Results

Only the response times from trials on which a correct judgement was made were included in the analysis of response times. In addition, if a response time to a particular stimulus in a given condition was more than 2.5 standard deviations from the mean response time, it was not included in the analysis. No participant had more than 1 outlier. The participants’ means were then considered in an analysis of variance. The results replicated those of previous studies in that the participants required more time for the noncanonical than the canonical condition (with means of 1195 ms, $SD = 576$, vs. 829, $SD = 302$, respectively), $F(1, 28) = 26.37$, $p = .0001$. In general, depressed participants were slower than control participants (with means of 1169 ms, $SD = 593$, vs. 855 ms, $SD = 299$, respectively, $F(1, 28) = 4.85$ and $p < .05$). Critically, for present purposes, there was no interaction between condition and diagnosis, $F(1, 28) = 1.05$, $p = .31$. These findings are illustrated in Figure 4.

In addition, the MDD participants tended to make more errors than the controls, with means of 10.1% ($SD = 4.47$) and 7.5% ($SD = 6.07$) respectively, $F(1, 28) = 3.62$, $p = .07$. Furthermore, participants made more errors in the noncanonical condition (with means of 14.6%, $SD = 6.92$, vs. 3.0%, $SD = 3.42$, in the canonical condition), $F(1, 28) = 74.13$, $p < .0001$. As in the
response time data, there was no evidence of an interaction between condition and diagnosis for error rates, \( F(1, 28) = 2.70, p = .11 \). However, in general, errors did not increase when response times decreased, and thus the response time findings cannot be ascribed to a speed-accuracy trade-off.

Discussion

This experiment replicated previous studies that used this task, finding that noncanonical views of objects require more processing than canonical views. We saw this not only in the response time data, but also in the error rates. The response times measured in this study were comparable to those found in past studies (Kosslyn et al., 1994). The results were also consistent with those from Experiments 1 and 2. The slowed responses by the MDD group do not appear to reflect impaired central processing, but rather arise from sensory/motor processes.

GENERAL DISCUSSION

In all three experiments, participants diagnosed with MDD generally required more time to respond than did the control participants, but increased task difficulty had similar effects in the two populations. These results were remarkably consistent over the three experiments, and provide evidence that impaired central processing does not underlie the observed slowed performance in MDD during imagery tasks; rather, this impairment seems to reflect encoding and/or response processes. The lack of an
interaction between increased difficulty and the population implies that central processing per se is not impaired in MDD.

The inference that central processing is preserved in participants with MDD could have important implications in the general study of cognition in major depression. In tasks in which central processing is not easily distinguished from sensory/motor processing, the sensory/motor deficit could mistakenly be attributed to a cognitive impairment. In fact, deficits seen in many cognitive tasks in depressed populations could be a result of simply a deficit in either encoding or response output. That is, a dysfunction in one component of processing can manifest itself as a global cognitive deficit. The present experiments suggest that many cognitive abilities are still preserved in participants with depression, specifically, in this case, image generation, image rotation, and identifying objects seen from different points of view.

The results of these experiments could also help explain why there is a growing number of studies of cognitive processing in patients with MDD that report inconsistent patterns of results. For example, Austin et al. (1992) and Ravnkilde, Videbech, Rosenberg, Gjedde, and Gade (2002), among others, found deficits in episodic memory and learning, whereas a number of studies, including Grant, Thase, and Sweeney (2001), and Fossati, Amar, Raoux, Ergis, and Allilaira (1999), have not observed such deficits. Such inconsistencies have also been found in studies of working memory (e.g., Landro, Stiles, & Sletvold, 2001, found deficits, but Grant et al., 2001 did not), and executive functioning (e.g., Grant et al., 2001; Martin, Oren, & Boone, 1991, found deficits, but Matsuo, Kato, & Kato, 2000, did not). Without being able to distinguish among the underlying processes used in the tasks, it remains unclear how much of the observed deficits in the performance of those with MDD can be attributed to sensory/motor processing vs. central processing itself. Hence, in order better to understand cognitive functioning in depression, it may be necessary to assess function by varying complexity and exploiting the logic of additive factors methodology.

The findings reported here also have implications for a wider set of theoretical issues about depression. First, there is a debate in the literature regarding activity in the left dorsolateral prefrontal cortex (DLPFC) in major depression or in the depressed state. Early positron emission tomography (PET) and electroencephalography (EEG) studies demonstrated hypoactivation of the frontal areas in baseline conditions, some specifically in the DLPFC, in participants with MDD or in a melancholic state (for PET, Baxter et al., 1989; Bench, Frackowiak, & Dolan, 1995; Bench, Friston, Brown, Frackowiak, & Dolan, 1993; Bench, Friston, Brown, Scott et al., 1992; Dolan et al., 1992; Martinot et al., 1990; for EEG, Henriques & Davidson, 1991; Tomarken & Davidson, 1994). More recently, however, EEG and functional magnetic resonance imaging (fMRI) studies have
demonstrated hyperactivation in left DLPFC, or other left frontal areas (for fMRI, Beauregard et al., 1998; for EEG, Drevets et al., 1992; George et al., 1995) or no differences between groups (Reid, Duke, & Allen, 1998). Because the cognitive tasks we employed recruit these regions, these tasks can perhaps elucidate the variability in this data. One possible explanation for the discrepancies in these results is heterogeneity of symptoms in MDD. Specifically, comorbid anxiety possibly could increase activation of the DLPFC and mask the effects of depression on the DLPFC (Eysenck & Calvo, 1992; Heller & Nitschke, 1997, 1998; Keller et al., 2000).

Second, the present findings bear on the treatment of depression, if visual mental imagery plays a major role in MDD. Individuals diagnosed with depression sometimes report negative images (Beck & Weishaar, 1989; Martin & Williams, 1990). For example, they sometimes report that stimuli evoke disturbing images more frequently than they did prior to the depressive episode (Beck et al., 1979). In addition, some forms of treatment for depression rely on the ability to form mental images (e.g., cognitive behavioural therapy). For example, “induced images” are used to demonstrate to depressed clients the relationship between their thinking and their feelings (Beck & Weishaar, 1989) and may serve as a diversion to automatic negative thoughts (Beck et al., 1979). The present results suggest that imagery and operations on imagery are not affected much, if at all, in MDD; thus, imagery-based treatment techniques appear sensible for this population.

In summary, in this study we found evidence that impaired central processing does not contribute to slowed responses in MDD. Rather, impaired performance is related to motor or sensory processes, or both.

REFERENCES


