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Effect of Antidepressant Medication Use on Emotional Information Processing in Major Depression

Tony T. Wells, Ph.D., Elise M. Clerkin, Ph.D., Alissa J. Ellis, Ph.D., and Christopher G. Beevers, Ph.D.

Department of Psychology, Oklahoma State University, Stillwater; the Department of Psychology, Miami University, Oxford, Ohio; the Semel Institute of Neuroscience and Human Behavior, University of California-Los Angeles; and the Department of Psychology, University of Texas at Austin

Abstract

Objective—Acute administration of antidepressant medication increases emotional information processing for positive information in both depressed and healthy participants. This effect is likely relevant to the therapeutic actions of these medications, but has not been studied in patients with Major Depressive Disorder (MDD) taking antidepressants as typically prescribed in the community.

Method—The authors examined the effects of antidepressant medication on selective attention for emotional stimuli using eye tracking in a sample of 47 participants (21 medicated; 26 non-medicated) with MDD and 47 matched, non-depressed controls. Participants completed a passive viewing eye tracking task assessing selective attention for positive, dysphoric, threatening, and neutral stimuli in addition to providing medication information and self-report measures of depression and anxiety severity. Results: Depressed participants currently taking antidepressant medication and non-depressed healthy control participants demonstrated greater total gaze duration and more fixations for positive stimuli, compared to non-medicated depressed participants. Depressed participants on medication (vs. depressed participants not on medication) also had fewer fixations for dysphoric stimuli.

Conclusions—Antidepressants, as prescribed in the community to depressed patients, appear to modify emotional information processing in the absence of differences in depression severity. These results are consistent with prior work and indicate a robust effect for antidepressants on positive information processing. They also provide further evidence for modification of information processing as a potential mechanism of action for antidepressant medication.

Introduction

There is now substantial theoretical and empirical support for the importance of information processing biases in the maintenance, and perhaps etiology, of major depression.^{1–3} Specifically, biased attention to depression-relevant material and avoidance of positive information are hypothesized to maintain the disorder. Recent research has also demonstrated that modifying these biases reduces symptoms of depression.^{4–6}

According to a recent cognitive neuropsychological model of depression,⁷ antidepressant medications—particularly those that target serotonin and norepinephrine—may exert their

Corresponding Author: Tony T. Wells, Department of Psychology, Oklahoma State University, 116 N. Murray, Stillwater, OK 74048, USA, tony.wells@okstate.edu.

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mechanism of action by modifying emotional information processing. In turn, modified emotional information processing is thought to lead to downstream antidepressant effects. This model helps to explain why antidepressant medication use is not immediately associated with amelioration of depressed mood. As Harmer and colleagues note: “Rather than acting as direct ‘mood enhancers,’ antidepressants may re-tune how we process personal and socially relevant affective information” (p. 107).⁷

This model follows from a series of studies suggesting that a) antidepressants influence emotional information processing early in treatment; b) changes in emotional information processing occur earlier than and in the absence of changes in subjective mood; and c) early changes in information processing are associated with eventual therapeutic improvement (see review by Harmer and colleagues).⁷ Thus far, much of this research has been conducted among healthy control participants. For example, Browning and colleagues randomly assigned 32 healthy volunteers to receive either one dose of citalopram or a placebo pill.⁸ Consistent with the cognitive neuropsychological hypothesis, individuals who received the antidepressant medication demonstrated increased attention to positive stimuli, as assessed with a visual probe task.

More recently, researchers have begun to evaluate this phenomenon among individuals with depression. For instance, Harmer and colleagues conducted a double-blind, placebo-controlled study evaluating patients with depression and healthy control participants.⁹ In line with the cognitive neuropsychological model, depressed patients who received a placebo displayed reduced recognition of positive facial expressions and memory for positive information, as well as slowed speed to respond to positive personality adjectives when compared to healthy controls. Importantly, these information processing effects in the depressed patients were reversed with the administration of just a single dose of an antidepressant (reboxetine). However, there were not corresponding reductions in subjective ratings of mood or anxiety after this initial administration.

The current study builds upon prior research to focus specifically on the relationship between antidepressant medication use and selective attention to emotional visual stimuli using eye tracking technology, among a sample of participants with major depressive disorder (MDD), as well as a non-depressed control group. This research adds to the small number of studies that have empirically examined the cognitive neuropsychological model of depression in a clinical sample. The inclusion of a non-depressed control group is also valuable for comparative purposes insofar as it allows us to more clearly delineate “normal” attention for emotional information.

Our use of an eye tracking paradigm is particularly valuable because it allows for multiple, dynamic measures of selective attention.¹⁰ This is critical because it enables us to capture the more elaborative stages of attention that are particularly relevant for patients with MDD.¹¹ Eye tracking also specifically provides an assessment of overt attention, since eye movements are necessarily associated with shifts in attention; whereas, the dot probe task used in prior studies does not always elicit eye movements and may measure both overt and covert shifts in attention.^{12,13} Ours is also the first to examine the effects of antidepressant medication, as prescribed in the community, on emotional information processing.

In this study, we used eye tracking to measure selective attention (total gaze duration, mean number of fixations, mean fixation duration) for dysphoric, threatening, positive, and neutral emotional scenes in a sample of community participants with MDD (both medicated and unmedicated) and a never depressed control group. Consistent with prior work,^{8,9} we hypothesized that antidepressant medication use (vs. non-use) would be associated with increased selective attention for positive stimuli. Further, we predicted that there would not

be significant group differences between the MDD-medicated group and Control group on selective attention for positive information, consistent with the idea that antidepressant medication use normalizes emotional information processing.

Method

Participants

We recruited 49 depressed and 47 never depressed participants using internet, television, and radio advertisements. Two participants were dropped from analyses due to inadequate eye tracking data resulting in a total sample of 94 participants including 21 depressed and currently taking antidepressant medication (MDD-medicated), 26 depressed participants not currently taking antidepressant medication (MDD-unmedicated), and 47 participants with no history of depression and not taking antidepressant medication (Control). Control participants were matched as closely as possible with MDD participants for age and years of education.

Inclusion criteria for depressed participants were 1) a DSM-IV diagnosis of major depressive disorder (MDD) and 2) Beck Depression Inventory II (BDI-II) score greater than 20. Inclusion criteria for Control participants were 1) no history of MDD and 2) BDI-II score less than 13. Inclusion criteria for all participants were: 3) aged between 22 and 55 years; 4) normal or corrected to normal vision; and 5) ability to speak, read, and understand English. Exclusion criteria for all participants were: 1) current or past DSM-IV diagnosis of substance or alcohol abuse in past 6 months; 2) current or past DSM-IV diagnosis of substance or alcohol dependence, Bipolar Disorder, Psychotic Disorder, Obsessive-Compulsive Disorder, Social Phobia, Panic Disorder, PTSD, and Generalized Anxiety Disorder; 3) a history of epilepsy or head trauma. After complete description of the study to participants, written informed consent was obtained. All procedures were approved by the institutional review board of the University of Texas at Austin.

Assessments

Mini International Neuropsychiatric Interview (MINI).¹⁴—The electronic version of the MINI was used as a screening interview to determine provisional study eligibility. The MINI is a short, structured screening interview that provides *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition and International Classification of Diseases, 10th edition (ICD-10) psychiatric disorder diagnoses.

Structured Clinical Interview for DSM-IV (SCID).¹⁵—The patient version of the SCID was administered on the day of study participation to provide psychiatric diagnoses for inclusion/exclusion criteria. Two assessors were doctoral graduate students with masters degrees in clinical psychology and at least two years of clinical training and assessment experience. The third assessor was a full time research assistant with a bachelor's degree in psychology who completed more than 40 hours of training in the administration of the SCID. Twenty percent of all interviews were rated by an independent assessor who was a doctoral student with a masters degree in clinical psychology and four years of assessment experience. Agreement for MDD diagnosis between study interviewers and the independent assessor was excellent ($k = 1.00$, $p < 0.0001$).

Beck Depression Inventory-II (BDI-II).¹⁶—The BDI-II is a widely used, 21-item self-report questionnaire that assesses depression severity. The BDI-II has shown validity among psychiatric outpatient and inpatient samples.¹⁷

Beck Anxiety Inventory (BAI).¹⁸—The BAI is a widely used, 21-item self-report questionnaire that assesses symptoms of anxiety. The BAI demonstrates good internal and test-retest reliability and convergent validity with other measures of anxiety.^{18,19}

Eye Tracking Task

On each trial in this task, four images selected from the International Affective Picture System (IAPS)²⁰ were presented simultaneously with one image appearing in each quadrant (upper left, lower left, etc.) of a 20 in LCD computer monitor. On every trial, one image was selected from each of the following stimulus categories: dysphoric, threat, positive, or neutral (see Supplementary Figure 1). The method for selecting and categorizing these images has been described previously.¹¹ The location of each image was randomly assigned for each participant by the stimulus presentation software (E-Prime 2.0) with the constraint that each stimulus category must appear in each of the four positions three times across 12 trials. In addition to the 12 study trials, 4 filler trials comprised completely of neutral images were presented to obscure the nature of the task, resulting in a total of 16 trials. Presentation order of stimuli was randomized for each participant. Each trial lasted 30 seconds. Trials were preceded by a centrally-presented fixation cross which remained on screen until the participant fixated within approximately 1° of visual angle of the cross for 1 second.

Participants sat approximately 60 cm from the screen. Each image measured 14.2 × 10.7cm (13.5° × 10.2° visual angle). The horizontal distance between the centers of images was 20.7 cm (19.6° visual angle) and the vertical distance between the centers of images was 15.5 cm (14.8° visual angle).

Eye Tracking System

Line of visual gaze was assessed using a remote optics eye tracking system model R6 from Applied Science Laboratories (ASL; Bedford, MA). Head location was fixed using a chin rest and forehead bar. The location of gaze was sampled every 16.7 ms (60 Hz). Eye movements that were stable for more than 100 ms within a 1° of visual angle were classified as a fixation. The total area of each stimulus on a trial was identified as an area of interest (AOI). For each AOI, the following selective attention indices were calculated with GazeTracker software (ASL, Bedford, MA): total gaze time per trial, number of fixations per trial, and mean fixation duration. Greater gaze time represents increased sustained attention. Greater number of fixations represents repeated attentional engagement whereas greater fixation duration represents increased attentional capture or difficulty disengaging attention.

Procedure

After providing informed consent, participants were administered the SCID to determine study eligibility. Eligible participants completed a demographic form, BDI-II, BAI, and provided the following information regarding antidepressant use: current use (yes/no), name of medication, medication dose, length of time on medication. For eye tracking, participants were seated in a height-adjustable chair, which was adjusted to minimize discomfort while participant head location was fixed with the chinrest and forehead bar. Camera adjustments were made by the experimenter to best capture pupil and corneal reflection of the participant's right eye. A 9-point calibration was completed to confirm recording line of visual gaze within 1° of visual angle for each calibration point. Calibration was repeated until this criterion was met.

Following successful calibration, participants were instructed verbally and via computer screen to view the images naturally, as if watching television or viewing a photo album. The only constraint was that they view the images at all times during each trial. To minimize

demand effects, instructions emphasized that the study measured processing of emotional information without specifically mentioning the measurement of eye movements. Participants were instructed to look at the fixation cross preceding each trial to standardize the starting location of their gaze. An experimenter located in an adjacent room monitored the stimulus presentation and eye tracking quality throughout the task.

Results

Participant Characteristics

Descriptive statistics for the sample are presented in Table 1. There were no significant differences between groups (Control, MDD-medicated, MDD-unmedicated) in age or years of education. The MDD-medicated and MDD-unmedicated groups did not differ in depression severity, anxiety severity, number of depressive episodes, or length of current depressive episode. Both the MDD-medicated and MDD-unmedicated groups reported greater depression and anxiety severity than the Control group. The majority of participants were women and there were no differences between Control (n women = 38), MDD-medicated (n women = 17), and MDD-unmedicated (n women = 22) groups in proportion of women, $\chi^2(2, N = 94) = 0.18, p = .92$. There was substantial variability in length of time MDD-medicated participants had been on their primary antidepressant medication (range: 1.5 – 520 weeks). Medication information provided by MDD-medicated participants can be seen in Supplemental Table 1. A majority of MDD-medicated participants ($n = 16$) reported an SSRI or SNRI as their primary medication. Three participants reported bupropion as their primary medication and two participants endorsed currently taking an antidepressant medication but did not provide any information about their medication. Analyses utilizing data only from participants reporting an SSRI or SNRI as their primary medication were nearly identical to results utilizing the full sample, thus results from the full sample are reported below.

Effects of medication status on eye tracking indices

Total mean gaze duration—A 3 (group: Control, MDD-medicated, MDD-unmedicated) by 4 (stimulus type: dysphoric, threat, positive, neutral) ANOVA revealed a significant main effect for stimulus type on mean gaze time, $F(3, 376) = 18.22, p < .001, \eta_p^2 = .13$, as well as a significant interaction between group and stimulus type, $F(6, 376) = 3.24, p = .004, \eta_p^2 = .05$. There was no main effect for group on mean gaze time, $F(2, 376) < 1, p = .78$. These results can be seen in Figure 1. As predicted, planned comparisons revealed that the group by stimulus type interaction was driven by longer total gaze duration for positive images in the MDD-medicated group and in the Control group compared to the MDD-unmedicated group. There was no difference between the Control and MDD-medicated groups in total gaze duration for positive images. Group differences for dysphoric, threat, and neutral images were non-significant. These results can be seen in Table 2.

To explore potential effects of time course on total gaze duration, the 30 s trial was divided into six 5-second epochs and total gaze time was calculated for each epoch. A 3 (group: Control, MDD-medicated, MDD-unmedicated) by 4 (stimulus type: dysphoric, threat, positive, neutral) by 6 (time) repeated measures ANOVA revealed a significant main effect for time on total gaze duration, $F(5, 360) = 17.31, p < .001, \eta_p^2 = .19$, and a significant interaction between stimulus type and time, $F(15, 1086) = 2.40, p = .002, \eta_p^2 = .03$. The stimulus type by time interaction can be seen in Figure 2. The group by stimulus type interaction was identical to the group by stimulus type interaction reported above for the full 30 s trial. The time by group interaction and the 3 way interaction on total gaze time were not significant, both $p > .30$.

Despite the non-significant interactions between time and group, due to our significant results of group on total gaze time for positive stimuli, we conducted exploratory post hoc tests examining time course effects of group on total gaze time for positive stimuli. There were significant differences between groups for total gaze time for positive stimuli at the 5th (seconds 21–25), $F(2, 93) = 7.25, p = .001$, and 6th (seconds 26–30), $F(2, 93) = 3.53, p = .034$, epochs and approaching significance at the 4th epoch, $F(2, 93) = 2.39, p = .097$. These results were driven by longer gaze times for positive stimuli by the Control and MDD-medicated groups compared to the MDD-unmedicated group. These results can be seen in Supplementary Figure 2. There were no significant differences on total gaze time for positive stimuli between groups for the 1st, 2nd, or 3rd epochs, all $p > .15$.

Mean number of fixations—Due to a programming error, mean number of fixations and mean fixation duration (see below) could not be calculated for 8 of the MDD-medicated participants. A 3 (group: Control, MDD-medicated, MDD-unmedicated) by 4 (stimulus type: dysphoric, threat, positive, neutral) ANOVA revealed a pattern of results similar to the mean gaze time findings. There was a significant main effect for stimulus type on number of fixations, $F(3, 343) = 21.27, p < .001, \eta_p^2 = .16$, as well as a significant interaction between group and stimulus type, $F(6, 343) = 4.23, p < .001, \eta_p^2 = .07$. There was no main effect for group on number of fixations, $F(2, 343) < 1, p = .93$. These results can be seen in Figure 3.

Similar to the total gaze duration results and consistent with hypotheses, planned contrasts for number of fixations revealed that the group by stimulus type interaction was driven by a greater mean number of fixations on positive images in the MDD-medicated group and in the Control group compared to the MDD-unmedicated group. There was no difference between the Control and MDD-medicated groups in number of fixations for positive images. The MDD-medicated group also made fewer fixations on dysphoric images compared to the MDD-unmedicated group. There were no significant differences between Control and MDD-unmedicated groups or between Control and MDD-medicated groups on number of fixations for dysphoric images. Again, differences for threat and neutral images were non-significant. These results can be seen in Table 2.

Mean fixation duration—A 3 (group: Control, MDD-medicated, MDD-unmedicated) by 4 (stimulus type: dysphoric, threat, positive, neutral) ANOVA revealed no significant main effects for group, $F(2, 343) = 1.82, p = .16$, or stimulus type, $F(3, 343) = 1.72, p = .16$, and no significant interaction between group and stimulus type, $F(6, 343) = 1.37, p = .23$, on mean fixation duration. Due to the lack of significant main effects or interaction, post hoc analyses were not performed for mean fixation duration.

Discussion

The current study tested the cognitive neuropsychological model of depression by evaluating the association between antidepressant medication use and selective attention for emotional information among individuals with Major Depressive Disorder (MDD), as well as a non-depressed control group who was not taking antidepressant medications. As hypothesized, among depressed participants, we found an association between antidepressant medication use (vs. non-use) and greater selective attention for positive stimuli. Specifically, individuals in the MDD-medicated group had longer total gaze duration and more fixations for positive images compared to the MDD-unmedicated group. The control group also demonstrated longer total gaze duration compared to the MDD-unmedicated group. Importantly, the MDD-medicated group did not differ from the control group in selective attention for positive stimuli, suggesting that antidepressant medication use normalizes information processing. We did not find differences between our groups in gaze duration for dysphoric

stimuli, but we did find that the MDD-medicated group had fewer fixations for dysphoric stimuli compared to the MDD-unmedicated depressed group.

Our results are consistent with prior research demonstrating an association between antidepressant medication use and changes in information processing for positive emotional stimuli.⁷⁻⁹ Previous studies have investigated the effects of a single dose of antidepressant medication vs. placebo on information processing whereas the present study is the first to examine the effects of antidepressant medication use as prescribed in the community on information processing in participants with major depression. The association between antidepressant use and emotional information processing for positive stimuli has now been observed across healthy and depressed samples and across several different information processing tasks. The effect has been observed after a single administration of an antidepressant in prior studies and in our study with depressed individuals on a consistent regimen of antidepressant medication. The fact that the effect of antidepressant medication use on emotional information processing for positive stimuli has now been observed across samples, tasks, and methods of medication administration, in combination with the large effect sizes observed in the present study, suggests that this effect is robust.

These findings add to the growing evidence that antidepressant medications exert their antidepressant effects through modification of emotional information processing rather than direct elevation of mood.⁷ Prior research also suggests that gaze bias plays a critical role in vulnerability to depression.² Given the fact that antidepressant medications appear to influence gaze bias, and gaze bias appears to confer vulnerability to depression, it will be necessary for future work to clarify the extent to which changes in gaze bias mediate the relationship between antidepressant use and subsequent reductions in depressed mood.

The present study must be interpreted in light of some limitations. Key limitations to this project are 1) the exclusion of anxiety disorders from our sample of depressed patients, which limits the external generalizability of study findings; and 2) the non-random assignment to medication condition, and the inclusion of multiple classes of antidepressant medications, which limit the internal validity of study findings. While there are clear limitations to these design choices, our more naturalistic approach does enhance the ecological validity of the present study. Indeed, participants in the present study were prescribed a variety of antidepressants (as opposed to being given a single and/or identical dose of study medication), which better matches the reality of antidepressant medication use in the general population. Moreover, eye tracking technology is a more ecologically valid tool than the indicators of selective attention that have been utilized in the past (e.g., probe detection) insofar as it provides critical information about dynamic stages of attention. Along with prior experimental research,^{8,9} the results of the present study provide further evidence for modification of information processing as a potential mechanism of action for antidepressant medication.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Mean total gaze duration by 5 s epoch and stimulus type.

Table 1

Demographic and clinical characteristics of the sample.

	Control (<i>n</i> = 47)		MDD-medicated (<i>n</i> = 21)		MDD-unmedicated (<i>n</i> = 26)	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Age (years)	33.6 ^a	11.2	37.2 ^a	12.8	31.3 ^a	8.7
Education (years)	14.3 ^a	1.3	14.3 ^a	1.3	14.0 ^a	1.7
BDI-II	2.0 ^a	3.2	27.6 ^b	6.6	28.9 ^b	9.7
BAI	3.2 ^a	5.5	11.0 ^b	5.2	14.2 ^b	6.5
Depressive episodes	--	--	6.5 ^a	8.5	6.2 ^a	4.9
Length of current depressive episode (weeks)	--	--	29.1 ^a	37.4	25.0 ^a	24.1
Time on current medication (weeks)	--	--	139.8	141.2	--	--

Note: Means with different superscripts are significantly different at $p < .05$ between groups.

Table 2

Eye tracking results.

	Control (<i>n</i> = 47)	MDD-medicated (<i>n</i> = 21)[†]	MDD-unmedicated (<i>n</i> = 26)
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)
Total mean gaze duration (seconds)			
Neutral	5.05 ^a (1.46)	4.77 ^a (1.54)	5.03 ^a (1.85)
Dysphoric	5.99 ^a (1.47)	5.84 ^a (1.95)	6.60 ^a (1.74)
Positive	7.46 ^a (2.63)	7.84 ^a (2.64)	5.79 ^b (2.04)
Threat	5.33 ^a (1.60)	5.17 ^a (2.28)	5.72 ^a (1.93)
Mean number of fixations			
Neutral	20.97 ^a (4.58)	20.03 ^a (4.36)	21.12 ^a (6.30)
Dysphoric	24.55 ^{ab} (4.43)	22.47 ^a (5.39)	26.30 ^b (5.62)
Positive	27.49 ^a (5.91)	31.68 ^a (10.14)	23.64 ^b (6.79)
Threat	21.48 ^a (4.99)	19.32 ^a (5.22)	22.53 ^a (6.35)

Note: Means with different superscripts are significantly different at $p < .05$ between groups.

[†] MDD-medicated $n = 13$ for mean number of fixations analyses.