Tsourtos, George; Thompson, J. C.; Stough, Con K.
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Psychological Medicine, 2002; 32 (2):259-265.

Psychological Medicine, 2002, **32**, 259–265.
DOI: 10.1017/S0033291701005001 Printed in the United Kingdom

Evidence of an early information processing speed deficit in unipolar major depression

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ABSTRACT

Background. Slowing of the speed of information processing has been reported in geriatric depression, but it is not clear if the impairment is present in younger patients, if motor retardation is responsible, or if antidepressant medications play a role.

Method. Twenty unmedicated unipolar depressed inpatients were compared with 19 medicated depressed in-patients and 20 age-, sex- and verbal IQ-matched controls on inspection time (IT), a measure of speed of information processing that does not require a speeded motor response. We also examined the relationship between IT and current mood and length of depressive illness.

Results. Unmedicated depressed patients showed slowing of information processing speed when compared to both medicated depressed patients and controls. The latter two groups were not significantly different from each other. Slowing of IT was not associated with current mood, but was negatively correlated with length of illness since first episode. No differences in IT were found between patients receiving medication with anticholinergic effects and patients receiving medication with no anticholinergic effects.

Conclusions. The findings indicate that unipolar depression is associated with a slowing of speed of information processing in younger patients who have not received antidepressant medication. This does not appear to be a result of motor slowing.

INTRODUCTION

Patients suffering depression often report the subjective experience of a slowing in mental speed (O'Connor *et al*. 1990). Cognitive slowing may contribute to neuropsychological impairment associated with unipolar major depression (MD), as well as depression secondary to other illnesses (Brebion *et al*. 2000; Fann *et al*. 2001). For example, Brown *et al*. (1994) reported that elderly ($\geqslant 65$ years old) depressed patients showed slower performance on a range of neuropsychological tests than age-matched controls. Nebes *et al*. (2000) reported that slowing of information speed, as well as working memory impairments, medicated neuropsychological impairment in patients with geriatric depression. Both motor and cognitive speed appear to be impaired in depression (Sobin & Sackheim, 1997; Calgiuri & Ellwanger, 2000), although Elliott *et al*. (1996) found that middle-aged (mean age 49 years) depressed patients were impaired on a measure of cognitive speed but not motor speed.

In contrast to these findings, Purcell *et al*. (1997) reported that younger patients (mean age 37 years) were impaired on measures of attentional set-shifting and planning, but not cognitive speed. It was concluded by Purcell *et al*. (1997) that younger patients with depression do not show the cognitive slowing that is reported in middle-aged and older patients. A problem with this interpretation is that the cognitive speed measure used by both Elliott *et al*. (1996)

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and Purcell *et al.* (1997) was time to respond ('thinking time') during a planning task (the Cambridge Neuropsychological Test Automated Battery (CANTAB) Tower of London). As this measure involves a number of cognitive operations including processing speed, it is unclear if one can conclude from Purcell *et al.*'s (1997) study that younger depressed patients do not show cognitive slowing. Tarbuck & Paykel (1995) reported that older depressed patients (mean age 69 years) were slower than younger depressed patients (mean age 41 years) on a choice reaction time (RT) measure. However, RT improved to a similar extent in both groups following recovery, indicating that both age and depression may affect information processing speed, but these variables do not interact to produce cognitive slowing in older depressed but not younger depressed. The aim of the present study was therefore to examine if speed of information processing was slowed in young, unipolar depressed individuals.

Most measures of information processing speed rely on reaction item (RT) as the dependent variable. Measures of RT can often be confounded by changes to motor speed. While many methodologies allow for the separation of movement time (MT) and decision time (DT) from RT, DT still measures the speed of both the perception and encoding of a stimulus, and the initiation of a motor action. The DT/MT paradigm is also constrained by subjects being able to adopt varying speed-accuracy trade-off strategies, as accuracy can be increased at the expense of response time. Unlike RT procedures the inspection time (IT) procedure is widely regarded as a measure of the speed of early stages of information processing that is not sensitive to motor speed, speed-accuracy trade-offs or other cognitive strategies (Nettelbeck, 1987; Deary & Stough, 1996). Avoiding tasks in which strategies can improve performance is crucial in assessing cognition in depression, as depressed individuals are often impaired in the deployment of effective cognitive strategies (Channon & Green, 1999). IT is a measure defined as the minimum duration of stimulus presentation required for near perfect response on a two-choice visual discrimination task. The stimulus duration is controlled by superimposing a backward mask over the stimulus which prevents extended iconic sampling (Nettelbeck, 1987). Subjects are instructed to take as long as necessary to respond, and to focus on accuracy. A preliminary study by Tsourtos *et al.* (1995) reported that a mixed group of psychiatric in-patients diagnosed with either depression, schizophrenia, mania or anxiety disorder had significantly longer ITs than a healthy control group.

The present study examined IT performance in unipolar depressed in-patients of a similar age to those in the study by Purcell *et al.* (1997). It was hypothesized that the depressed patients would be impaired relative to age-, sex- and IQ-matched control subjects. Many studies of the neuropsychological profile of depression have included medicated and unmedicated patients within the same group (Austin *et al.* 1992; Tarbuck & Paykel, 1996; Purcell *et al.* 1997). In order to examine if medication has an effect on IT in depression, medicated and unmedicated subjects were grouped separately in the present study. As our previous research has indicated that anticholinergic drugs can impair IT (Thompson *et al.* 2000; Waterham *et al.* 2002), we compared patients receiving antidepressants with anticholinergic effects to those receiving antidepressants with minimal anticholinergic actions. Depressive symptoms and history have also been reported by some studies to be related to cognitive impairment in some studies (Austin *et al.* 1992) but not others (Purcell *et al.* 1997; Schatzberg *et al.* 2000), thus the relationship between level of depression, depressive history and IT were examined.

METHOD

Subjects

Twenty unmedicated and 19 medicated depressed in-patients from a psychiatric ward in a general hospital in Adelaide, South Australia who were clinically diagnosed with (MD) according to the DSM-III-R criteria, together with 20 healthy controls, match for age, sex and IQ participated. Patients diagnosed with any history of substance abuse, neurological injury, or concurrent psychiatric disorder were excluded. An initial informal interview with the control subjects was used to establish any evidence of substance abuse, neurological injury, or family history of psychiatric illness. The vocabulary subscale from the Weschler Adult Intelligence Scale – Revised (WAIS-R; Weschler, 1987) was

Table 1. *Means* (S.D.) *of the demographic variables*

	Age	Sex	Vocabulary
Unmedicated ($N = 20$)	39·4 (13·6)	12 F	39·6 (13·3)
Medicated ($N = 19$)	36·1 (12·8)	15 F	39·5 (13·0)
Controls ($N = 20$)	35·8 (13·7)	14 F	40·3 (11·4)

Table 2. *Medication of depressed patients in the non-cholinergic and anticholinergic medication groups*

Subject No.	Medication	Dose/day mg
Non-cholinergic		
1	Fluoxetine	20
2	Moclobemide	150
3	Fluoxetine	20
4	Moclobemide	500
5	Moclobemide	150
6	Fluoxetine	20
7	Tranylcypromine	70
8	Sertraline	50
9	Fluoxetine	20
10	Lithium	500
Anticholinergic		
1	Thioridazine	50
2	Dothiepin	75
3	Chlorpromazine	200
4	Diazepam	75
5	Dothiepin	225
6	Imipramine	150
7	Amitriptyline	25
8	Desipramine	125
9	Desipramine	150

used as an estimate of verbal intelligence (IQ). Vocabulary subscale scores load the highest of any subscale on Full Scale IQ and are the best single subscale estimate of IQ (Sprandel, 1995). Mean (and standard deviation) age, sex and vocabulary scores are presented in Table 1. The three groups were not significantly different for age ($F_{2,57} = 0{\cdot}34$, NS), sex ($F_{2,57} = 0{\cdot}93$, NS), or vocabulary scores ($F_{2,57} = 0{\cdot}03$, NS). All subjects had normal or corrected normal visual acuity assessed using a Snellen chart, and reported free of ocular pathology. The medicated group consisted of nine patients who were treated with anticholinergic antidepressants and 10 patients with non-cholinergic antidepressants (see Table 2).

Measures and procedures

Clinical measures administered included self-ratings of depression using Zung's (1965) 20-item scale (standardized scores range between 25–100) of depression experienced in the past, and a visual analogue scale (VAS, scores range between 0–10) measuring the extent of depression currently experienced. Additional information was gathered about the patient's length of illness both current episode (weeks) and from initial onset (number of weeks since first episode). The type, dosage (mg/day) and length of medication administered to the medicated group was also retrospectively recorded from hospital drug charts after drug administration.

Inspection time

An IBM compatible PC with a 14 inch monitor was used to display the monochrome visual IT task with an accompanying 12×12 cm two response choice panel. The two buttons were 17 mm in diameter and spaced 107 mm apart. To measure IT, a small central circular cue appeared prior to the stimulus for 500 ms. The stimulus was composed of two vertical lines, one 29 mm in length, the other, 21 mm. The lines were positioned 16 mm apart and connected at the top by a horizontal line. A pair of vertical lightning rod shaped lines 29 mm in length representing the mask ('flash'), was presented immediately after the stimulus for 500 ms. The response–stimulus interval was 2000 ms. Subjects indicated which was the shorter of the two lines by pressing the appropriate response button, (left button for left line and vice versa). Four blocks of 20 trials were presented in descending order at exposure durations of 180 ms, 140 ms, 100 ms and 60 ms. Four unmasked (attention check) trails with an exposure duration of 300 ms were included in each block of trials. Ten practice trials with a set exposure duration of 500 ms were given prior to the 80 experimental trials. Participants were cautioned not to confuse the stimulus with the backward mask that followed. Where it would be difficult to judge which of the two lines was the shortest, subjects were instructed to make their best guess. An emphasis on accuracy rather than speed was conveyed. IT scores were calculated at the 87·5% accuracy level using the Probit analysis program. For subjects who made two or fewer errors in the 60 ms block of trials, a further block of 20 trials was administered at 40 ms. The computer task was completed under ten minutes by all subjects and the experiment in

its entirety was completed in no more than 30 min.

RESULTS

Table 3 displays the summary statistics of all variables for the three groups; unmedicated depressives, medicated depressives and healthy controls.

Level and history of depression

Current depression measured by the visual analogue scales was significantly different across the three groups ($F_{2,56} = 21{\cdot}8$, $P < 0{\cdot}001$). *Post hoc* analyses indicated a significant difference between the unmedicated group and the controls ($P < 0{\cdot}001$) and between the medicated group and controls ($P < 0{\cdot}001$), and a trend towards a significant difference between the unmedicated and medicated groups ($P = 0{\cdot}06$). Level of depression measured by the Zung was also significantly different across the three groups ($F_{2,56} = 45{\cdot}1$, $P < 0{\cdot}0001$). There was a significant difference between the control group and the unmedicated ($P < 0{\cdot}001$) and medicated ($P < 0{\cdot}001$) groups, but not between the two depressed groups ($P > 0{\cdot}05$). There was a trend towards significant difference between the two groups in length of illness (first episode) ($z = 1{\cdot}9$, $P = 0{\cdot}05$, Mann–Whitey), however, length of illness (current episode) was not significant ($z = 1{\cdot}3$, $P \geqslant 0{\cdot}05$).

Inspection time (IT)

IT was significantly different across the three groups ($F_{2,56} = 7{\cdot}4$, $P < 0{\cdot}005$). *Post hoc* comparisons revealed that there was a significant difference in IT between the control and unmedicated depressed groups ($P < 0{\cdot}005$), but not the control and medicated depressed group ($P > 0{\cdot}05$). The unmedicated group was significantly slower than the medicated group ($P > 0{\cdot}05$). There was no correlation between IT and level of depression from the Zung Depression Scale (Spearmans's $\rho = 0{\cdot}23$, $P > 0{\cdot}05$), or level of depression measured by the VAS ($\rho = 0{\cdot}21$, $P > 0{\cdot}05$). There was a significant negative correlation between length of depression from first depressive episode and IT ($\rho = -0{\cdot}40$, $P < 0{\cdot}05$), while IT and duration of current depressive episode ($\rho = -0{\cdot}33$, $P = 0{\cdot}06$) showed a trend towards a negative correlation.

Anticholinergic *v.* non-cholinergic medication

There was no significant difference between patients medicated on drugs with anticholinergic effects and patients on medications with non-cholinergic effects ($T_{18} = 0{\cdot}8$, $P > 0{\cdot}05$). The mean (and S.D.) IT for the anticholinergic administered subjects with MD was 106·3 (44·0) ms and for the non-cholinergic administered patients with MD was 93·9 (21·7) ms. Four of the patients in the anticholinergic group were receiving additional psychotropic medication (chlorpromazine, thioridazine) and two of the patients in the non-cholinergic group also received additional medication (clonazepam, xanax). Removal of these subjects did not substantially change mean (and S.D.) IT for either group (anticholinergic mean = 103·2 (47·6), non-cholinergic mean = 91·8 (19·8)).

DISCUSSION

Our results indicated that speed of information processing, as measured by IT, is impaired in young, unmedicated, unipolar depressed patients. This finding is consistent with the hypothesis that young depressed individuals do show cognitive slowing. Medicated, depressed patients were not significantly slower on the IT task than control subjects, but they were

Table 3. *Summary scores for inspection time* (*IT*), *depression scales*, *and history of depression*

		Depression score		Length of depression (weeks)	
	IT (ms) Mean (S.D.)	VAS Mean (S.D.)	Zung Mean (S.D.)	First episode Median	Current Median
Unmedicated ($N = 20$)	121·3 (42·3)	6·8 (2·7)	68·7 (12·4)	27	14
Medicated ($N = 19$)	95·1 (26·8)	4·8 (3·3)	66·6 (11·9)	100	16
Controls ($N = 20$)	82·2 (17·5)	1·2 (1·9)	36·8 (11·2)		

significantly faster than unmedicated depressed patients. These data suggest that the slowing of cognition associated with depression may be partly alleviated by medication, however as the present study was cross-sectional it is not possible to be certain of this conclusion. It could be argued that the relationship between depression and cognitive impairment may simply be a reflection of reduced effort and motivation in depression (Cohen *et al.* 1982). However, the IT task is very simple, requires only minimal effort and does not appear to be sensitive to manipulation of motivation level (Simpson & Deary, 1997). The IT task also minimizes the use of strategies to aid performance (e.g. speed/accuracy trade-off), thus it would appear unlikely that the impairment of the depressed patients was due to an inability to employ effective strategies (Channon & Green, 1999).

Purcell *et al.* (1997) argued that young, unipolar depressed patients do not show cognitive slowing, and that the impairments in speeded performance reported by Brown *et al.* (1994) and Elliott *et al.* (1996) were associated with the age of the depressed participants. However, Purcell *et al.*'s (1997) study combined a sample of unmedicated and medicated patients, which may have contributed to the negative finding. Furthermore, the measure of cognitive speed used in Purcell *et al.*'s (1997) study was a complex task and may have been sensitive to a number of cognitive factors aside from processing speed. The IT measure used in the present study is regarded by many as a relatively pure measure of information processing speed (Nettlebeck, 1987; Krantzler & Jensen, 1989; Deary & Stough, 1996). Supporting the interpretation of the present measure of IT as a measure of general processing speed is evidence of correlations with choice reaction time, auditory IT, and other mental speed measures (Deary *et al.* 1989; Vickers, 1995). In addition, a recent meta-analysis of over 90 studies indicated the IT explains approximately 25% of psychometric IQ scores (Grudnik & Krantzler, 2002). The impairment in IT in unmedicated depressed patients in the present study, who were in the same age range as those of Purcell *et al.* (1997), indicates that processing speed deficits are an important aspect of the neuropsychological profile of younger depressed patients as well as geriatric depressed patients.

There was no significant correlation between IT and self-ratings of depressed mood or scores on the Zung depression scale in the present results. Austin *et al.* (1992) reported that levels of depression were significantly correlated with memory impairments, while others have found no relationship between depression levels and cognition (Purcell *et al.* 1997; Schatzberg *et al.* 2000). The results of the present study indicated that although the unmediated depressed patients showed higher levels of currently depressed mood, this did not appear to explain the differences between the groups in speed of information processing. However, the present study used self-report measures of depressed mood, which may be of questionable reliability, and a more thorough examination of this issue should use a measure such as the Hamilton Depression Rating Scale or the like. Among the depressed patients in the present study, length of depressive illness since first episode, and to a lesser degree length of current illness, was associated with shorter IT. These findings may simply be a reflection of the fact that medicated depressed patients tended to have had a depressive illness for longer, and had shorter ITs. The findings do suggest that cognitive slowing may not simply be a consequence of long-term medication effects, or a sign of neurodegeneration that may follow a long history of depression. However, longitudinal data is required to help clarify this issue.

There was no significant difference between medicated patients receiving antidepressants with anticholinergic effects and those on antidepressants with minimal cholinergic effects. However, the sample size for this comparison was small and interpretation of this negative result should be made with caution. Further examination of the effects of antidepressants on the cognitive function of depressed individuals is clearly necessary. Selective anticholinergic drugs such as scopolamine and mecamylamine impair IT performance in healthy subjects (Thompson *et al.* 2000; Waterham *et al.* unpublished observations). Our laboratory has preliminary findings that the anticholinergic antidepressant amitriptyline impairs IT in healthy subjects. However, there have been mixed findings of the effects of medication on neuropsychological function in depressed patients (Glass *et al.* 1981; Abas *et al.* 1990). A study by Spring *et al.* (1992)

indicated that the adverse cognitive effects of amitriptyline were observed only after depressive symptoms had improved. The direct anticholinergic effects of the antidepressants prescribed in the present study, however, may be considerably less than that of selective cholinergic anatagonists such as scopolamine and mecamylamine. Monoamine or other neuromodulatory changes may also counterbalance the possible adverse anticholinergic effects of some antidepressant drugs.

A number of pathologies have been suggested as the basis of neuropsychological impairments in depression, including medial temporal (Mayberg *et al*. 1999) and frontostriatal (Purcell *et al*. 1997) dysfunction. Hypothalamic–pituitary–adrenal (HPA) axis abnormalities have also been attributed a role (McAllister-Williams *et al*. 1998; Holsboer, 2000). There is some suggestion that the mood-alleviating effects of antidepressants are in part mediated by effects on corticosteriodal systems (Barden *et al*. 1995). While the neurobiological basis of speed of information processing appears to involve cholinergic systems, other neuromodulatory systems such as those involving glucocorticoids may play some part. In conclusion, this study has shown that information processing speed is slowed in young, unmedicated depressed patients. Cognitive slowing should thus be considered in future studies of the neuropsychological profile of depression, and IT is a quick, simple and easily administered measure. Medication status should also be considered when examining cognitive function in depression.

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