Psychological Medicine, 2001, **31**, 1269–1277. © 2001 Cambridge University Press
DOI: 10.1017/S0033291701004597 Printed in the United Kingdom

Backward masking task performance in stable, euthymic out-patients with bipolar disorder

G. M. MacQUEEN,[1] L. T. YOUNG, T. M. GALWAY AND R. T. JOFFE

From the Mood Disorders Program, McMaster University, Hamilton, Ontario, Canada

ABSTRACT

Background. Several studies have suggested that visual backward masking (VBM) impairment is present in patients with bipolar disorder, but the clinical features, such as current symptoms, treatment status and past burden of illness that may contribute to the impairment have not been well described. This study examined well-characterized euthymic patients on two VBM tasks to ascertain the extent of VBM impairment in this group and the clinical correlates of this impairment.

Method. Twenty-eight euthymic patients with a DSM-IV diagnosis of bipolar disorder were matched by age, sex and IQ with 28 non-psychiatric control subjects. Both groups completed two VBM tasks; one required subjects to locate the target stimulus, one required identification of the target stimulus. Reaction times and error rates across a range of target-mask inter-stimulus intervals were assessed.

Results. Patients were significantly slower and had more errors on both VBM tasks. There was a significant relation between reaction times on the identification task and past burden of illness, particularly past number of depressions. There was no discernible impact of treatment status on reaction time or performance, including no difference in lithium-treated *versus* not treated subjects.

Conclusions. These results are consistent with previous reports of neuropsychological deficits in euthymic bipolar disorder patients. The potential benefit to employing tasks such as VBM is that it may provide a method for relating clinical variables such as illness burden with known neural pathways in order to elucidate better the pathophysiology leading to impaired cognitive performance in patients with bipolar disorder.

INTRODUCTION

Several studies have recently found that patients with bipolar disorder have detectable impairments in neuropsychological performance that persist into the euthymic state (Tham *et al.* 1997; Kessing, 1998; VanGorp *et al.* 1998; Ferrier *et al.* 1999; Rubinsztein *et al.* 2000). Impairment has been demonstrated in global cognitive functioning (Kessing, 1998), visuospatial recognition memory (Rubinsztein *et al.* 2000), verbal memory (VanGorp *et al.* 1998), general intelligence, reasoning and the trail-making test (Tham *et al.* 1997). Notably, past number of depressions was particularly predictive of cognitive functioning in the bipolar disorder patients (Kessing, 1998; VanGorp *et al.* 1998; see also Denicoff *et al.* 1999). These data suggest that progressive neural changes occurring over the course of bipolar disorder may result in impaired cognitive performance, but the tests utilized to date do not readily allow for further study of specific neural substrates that may be compromised in these patients.

There are available methods of assessing information processing in patients that map onto known neural processing pathways. Visual backward masking (VBM) is one such paradigm, and it has been extensively employed to study information processing deficits in patients with

[1] Address for correspondence: Dr Glenda M. MacQueen, 4N77A, Department of Psychiatry and Behavioural Neurosciences, McMaster University Medical Centre, 1200 Main Street West, Hamilton, Ontario, Canada, L8N 2Z5.

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

schizophrenia (Braff, 1981; Saccuzzo & Braff, 1981; Green & Walker, 1986; Schuck & Lee, 1989; Rund *et al*. 1996). To a lesser extent the performance of patients with bipolar disorder has been examined, but these studies have been largely confined to acutely manic, psychotic patients (Schubert, 1985; Green & Walker, 1986; Saccuzzo & Braff, 1986) or patients who were not yet discharged following a manic episode (Fleming & Green, 1995). Other studies in BD focused on the neural pathways that may underlie impairment in BD but were conducted on chronic, hospitalized patients, most of whom required antipsychotic medication at the time of assessment (Green *et al*. 1994*a*, *b*). Thus, the performances of euthymic, stable patients have not been evaluated to date, neither has the effect of clinical course variables such as past episodes or current treatment and symptom levels on VBM performance been well described.

In VBM a briefly presented informative stimulus (the target) is quickly replaced in the visual field by a non-informative mask. Two potential mechanisms have been proposed to account for the masking phenomenon: integration and interruption (Turvey, 1973). Integration occurs when the visual representation, or icon, of the target fuses with the icon of the mask, producing a confused, composite image that has been compared to a double-exposed photograph. Interruption occurs when late processing of the target is disrupted by the mask so that further processing of the target does not occur. Both integration and interruption appear to be involved in masking, with the relative contribution of either determined by the temporal and spatial relationships of the target and mask.

Breitmeyer & Ganz (1976) and Breitmeyer (1980, 1984) developed a model to integrate behavioural data on VBM with known neurophysiological mechanisms of visual perception. The model proposed that transient (magnocellular) visual channels are involved in processing the stimulus for spatial location, while sustained (parvocellular) visual channels are required for further processing stimulus features such as identity of the stimulus. Masking by interruption occurs when the transient channels of the mask interrupt activity in the sustained channels of the target; masking by integration is thought to occur when activity in the sustained channels of both the target and mask are combined. It is therefore possible to modify a masking task so that either the transient or sustained channels are primarily activated, which in turn can influence different visual pathways. Requiring a subject to locate *versus* identify the target in VBM may discriminate the function of discrete anatomical attention pathways efferent to the primary visual cortex (V1). An object recognition stream originates in V1 and projects to the inferior temporal cortex via sub-regions of areas V2 and V4 (Ungerleider & Haxby, 1994). More important for spatial performance and localization is the dorsal stream, also projecting from V1 into the posterior parietal region and subsequently to the prefrontal cortex (see Desimone & Duncan, 1995 for a review). Green *et al*. (1994*a*, *b*) suggested that the deficits experienced by patients with bipolar disorder are related to a failure to attenuate the transient channel activity of the mask, resulting in interruption of the sustained channels of the targets. They further suggest that processes requiring transient channel activity are right hemisphere-reliant processes such as locating objects in space, but as discussed, this pattern of results has not been confirmed in stable patients with bipolar disorder.

In this study we systematically manipulated reliance on sustained or transient channels by requiring a large group of stable, euthymic bipolar patients to perform VBM tasks requiring either locating or identifying the target stimulus. Speed and accuracy of patients on these tasks were compared to performance of non-psychiatric control subjects. We also specifically examined whether clinical variables including residual symptoms, past illness burden or medication use influenced performance on the VBM tasks.

METHOD

Subjects

Twenty-eight stable out-patients with a diagnosis of bipolar disorder I without rapid cycling confirmed by the Structured Clinical Interview for DSM-IV (SCID) (Spitzer & Williams, 1987) were recruited from the Mood Disorders Program of McMaster University. All subjects were euthymic for a minimum of 1 month prior

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

to assessment (range 1–33 months). Twenty-eight age, sex and IQ matched non-psychiatric control subjects were recruited according to hospital guidelines. All subjects provided written informed consent. Inclusion criteria for both groups included: (1) age between 21 and 60; and (2) ability to give informed consent. Exclusion criteria for both groups included: (1) substance abuse or dependence within the last 6 months or a history of lifetime substance dependence; (2) treatment with electroconvulsive therapy within the past 2 years; (3) history of closed head injury resulting in loss of consciousness; (4) evidence of a reading deficit as assessed by performance on the vocabulary portion of the Wide Range Achievement Test; (5) untreated active medical illness (e.g. diabetes, hyperthyroidism, hypertension); (6) co-morbid anxiety disorder as assessed by the SCID; (7) treatment with antipsychotic medication in 4 weeks prior to the study. In addition, control subjects were excluded if they had a history of any psychiatric illness as assessed by the SCID.

Diagnostic assessment

The SCID was administered by a trained rater to each subject for diagnosis, clarification of number of past episodes and assessment of co-morbidity. Medical chart records, further patients interviews, family members, treating clinicians and life charts, where available, were also consulted to determine the most accurate estimates of past number of episodes and level of functioning.

Symptom assessment

The Hamilton Rating Scale for Depression (Ham-D) (Hamilton, 1960) and Young Mania Rating Scale (YMS) (Young *et al*. 1978) were administered during out-patient treatment in order to determine when euthymia had been obtained. Euthymia was defined as a Ham-D score $\leqslant 7$ and a YMS score $\leqslant 5$, and patients were required to have been euthymic for 1 month prior to participating in the experiment. Ham-D and YMS scores were obtained on the day of cognitive testing. Subjects and controls were rated by one investigator for whom reliable correlations with standard assessment techniques were periodically determined. Clinical ratings were conducted prior to subjects completing the VBM tasks.

Cognitive assessment

Because the computer task involved presentation of letters as stimuli, all subjects were required to be able to read at the grade six level as assessed by the Wide Range Achievement Test (WRAT). Subjects also received the National Adult Reading Test (NART) to determine pre-morbid IQ levels for the purposes of matching patients and controls. Subjects estimated perceived impairment in cognitive function by completing the Cognitive Failures Questionnaire (Broadbent *et al*. 1982). The CFQ is a 38 self-report questionnaire that assesses perception of recent failures in cognitive function (e.g. Do you often forget where you have put your keys?).

Computer assessment

The backward masking tasks were presented on an IBM compatible microcomputer with an SVGA monitor and circuitry capable of millisecond timing. Visual angles subtended by the stimuli were approximately 0·57° on the vertical and horizontal dimensions. The masking task was divided into two parts, a locating task and an identification task. For both parts, the target stimuli were letters (O, S, Q, C) presented at one of four possible target locations (up, down, left or right, approximately 2·2° of visual angle away from fixation); the mask consisted of three overlapping Xs and appeared only in the four possible target locations. In the locating task, subjects identified the location of the target by pointing in the correct direction using a joystick; they were not required to identify the target. This condition optimizes transient channel responses. In the identification component, subjects verbally reported the identity of the presented letter using a voice key linked to software capable of millisecond timing. A technician entered the stated letter to record correct and incorrect responses. This condition maximized sustained channel responding. Prior to each task, a series of 12 practice trials was given in which subjects reported the location of the target in the absence of any mask in order to orientate them to the requirements of the task.

Trials began with presentation of a fixation point for 400 ms that terminated 200 ms before target presentation. The target appeared for 16·5 ms followed by a 16·5 ms mask. The target-mask ISIs were 16·5, 33, 49·5, 66 and 79·5 ms

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

and 96 ms. There were 12 trials at each ISI for both the locating and the identification components of the task, with an overall total of 168 trials, including all practice trials. Percentage of incorrect responses and mean reaction time (RTs) at each ISI constituted the dependent measures.

Statistical analysis

Demographic characteristics of the patient and control groups were compared using independent *t* tests (SPSS, 1996). Repeated measures analyses of variance compared RTs and error rates between groups at each ISI. Analyses of variance were also used to compare performance of subjects who were or were not treated with lithium, and performance of subjects who were or were not treated with anticonvulsants. Pearson's *r* was used to test for associations with RTs and error rates against number of past episodes of depression and mania. To provide a conservative analysis of the effect of past illness episodes, patients who reported very large numbers of episodes (>20) were assigned 20 as a maximum number of past depressions or manias (maximum 40 episodes total). Linear regression models were utilized to determine whether age, current symptom levels or past illness burden accounted for a significant portion of the variance in RTs and error rates.

RESULTS

Characteristics of the sample

The average age of bipolar patients was 42·2, while controls were an average of 41·8 years of age (see Table 1). There were 15 women and 13 men in each group. Patients had an average full scale IQ of 112·3, while controls had an average of 116·0. The patients had an average of 9·8 previous episodes of depression and 8·1 previous episodes of mania. The Ham-D scores at the time of testing averaged 2·8 for the patients and 1·5 for controls. The YMS scores averaged 1·4 and 0·7 for patients and controls respectively.

Table 1. *Demographic and clinical characteristics of the patient and control subjects. Values are shown as mean* (*standard deviation*) *unless otherwise stated*

Variable	Patients Mean (S.D.)	Controls Mean (S.D.)
Age	42·2 (12·3)	41·8 (8·6)
Gender (F/M)	15/13	15/13
IQ	112·3 (9·2)	116·0 (8·1)
CFQ	44·8 (20·2)	31·7 (13·5)*
Ham-D	2·8 (1·9)	1·5 (2·3)
YMS	1·39 (2·1)	0·69 (1·4)
Past depressions, *N* (%)	9·8 (5·9)	0 (0)
Past manias, *N* (%)	8·1 (3·7)	0 (0)
Receiving psychotropic medication, *N* (%)	17 (60·7)	0 (0)
Receiving lithium, *N* (%)	14 (50)	0 (0)

* $P < 0{\cdot}05$.

Performance on backward masking

Repeated measures analyses of variance showed that patients were significantly slower than controls to respond across ISIs on the identification task ($F(49,1) = 11{\cdot}9$; $P = 0{\cdot}001$) and made more errors than controls on this task ($F(49,1) = 12{\cdot}7$, $P < 0{\cdot}001$; see Fig. 1). As well, their performance differed from control subjects on the locating task, in that they were slower ($F(54,1) = 25{\cdot}5$, $P < 0{\cdot}001$) and made more errors ($F(54,1) = 8{\cdot}3$, $P = 0{\cdot}006$; Fig. 2). RTs and error rates for both tasks were significantly dependent on ISIs, with both patients and controls becoming faster and more accurate as ISIs increased. An interaction between ISI and group was apparent for the locating task ($F(245, 5) = 7{\cdot}9$; $P < 0{\cdot}001$), as subjects with bipolar disorder had higher error rates at the short ISIs that converged to those of controls at longer ISIs. Although there was a similar trend for the identify task, the interaction was not statistically significant ($F(245, 5) = 1{\cdot}67$; $P = 0{\cdot}14$) as patient error rates did not converge to control levels even at longer ISIs.

Correlation between speed and accuracy and illness characteristics

The most striking correlation was that number of past depressions significantly and specifically predicted RTs at each ISI of the identification task (r^2 values for ISI 16·5 ms = 0·43; ISI 33 ms = 0·56; ISI 49·5 ms = 0·44; ISI 66 ms = 0·62; ISI 79·5 ms = 0·52; ISI 96 ms = 0·47; all *P* values < 0·03). There were trends for a relation between error rates on the identification task. There was no such relation evident for number of past episodes (either depressions or manias) and either reaction time or error rate for the

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

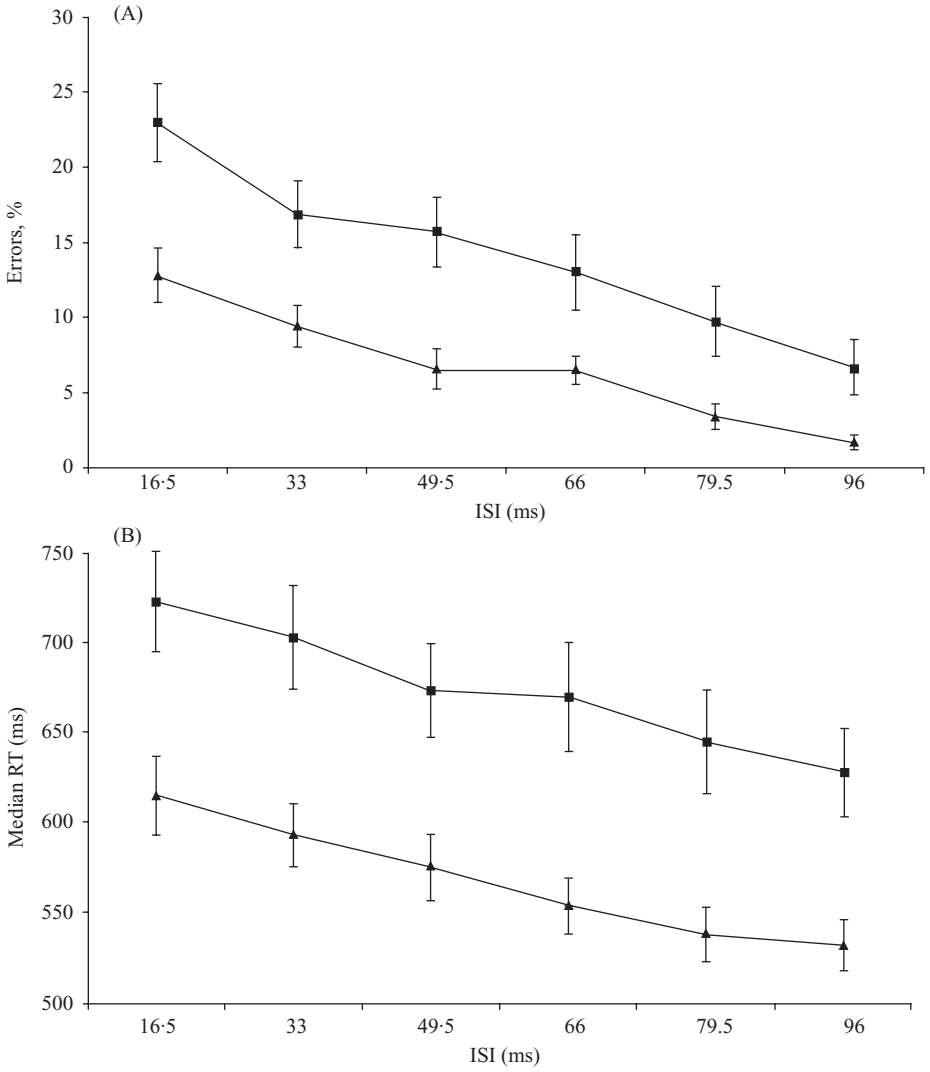


FIG. 1. Error rates (A) and reaction times (B) for patients (■) and controls (▲) on the VBM task requiring subjects to identify verbally the target letter at various interstimulus intervals.

location task. The relation between past episodes and performance on the identification task was not a function of age or current symptoms as there was no correlation with age or current symptoms with RT or accuracy data at any ISI on either the location or identification task. Furthermore, a linear regression did not reveal a significant effect of either age or current symptom level, but a significant portion of the variance in patient reaction time performance on the identification task was accounted for by past numbers of depressive episodes ($B = 4{\cdot}7$, $t = 3{\cdot}2$, $P = 0{\cdot}004$). Despite a wide range of times since entering euthymia (1–33 months) we could find no evidence that time since entering euthymia predicted performance on any aspects of the tasks.

Notably, perception of cognitive impairment as reflected by the CFQ scores was significantly higher for patients than controls, despite the patients' euthymic status ($t = 2{\cdot}53$, $P = 0{\cdot}015$). Furthermore, patients' self reports of cognitive impairment were weakly correlated with total number of past illness episodes ($r^2 = 0{\cdot}41$, $P =$

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

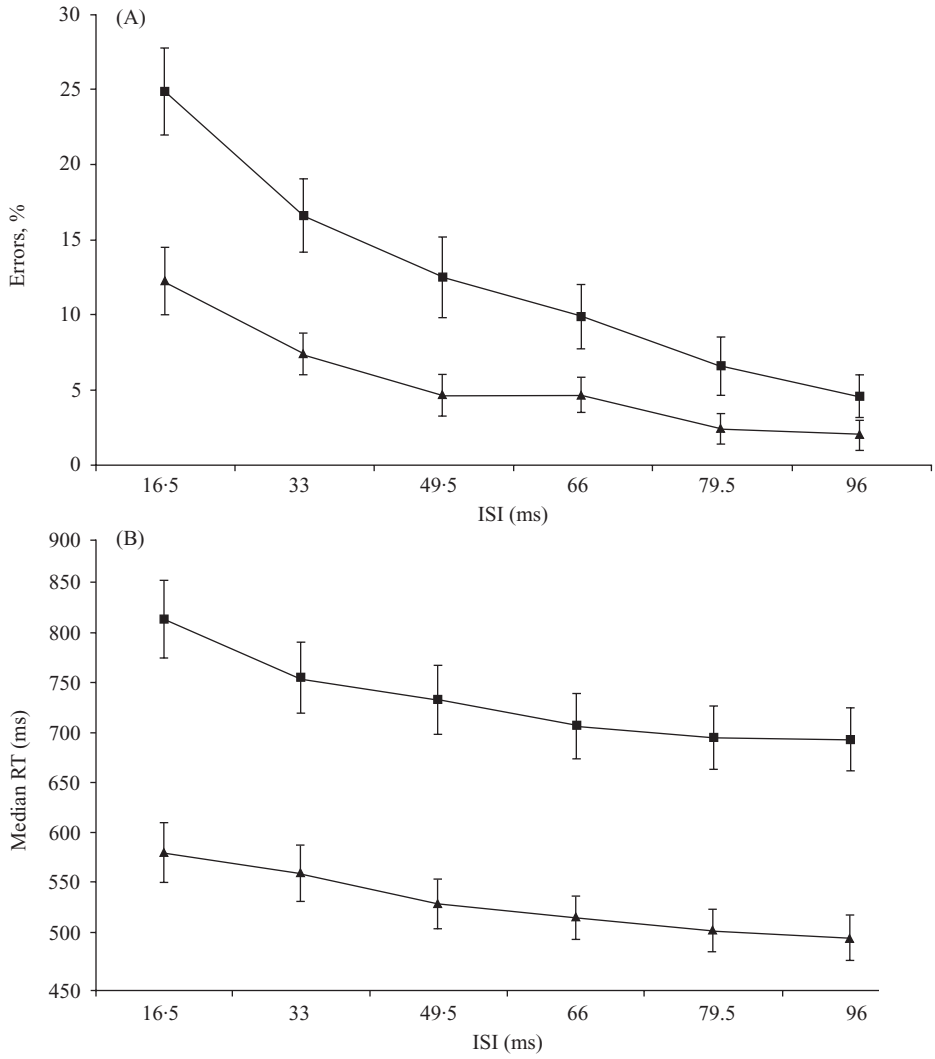


FIG. 2. Error rates (A) and reaction times (B) for patients (■) and controls (▲) on the VBM task requiring subjects to locate the target letter at various interstimulus intervals.

0·06); further examination revealed a strong association between CFQ and number of past episodes of depression ($r^2 = 0{\cdot}52$, $P = 0{\cdot}013$) but no relation between CFQ and number of past manias ($r^2 = 0{\cdot}17$, $P = 0{\cdot}45$). CFQ scores did not, however, predict performance on the VBM tasks for either reaction times or accuracy in the location or identification task.

Effect of medication

Subjects were naturalistically treated with lithium, anticonvulsants and antidepressants. To examine whether there was any evidence that current treatment with conventional mood stabilizers (lithium, valproate, carbamazepine) either improved or worsened reaction time and accuracy performance, we compared subjects treated with these agents ($N = 17$) compared with subjects who were not receiving any of these agents ($N = 11$) on repeated measures analyses of variance of RTs and error rates. There was no evidence of either a positive or negative effect of these agents on the location task for either speed ($F(26,1) = 0{\cdot}47$, $P = 0{\cdot}5$) or accuracy ($F(26,1) = 0{\cdot}89$, $P = 0{\cdot}35$), neither was an effect apparent on speed ($F(22,1) = 0{\cdot}79$, $P =$

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

0·39), or accuracy ($F(22,1) = 0·02$, $P = 0·83$) for the identity task.

We next used repeated measures analyses to determine whether there was a discernible difference between lithium treated and not treated subjects, as lithium as been specifically identified as an agent that may influence cognitive performance. There were no differences in reaction times or accuracy for lithium treated and not treated patients (all P values $> 0·20$). Repeated measures analyses were used to determine whether treatment with the valproate or tegretol influenced task performance. There was no discernible effect of these agents on reaction time or accuracy in either the location or identification task (all P values $> 0·20$). Similarly, there was no detectable effect of antidepressant treatment for either the location or identification task (all P values $> 0·20$).

DISCUSSION

Although patients in this study were stable, euthymic out-patients with no significant comorbidity, they were significantly impaired on VBM tasks as assessed by both speed and accuracy. This impairment was not predicted by residual symptoms as reflected on the Ham-D and YMS scores. These data suggest that impaired performance on VBM tasks is not a state function in BD patients, and are consistent with other recent studies that found impaired neuropsychological performance in BD patients. Past burden of illness, particularly past number of depressions was strongly predictive of reaction time performance on the identification task and this relation was apparent across all ISIs.

The relation of past number of depressions to reaction time performance suggests that this impairment may evolve over the course of the disorder, rather than being present early in the course of illness, or detectable as a vulnerability marker in the pre-morbid state. It is striking that of four recent reports of neuropsychological impairment in euthymic BD patients, all have reported a relation between performance and past burden of illness. Rubinsztein and colleagues (2000) found that past number of months hospitalized predicted impairment in performance on a delayed matching to sample task. Kessing found that patients with recurrent episodes were more impaired than those with a single episode, who were not different than non-psychiatric controls. Furthermore, he reported that it was number of past depressive, not manic, episodes that predicted cognitive function (Kessing, 1998). VanGorp and colleagues found a negative correlation between lifetime months of either depression or mania and verbal memory and tests of executive function. (VanGorp *et al.* 1998). Finally, Tham and colleagues found that number of past hospitalizations was correlated with tests of general intelligence and reasoning as well as with performance on Trails A and B (Tham *et al.* 1998). Definitive studies examining neuropsychological functioning in patients at high risk for BD, family probands of BD patients or longitudinal studies of patients beginning in a first episode have not been conducted and would be helpful in discerning whether, in fact, the cognitive changes observed in BD patients do emerge over time. In the absence of such information, it is impossible to rule out the possibility that cognitive deficits and multiple episodes are co-occurring markers of a severe form of BD with no causal relation existing between episode frequency and cognitive function.

While the performance of patients was generally impaired in the VBM tasks, the specificity of the relation between past depressions and the identification task performance may point toward a neural pathway that is particularly sensitive to illness burden. The identification task should be more dependent than the location task on function of the sustained visual channels that rely on Type B ganglion cells of the retina. The Type B ganglion cells project to the parvocellular layers of the lateral geniculate, through multiple layers of the visual cortex and terminate in the inferotemporal cortex, a region that has been implicated by imaging studies of patients with mood disorders (Altshuler *et al.* 2000 for a summary). In contrast, while patients' RTs and error rates for the location task were also higher than controls, a similar relation with past illness did not emerge for the task that is more dependent upon the magnocellular-parietal visual processing stream. Our data did not reveal a relative sparing in performance for one task *versus* the other as patients were impaired relative to controls on both the identify and locate tasks. This is consistent with McClure's recent summary suggesting that masking by

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

both interference and interruption is impaired in bipolar disorder (McClure, 1999). The data, however, do not support the previous data suggesting that visual processing in bipolar disorder is particularly vulnerable in conditions emphasizing transient channel activity (Green *et al.* 1994*b*), although it is important to note that these investigators emphasized that they were examining patients in the manic state. A recent study found differences in neuropsychological performance of manic or mixed bipolar patients *versus* depressed bipolar patients, emphasizing that neuropsychological performance during manic periods may not reflect permanent deficit (Sweeney *et al.* 2000). In Sweeney *et al.*'s study, spatial attention, which is dependent upon right parietal function, was impaired only in the manic/mixed group. Episodic memory, which is dependent upon infero-temporal lobe integrity, was impaired in all groups of bipolar patients assessed, suggesting that impairment in this region may be apparent regardless of mood state while impairment in right parietal function may be associated with the mixed or manic state. Notably, Rubinsztein *et al.* (2000) also found persistent deficits in euthymic bipolar patients on tasks dependent on temporal lobe function.

Although McClure (1999) recently suggested that lithium treatment has a detrimental effect on performance in masking tasks, we found no evidence of such a difference, and to our knowledge, no previous backward masking studies systematically compared patients treated or not treated with lithium. In masking studies in which patients were taking lithium, no effect was made to eliminate potentially confounding variables such as mood state and concurrent treatment with antipsychotic medication. Clinically, concurrent treatment with lithium and conventional antipsychotic medication is thought to potentiate the neurocognitive effect of each drug and perhaps one of the reasons that we found no apparent effect of lithium treatment is that no subjects were taking antipsychotic medications at the time of testing. There has been controversy regarding the effect of lithium on cognition in general, but a number of studies have failed to find evidence that long-term lithium treatment impairs performance on neuropsychological tasks (Smigan & Perris, 1983; Calev *et al.* 1986; Engelsmann *et al.* 1988) although psychomotor speed may be affected (Squire *et al.* 1980). In recent studies, VanGorp *et al.* (1998) did not find an association with current lithium level and performance on a cognitive test battery, neither did Rubinsztein *et al.* (2000) find a difference on either simultaneous nor delayed matching to sample tasks when euthymic bipolar patients treated or not treated with lithium were compared.

It is difficult to compare performance of these euthymic bipolar patients with those of patients with schizophrenia. Certainly, it is clear at this point that masking deficits are not restricted to patients with psychotic disorders, or to patients in a psychotic state. It is less clear whether bipolar patients have a response pattern that overall is distinguishable from patients with schizophrenia, in part because stable bipolar patients have not been compared to stable outpatients with schizophrenia. Further complicating such comparisons is the fact that most patients with schizophrenia require antipsychotic medication, while many bipolar patients, such as the patients here, are treated primarily with mood stabilizers. Differences in treatment would have to be controlled before conclusions can be reached regarding profiles that may be specific to these disorders.

This study is limited by the fact that medication status was not rigidly controlled and therefore it is not possible to rule out the possibility that a systematic bias obscured a real difference between treated and untreated patients, or patients on different medications. Furthermore, although patients must have had a period of euthymia that was sustained for at least 1 month, it is possible that if patients were tested after a longer period of symptom quiescence, performance would have been enhanced.

Despite the potential limitations, these data add to the growing literature on neuropsychological changes in euthymic bipolar disorder. They are consistent with previous studies demonstrating a relation between past depressions and performance, and they expand on these studies by describing an association between this illness variable and performance on a task of early information processing for which there are known neural substrates. Furthermore, these data add to the limited information available on the effect of mood stabilizing medication on neuropsychological performance, a factor that has not received adequate sys-

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press

tematic investigation. The functional significance of the cognitive changes observed in euthymic patients with bipolar disorder remains to be established, as does the extent to which these changes are reversible. Studies such as this, however, may provide the foundation upon which future studies addressing both underlying mechanism and clinical significance can be determined.

G. MacQueen is a New Investigator of the Canadian Institutes of Health Research. L. T. Young is a career scientist with the Ontario Ministry of Health. This work was supported by the Ontario Mental Health Foundation and the Stanley Foundation.

REFERENCES

Altshuler, L., Bartzoxis, G., Grueder, T., Curran, J., Jimenez, T., Leight, K., Wilkins, J., Gerner, R. & Mintz, R. (2000). An MRI study of temporal lobe structures in men with bipolar disorder or schizophrenia. *Biological Psychiatry* **48**, 147–162.

Braff, D. L. (1981). Impaired speed of information processing in nonmedicated schizotypal patients. *Schizophrenia Bulletin* **7**, 499–508.

Breitmeyer, B. G. & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual pattern masking, saccadic suppression, and information processing. *Psychological Review* **83**, 1–36.

Breitmeyer, B. G. (1980). Unmasking visual masking: a look at they 'why' behind the veil of the 'how'. *Psychological Review* **1**, 52–69.

Breitmeyer, B. G. (1984). *Visual Masking: An Integrative Approach*. Oxford University Press: New York.

Broadbent, D. E., Cooper, P. F., Fitzgerald, P. & Parkes, K. R. (1982). The cognition failures questionnaire (CFQ) and its correlates. *British Journal of Clinical Psychology* **21**, 1–16.

Calev, A., Korin, Y., Shapira, B., Kugelmass, S. & Lerer, B. (1986). Verbal and non-verbal recall by depressed and euthymic affective patients. *Psychological Medicine* **16**, 789–794.

Denicoff, K. D., Ali, S. O., Mirsky, A. F., Smith-Jackson, E. E., Leverich, G. S., Duncan, C. C., Connell, E. G. & Post, R. M. (1999). Relationship between prior course of illness and neuropsychological functioning in patients with bipolar disorder. *Journal of Affective Disorders* **56**, 67–73.

Desimone, R. & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience* **18**, 193–222.

Engelsmann, F., Katz, J., Ghadirian, A. M. & Schachter, D. (1988). Lithium and long-term memory: a long-term follow up study. *Journal of Clinical Psychopharmacology* **8**, 207–211.

Ferrier, I. N., Stanton, B. R., Kelly, T. P. & Scott, J. (1999). Neuropsychological function in euthymic patients with bipolar disorder. *British Journal of Psychiatry* **175**, 246–251.

Fleming, K. & Green, M. F. (1995). Backward masking performance during and after manic episodes. *Journal of Abnormal Psychology* **104**, 63–68.

Green, M. F. & Walker, E. (1986). Symptom correlates of vulnerability to backward masking in schizophrenia. *American Journal of Psychiatry* **143**, 181–186.

Green, M. F., Nuechterlein, K. H. & Mintz, J. (1994*a*). Backward masking in schizophrenia and mania. I. Specifying a mechanism. *Archives of General Psychiatry* **51**, 939–944.

Green, M. F., Nuechterlein, K. H. & Mintz, J. (1994*b*). Backward masking in schizophrenia and mania. II. Specifying the visual channels. *Archives of General Psychiatry* **51**, 945–951.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology Neurosurgery and Psychiatry* **23**, 56–72.

Kessing, L. V. (1998). Cognitive impairment in the euthymic phase of affective disorder. *Psychological Medicine* **28**, 1027–1038.

McClure, R. K. (1999). Backward masking in bipolar affective disorder. *Progress in Neuropsychopharmacology and Biological Psychiatry* **23**, 195–206.

Rubinsztein, J. S., Michael, A., Paykel, E. S. & Sahakian, B. J. (2000). Cognitive impairment in bipolar affective disorder. *Psychological Medicine* **30**, 1025–1036.

Rund, B. R., Oie, M. & Sundet, K. (1996). Backward-masking deficit in adolescents with schizophrenic disorders or attention deficit hyperactivity disorder. *American Journal of Psychiatry* **153**, 1154–1157.

Saccuzzo, D. P. & Braff, D. L. (1986). Information-processing abnormalities: trait- and state-dependent components. *Schizophrenia Bulletin* **12**, 447–459.

Schubert, D. L., Saccuzzo, D. P. & Braff, D. L. (1985). Information-processing in borderline patients. *Journal of Nervous and Mental Disease* **173**, 26–31.

Schuck, J. R. & Lee, R. G. (1989). Backward masking, information processing, and schizophrenia. *Schizophrenia Bulletin* **15**, 491–500.

Smigan, L. & Perris, C. (1983). Memory functions and prophylactic treatment with lithium. *Psychological Medicine* **13**, 529–536.

Spitzer, R. & Williams, J. (1987). *Structured clinical interview for DSM-III-R*. Biometrics research Department, NY State Psychiatric Institute: New York.

SPSS (1996). *SPSS for Windows, Version 7.5*. SPSS Inc., Chicago, IL.

Squire, L. R., Judd, L. L., Janowsky, D. S. & Huey, L. Y. (1980). Effects of lithium carbonate on memory and other cognitive functions. *American Journal of Psychiatry* **137**, 1042–1046.

Sweeney, J. A., Kmiec, J. A. & Kupfer, D. J. (2000). Neuropsychiatric impairments in bipolar and unipolar mood disorders on the CANTAB cognitive battery. *Biological Psychiatry* **48**, 674–684.

Tham, A., Engelbrektson, K., Mathe, A. A., Johnson, L., Olsson, E. & Aberg-Wistedt, A. (1997). Impaired neuropsychological performance in euthymic with recurring mood disorders. *Journal of Clinical Psychiatry* **58**, 26–29.

Turvey, M. T. (1973). On peripheral and central processes in vision: inferences from an information-processing analysis of masking with patterned stimuli. *Psychological Reviews* **80**, 1–52.

Ungerleider, L. G. & Haxby, J. (1994). What and where in the human brain. *Current Opinions in Neurobiology* **4**, 157–651.

VanGorp, W. G., Altshuler, L., Theberge, D. C., Wilkins, J. & Dixon, W. (1998). Cognitive impairment in euthymic bipolar patients with and without prior alcohol dependence. *Archives of General Psychiatry* **55**, 41–46.

Young, R. C., Biggs, J. T. & Ziegler, V. E. (1978). A rating scale for mania: reliability, validity and sensitivity. *British Journal of Psychiatry* **133**, 429–435.

https://doi.org/10.1017/S0033291701004597 Published online by Cambridge University Press