Psychological Medicine, 2004, **34**, 555–561.
DOI: 10.1017/S0033291703001028 Printed in the United Kingdom

BRIEF COMMUNICATION

Continuation cognitive-behavioural therapy maintains attributional style improvement in depressed patients responding acutely to fluoxetine

T. PETERSEN,[1] R. HARLEY, G. I. PAPAKOSTAS, H. D. MONTOYA, M. FAVA AND J. E. ALPERT

From the Depression Clinical and Research Program, Massachusetts General Hospital, Boston, MA, USA

ABSTRACT

Background. Little is known about how continuation and maintenance cognitive-behavioural therapy (CBT) influences important psychological constructs that may be associated with long-term outcome of major depressive disorder. The goal of this study was to examine whether CBT would help maintain attributional style changes experienced by patients during acute phase fluoxetine treatment.

Method. Three hundred and ninety-one patients with major depressive disorder were enrolled in an open, fixed-dose 8 week fluoxetine trial. Remitters to this acute phase treatment ($N=132$) were randomized to receive either fixed-dose fluoxetine (meds only) or fixed-dose fluoxetine plus cognitive-behavioural therapy (CBT + meds) during a 6-month continuation treatment phase. The Attributional Style Questionnaire (ASQ) was completed by patients at three time points – acute phase baseline, continuation phase baseline and continuation phase endpoint. Analysis of covariance was used to compare continuation phase ASQ composite score changes between groups.

Results. Patients in both treatment groups experienced significant gains in positive attributional style during the acute phase of treatment. Continuation phase ASQ composite change scores differed significantly between treatment groups, with the CBT + meds group maintaining acute phase positive attributional style changes, and the meds only group exhibiting a worsening of attributional style. The two treatment groups did not significantly differ in rates of relapse and final continuation phase visit HAMD-17 scores.

Conclusions. In this sample, the addition of CBT to continuation psychopharmacological treatment was associated with maintenance of acute treatment phase attributional style gains. Further research is needed to evaluate the role of such gains in the long-term course of depressive illness.

INTRODUCTION

Recent research suggests that cognitive behavioural therapy (CBT) may have an important role in the successful long-term treatment of unipolar depression. In particular, recent studies have placed an emphasis on the use of various forms of CBT adapted for the 4–6 month continuation phase of treatment for the prevention of depressive relapse and recurrence (Fava *et al.* 1994, 1996, 1998*a*, *b*, *c*; Jarrett *et al.* 1998; Fava, 1999; Paykel *et al.* 1999; Teasdale *et al.* 2000). In general, these studies suggest that the addition of some form of CBT during the continuation phase of treatment confers a better long-term course than either medication only or

[1] Address for correspondence: Dr Timothy Petersen, Massachusetts General Hospital, 15 Parkman Street, WAC 812, Boston, MA 02114, USA.
(Email: tpetersen@partners.org)

no treatment control conditions. These studies have focused on traditional outcome measures such as relapse and recurrence, defined with use of instruments such as the Hamilton Depression Rating Scale and Clinical Interview for Depression. These instruments measure core symptoms of depression such as depressed mood, lack of interest and neurovegetative disturbance (e.g. insomnia), but do not adequately measure other cognitive constructs that may evidence changes during the course of treatment for depression.

One such cognitive construct is attributional style. The concept of attributional style (AS) stems from the work of Abramson *et al*. (1978), who advanced the reformulated learned helplessness model of depression. This model claims that a tendency to make internal, stable and global explanations for bad/negative events is a risk factor for depression. For example, a patient would be displaying a negative attributional style if, upon meeting up with a friend who acts hostile, the patient attributes the friend's behaviour as due to him/herself, predicts that the hostility will be present in the future and that the hostility will affect all areas of his/her life. The Attributional Style Questionnaire (ASQ) (Peterson *et al*. 1982), a patient self-report instrument, was designed to measure this construct and has been explored in several studies of depressed patients. The ASQ has been found to be a reliable and valid instrument (Peterson *et al*. 1988).

Some studies of depressed out-patients have investigated the effect of CBT on attributional style. In general, these findings suggest that CBT is associated with improved attributional style (Seligman *et al*. 1988; Barber & DeRubeis, 2001), and that improvement in AS is possibly mediated by changes in other symptoms or constructs (Tennen & Herzberger, 1987; Johnson *et al*. 1998; Bruder-Mattson & Hovanitz, 1990). In addition, AS appears to be stable across the life span (Burns & Seligman, 1989; Tiggemann *et al*. 1991) and predicts poor long-term functioning (Peterson *et al*. 1988). However, not all studies have confirmed the relationship between AS and depression (Hargreaves, 1985; Follette & Jacobson, 1987). Nonetheless, AS is one of the most studied psychological constructs in relation to affective disorders, and seems to play an important role in accounting for cognitive vulnerability to depression. In addition, the tendency to make negative attributions for events may contribute to the onset and maintenance of depression.

Given recent research suggesting that the addition of CBT to pharmacotherapy during the continuation and maintenance phases of treatment may better prevent relapse and recurrence than medication does alone, it is particularly important to examine whether this advantage is attributable to changes in cognition. For instance, changes in AS may prove to be important in prevention of relapse and recurrence. Thus, the objective of this study was to examine the role of CBT in maintaining AS changes experienced by patients responding acutely to fluoxetine.

METHOD

Three hundred and ninety-one out-patients (55% female, mean age $39{\cdot}8 \pm 10{\cdot}6$) with major depressive disorder were treated during the acute phase of the protocol with fluoxetine 20 mg/day for 8 weeks after a washout period of 2 weeks for antidepressants (5 weeks for fluoxetine) and 1 week for any other psychotropic medication. No subjects received placebo, and no other psychotropic drugs were allowed. During the acute treatment phase, the HAMD-17 (Hamilton, 1960) was completed at baseline and weeks 2, 4, 6, and 8. The details and results of the acute phase of this study have been described elsewhere (Fava *et al*. 2000).

Subjects entering the acute phase of treatment included both male and female out-patients, ages 18 to 65 who met DSM-III-R criteria for a current episode of major depressive disorder determined by structured clinical interview (Spitzer *et al*. 1989) and who had an initial HAMD-17 score of $\geqslant 16$. Subjects were also required to meet at least one of the following criteria: history of three or more major depressive episodes, with the prior episode no more than 2·5 years before the onset of the current episode; diagnosis of current episode as chronic (onset of continuous depressive symptoms $\geqslant 36$ months prior to study); history of poor inter-episode recovery; or both MDD and dysthymia. Exclusion criteria included failure to respond to fluoxetine 60 mg during any depressive episode, or treatment resistance, defined as failure to

respond during the course of the current episode to at least one adequate antidepressant trial. 'Adequacy' was defined as $\geqslant$6 weeks of treatment with either $\geqslant$20 mg of fluoxetine (or its selective serotonin reuptake inhibitor equivalent), $\geqslant$150 mg of imipramine (or its tricyclic equivalent), or $\geqslant$60 mg of phenelzine (or its monoamine oxidase inhibitor equivalent). Other exclusion criteria included: pregnancy or breastfeeding; serious suicidal risk; serious or unstable medical illness; history of seizure disorder; organic mental disorders; substance use disorders (including alcohol) active within the last year; schizophrenia; delusional disorder; mood congruent or incongruent psychosis; psychotic disorders not elsewhere classified; bipolar disorder; current use of other psychotropic drugs; current psychotherapy; or, clinical or laboratory evidence of hypothyroidism. Subjects provided written informed consent (IRB approved) after the protocol was fully explained and all questions were answered.

'Remission' was defined, at the end of the acute phase, as a HAMD-17 score of $\leqslant$7 for at least 3 weeks (Frank *et al.* 1991). A total of 132 patients who met criteria for remission entered a 28-week single-blind, continuation treatment phase. Patients had their acute fluoxetine dose of 20 mg increased to 40 mg/day at the first continuation visit and were randomized to CBT or medication (meds) management. Thus, there were two continuation phase treatment groups – CBT + meds and meds only. Cognitive therapy was conducted by highly trained doctoral-level psychologists according to a treatment manual adapted from Beck and associates (1979) and Mercier and Leahy (M. A. Mercier and R. L. Leahy, Cognitive therapy of dysthymia: a treatment manual (unpublished manuscript) Columbia-Presbyterian Hospital, New York 1992). This psychotherapy consisted of 12 weekly sessions followed by seven biweekly sessions. Therapy was modified to address residual symptoms specifically and to enhance patient coping skills. Psychopharmacologists were instructed not to make cognitive or behavioural interventions (Pava *et al.* 1994) and followed a standard protocol for medication management visits (Fawcett *et al.* 1987).

Subjects completed the Attributional Style Questionnaire (ASQ) at three time points –acute phase baseline, continuation phase baseline and continuation phase endpoint. Three hundred and twenty-three patients completed the ASQ at acute baseline. Fifty-seven patients in the meds only group and 49 patients in the CBT + meds group completed the ASQ at both continuation baseline and endpoint visits. The ASQ asks subjects to make causal attributions for 12 hypothetical good and bad events. The subject then rates each cause on a seven-point scale for internality, stability and globality. Ratings are summed across the three causal dimensions separately for good and bad events to create a composite positive (CP) and composite negative (CN) explanatory style score, which can range from 3 to 21. An overall score is derived by subtracting CN from CP (the CPCN measure). All subjects were administered the HAMD-17 at each study visit. Patients were assessed by raters blinded to treatment status at monthly intervals for up to 28 weeks following randomization, or until a relapse occurred, defined as meeting criteria for a new episode of major depressive disorder at any continuation visit or scoring $\geqslant$15 on the HAMD-17 at two consecutive visits. Relapse was confirmed by a follow-up visit 1 week later with another clinician, also blind to treatment status.

The primary study endpoint was depressive relapse. Kaplan–Meier survival analysis was utilized for time-to-relapse or study discontinuation, with observations censored after 28 weeks, following completion of this phase of the study. The Mantel–Cox (log-rank) test was employed to compare survival curves between study conditions. Paired *t* tests were used for within group comparisons of ASQ and HAMD-17 changes during the acute treatment phase. Analysis of covariance was used to compare continuation phase ASQ change values between treatment groups, with continuation baseline ASQ scores as covariates. Unpaired *t* tests and chisquare analyses were used to compare demographic and clinical characteristics between groups. All analyses were conducted on an intent-to-treat basis, with last observation carried forward. All tests were two-tailed, with the threshold for statistical significance set at $P<0{\cdot}05$.

RESULTS

The primary outcome (rate of relapse) for the continuation treatment phase of this study is

https://doi.org/10.1017/S0033291703001028 Published online by Cambridge University Press

Table 1. *Demographic and clinical characteristics of randomized patients*

Characteristic	CBT + meds ($N=66$) Mean	(S.D.)	Meds only ($N=66$) Mean	(S.D.)
Age, years	38·8	(10·6)	41·0	(10·0)
Age at first episode, years	22·5	(14·0)	25·3	(13·7)
Duration of current episode, years	2·8	(5·1)	3·7	(6·1)
Prior episodes, *N*	5·6	(9·2)	4·4	(5·9)
HAMD-17				
Acute baseline	19·2	(3·3)	18·3	(2·4)
Cont. baseline	4·7	(2·2)	4·5	(2·1)
Cont. endpoint	4·9	(3·8)	5·5	(3·9)
Change during continuation phase	0·2	(4·0)	1·0	(4·1)
	Frequency	%	Frequency	%
Female*	42	64	30	45
Caucasian	63	95	61	92
Ever married	34	52	34	52
Post-secondary education ⩾4 years	38	58	37	56
Current GAD	7	8	13	20
Current social phobia	16	25	18	27
Current panic disorder	3	5	1	2

* $P<0·05$.

Table 2. *ASQ scores*

	Acute baseline	Continuation baseline	Continuation endpoint
Composite positive (CP)			
Meds + CBT	13·85 ± 2·3	14·74 ± 2·1	14·62 ± 2·5
Meds only	14·35 ± 2·2	15·21 ± 2·4	12·61 ± 1·9
Composite negative (CN)			
Meds + CBT	12·99 ± 3·1	12·76 ± 2·0	12·30 ± 2·1
Meds only	12·45 ± 2·8	12·29 ± 2·1	15·37 ± 1·5
Composite overall (CPCN)			
Meds + CBT	0·86 ± 3·6	1·98 ± 3·1	2·32 ± 3·9
Meds only	1·90 ± 3·8	2·92 ± 3·1	−2·76 ± 2·3
	ASQ continuation change scores		
	CP	CN	CPCN
Meds + CBT	−0·12	−0·46	0·34
Meds only	−2·60	3·08	−5·68

reported elsewhere (Perlis *et al*. 2002), and is summarized as follows. No difference was found in rates of relapse, rates of discontinuation, change in symptoms or change in well being between the two continuation treatment groups. Rates of relapse were 6% (4/66) and 8% (5/66) for the CBT + meds and meds only groups, respectively.

Table 1 depicts demographic and clinical characteristics for the CBT + meds and meds only treatment groups. As can be seen, the only significant difference found between study groups was proportion of females ($P<0·05$). The groups did not differ on any other variables including age, duration of current episode, and number of previous episodes. Table 1 also depicts HAMD-17 scores for each group at acute baseline, continuation baseline, and continuation endpoint as well as change in HAMD-17 scores during the continuation treatment phase. HAMD-17 scores and degree of change did not significantly differ between treatment groups.

Table 2 presents ASQ composite positive, composite negative and composite overall scores for each treatment group at three time points – acute baseline, continuation baseline and continuation endpoint. With respect to differences in ASQ composite scores between treatment groups, CP, CN and CPCN scores were significantly different between groups at continuation phase endpoint ($P=0·004$, $P<0·0001$, and $P<0·0001$), but not significantly different at the other two measurement points (acute phase baseline, continuation phase baseline). With respect to changes in ASQ scores within groups, patients in the meds only group experienced statistically significant gains in CP scores during the acute treatment phase ($P=0·01$), but no significant changes in CN or CPCN scores ($P>0·05$). During the continuation treatment phase, patients in the meds only group experienced statistically significant losses in CP and CPCN scores and gains in CN scores ($P=0·007$, $P<0·0001$, $P=0·0007$). Patients in the CBT + meds group experienced statistically significant gains in CP and CPCN scores during the acute treatment phase ($P=0·003$, $P=0·04$), but no significant change in CN scores ($P>0·05$). During the continuation treatment phase, patients in the CBT + meds group did not experience any significant changes in any of the ASQ composite scores.

Using analyses of covariance, continuation phase ASQ change scores (for all composite indices) were found to be different between treatment groups, while controlling for continuation baseline levels of attributional style (CP, $F=2·628$, $P=0·0330$; CN, $F=3·163$, $P=0·0188$; CPCN, $F=2·823$, $P=0·0211$). As a secondary

https://doi.org/10.1017/S0033291703001028 Published online by Cambridge University Press

analysis, treatment groups were collapsed and patients were then divided into those who did or did not lose acute phase AS gains. We compared relapse rates and HAMD-17 scores (continuation baseline, endpoint, and change scores) between these two groups and did not find any significant differences ($P > 0{\cdot}05$ for all comparisons).

DISCUSSION

Results of the current study suggest that the addition of CBT to continuation medication management, in remitters following acute treatment with the antidepressant fluoxetine, is associated with the maintenance of acute phase attributional style gains. We found, specifically, that all patients exhibited gains in positive attributional style scores during acute phase treatment, but no significant changes in negative attributional style scores. During continuation treatment, the CBT + meds group maintained improvement in positive attributional style scores, while the meds only group demonstrated a decrease in positive and increase in negative attributional style scores.

Remitted depressed subjects showed gains in positive attributional style scores during acute phase treatment with fluoxetine 20 mg alone. A significant increase in positive attributional style scores indicates that remitted depressed outpatients demonstrated greater likelihood of attributing positive events (as presented in the ASQ) to internal, stable and global factors at the end of acute phase treatment than they did at initiation of treatment. Given the improvement in positive attributional style during acute treatment, we were surprised that negative attributional style scores did not also significantly improve. This result is contrary to some studies (Sweeney *et al*. 1986) that suggest negative attributional style is more responsive than positive attributional style to both antidepressant and psychological treatments. It is possible that the baseline level of negative attributional style scores was such that even with remission (determined by HAMD-17), we observed a floor effect (e.g. scores were not particularly unhealthy at baseline, thus leaving little room for improvement). Another possibility is that an 8-week acute phase treatment is not sufficient time to improve negative attributional style. Finally, it could be that high levels of negative AS are more difficult to modify than are low levels of positive AS. However, those patients in the meds only continuation treatment group did not experience a late-onset AS improvement, even with a dose increase. This does not support the idea that more intensive treatment dosing results in change in negative attributional style.

The most intriguing finding of this study occurred during the continuation treatment phase. Specifically, the meds only group demonstrated 'unhealthy' changes in both negative and positive attributional style scores, while the CBT + meds group maintained acute treatment phase AS score gains. There are several possible explanations. It is possible that antidepressants treat both traditional depressive symptoms and corresponding cognitive vulnerabilities (e.g. negative AS), but that CBT is required to consolidate these gains after remission. In other words, perhaps antidepressants are able to initially relieve a broad constellation of depressive phenomena but following acute phase treatment, cognitive vulnerabilities return and require the addition of a treatment (CBT) more specifically targeting these vulnerabilities. A variant of this explanation is that within this sample a placebo effect occurred specific to attributional style, but not to classic depressive symptoms (e.g. mood, sleep, etc.), perhaps reflecting nonspecific supportive factors and expectations inherent in participating in a clinical trial. The placebo effect dissipated after acute phase treatment and patients essentially experienced a 'relapse' into more characteristic attributional style. In either case, our results suggest that the addition of CBT during continuation phase treatment is necessary to maintain and consolidate positive changes.

In a review of recent literature focused on attributional style, several studies bear mention. Johnson *et al*. (1998) examined change in modified forms of the ASQ over the course of 12–14 weeks of antidepressant treatment for 52 psychiatric in-patients. Findings from this study partly support the Needles & Abramson (1990) depression recovery model, which proposes that stable, global attributional style for positive events, along with increased frequency of positive events, predicts decreases in hopelessness which, in turn, decreases depressive symptoms. The Johnson *et al*. trial was an uncontrolled,

open, acute phase study. Our study differs in that we examined out-patients who had all begun antidepressant treatment at the same time point, and underwent continuation phase treatment randomization. Without randomization to two treatment arms (one including CBT and one not), previous authors would be unable to evaluate the contribution of CBT to attributional style changes.

Spangler *et al.* (1993) examined whether hopelessness depression could be identified as a specific subtype (based on congruency between attributional diathesis and negative life stressor) and whether such patients would experience symptoms consistent with this subtype. This research group found mixed evidence for the hopelessness theory. Unfortunately, in the current study we did not assess occurrence of life events/stressors during acute and continuation treatments, and this can be seen as a clear limitation to our findings. Without data on life events/stressors, we cannot account for reduction in depressive symptoms during the acute treatment phase by using Abramson's model. In fact, acute phase improvement in ASQ scores that we observed could be mediated by several other variables, which include life stressors, degree of social support, etc. In addition, recent research (Simons *et al.* 1993) indicates that a negative attributional style may contribute to the generation of negative life events. Without these data we are unable to examine this relationship as well. Testing more complex models, by examining AS in combination with other variables such as life events, are needed to elucidate the relative contribution of AS to treatment outcome.

In this study there was no difference in relapse rates or HAMD-17 scores between the CBT + medication and medication alone groups despite the significantly divergent trajectories of ASQ scores. Possible reasons for the lack of effect of CBT on relapse have been discussed elsewhere (Perlis *et al.* 2002), including the increase in fluoxetine dose to 40 mg following remission, which may have further reduced the vulnerability to relapse. Another possible explanation for lack of difference in relapse rates is that CBT may effect change in attributional style without having any meaningful effect on long-term clinical consequences. Nevertheless, the results of this study suggest that assessment of psychological constructs such as attributional style may provide informative and, perhaps, more sensitive measure of the impact of continuation treatments than traditional outcome measures. Further studies delineating the relevance of maintaining gains in these psychological constructs to quality of life will be of much interest.

This study was supported, in part, by a grant from Eli Lilly and Company.

REFERENCES

Abramson, L. Y., Seligman, M. E. P. & Teasdale, J. D. (1978). Learned helplessness in humans: critique and reformulation. *Journal of Abnormal Psychology* **87**, 49–74.

Barber, J. P. & DeRubeis, R. J. (2001). Change in compensatory skills in cognitive therapy for depression. *Journal of Psychotherapy Practice & Research* **10**, 8–13.

Beck, A. T., Rush, A. J., Shaw, B. F. & Emery, G. (1979). *Cognitive Therapy of Depression*. Guilford Press: New York.

Burns, M. O. & Seligman, M. E. P. (1989). Explanatory style across the life span: evidence for stability over 25 years. *Journal of Personality and Social Psychology* **56**, 471–477.

Bruder-Mattson, S. F. & Hovanitz, C. A. (1990). Coping and attributional styles as predictors of depression. *Journal of Clinical Psychology* **46**, 557–665.

Fava, G. A. (1999). Subclinical symptoms in mood disorders. Pathological and therapeutic implications. *Psychological Medicine* **29**, 47–61.

Fava, G. A., Grandi, S., Zielezny, M., Canestrari, R. & Morphy, M. A. (1994). Cognitive behavioral treatment of residual symptoms in primary major depressive disorder. *American Journal of Psychiatry* **151**, 1295–1299.

Fava, G. A., Grandi, S., Zielezny, M., Rafanelli, C. & Canestrari, R. (1996). Four outcome for cognitive behavioral treatment of residual symptoms in major depression. *American Journal of Psychiatry* **153**, 945–947.

Fava, G. A., Rafanelli, C., Cazzaro, M., Conti, S. & Grandi, S. (1998*a*). Well-being therapy. *Psychological Medicine* **28**, 475–480.

Fava, G. A., Rafanelli, C., Grandi, S., Canestrari, R. & Morphy, M. A. (1998*b*). Six year outcome for cognitive behavioral treatment of residual symptoms in major depression. *American Journal of Psychiatry* **155**, 1443–1445.

Fava, G. A., Rafanelli, C., Grandi, S., Conti, S. & Belluardo, P. (1998*c*). Prevention of recurrent depression with cognitive behavioral therapy. *Archives of General Psychiatry* **55**, 816–820.

Fava, M., Alpert, J. E. & Nierenberg, A. A. (2000). Double-blind study of high dose fluoxetine versus lithium or desipramine augmentation of fluoxetine in partial and nonresponders to fluoxetine. Report present at the 153rd Annual Meeting of the American Psychiatric Association, Chicago, IL, May 2000.

Fawcett, J., Epstein, P., Fiester, S. J., Elkin, I. & Autry, J. H. (1987). Clinical management: imipramine/placebo administration manual. *Psychopharmacological Bulletin* **23**, 309–323.

Follette, V. M. & Jacobson, N. S. (1987). Importance of attributions as a predictor of how people cope with failure. *Journal of Personality and Social Psychology* **52**, 1205–1211.

Frank, E., Prien, R. F., Jarrett, R. B., Keller, M. D., Kupfer, D. J., Lavori, P. W., Rush, A. J. & Weissman, M. M. (1991). Conceptualization and rationale for consensus definitions of terms in major depressive disorder: remission, recovery, relapse, and recurrence. *Archives of General Psychiatry* **48**, 851–855.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry* **23**, 56–62.

Hargreaves, I. R. (1985). Attributional style and depression. *British Journal of Clinical Psychology* **24**, 65–66.

Jarrett, R. B., Basco, M. R., Riser, R., Ramanan, J., Marwill, M. & Rush, A. J. (1998). Is there a role for continuation phase cognitive therapy for depressed outpatients? *Journal of Consulting Clinical Psychology* **66**, 1036–1040.

Johnson, J. G., Han, Y. S., Douglas, C. J., Johannet, C. M. & Russell, T. (1998). Attributions for positive life events predict recovery from depression among psychiatric inpatients: an investigation of the Needles and Abramson model of recovery from depression. *Journal of Consulting and Clinical Psychology* **66**, 369–376.

Needles, D. J. & Abramson, L. Y. (1990). Positive life events, attributional style, and hopefulness: testing a model of recovery from depression. *Journal of Abnormal Psychology* **99**, 156–165.

Pava, J. A., Fava, M. & Levenson, J. A. (1994). Integrating cognitive therapy and pharmacotherapy in the treatment and prophylaxis of depression: a novel approach. *Psychotherapy & Psychosomatics* **61**, 211–219.

Paykel, E. S., Scott, J., Teasdale, J. D., Johnson, A. L., Garlan, A., Moore, R., Jenaway, A., Cornwall, P. L., Hayhurst, H., Abbott, R. & Pope, M. (1999). Prevention of relapse in residual depression by cognitive therapy. *Archives General Psychiatry* **56**, 829–835.

Perlis, R. H., Nierenberg, A. A., Alpert, J. E., Pava, J., Matthews, J. D., Buchin, J., Sickinger, A. H. & Fava, M. (2002). Effects of adding cognitive therapy to fluoxetine dose increase on risk of relapse and residual depressive symptoms in continuation treatment of major depressive disorder. *Journal of Clinical Psychopharmacology* **22**, 474–480.

Peterson, C., Seligman, M. E. P. & Valliant, G. E. (1988). Pessimistic explanatory style is a risk factor for physical illness: a thirty-five year longitudinal study. *Journal of Personality of Social Psychology* **55**, 23–27.

Peterson, C., Semmel, A., von Bayer, C., Abramson, L. Y., Metalsky, G. I. & Seligman, M. E. P. (1982). The attributional style questionnaire. *Cognitive Therapy and Research* **6**, 287–299.

Seligman, M. E. P., Castellon, C., Cacciola, J., Schulman, P., Luborsky, L., Ollove, M. & Downing, R. (1988). Explanatory style change during cognitive therapy for unipolar depression. *Journal of Abnormal Psychology* **97**, 13–18.

Simons, A. D., Angell, K. L., Monroe, S. M. & Thase, M. E. (1993). Cognition and life stress in depression: cognitive factors and the definition, rating, and generation of negative life events. *Journal of Abnormal Psychology* **102**, 584–591.

Spangler, D. L., Simons, A. D., Monroe, S. M. & Thase, M. E. (1993). Evaluating the hopelessness model of depression: diathesis-stress and symptom components. *Journal of Abnormal Psychology* **102**, 592–600.

Spitzer, R. L., Williams, J. B. W. & Gibbon, M. B. (1989). *Structured clinical interview for DSM-III-R – Patient Version* (*SCID-P*). Biometrics Research Department, New York State Psychiatric Institute: New York.

Sweeney, P. D., Anderson, K. & Bailey, S. (1986). Attributional style in depression: a meta-analytic review. *Journal of Personality and Social Psychology* **50**, 974–991.

Teasdale, J. D., Segal, Z., Williams, J. M. G., Ridgeway, V. A., Soulsby, J. M. & Lau, M. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting Clinical Psychology* **68**, 615–623.

Tennen, H. & Herzberger, S. (1987). Depression, self-esteem, and the absence of self-protective attributional biases. *Journal of Personality and Social Psychology* **52**, 72–80.

Tiggemann, M., Winefield, A. H., Winefield, H. R. & Goldney, R. D. (1991). The stability of attributional style and its relation to psychologic distress. *British Journal of Clinical Psychology* **30**, 247–255.