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Explanatory Style Change During Cognitive Therapy for Unipolar Depression

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We administered the Attributional Style Questionnaire to 39 unipolar depressed patients at the beginning and end of cognitive therapy and at one-year follow-up, and we administered it to 12 bipolar patients during a depressed episode. A pessimistic explanatory style for bad events correlated with severity of depression for unipolars at cognitive therapy intake ($r = .56$, $p < .0002$), termination ($r = .57$, $p < .0008$), and one-year follow-up ($r = .64$, $p < .0005$) and among the bipolars ($r = .63$, $p < .03$). Explanatory style and depressive symptoms significantly improved by the end of cognitive therapy and remained improved at one-year follow-up. For the unipolars in cognitive therapy, explanatory style change from intake to termination correlated with change in depressive symptoms from intake to termination ($r = .65$, $p < .0001$). These results suggest that explanatory style may be one of the mechanisms of change for unipolar depressive patients undergoing cognitive therapy.

The reformulation of the learned helplessness model of depression claims that a tendency to make internal, stable, and global explanations for bad events is a risk factor for depression (Abramson, Seligman, & Teasdale, 1978). Although this model has been explored in a variety of populations, its clinical relevance is best tested among carefully diagnosed depressed patients (Peterson & Seligman, 1984). Sweeney, Anderson, and Bailey (1986), in a meta-analysis of 104 studies of explanatory style and depression, cited 12 studies that used psychiatric patients. These patient studies, taken together, show the predicted correlation of explanatory style and depressive symptoms (e.g. Eaves & Rush, 1984; Hamilton & Abramson, 1983; Persons & Rao, 1985; Raps, Peterson, Reinhard, Abramson, & Seligman, 1982).

In this study we replicate and extend the aforementioned patient studies to both carefully diagnosed unipolar and bipolar patients suffering a depressive episode. These two patient groups are also contrasted with a matched nonpatient sample. In addition, the unipolars underwent cognitive therapy, and we report here the association of explanatory style and depression at intake, termination, and follow-up one year after the completion of therapy.

Method

Subjects and Procedure

The sample consisted of 39 depressed unipolar patients undergoing outpatient therapy at the Center for Cognitive Therapy at the University of Pennsylvania and 12 depressed bipolar patients experiencing a depressive episode. We obtained the bipolars through the University of Pennsylvania inpatient and outpatient units. Eight of the 12 bipolars were on psychotropic medication (including lithium), and at intake, 16 of the 39 unipolars were receiving psychotropic medication (antidepressants and/or anxiolytics).

Prior to their second therapy session or second week of hospitalization, we gave each patient a Schedule for Affective Disorders and Schizophrenia (SADS; Endicott & Spitzer, 1978) interview as well as our test battery. The SADS interviews were conducted by master's level clinicians with previous psychodiagnostic experience. All were trained in conjunction with and following the training procedures of the Endicott SADS group. A sample of taped interviews was reviewed and calibrated by Endicott.

The criteria for inclusion as a unipolar depressed patient were as follows: (a) a diagnosis of major depressive disorder according to the Research Diagnostic Criteria (RDC; Spitzer, Endicott, & Robins, 1978); (b) no diagnosis of bipolar depression, cyclothymia, or schizophrenia; and (c) a score of 10 or more on the short form of the Beck Depression Inventory (BDI; Beck & Beck, 1972). Of the 39 unipolars, 25 had a primary depression and 14 had a secondary depression. Because we found no significant difference on any of our variables at any time for this distinction, we have collapsed across the primary–secondary distinction throughout. The criteria for inclusion as a bipolar depressive were a current episode of major depression plus a past episode of mania or hypomania.

The unipolars consisted of 27 women and 12 men, with a mean age of 36 years. The bipolars consisted of 8 men and 4 women, with a mean age of 38 years. Table 1 presents the age, sex, education, marital status, and employment of this sample and of a nonpatient control group.

In addition to looking at continuous differences in explanatory style within both groups of depressed patients, we also compared a group of 10 nonpatient controls recruited through the Philadelphia media to take our diagnostic interview and test battery. Six of the nonpatient controls were women and 4 were men. They had a mean age of 37 years; their education, marital status, and employment is shown in Table 1. This is one of a large number of nonpatient control groups we have tested over the years. Because they were matched for age and education at time of testing, and their explanatory style and depression scores were

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Table 1
Demographic Breakdown of 39 Unipolar Depressed Patients, 12 Bipolar Depressed Patients, and 10 Nonpatient Controls

Variable	Unipolars		Controls		Bipolars	
	n	%	*n*	%	*n*	%
Education						
Non-high-school graduate	2	5	0	0	0	0
High school graduate	4	10	0	0	2	17
Some college	10	26	2	20	4	33
College graduate	12	31	3	30	3	25
Graduate degree	11	28	1	10	3	25
No information	0	0	4	40	0	0
Marital status						
Never married	16	41	3	30	5	42
Currently married	14	36	4	40	3	25
Divorced/separated/ widowed	9	23	3	30	4	33
No information	0	0	0	0	0	0
Race						
White	33	85	10	100	9	75
Black	4	10	0	0	3	25
Other	2	5	0	0	0	0
No information	0	0	0	0	0	0
Employment						
Employed	17	44	7	70	9	75
Unemployed	14	36	1	10	2	17
Retired	1	3	0	0	0	0
Student	7	18	2	20	1	8
No information	0	0	0	0	0	0
Sex						
Female	27	69	6	60	4	33
Male	12	31	4	40	8	67
Mean age	36		37		38	

Note. Percentages not adding to 100% are due to rounding.

quite typical of controls, we include these groups here as typical reference points for ASQ and BDI scores. They are not intended as an extensive, nonhospitalized control because most of our hypothesis testing in this study requires within-group and cross-time comparisons rather than static between-groups comparisons.

The unipolar depressed patients have been followed since the start of cognitive therapy. Of the 39 cognitive therapy patients, 31 had terminated and agreed to be reinterviewed at termination of therapy. They received cognitive therapy approximately once per week during about a 6-month period for a mean of 22.5 sessions. Therapy was carried out in the manner outlined by Beck, Rush, Shaw, and Emery (1979).

Each patient who terminated was given the SADS-C (Change) interview and our test battery within 1 month of termination. One year following termination, each patient was again given the SADS and the test battery. We report here the results for all 39 unipolar and 12 bipolar patients on intake, the termination results for 31 of the 39 cognitive therapy patients for whom we have explanatory style and BDI information, and the results for 29 patients who reached one-year follow-up (we have explanatory style data for only 26 of the 29). Because the present research is part of an ongoing project, we will eventually report on 2- to 4-year follow-up of the present cohort. Because the effects are highly consistent and statistically robust at this stage, it seems useful to disseminate the termination and follow-up results now.

Test Battery

BDI (short form). At each session, patients filled out the short form of the BDI. This is a 13-item self-report instrument designed to assess the severity of depressive symptoms. Scores can range from 0 to 39, with a score of 10 or above corresponding roughly to a clinically significant depression.

Demographic Questionnaire. The patients filled out a demographic questionnaire, the results of which are summarized in Table 1.

Attributional Style Questionnaire (ASQ). The patients completed the ASQ, which asks subjects to make causal attributions for 12 hypothetical good and bad events. The subject then rates each cause on a 7-point scale for internality, stability, and globality (Peterson & Seligman, 1984; Peterson et al., 1982; Seligman, Abramson, Semmel, & von Baeyer, 1979). 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). We will report statistics primarily for the CN measure, because this has consistently been the most valid correlate and predictor of depression (Peterson & Seligman, 1984).

SADS. The SADS assesses a number of dimensions of psychopathology for the worst week of the current episode of psychiatric illness and for the week prior to evaluation. The SADS-C is designed to measure change and includes a subset of these dimensions for the week prior to evaluation. We used two summary scores from these instruments. First, we used an Anxiety Scale comprised of three 6-point scales indicating severity of anxiety (psychic, somatic, and phobic) for the week prior to evaluation. We dichotomized patients in the unipolar group to compare high- versus low-anxiety patients. Second, we extracted a Hamilton Depression Rating Scale (Ex-Ham) from the SADS interviews to create a clinician rated measure of depression. The Ex-Ham includes the following items: depressive mood, guilt, suicidal tendencies, somatic anxiety, psychic anxiety, obsessions–compulsions, insomnia, appetite loss, weight loss, somatic preoccupation, depersonalization, agitation, psychomotor retardation, worse in morning, worse in evening, suspiciousness, and impairment in functioning.

Health Sickness Rating Scale (HSRS). The HSRS is a 100-point scale designed to assess overall mental health. A single number integrates seven criteria of mental health. The clinician rates the patient on the basis of current status at the time of evaluation. The HSRS has been reported to be reliable and correlate with severity of depression, adequacy of personality functioning, and treatment outcome (Luborsky & Bachrach, 1974).

Global Assessment Scale (GAS). The GAS is a stripped-down variant of the HSRS, used to rate overall functioning on a 100-point scale (Endicott, Spitzer, Fleiss, & Cohen, 1976).

Results

The overall results are easily summarized. Unipolar and bipolar depressives had both a pessimistic explanatory style and a more pessimistic style than did nonpatient controls. The more severe the depression, the worse the explanatory style. For the depressive unipolars, explanatory style and severity of depression improved during cognitive therapy and remained improved at one-year follow-up. Explanatory style and depressive symptoms moved in lockstep from intake to termination to follow-up. As explanatory style became more optimistic during therapy, patients became less depressed and remained less depressed at therapy termination and at one-year follow-up. Finally, recovered patients with an optimistic explanatory style at

Table 2
Explanatory Style and BDI Means and Standard Deviations

Group	BDI	CN	CP	CPCN
		Intake		
Unipolars (n = 39)				
M	15.8	14.4	14.4	.1
SD	4.9	2.1	2.1	3.2
Bipolars (n = 22)				
M	15.8	13.5	14.4	.9
SD	6.5	2.5	1.1	2.5
Controls (n = 10)				
M	4.4	12.4	16.3	3.9
SD	3.2	2.2	1.8	3.0
		Termination		
Unipolars (n = 31)				
M	5.8	12.9	15.8	2.9
SD	4.6	2.0	1.9	2.7
		Follow-up		
Unipolars (n = 26)				
M	6.7	13.4	15.6	2.3
SD	5.2	2.2	2.0	3.2

Note. BDI = the short form of the Beck Depression Inventory. CP = composite positive (explanatory style for good events). CN = composite negative (explanatory style for bad events). CPCN = CP − CN.

termination were less depressed one year later than were those with a pessimistic style.

Reliability

The reliability of the ASQ composites, estimated by Cronbach's (1951) alpha, was satisfactory. Alphas at intake, termination, and follow-up were .73, .73, and .76 for CN and .74, .72, and .77 for CP, respectively.

Cross-Sectional Relation of Explanatory Style and Depression

For the unipolar depressed patients, we found a significant correlation between the explanatory style for bad events (CN) and severity of depression, as measured by the BDI, at intake (r = .56, p < .0002, n = 39), termination (r = .57, p < .0008, n = 31), and one-year follow-up (r = .64, p < .0005, n = 26). Broken down by individual dimensions, internal (r= .36, p < .02), stable (r = .43, p < .006), and global (r = .52, p < .0007) all correlated with depression at intake. At termination, internal (r = .39, p < .03), stable (r = .41, p < .02), and global (r = .49, p < .005) dimensions and, at follow-up, internal (r = .41, p < .04), stable (r = .44, p < .02), and global (r = .63, p < .0005) dimensions all correlated with BDI. See Tables 2 and 3 for results.

We also found a strong association between CN and severity of depression for the bipolar patients (r = .63, p < .03, n = 12). The internal dimension did not correlate significantly with depression (r = .32), but the stable (r = .73, p < .007) and global (r = .62, p < .03) dimensions did. The 10 nonpatient controls showed correlations in the predicted direction, but they were nonsignificant, probably because of the restricted range of BDI scores and small sample size (see Sweeney et al., 1986, for a meta-analysis of 76 studies of nonpatient samples, which together strongly suggest that when the sample is large enough, the correlation is robust).

More important, the more the unipolar patients' explanatory style improved in the course of cognitive therapy, the more their depression improved. The correlation between CN change and BDI change from intake to termination was .65 (p < .0001, n = 31). This correlation was not an artifact of self-report because change in the clinicians' measures of depression, extracted from the SADS (Ex-Ham), also correlated with CN change (r = .52, p < .003, n = 30).

Explanatory Style and Clinician-Rated Measures of Depression

So far, with the exception of the last statistic, we have presented correlations between two self-report measures, the BDI and the ASQ. We also measured depression by clinicians' ratings using three rating scales—the Hamilton Depression Scale extracted from the SADS interview (Ex-Ham), the HSRS, and the GAS. The clinician was blind to the ASQ scores. Each of these measures tended to correlate with explanatory style but not to the same robust and consistent degree that the BDI correlated with explanatory style. So, for example, among the unipolar depressed patients at intake, the Ex-Ham measure correlated .40 with CN (p < .01, n = 39), but the GAS and HSRS showed no correlation. At termination, CN correlated with the Ex-Ham at .40 (p < .03, n = 30), the GAS at −.38 (p < .04, n = 30), and the HSRS at −.30 (p < .11, n = 30). At follow-up, CN correlated .48 with the Ex-Ham (p < .02, n = 25), −.60 with the GAS (p < .002, n = 25), and −.56 with the HSRS (p < .004, n = 25). Overall then, we conclude that clinicans' ratings of severity of depression corresponded roughly with explanatory style in the same way that patients' self-reports of depressive symptoms did (see Table 3 for results).

Comparisons Among Diagnostic Groups

Does explanatory style differ among diagnostic groups? There are several relevant comparisons: unipolar depressives at intake versus nonpatient controls, unipolar depressives at intake versus bipolars during a depressed state, and bipolars versus nonpatient controls. There are two other comparisons worth making within the unipolar depressives group: those with versus those without melancholia (endogenous vs. nonendogenous) and those with versus those without anxiety.

Our data have so far shown that the more severe the symptoms of depression among unipolar depressives, the worse the explanatory style. It is useful to bolster this finding by comparing unipolars with nonpatient controls. Toward this end we applied our procedures (including the SADS interview) to a group of 10 controls matched for sex, age, and education to our patients. None of the nonpatient controls was given a current RDC diagnosis of an affective disorder. A t-test analysis revealed

Table 3
Explanatory Style Correlations With Depression

Group	CN		CP		CPCN	
	r	*p*	*r*	*p*	*r*	*p*
Intake						
Unipolars (n = 39)						
BDI	.56	.0002	−.18	*ns*	−.49	.002
Ex-Ham	.40	.01	.12	*ns*	−.19	*ns*
GAS	−.04	*ns*	−.20	*ns*	−.11	*ns*
HSRS	−.04	*ns*	−.02	*ns*	.02	*ns*
Bipolars (n = 12)						
BDI	.63	.03	.31	*ns*	−.49	.11
Ex-Ham	.06	*ns*	.14	*ns*	0	*ns*
GAS	.59	.04	.16	*ns*	−.53	.08
HSRS	.62	.03	.43	*ns*	−.44	*ns*
Controls (n = 10)						
BDI	.41	*ns*	−.12	*ns*	−.37	*ns*
Ex-Ham	.19	*ns*	−.06	*ns*	−.17	*ns*
GAS	−.50	*ns*	−.06	*ns*	.33	*ns*
HSRS	−.34	*ns*	0	*ns*	.25	*ns*
Termination						
Unipolars (n = 31)						
BDI	.57	.0008	.15	*ns*	−.32	.08
Ex-Ham	.40	.03	.09	*ns*	−.24	*ns*
GAS	−.38	.04	.12	*ns*	.36	.05
HSRS	−.30	.11	.06	*ns*	.26	*ns*
Follow-up						
Unipolars (n = 26)						
BDI	.64	.0005	−.25	*ns*	−.60	.001
Ex-Ham	.48	.02	−.17	*ns*	−.44	.03
GAS	−.60	.002	.47	.02	.72	.0001
HSRS	−.56	.004	.39	.05	.64	.0006

Note. BDI = the short form of the Beck Depression Inventory. Ex-Ham = Hamilton Depression Rating Scale. GAS = Global Assessment Scale. HSRS = Health Sickness Rating Scale. CP = composite positive (explanatory style for good events). CN = composite negative (explanatory style for bad events). CPCN = CP minus CN.

that unipolars had a significantly worse CN at intake than did nonpatient controls, $t(47) = 2.6$, $p < .01$, as well as worse BDI scores, $t(47) = 7.0$, $p < .0001$. (All t-test analyses were two-tailed.) See Table 2 for means and standard deviations.

The depression and explanatory style of the bipolars did not differ significantly from the depression and explanatory style of the unipolar patients. The differences in depression and explanatory style between the unipolars and controls, however, were statistically more consistent than those between the bipolars and controls, probably because of the larger size of the unipolar group. A t-test analysis revealed that the bipolars had significantly worse BDI scores than did the controls, $t(18.3) = 5.5$, $p < .0001$, and a worse explanatory style as measured by CPCN, $t(20) = 2.5$, $p < .02$, though the CN measure was not significant but in the right direction, $t(20) = 1.1$. See Table 2 for means and standard deviations.

We divided the 39 unipolar patients into endogenous (n = 16), probably endogenous (n = 16), and nonendogenous (n = 7) groups by using RDC criteria at intake. We found no differences in explanatory style or BDI among these subgroups. We then collapsed the probably endogenous patients with either of the other two groups and again found no differences.

We divided the 39 unipolar patients at the mean of the Anxiety Scale derived from the SADS interview at intake. We found no difference in BDI score, but we did find a difference in the Ex-Ham, $t(25.5) = 5.0$, $p < .0001$, and CN, $t(37) = 2.4$, $p < .02$. The anxious unipolars had worse explanatory style than did the nonanxious depressives. They were also more severely depressed according to the SADS criteria.

Changes in Depression and Explanatory Style Over Time

We had measures of depression, assessed by the BDI, for 31 of the 39 unipolars at termination of cognitive therapy. A paired comparison revealed that depressive symptoms were significantly lowered by the end of therapy (mean change = 9.8), $t(5.3) = 10.3$, $p < .0001$. All 31 patients were less depressed at termination than at intake.

We had measures of explanatory style for 31 of the 39 unipolars at the end of cognitive therapy (mean CN change = 1.40), $t(2.5) = 3.1$, $p < .004$. Note that explanatory style at intake did not predict response to therapy, by itself or after partialing out intake BDI.

BDI scores were very stable from termination to follow-up ($r = .62$, $p < .0003$, $n = 29$), implying lasting benefits of cognitive therapy for unipolar depressives. The explanatory style for bad events was also highly stable over the one-year period from termination to follow-up ($r = .65$, $p < .0003$, $n = 26$).

Explanatory Style Predictions of Depression

Explanatory style showed nonsignificant trends predicting later depression. CN change from intake to termination marginally predicted BDI at follow-up ($r = -.30$, $p < .12$, $n = 29$). This was not significant, but it is in the right direction. Also, the better the explanatory style at the end of therapy, the less depressed individuals seemed to be at one-year follow-up. CN at termination nonsignificantly predicted BDI at follow-up, partialing out BDI at termination ($F = 2.3$, $p < .14$, $n = 29$). CN at termination correlated significantly with severity of depression at follow-up ($r = .54$, $p < .002$, $n = 29$). Even though these results are nonsignificant trends, we report them because of the importance of predicting relapse. These results therefore need to be investigated further.

Discussion

Five important findings emerged from this study. The first major finding was that the more internal, stable, and global the explanations were for bad events, the more severe were the depressive symptoms for unipolar patients at therapy intake, termination, and one-year follow-up and for bipolar patients during a depressive episode. In addition, unipolars and bipolars had a more pessimistic explanatory style than did the nonpatient

control group. These findings replicate the 12 patient studies that Sweeney et al. (1986) meta-analyzed.

The second major finding was that explanatory style and severity of depression changed in lockstep with each other—the more explanatory style improved during cognitive therapy, the more depressive symptoms disappeared. This raises the possibility that explanatory style may be the active therapeutic mechanism by which cognitive therapy alleviates unipolar depression (Elkin, Imber, Sotsky, & Watkins, 1986; Seligman, 1980). It may be the case, however, that changes in explanatory style are a consequence of relief from depression or that some third variable, like change in expectations, changes both.

The third finding concerned changes in explanatory style and depression over time. Both explanatory style and depressive symptoms improved dramatically during the course of cognitive therapy for unipolars. Furthermore, the effects were long lasting—explanatory style and depressive symptoms were highly stable in the one year following the end of therapy. It is possible that these changes were wrought by cognitive therapy. Because an untreated control group was not used, however, and the time periods were substantial, it is possible that much of this change was due to a spontaneous recovery of depression. Whatever the cause, the changes in depression and explanatory style were stable.

The fourth finding was that there were no differences in explanatory style for unipolars versus bipolars or for endogenous versus nonendogenous unipolars. This finding seems neutral to the reformulated learned helplessness model. Both endogenous and bipolar depression probably have a considerable genetic diathesis. It is possible that depressive explanatory style is heritable as well as acquired, although this is untested. Explanatory style, however, did discriminate between the anxious and nonanxious unipolars. The anxious unipolars had a more pessimistic style and more severe depressive symptoms. This difference in explanatory style may reflect a more severe depression, rather than an effect of anxiety.

The fifth finding was that explanatory style change during therapy marginally predicted depression at one-year follow-up. Furthermore, explanatory style at termination marginally predicted depression at follow-up, partialing out the effects of depression at termination. This fifth finding, if replicated, implies that we may be able to identify in advance those patients who are at greatest risk for relapse even after successful therapy for depression. There are several strategies for dealing with such high-risk individuals. Such individuals could be more carefully monitored following the end of therapy or given intermittent booster sessions, or perhaps therapy should not be terminated until both the depressive symptoms have remitted and explanatory style has reached a more optimistic level.

How do these findings bear on the claim that explanatory style is a trait (Peterson & Seligman, 1984)? They provide mixed support. On the one hand, explanatory style is quite stable in the absence of therapy. During the one year from termination to follow-up, the correlation of CN was .65. On the other hand, explanatory style changed substantially in therapy and in lockstep with change in depression. Traits may be changeable in therapy, and this is a major aim of extended psychotherapy. At this point, the most we can say is that cognitive therapy (and possibly the onset of depression) change explanatory style, but explanatory style appears traitlike in the absence of those factors.

These five findings are essentially correlational, yet the reformulated learned helplessness model of depression is a causal theory. It claims that an internal, stable, and global explanatory style for bad events precedes, and is a risk factor for, later depression. There are four noncausal interpretations that are compatible with this correlational data. First, depression may cause a bad explanatory style, and relief from depression enhances explanatory style. Second, some third variable, like catecholamine changes, causes both a bad style and depression. Third, the whole business may be a tautology. Pessimistic explanations may be a part of how we diagnose depression. The fourth interpretation is that a bad explanatory style is a premorbid symptom of depression that later blossoms into full-blown depression. This last hypothesis is hard to differentiate from our risk-factor interpretation but is a variant of the third variable hypothesis, which claims that another factor causes both the premorbid explanatory style and depression. These alternatives are not tested here, but Peterson and Seligman (1984) and Brewin (1985) reviewed the evidence suggesting a causal role for explanatory style.

One strategy for testing the causal role is to change explanatory style in therapy and see if depression changes as a consequence. Seligman (1980) proposed that the mechanism of change in cognitive therapy was change in explanatory style. The observations in this study, however, are not fine-grained enough to test this definitively. It is entirely possible that depressive symptoms remitted before explanatory style improved during therapy and that explanatory style change was a result and not a cause of successful cognitive therapy. To test this, it would be necessary to measure session by session changes in depressive symptoms, explanatory style, and amount of therapy actually delivered. If change in explanatory style occurred in sessions in which much therapy was delivered and was followed by changes in depressive symptoms, a causal role for changing explanatory style in alleviating depression would be confirmed. We are currently engaged in such a study.

In conclusion, we have found that unipolar and bipolar depressives share an insidious way of explaining bad events that distinguishes them from less depressed patients and nondepressed individuals. During cognitive therapy, explanatory style and depression changed in lockstep from intake to termination, and as explanatory style became more optimistic, the patient became less depressed. Finally, recovered patients with an optimistic style at termination may be less likely to be depressed one year later than those with a pessimistic style. We suggest that habitually explaining the causes of bad events as internal, stable, and global puts people at risk for depression and that undoing this pessimistic explanatory style may be a curative element in the cognitive therapy of depression.

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Call for Nominations: *Journal of Experimental Psychology: General*

The Publications and Communications Board has opened nominations for the editorship of the *Journal of Experimental Psychology: General* for the years 1990–1995. Sam Glucksberg is the incumbent editor. Candidates must be members of APA and should be available to start receiving manuscripts in early 1989 to prepare for issues published in 1990. Please note that the P & C Board encourages more participation by women and ethnic minority men and women in the publication process, and would particularly welcome such nominees. To nominate candidates, prepare a statement of one page or less in support of each candidate. Submit nominations no later than February 15, 1988 to

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Other members of the search committee are James J. Jenkins, Jean Mandler, J. E. R. Staddon, and Saul Sternberg.