

BRIEF REPORTS

The Prevalence and Impact of Large Sudden Improvements During Adolescent Therapy for Depression: A Comparison Across Cognitive–Behavioral, Family, and Supportive Therapy

Scott T. Gaynor, V. Robin Weersing, David J. Kolko, Boris Birmaher, Jungeun Heo, and David A. Brent
University of Pittsburgh School of Medicine

This study assessed the treatment specificity and impact on outcome of large, abrupt symptomatic improvements occurring prior to and during cognitive–behavioral, family, and supportive therapy. Eighty-seven depressed adolescents receiving at least 8 therapy sessions were included. Abrupt large decreases in depressive symptoms were identified by changes in weekly Beck Depression Inventory scores. Overall, 28% experienced a pretreatment gain and 39% a sudden within-treatment gain. Both types of gains were associated with superior outcome on self-report and interviewer ratings of depression. Among those participants failing to experience a pretreatment or sudden within-treatment gain, cognitive–behavioral therapy produced the superior outcomes. These findings suggest pretreatment and sudden within-treatment gains are important therapeutic events worthy of further investigation.

Meta-analyses support the efficacy of cognitive–behavioral therapy (CBT) in the treatment of depressed adults (Barlow, 1994) and adolescents (Lewinsohn & Clarke, 1999). Despite these positive findings, there is little information on whether CBT works through specific cognitive mechanisms (Kopta, Lueger, Saunders, & Howard, 1999; Whisman, 1993). For example, we have found that depressed teens treated with CBT demonstrated greater change in both depressive symptoms and cognitive distortions than those treated with systemic behavioral family therapy (SBFT) and nondirective supportive therapy (NST). However, changes in depressive symptoms were not mediated by changes in cognitive distortions, and alternative cognitive measures failed to reveal treatment specific effects, leaving the mechanisms of CBT action unclear (Kolko, Brent, Baugher, Bridge, & Birmaher, 2000).

Scott T. Gaynor, V. Robin Weersing, David J. Kolko, Boris Birmaher, Jungeun Heo, and David A. Brent, Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine.

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Correspondence concerning this article should be addressed to Scott T. Gaynor, who is now at the Department of Psychology, Western Michigan University, 1903 West Michigan Avenue, Kalamazoo, Michigan 49008-5439 or David A. Brent, Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, 3811 O'Hara Street, Suite 112, Pittsburgh, Pennsylvania 15213. E-mail: scott.gaynor@wmich.edu

Evaluating patterns of symptomatic change occurring over the course of treatment may also reveal treatment specific effects, thereby pointing toward mechanisms of action. Recent time-course investigations have explored the amount of early response to depression treatment and the impact of sudden symptomatic gains during treatment. The relevant findings are briefly reviewed later in the article.

Across multiple studies it appears that approximately two thirds of change in CBT for adult depression occurs within the first 4 weeks (Ilardi & Craighead, 1994). Data from multiple outcome studies with depressed adolescents have not yet become available. However, we found that across CBT, NST, and SBFT approximately one third of depressed adolescents experienced a greater than 50% change in Beck Depression Inventory (BDI; Beck, Steer, & Garbin, 1988) score prior to the second therapy session. In addition, these rapid responders experienced superior outcome regardless of treatment condition (Renaud et al., 1998). Among adult participants, Fennell and Teasdale (1987) also have reported equivalent pretreatment to Week 1 rates of rapid response in CBT and treatment as usual (TAU). However, by Week 2 of treatment (after three sessions) CBT rapid responders showed marked additional improvement compared with TAU rapid responders. This difference was sustained as CBT rapid responders demonstrated better outcome compared with TAU rapid responders and nonrapid responders in both CBT and TAU (Fennell & Teasdale, 1987). These results converge in pointing to the importance of rapid response for outcome. In addition, Fennell and Teasdale's (1987) findings suggest the possibility of significant CBT-specific changes occurring over brief intervals within the course of treatment.

Research on sudden within-treatment gains supports this suggestion. Tang and DeRubeis (1999) identified *sudden gains*—significant decreases in depressive symptoms from one session to the next—occurring during CBT for adult depression. The results indicated that 39% of patients had sudden gains (with an average

of 11.2 BDI points), which were associated with superior outcome at termination and follow-up. Interestingly, the average termination BDI of those without a sudden gain ($M = 16.9$, $SD = 13.0$) was quite similar to the mean of the placebo + clinical management condition ($M = 11.0$, $SD = 8.5$) in the National Institute of Mental Health-sponsored Treatment of Depression Collaborative Research Project (Elkin, 1994). The similarity in outcome between CBT patients without a sudden gain and a supportive-placebo condition suggests the efficacy of CBT may be compromised in the absence of a sudden gain.

However, in the presence of a sudden gain, Tang and DeRubeis (1999) found evidence that cognitive change, occurring in the session prior to the sudden gain, appeared to trigger the sudden symptomatic improvement. These findings provide a rationale for suggesting that sudden gains might be critical to CBT and more prevalent in CBT than other approaches. Specifically, if cognitive change produces sudden gains, then CBT would be expected to be more effective at producing sudden gains than approaches that do not emphasize the importance of modifying and/or restructuring negative thoughts (e.g., supportive therapy).

In the present study, the methodology of Tang and DeRubeis provided a common basis for *pretreatment gain* (PG)¹ and *sudden within-treatment gain* (SG) identification using data from a comparative efficacy trial of CBT versus NST and SBFT (Brent et al., 1997). This data set allowed for the potential extension of the findings of Tang and DeRubeis to an adolescent population. In addition, the specificity and impact of PG and SG could be examined across two “active” interventions (i.e., CBT and SBFT) and an intervention designed to control for nonspecific factors (i.e., NST; see Brent et al., 1996). The following specific research questions were explored:

1. How therapy-specific are PG and SG? Because PGs, by definition, occur before therapy begins, these should be evenly distributed across interventions. However, if SGs are the result of events idiosyncratic to CBT (e.g., as suggested by Tang & DeRubeis, 1999), then the number of SGs, and the number of participants experiencing SGs, should be greatest in CBT.
2. What is the impact on treatment outcome of PG and SG? Using different identification criteria, previous research has associated rapid response with positive outcome across treatment modalities (Renaud et al., 1998), which should hold herein. In addition, on the basis of the findings of Tang and DeRubeis (1999), it was expected that those experiencing SGs would demonstrate enhanced treatment effects compared with their no-SG counterparts. Moreover, on the basis of outcome differences between those with and without SGs in Tang and DeRubeis's adult CBT sample, those who did not experience SGs in CBT were expected to show equal endpoint functioning to those in NST and SBFT.

Method

The data were obtained from a clinical trial of psychosocial treatments for depressed adolescents (Brent et al., 1997). One hundred and seven participants, between the ages of 13 and 18, met criteria for a *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., rev.; American

Psychiatric Association, 1987) diagnosis of major depressive disorder, had a BDI score of at least 13, and agreed to randomization. In the present study, following Tang and DeRubeis (1999), we added the requirement that participants receive eight or more therapy sessions. Overall, 87 participants met this additional criterion, 32/37, 27/35, and 28/35 from CBT, SBFT, and NST, respectively. Of those included, 63% were clinically referred; the remaining 37% were recruited through advertisements. Table 1 describes additional sample characteristics. Those who were excluded ($n = 20$) did not differ from those included on any of the variables listed in Table 1, with the exception of the inclusion criterion variable—number of sessions attended, $t(105) = 18.03$, $p < .0001$.²

Acute treatment consisted of 12–16 weekly sessions of CBT, NST, or SBFT, with all teens and families receiving psychoeducation about depression. CBT was based on the approach of Beck, Rush, Shaw, and Emory (1979). The adolescent was socialized into the treatment model emphasizing collaborative empiricism. The therapist and adolescent then worked together to test pessimistic beliefs and attitudes and improve problem-solving, affect regulation, and social skills. SBFT was based on functional family therapy (Alexander & Parsons, 1982) and the behavioral-family systems approach (Robin & Foster, 1989). The treatment included providing information about parenting and development, explaining the treatment model, identifying negative familial patterns, and teaching problem-solving and communication strategies to alter dysfunctional interaction patterns. The NST treatment model emphasized the importance of establishing rapport and aiding the adolescent in identifying recent circumstances and associated affective states that contribute to depression. However, these tasks occurred in the absence of clear therapeutic directives or specific skills training (see Brent et al., 1996, for detailed treatment descriptions).

Therapists were master's-level clinicians with a median of 10 years of experience in the treatment of adolescents. Each therapist received 6 months of modality-specific training and provided treatment only for that modality. Ratings of 25% of the sessions (by internal and external consultants) suggested good protocol adherence, with over 90% of the sessions rated as “acceptable” according to treatment specific scales. Moreover, the raters readily distinguished between the treatment conditions (see Brent et al., 1997, for additional detail).

¹ The sudden within-treatment gain analyses are entirely novel and were not a part of the Renaud et al. (1998) study. The pretreatment gain assessment is conceptually similar to the previous investigation of rapid response; however, the current analysis is different in several ways germane to the present purposes. First, the assessment of pretreatment change is more restrictive than the assessment of rapid response in that it includes only changes occurring from intake to the first session rather than to the second session. Second, here we are using only a sample of participants who completed a minimum course of treatment (eight sessions) rather than using the intent-to-treat sample. Third, herein we used the criteria of Tang and DeRubeis (1999) to provide a common approach to the identification of both pretreatment and sudden within-treatment gains, a direct comparison that has yet to be conducted in the literature.

² Those excluded from the present study also appeared more hopeless than those included according to the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974), $t(102) = 2.0$, $p < .05$. There was also a trend suggesting that a larger proportion of those excluded came to the study from clinic referral, 85% versus 63%, $\chi^2(1, N = 107) = 3.51$, $p < .07$. As described in Brent et al. (1997), protocol deviations occurred because of failure to return for treatment after randomization, dropout after starting treatment, or removal from the trial for clinical reasons (e.g., suicidality or discovery of preexisting clinical conditions, such as substance abuse or bipolar disorder, missed during the assessment). Given this range of possible reasons for participants' failure to complete eight sessions, the differences found are not entirely surprising.

Table 1
Sample Demographic and Treatment Variables

Variable	CBT (<i>n</i> = 32)		SBFT (<i>n</i> = 27)		NST (<i>n</i> = 28)		Statistic	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
Age (years)	15.6	1.3	15.4	1.4	15.7	1.5	$F(2, 84) = 0.46$	.63
SES	39.8	12.0	41.1	11.5	39.1	16.2	$F(2, 84) = 0.15$	.86
No. of sessions	13.2	1.7	12.0	1.6	12.9	2.0	$F(2, 84) = 3.15$	.05 ^a
Pretreatment BDI	24.0	8.1	22.7	8.3	25.1	7.5	$F(2, 84) = 0.59$	.56
Pretreatment Dep 13	2.8	0.4	2.7	0.5	2.8	0.4	$F(2, 84) = 0.24$	.79
Pretreatment CGAS	58.1	7.4	55.2	8.1	57.5	8.9	$F(2, 84) = 1.01$	.37
Sex (% female)	72		81		75		$\chi^2(2) = 0.76$	.69
Ethnicity (% White)	75		93		93		$\chi^2(2) = 6.56$	.08

Note. The inclusion requirement that participants have attended a minimum of eight sessions ensured that at least a minimum amount of therapy was received. However, it is important to ensure that the difference between CBT and SBFT did not decrease the likelihood of finding change in the SBFT cell. Thus, within the SBFT cell, a median split, based on number of sessions attended, was conducted and the amount of BDI and Dep 13 change compared. These analyses revealed no differences (see also Footnote 3). CBT = cognitive-behavioral therapy; SBFT = systemic behavioral family therapy; NST = nondirective supportive therapy; SES = socioeconomic status; BDI = Beck Depression Inventory; Dep 13 = 13 depression items from the School-Age Schedule For Affective Disorders and Schizophrenia; CGAS = Children's Global Assessment Scale.

^a Tukey-Kramer HSD tests revealed a significant difference between CBT and SBFT, $p < .05$.

Depression was assessed, by interviewers blind to treatment condition, at pretreatment, posttreatment, and 3-, 6-, 9-, 12-, and 24-month follow-up, using the 13 depression items (Dep 13) from the School-Age Schedule for Affective Disorders and Schizophrenia (K-SADS; Puig-Antich & Ryan, 1986). At all assessments and prior to every treatment session, youths also completed the BDI. General psychosocial functioning was rated by K-SADS interviewers on the Children's Global Assessment Scale (CGAS; Shaffer et al., 1983). Socioeconomic status (SES) was measured with the Hollingshead Index of Social Status (Hollingshead, 1975).

To be identified as an SG, (a) a change of at least 7 BDI points had to have occurred between two consecutive sessions, (b) this change had to represent at least 25% of the pregain session BDI score, and (c) the BDI mean of the three pregain sessions had to significantly exceed the mean BDI of the three postgain sessions (with alpha set at .05; see Tang and DeRubeis, 1999, for a discussion of their rationale for establishing the three criteria). On the basis of the third criterion, we did not identify SGs occurring prior to the first or last therapy sessions but did assess SGs occurring in the second and second-to-last sessions. Changes occurring from intake to prior to the first session were identified as a PG. Criteria 1 and 2 from Tang and DeRubeis were maintained, assuring that PGs were large in overall and relative magnitude. The lack of preceding data points precluded use of Criterion 3. Thus, to ensure changes were not a transient fluctuation, we required that at least 50% of the PG be maintained during the next two assessments. To capture the stability of gains, we identified *reversals* when greater than 50% of the BDI improvement from the gain was lost (cf. Tang & DeRubeis, 1999).

Subdividing groups into those with and without PG and SG necessarily reduced the number of participants per group. Given concerns about statistical power and the absence of previously published data on the prevalence and impact of sudden changes across treatments, we set alpha at .05 for all analyses. Pearson's chi-square tests were used to compare the prevalence of gains across the three treatment cells, with significant results followed up with two-group comparisons using Fisher's exact tests. Group differences in the size of gains were assessed using one-way analyses of variance (ANOVAs). To explore the impact of gains on acute treatment outcome, we conducted 3 (treatment; CBT, SBFT, NST) $\times$ 2 (gain; gain, no gain) $\times$ 2 (time; pretreatment, termination) ANOVAs with repeated measures on the time factor (BDI, Dep 13, CGAS).³

We also assessed the impact of gain status by calculating effect sizes (Cohen, 1992) comparing the amount of change demonstrated from pretreatment to posttreatment on the BDI, Dep 13, and CGAS, between gain and no-gain groups. Finally, we explored the effects of PG and SG on clinically significant change. Clinical significance was assessed with the BDI at each session using the approach outlined by Jacobson and Truax (1991).⁴ Participants were considered to have attained clinically significant change when (a) the BDI cutoff of less than 10 was met, (b) the reliable change index was significant at the .05 level, and (c) both (a) and (b) continued to be met at all future BDI points during acute treatment. The rate of clinically significant change across treatments and gain groups was assessed using Kaplan-Meier tests of survival, with survival defined as the length of time during acute treatment until reaching clinically significant change (Greenhouse, Stangl, & Bromberg, 1989).

Results and Discussion

Prevalence and Magnitude of Gains

As shown in Table 2, 28% of participants experienced a PG and 39% a SG. The distribution (Table 2) and size (Table 3) of PGs and SGs were similar across treatments. However, removing partici-

³ Because the treatment package included some flexibility in termination point (i.e., treatment length ranged from 12–16 weekly sessions), we used each participant's final BDI. This approximates the analytic strategy used by Tang and DeRubeis (1999) who, because they only compared SG and no-gain groups receiving CBT, conducted a *t* test using endpoint BDIs. In addition, ANCOVAs, with number of sessions entered as a covariate, were also conducted. Results of these analyses did not alter our conclusions.

⁴ The BDI cut-off score was set at less than 10, a common marker of return to the normative range used in both adult and adolescent samples. The standard error of the difference used in calculating the reliable change index was derived from the pretreatment BDI mean and standard deviation from the current sample ($M = 25.14$, $SD = 8.57$), along with the mean, standard deviation, and test-retest reliability data from a large normative sample ($M = 7.17$, $SD = 7.50$; test-retest = .67; Roberts, Lewinsohn, & Seeley, 1991).

Table 2
Prevalence of Pretreatment Gain (PG), Sudden Within-Treatment Gain (SG), and Their Combination (PG + SG), With Participants Experiencing Reversals Included and Removed

Group	Total (<i>n</i> = 87)		CBT (<i>n</i> = 32)		SBFT (<i>n</i> = 27)		NST (<i>n</i> = 28)		$\chi^2(2)$
	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>	%	
Participants (with reversals included)									
PG	24	28	9	28	6	22	9	32	0.69, <i>p</i> = .71
SG	34	39	16	50	7	26	11	39	3.60, <i>p</i> = .17
PG + SG	48	55	20	63	12	44	16	57	2.00, <i>p</i> = .37
Participants (with reversals removed)									
PG	19	22	8	25	3	11	8	29	2.75, <i>p</i> = .25
SG	26	30	13	41	3	11	10	36	6.76, <i>p</i> < .03
PG + SG	35	40	16	50	5	19	14	50	7.68, <i>p</i> < .02

Note. Chi-square tests compared the three treatment groups. CBT = cognitive-behavioral therapy; SBFT = systemic behavioral family therapy; NST = nondirective support therapy.

pants who experienced a reversal revealed a significant difference in SGs, $\chi^2(2, N = 26) = 6.76, p < .03$, with fewer sustained SGs in SBFT compared with CBT ($p < .02$) and NST ($p < .06$). Combining PG and SG groups resulted in identification of 55% of the sample as having experienced a gain. Again, removing participants who experienced a reversal revealed a significant difference, $\chi^2(2, N = 35) = 7.68, p < .02$, with CBT ($p < .02$) and NST ($p < .02$) groups demonstrating more sustained gains than those in SBFT. Thus, consistent with predictions, PGs were not differentially distributed across groups. SGs, on the other hand, were more prevalent in individual therapy (CBT or NST) than family therapy. It is possible that one-on-one therapy may lead to especially productive interventions and curative interactions promoting SGs, whereas a focus on the family may be less likely to yield quick and dramatic effects at the individual level.

The lack of a difference between CBT and NST failed to support the prediction that CBT would be uniquely likely to generate SGs. Thus, in an attempt to find general predictors of SGs, we assessed whether pretreatment clinical or demographic variables differentiated those with and without SGs. No significant differences were

found.⁵ It appears that to better understand the events resulting in a SG, a more fine-grained analysis of the therapeutic time course may be necessary. Evidence of cognitive change should be sought (as it is still possible to suggest that in-session cognitive changes produced the SGs, but did so independent of CBT), but other possibilities, such as the amount of in-session affect, the therapist's recognition of and response to in-session instances of problematic behavior, the completion of extrasession homework, and the timing of important life events and daily stressors should also be explored.

Impact of Gains on Acute Treatment Outcome

The PG group reported significantly greater reductions on the BDI compared with the no-PG group, Time $\times$ Gain group effect, $F(1, 81) = 6.86, p < .01$. Similarly, the SG group demonstrated greater BDI reductions, Time $\times$ Gain group effect, $F(1, 81) = 8.97, p < .01$, and CGAS improvements, Time $\times$ Gain group effect, $F(1, 78) = 5.59, p < .03$, compared with the no-SG group. In addition, the Dep 13 results revealed a significant Time $\times$ Treatment $\times$ Gain group interaction between the SG and no-SG groups, $F(1, 77) = 5.30, p < .03$. Follow-up contrasts showed CBT producing superior outcome compared with SBFT, $F(1, 77) = 7.32, p < .01$, and NST, $F(1, 77) = 6.89, p < .02$, among those not experiencing SGs. In the combined PG + SG group, those who experienced a gain demonstrated significantly greater improvement compared with those without a gain on the BDI, Time $\times$ Gain group effect, $F(1, 81) = 18.42, p < .0001$, CGAS, $F(1, 77) = 5.58, p < .03$, and Dep 13, Time $\times$ Gain group effect, $F(1, 77) = 6.52, p < .02$ (see Figure 1, top panel). In

Table 3
Size of Pretreatment Gain (PG), Sudden Within Treatment Gain (SG), and Their Combination (PG + SG)

Group	PG + SG group		SG group		PG group	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Size of gain	10.5 ^a	4.0	10.1 ^a	4.1	11.0	3.9
CBT	10.7	3.9	10.8	4.0	10.4	3.9
SBFT	10.4	4.3	10.7	5.4	9.8	2.0
NST	10.2 ^a	4.2	8.3 ^a	2.5	12.2	4.8

Note. Overall there were 87 participants, 32, 27, and 28 in cognitive-behavioral therapy (CBT), systemic behavioral family therapy (SBFT), and nondirective support therapy (NST), respectively.

^a One member of the NST group had a sudden gain of 47 Beck Depression Inventory points. The data point from this participant was not included in the table. Including this data point yields the following: PG + SG $M = 11.0, SD = 6.1$, with NST $M = 12.0, SD = 9.2$; and SG $M = 11.1, SD = 7.2$, with NST $M = 11.8, SD = 11.9$.

⁵ Demographic variables included age, SES, sex, and ethnicity. Clinical variables included depression (BDI, Dep 13), general psychosocial functioning (CGAS), hopelessness (BHS), cognitive distortions (Children's Negative Cognitive Errors Questionnaire; Leitenberg, Yost, & Carroll-Wilson, 1986), parent- and child-rated treatment credibility, and referral source (advertisement vs. clinic; see Brent et al., 1997, for detailed description of these measures). None of these demographic or clinical characteristics were significantly different between those with and without sudden gains.

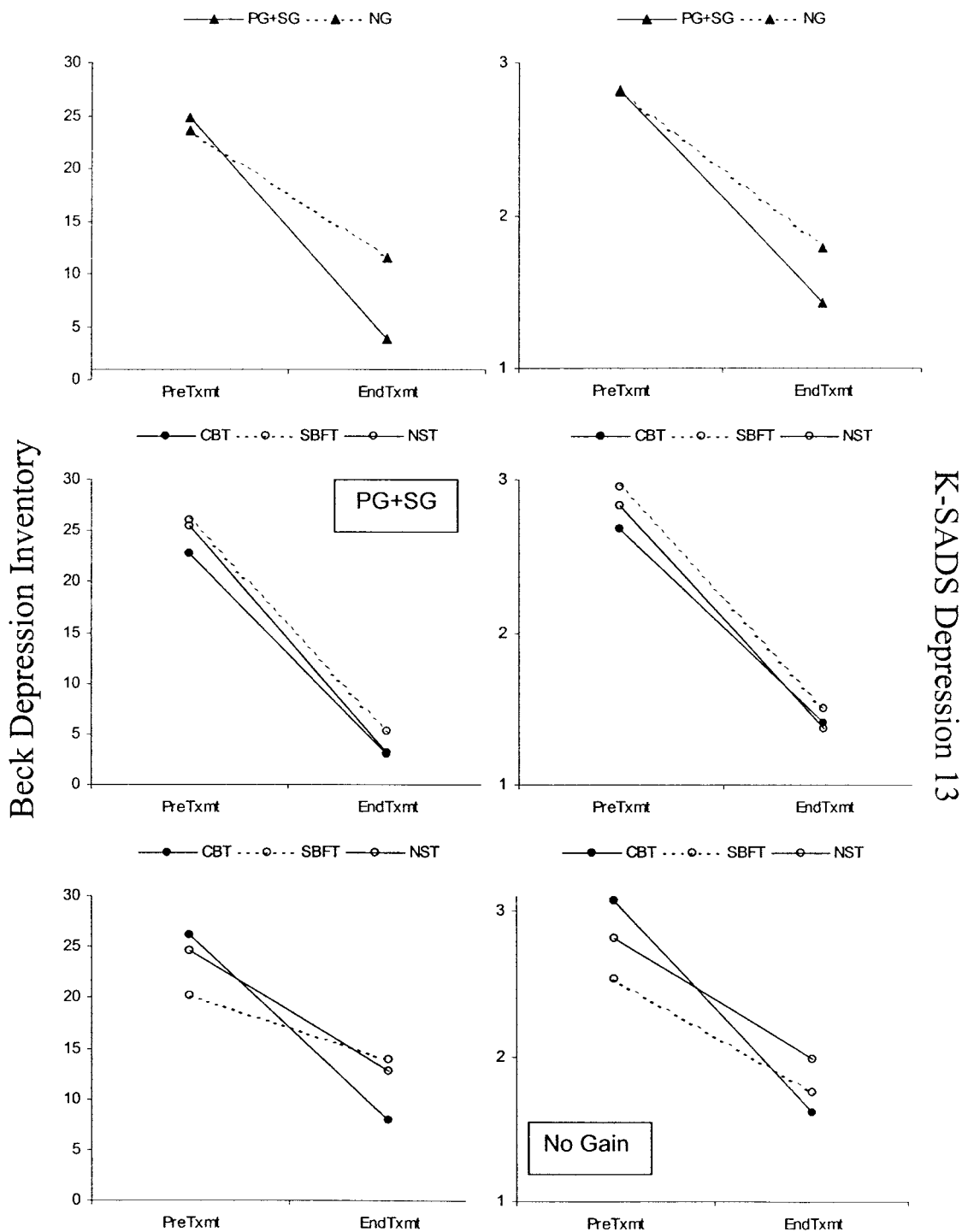


Figure 1. Pretreatment and endpoint Beck Depression Inventory and Depression 13 data illustrating the Time $\times$ Gain group interaction (upper panel) and the Time $\times$ Gain Group $\times$ Treatment Group interaction (middle and lower panel). For visual clarity, we stratified the middle and lower panels by gain status. The middle panel presents the cognitive-behavioral therapy (CBT), systemic behavioral family therapy (SBFT), and nondirective supportive therapy (NST) data for participants experiencing pretreatment gain (PG) or sudden within-treatment gains (SG; combined is PG + SG). The lower panel presents the CBT, SBFT, and NST data for participants not experiencing a gain of either type (No Gain). K-SADS Depression 13 = 13 depression items from the School-Age Schedule for Affective Disorders and Schizophrenia; PreTxmt = pretreatment; EndTxmt = end treatment.

addition, both depression measures suggest the effect of treatment group was important only among those who failed to demonstrate a gain, Time $\times$ Treatment Group $\times$ Gain Group effect: (BDI), $F(2, 81) = 3.54, p < .03$; (Dep 13), $F(2, 77) = 3.90, p < .03$; see Figure 1, bottom two panels. Specifically, among those who failed to experience a gain, follow-up contrasts favored CBT over SBFT: (BDI), $F(1, 81) = 10.76, p < .01$; (Dep 13), $F(1, 77) = 6.94, p < .02$, and NST: (Dep 13), $F(1, 77) = 5.49, p < .03$.

As presented in Table 4, effect size calculations comparing the amount of change in the PG + SG group with the no-gain groups on the BDI, Dep 13, and CGAS resulted in medium to large estimates, suggesting greater change occurred in the gain group. These results are similar to the large effect size found when comparing change in adult SG and no-SG groups receiving CBT (cf. Tang & DeRubeis, 1999). However, comparing CBT gain and CBT no-gain adolescent participants yielded small effect sizes across measures. To the contrary, large effects favoring those who experienced gains were found for SBFT and NST (see Table 4).⁶

Consistent with predictions, gain groups showed greater reductions in depressive symptoms. However, contrary to predictions, outcome in CBT was robust in the absence of a sudden large improvement. By implication, this finding suggests a potentially important role for the various CBT-specific skills. That is, in the absence of a gain, activation of adaptive behavioral repertoires and learning to challenge negative thoughts may be especially critical to producing a positive treatment response. This conclusion is at odds with the results of Tang and DeRubeis (1999) in which the efficacy of CBT was markedly reduced in the absence of a sudden gain. There is no clear data-based explanation for the differential findings. The initial BDI data did not suggest that the adults in the no-gain group ($M = 27.9, SD = 7.9$) were more depressed than their adolescent counterparts in the CBT no-gain group ($M = 26.2, SD = 9.5$). However, it is possible that other indicators of clinical severity (e.g., comorbidity, length of depressive episode, dysthymia superimposed on major depression) may differentiate these subsamples. It is also possible that developmental level plays an important role. For instance, CBT may be especially effective as an early life intervention when dysfunctional coping styles and behavior patterns are less well learned. In addition, the emphasis in CBT on the teen learning coping skills and improving self-

management (of depressive symptoms) may correspond well with ongoing developmental tasks related to establishing autonomy and increasing independence, thereby enhancing efficacy. However, this speculation is based on findings from two initial studies of sudden gains; replication of these age differences in response to CBT is required.

A Kaplan–Meier survival test across the three treatment groups approached significance, log-rank (1) = 5.7, $p < .06$, with pairwise analyses showing a greater prevalence of clinically significant change in CBT (75%) than SBFT (48%), log-rank (1) = 7.2, $p < .01$. In addition, as presented in Table 5, significant results were observed between the respective no-gain groups and the PG, log-rank (1) = 16.8, $p < .0001$; SG, log-rank (1) = 11.2, $p < .001$; and PG + SG groups, log-rank (1) = 21.3, $p < .0001$. The combination PG + SG group illustrates clearly that those who experienced a gain showed higher rates (83% to 41%) and more rapid achievement (median session 5 vs. 9.5) of clinically significant change compared with those without a gain. These results replicate the acute treatment remission rates of 70% and 41% reported by Tang and DeRubeis (1999) for adult SG and no-gain groups.

The temporal pattern of gains and the time to reaching clinically significant change have potential implications for treatment provision with depressed adolescents. The combined results suggest that one quarter to one third of depressed teens demonstrate a PG or rapid response (see also Renaud et al., 1998). In addition, almost 40% experienced a SG, with 85% of SGs occurring by completion of 5 therapy sessions and 100% by completion of 10 sessions. Thus, the first level of care for depressed teens may be the presentation of a credible treatment rationale with a set of associated therapeutic procedures, independent of any specific orientation. If after 5–10 sessions of such care significant improvements have not been noticed, specialized treatment in the form of CBT would appear recommended. This said, there does not appear to be any harm in providing CBT to all depressed youth. In addition, if the prophylactic effect of CBT can be more clearly established, across age groups and independent of gain group status, it would provide stronger justification for recommending CBT as a first level of care.

Outcome at Follow-Up

Given that the strongest findings emerged in comparisons between the PG + SG group versus the no-gain group, only these groups were included in the follow-up factorial between-subjects ANOVAs. On the BDI, there were significant gain group effects at 3 months, $F(1, 75) = 5.0, p < .03$; 6 months, $F(1, 71) = 4.18, p < .05$; and 12 months, $F(1, 75) = 5.05, p < .03$. However, ANCOVAs, adjusting for posttreatment level of depression, failed

Table 4
Effect Size Indices (and Cohen's, 1992, Conventions) From Comparisons of the Combined Pretreatment Gain and Sudden Within-Treatment Gain Group (PG + SG) With the No-Gain Groups, in Mean Change From Pre- to Posttreatment

Measure	Overall	CBT	SBFT	NST
BDI	0.94 (L)	0.20 (S)	1.15 (L)	1.38 (L)
Dep 13	0.55 (M)	−0.31 (S)	0.99 (L)	0.95 (L)
CGAS	0.50 (M)	0.20 (S)	0.48 (M)	0.90 (L)

Note. Effect size = $(M_{\text{pre-post difference of gain group}} - M_{\text{pre-post difference of no gain group}}) / SD_{\text{pooled}}$. Positive values indicate greater change in the PG + SG group than the no-gain group. CBT = cognitive-behavioral therapy; SBFT = systemic behavioral family therapy; NST = nondirective support therapy; BDI = Beck Depression Inventory; L = large; S = small; M = medium; Dep 13 = 13 depression items from the School-Age Schedule for Affective Disorders and Schizophrenia; CGAS = Children's Global Assessment Scale.

⁶ Only the effect size data from comparisons of the PG + SG group with the respective no-gain groups are presented. It is also possible to compare the PG and SG groups with their respective no-gain groups. However, the correlations between the effect sizes from the PG (vs. no-gain) and SG (vs. no-gain) comparisons were $r = .90, p < .0001$ and $r = .77, p < .003$, respectively, with the effect size indices from the PG + SG (vs. no-gain) comparisons. In other words, these additional effect size data point to similar conclusions. The PG (vs. no-gain) and SG (vs. no-gain) comparisons correlated at $.52, p < .09$.

Table 5
Number and Percentage of Participants Reaching Clinically Significant Change and Mean and Median Time to Clinically Significant Change Across Treatment and Gain Groups

Group	n	Clinical significance				
		No.	%	Mdn	M	SD
Overall	87	56	64	5.5	6.4	4.3
PG + SG	48	40	83	5.0	5.4	4.1
CBT	20	17	85	4.0	4.4	2.9
SBFT	12	9	75	10.0	8.7	4.0
NST	16	14	88	2.5	4.6	4.7
No PG + SG	39	16	41	9.5	8.9	3.8
CBT	12	7	58	6.0	7.4	3.7
SBFT	11	4	27	10.5	9.8	3.6
NST	12	5	42	11.0	10.4	4.0
SG	34	28	82	5.0	5.2	4.0
CBT	16	13	81	5.0	4.2	2.6
SBFT	7	5	71	8.0	7.4	3.6
NST	11	10	91	3.5	5.5	5.3
No SG	53	28	53	6.5	7.6	4.4
CBT	16	11	69	6.0	6.5	3.9
SBFT	20	8	40	11.5	10.0	3.7
NST	17	9	53	5.0	6.9	5.2
PG	24	21	88	3.0	4.6	4.2
CBT	9	9	100	3.0	3.8	3.3
SBFT	6	5	83	11.0	9.8	3.9
NST	9	7	78	1.0	2.0	1.5
No PG	63	35	56	6.0	7.5	4.1
CBT	23	15	65	6.0	6.1	3.2
SBFT	21	8	38	9.5	8.5	3.8
NST	19	12	63	10.5	8.6	4.9

Note. PG + SG = pretreatment gains (PG) and sudden within-treatment gains (SG) combined; CBT = cognitive-behavioral therapy; SBFT = systemic behavioral family therapy; NST = nondirective support therapy.

to yield significant results. The BDI data suggest that whereas the gain group continued to report improved functioning compared with the no-gain group, they did not differentially improve during the follow-up period. No follow-up differences were found on the Dep 13 or CGAS.

Limitations and Future Directions

Potential limitations need to be considered. First is limited statistical power. When we stratified our sample by gain status, the number of participants in each treatment cell was reduced. Thus, it remains possible that increased sample sizes would reveal group differences in the prevalence of sudden gains favoring CBT over NST. However, the current data suggest that extremely large samples would be required to detect such differences.

Another limitation is that we did not assess cognitive change in the session preceding the SG. In the absence of any data suggesting that SGs were more prevalent in CBT than other approaches, the current study appeared to be the most logical first step. One might hypothesize multiple mechanisms producing SGs (cognitive change in CBT, family change in SBFT, and remoralization in NST). However, given the difficulties finding robust and replicable mediators of change (see Kolko et al., 2000; Weersing & Weisz, 2002; Whisman, 1993), postulating unique mechanisms across treatments does not appear to be the most parsimonious

explanation for the lack of differences. This does not change the fact that SGs are likely a proxy for some other process or set of processes that we have yet to understand. Future research exploring therapist, client, and observer ratings of sessions appears important for isolating the critical pregain events.

In this investigation repeated measures of depression were analyzed for evidence of large week-to-week changes in symptomatic functioning occurring prior to or during CBT, NST, and SBFT. The findings suggest that sudden improvements mark an important event that bodes well for acute and long-term outcome with both adolescent and adult participants. The most striking finding was the differential ability of CBT to produce improvements in the absence of a PG or SG, whereas improvement in NST and SBFT depended much more on the participant experiencing a gain. Moreover, if PGs and SGs are a general therapy phenomenon, then a subset of participants (55% in the current sample) appears likely to experience positive outcomes regardless of treatment modality. Including these participants in analyses based on group averages would dilute group differences and, thereby, contribute to treatments appearing equally efficacious. Thus, the search for uniquely efficacious treatments or treatment specific mechanisms of action might profit by focusing on better understanding those not experiencing PG or SG.

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