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Sudden Gains During Psychological Treatments of Anxiety and Depression: A Meta-Analysis

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Objective: The present study quantitatively reviewed the literature on sudden gains in psychological treatments for anxiety and depression. The authors examined the short- and long-term effects of sudden gains on treatment outcome as well as moderators of these effects. ***Method:*** The authors conducted a literature search using PubMed, PsycINFO, the Cochrane Library, and manual searches. The meta-analysis was based on 16 studies and included 1,104 participants receiving psychological treatment for major depressive disorder or an anxiety disorder. ***Results:*** Effect size estimates suggest that sudden gains had a moderate effect on primary outcome measures at posttreatment (Hedges's $g = 0.62$) and follow-up (Hedges's $g = 0.56$). These effect sizes were robust and unrelated to publication year or number of treatment sessions. The effect size of sudden gains in cognitive-behavioral therapy was higher (Hedges's $g = 0.75$) than in other treatments (Hedges's $g = 0.23$). ***Conclusions:*** These results suggest that sudden gains are associated with short-term and long-term improvements in depression and anxiety, especially in cognitive-behavioral therapy.

Keywords: sudden gains, depression, anxiety disorders, meta-analysis, treatment

For many individuals, reductions in symptoms are not experienced gradually throughout the course of treatment. Rather, some individuals experience sudden reductions in symptoms that occur between consecutive sessions. These reductions have been termed *sudden gains*. Tang and DeRubeis (1999) were the first to quantitatively define sudden gains and suggested that a sudden gain needs (a) to be large in absolute terms, (b) to represent at least a 25% reduction from the pregain level of symptoms, and (c) to represent a stable reduction (i.e., the mean symptom level in the three pregain sessions needs to be significantly higher than the mean symptom level in the three postgain sessions).

Sudden gains were initially examined in the treatment for depression and were found to be a common phenomenon, occurring in more than 50% of responders to treatment (Tang & DeRubeis, 1999). Sudden gains were also found to be large in magnitude, accounting for more than 50% of the improvement in treatment (Tang & DeRubeis, 1999). Finally, sudden gains were found to be stable and were reversed in only 17% of cases (Tang & DeRubeis, 1999). Individuals who experienced sudden gains reported lower levels of depressive symptoms at posttreatment, 6 months following treatment and 18 months following treatment, compared with individuals who did not experience sudden gains (Tang & DeRubeis, 1999). These findings suggested that sudden gains were related to better treatment outcome at termination and at follow-up.

Since the initial identification of sudden gains, a large body of research has examined them in various treatments for depression. Sudden gains have been found in cognitive therapy (Busch, Kanter, Landes, & Kohlenberg, 2006; Hardy et al., 2005; Tang, DeRubeis, Hollon, Amsterdam, & Shelton, 2007; Vittengl, Clark, & Jarrett, 2005), individual cognitive-behavioral therapy (Tang, DeRubeis, Beberman, & Pham, 2005), group cognitive-behavioral therapy (M. A. R. Kelly, Roberts, & Ciesla, 2005), supportive-expressive therapy (Tang, Luborsky, & Andrusyna, 2002), behavioral activation (Hopko, Robertson, & Carvalho, 2009) and interpersonal psychotherapy (M. A. R. Kelly, Cyranowski, & Frank, 2007). In most studies, sudden gains were significantly associated with lower levels of posttreatment depression. Results regarding follow-up measurements have been mixed, with some studies finding that sudden gains predict lower levels of depression at long-term outcome (e.g., Gaynor et al., 2003; Hardy et al., 2005) and lower relapse rates (Tang et al., 2007), whereas others find no such effect (Tang et al., 2002; Vittengl et al., 2005). However, it is important to note that the studies with null findings had significant methodological differences compared with the studies in whichlong-term effects were found. Specifically, Tang et al. (2002) examined supportive-expressive psychotherapy, and

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Vittengl et al. (2005) examined sudden gains based on measurements that occurred every two sessions.

In recent years, there has been growing evidence that sudden gains are not only circumscribed to treatment for depression but also play an important role in treatments for other disorders. For instance, Stiles and colleagues (2003) detected sudden gains in the treatment of individuals who suffered from a variety of disorders and were treated with a diverse set of clinical approaches in a naturalistic setting. Sudden gains have also been found in treatments for anxiety disorders: in group cognitive-behavioral therapy and group exposure therapy for social anxiety disorder (Hofmann, Schulz, Meuret, Moscovitch, & Suvak, 2006), group cognitive-behavioral therapy for panic disorder (Clerkin, Teachman, & Smith-Janik, 2008), psychodynamic therapy for generalized anxiety disorder (Present et al., 2008), cognitive-behavioral therapy for posttraumatic stress disorder (Doane, Feeny, & Zoellner, 2010; K. A. Kelly, Rizvi, Monson, & Resick, 2009), and transdiagnostic cognitive-behavioral group therapy for anxiety disorders (Norton, Klenck, & Barrera, 2010). In the majority of these studies, sudden gains resulted in reduced symptoms at posttreatment, but this effect was not maintained at follow-up (e.g., Clerkin et al., 2008; K. A. Kelly et al., 2009).

Several proposed mechanisms of sudden gains have been suggested. Tang and DeRubeis (1999) found that cognitive changes frequently precede sudden gains and suggested that cognitive changes may serve as a causal factor in cognitive behavior treatment (CBT). Despite some support for this proposed mechanism (Norton et al., 2010; Tang et al., 2005), other studies did not find sudden gains to be preceded by cognitive changes (e.g., M. A. R. Kelly et al., 2005). Importantly, in supportive-expressive therapy, cognitive changes were unrelated to sudden gains, but greater therapist interpretation accuracy was found to precede the gains (Andrusyna, Luborsky, Pham, & Tang, 2006). Sudden gains were found to be unrelated to changes in the therapeutic alliance in the session before the gain (Tang & DeRubeis, 1999) and to early therapeutic alliance (Hardy et al., 2005). Similarly, other factors suggested to drive sudden gains but lacking research support include changes in self-esteem (M. A. R. Kelly et al., 2005), positive and negative life events (Hardy et al., 2005), and treatment adherence (Doane et al., 2010).

The criteria used to define sudden gains complicate the consistent examination of this phenomenon. The original criteria developed by Tang and DeRubeis (1999) and especially the third criterion regarding the stability of the gain have been criticized on the grounds of autocorrelation of the data (Hardy et al., 2005; Vittengl et al., 2005). Specifically, it has been noted that the three pregain measurements and the three postgain measurements used to determine the stability of the gain are not independent observations and may bias the corresponding *t* value if correlated. To adjust for this, Hardy and colleagues (2005) altered the critical *t* levels for the third sudden gains criterion. The third criterion has also been criticized as it does not allow for the examination of early sudden gains occurring between the first and second sessions. Thus, some researchers have modified this criterion by calculating each individual's standard deviation across all treatment sessions as a measure of individual variation. If a gain was 1.5 times larger than the standard deviation during treatment, then it was considered to fulfill the stability criterion (M. A. R. Kelly, Cyranowski, & Frank, 2007; M. A. R. Kelly, Roberts, & Bottonari, 2007; M. A. R. Kelly et al., 2005). Thus, variation of criteria used in different studies may affect the characteristics of sudden gains and their effects.

Several studies have examined sudden gains occurring after the first treatment session. Busch and colleagues (2006) found that individuals who experienced a sudden gain after the first session had lower levels of depression following treatment compared with individuals who did not experience sudden gains after the first session (including those who experienced a later sudden gain). This suggests that first-session gains may be more strongly related to outcome than later sudden gains. In contrast, Clerkin and colleagues (2008) found that in the treatment of panic disorder (PD), first-session gains were unstable and not related to PD symptoms at posttreatment, whereas later sudden gains were. Thus, the evidence regarding the effects of first-session gains has been mixed.

The aim of the present meta-analysis was to examine the short- and long-term effects of sudden gains on symptoms of depression and anxiety. We expected that sudden gains would be related to better treatment outcome at both posttreatment and follow-up measurements. Consistent with the original conceptualization of sudden gains as a phenomenon that is linked to changes in cognitive processes (Tang & DeRubeis, 1999; Tang et al., 2002), we expected the sudden gains effect to be stronger for CBT than non-CBT treatments. We further tested whether sudden gains differentially influence anxiety disorders as compared with depression, whether including first-session gains influenced the effect of sudden gains and whether variations in the sudden gains criteria moderate the effect of sudden gains.

Method

Identification and Selection of Studies

The PsycINFO, PubMed, and Cochrane databases (1840 to December 2010) were searched for all articles containing the words *sudden gains* in the title or abstract. An extensive manual review was also conducted of reference lists from all articles identified in order to locate additional relevant articles. Both the first and last author conducted independent searches and pooled the results. This initial search yielded 38 potential articles. Studies were included in the present review if they (a) were based on quantitative sudden gains criteria; (b) examined sudden gains during psychological treatment (e.g., cognitive behavior therapy, family therapy, psychodynamic psychotherapy, etc.; studies implementing pharmacological treatments were excluded); (c) were original reports (i.e., not reanalyses of previously reported sudden gains results); (d) measured symptoms at every session; and (e) examined time-limited treatment, defined as specifying a specific maximum number of sessions that all study participants adhered to. The rationale behind the fifth criterion was that in treatments that are unlimited in duration, differences in the number of sessions can affect the baseline probability to experience sudden gains (Lutz & Tschitsaz, 2007; Tang & DeRubeis, 1999). The first three authors (doctoral-level clinical psychologists) coded studies independently and compared results. In cases of differential coding, the first three authors discussed the proper coding until an agreement was reached. Sixteen studies with a total sample size of 1,104 participants met inclusion criteria and were included in the meta-

analysis (Table 1 presents these studies, and Figure 1 presents the study flow chart).

Publication Bias

To address the file-drawer problem, the procedures outlined by Rosenthal and Rubin (1988) and Rosenthal (1991) were used and the fail-safe *N* was calculated. The following formula was used:

$$X = \frac{K(K\bar{Z}^2 - 2.706)}{2.706},$$

where K is the number of studies in the meta-analysis and $\bar{Z}$ is the mean *Z* obtained from the K studies (Rosenthal, 1991). An effect is considered robust if the number of studies needed to reduce the effect size to a nonsignificant level exceeds 5K + 10 (Rosenthal, 1991).

In addition, a funnel plot was constructed to examine the publication bias. No publication bias results in a funnel plot that is symmetrical around the mean effect size. The publication bias was quantitatively assessed using Begg and Mazumdar's rank correlation test (Begg & Mazumdar, 1994). This method examines the relationship between the standardized effect size and its variance using Kendall's τ. The formula used was

$$z = \frac{t}{var(t)},$$

where *t* is Kendall's τ. If the *z* value exceeds 1.96, then the null hypothesis that no bias exists is rejected.

Study Characteristics

For each study, the following were coded: the primary disorder targeted by treatment, the treatment conditions, the number of sessions (i.e., dose of treatment), the sample size, the publication year, the mean age, the gender ratio, the criteria for sudden gains, the measure used to calculate sudden gains, the number of sudden gains detected, the percentage of individuals with sudden gains, and the percentage of reversal of sudden gains.[1] Dependent measures were the effect sizes for depression and anxiety measures.

Because some studies in this review encompassed more than one treatment, treatment condition was considered as the unit of analysis in the present review. Whenever possible, different treatment conditions were coded separately, and when data were not available, we collapsed across treatment conditions within a single study. For the 16 studies included in this review, data were available on 19 treatment conditions.

Effect Size

Controlled effect sizes (between-groups effect sizes) were calculated using Hedges's *g* (Rosenthal, 1991), which corrects for biases due to small sample sizes (Hedges & Olkin, 1985). This effect size can be interpreted using Cohen's (1988) convention of small (0.2), medium (0.5), and large (0.8) effects. When means and standard deviations were available, the following formula was used:

$$g = \frac{\bar{\Delta}_{SG} - \bar{\Delta}_{noSG}}{\sqrt{\frac{(N_{SG} - 1)SD^2_{SG} + (N_{noSG} - 1)SD^2_{noSG}}{(N_{total} - 2)}}} * \left(1 - \frac{3}{4(N_{SG} + N_{noSG}) - 9}\right),$$

where $\bar{\Delta}$ is the mean pre- to posttreatment change, the subscript *SG* refers to individuals with sudden gains, and the subscript no*SG* refers to individuals without sudden gains.

When means and standard deviations could not be obtained, Hedges's *g* was calculated using conversion equations for significance tests (e.g., *t*, *F*) (Rosenthal, 1991). Effect sizes were calculated with random effects models, which assume the studies in the meta-analysis are only a sample of the entire population of studies, and that significant heterogeneity exists between studies. When calculating averages, effect sizes were weighted by their sample size. When several measures of depression severity or anxiety severity were used in the same study, average Hedges's *g* effect sizes were calculated for depression and anxiety. Effect sizes for primary measures (depression measures in treatments for depression, and anxiety measures in treatments for anxiety) and secondary measures (anxiety measures in treatments for depression, and depression measures in treatments for anxiety) were then examined. All analyses were conducted with the program Comprehensive Meta Analysis 2.0.

Heterogeneity Between Studies and Moderator Analyses

The *Q* and I^2 statistics were used to assess heterogeneity and categorical moderators (Huedo-Medina, Sánchez-Meca, Marín-Martínez, & Botella, 2006). The *Q* statistic indicates the presence or absence of heterogeneity, and the I^2 assesses the degree of heterogeneity on a scale of 0–1, where 0 is complete homogeneity and 1 is complete heterogeneity (Huedo-Medina et al., 2006). When examining categorical moderators, the heterogeneity within each group (Q_{within}) was assessed, as well as the heterogeneity between the groups ($Q_{between}$). Significant between-groups heterogeneity indicates that the grouping variable (i.e., the moderator) is significant. Continuous moderators were assessed by meta-regression.

Results

Effects at Posttreatment

At posttreatment, the mean effect size of sudden gains for primary outcome measures was medium, suggesting that individuals who experienced sudden gains had significantly greater improvement during treatment compared with individuals who did not experience sudden gains (Hedges's $g = 0.62$, $SE = 0.09$, 95% CI [0.43, 0.80], $z = 6.63$, $p < .001$, $n = 19$ treatment conditions).

[1] *Reversals* were defined as a loss of 50% or more of the sudden gain at any point in treatment following the gain (Tang & DeRubeis, 1999). Percentage of reversals was calculated by dividing the number of reversed gains by the total number of sudden gains.

Table 1
Studies in the Meta-Analysis

Study	Primary disorder targeted by intervention	Treatment condition	Sessions	*N*	Sudden gains based on	Depression measures	Anxiety measures	Individuals with sudden gains	Percentage of reversals	First-session gains included
Busch et al., 2006	Depression	Cognitive therapy	20	38	BDI	BDI	—	42.1%	43.8%	Yes
Clerkin et al., 2008	PD	Cognitive behavior group treatment	12	30	PDSS	BDI-II	PDSS	43.3%	85.7%	Yes
Doane et al., 2010	PTSD	Prolonged exposure	10	23	PSS-SR	BDI	PSS-SR	52.2%	16.7%	No
Gaynor et al., 2003	Depression	Cognitive behavior treatment	12–16	32	BDI	BDI, K-SADS	—	50.0%	18.8%	No
Gaynor et al., 2003	Depression	Systemic behavioral family therapy	12–16	27	BDI	BDI, K-SADS	—	25.9%	57.1%	No
Gaynor et al., 2003	Depression	Nondirective supportive therapy	12–16	28	BDI	BDI, K-SADS	—	39.3%	9.1%	No
Hardy et al., 2005	Depression	Cognitive therapy	12–20	76	BDI-II	BDI-II	—	40.8%	32.3%	No
Hofmann et al., 2006	SAD	Exposure group therapy	12	59	LSAS	—	LSAS, SPAI	22.0%	—	No
Hofmann et al., 2006	SAD	Cognitive behavior group therapy	12	48	LSAS	—	LSAS, SPAI	14.6%	—	No
Hopko et al., 2009	Depression	Behavioral activation	9	26	BDI-II	BDI-II, HRSD	BAI	50.0%	30.8%	Yes
M. A. R. Kelly et al., 2005	Depression	Cognitive behavior group treatment	12	31	BDI	BDI	—	41.9%	53.8%	Yes
M. A. R. Kelly et al., 2007	Depression	Interpersonal treatment	12	185	BDI	BDI, HRSD	—	33.5%	53.0%	Yes
Norton et al., 2010	Anxiety disorder	Transdiagnostic group cognitive behavior therapy	12	98	STAI-S	—	STAI-S	17.3%	17.6%	No
Present et al., 2008	GAD	Brief psychodynamic-interpersonal	16	29	BAI	BDI	BAI, HAM-A	34.5%	40.0%	No
Stiles et al., 2003	Diverse	Diverse	16	135	CORE-SF	BDI	BAI	17.8%	43.0%	No
Tang and DeRubeis, 1999	Depression	Cognitive vehavior therapy	8–20	61	BDI	BDI	—	39.3%	16.7%	No
Tang et al., 2002	Depression	Supportive-expressive therapy	16–20	35	BDI	BDI	—	42.9%	47.0%	No
Tang et al., 2005	Depression	Behavioral activation and automatic thought techniques	12–20	37	BDI	BDI	—	45.9%	29.4%	No
Tang et al., 2005	Depression	Cognitive therapy	16–20	46	BDI	BDI	—	43.5%	40.0%	No
Tang et al., 2007	Depression	Cognitive therapy	12–16	60	BDI	BDI, HRSD	—	40.0%	37.5%	No

Note. BDI = Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961); PD = panic disorder; PDSS = Panic Disorder Severity Scale (Shear et al., 1997); BDI-II = Beck Depression Inventory–II (Beck, Steer, & Brown, 1996); PTSD = posttraumatic stress disorder; PSS-SR = PTSD symptom scale-self-report (Foa, Riggs, Dancu, & Rothbaum, 1993); K-SADS = School-Age Schedule for Affective Disorders and Schizophrenia (Puig-Antich & Ryan, 1986); SAD = social anxiety disorder; LSAS = Liebowitz Social Anxiety Scale (Liebowitz, 1987); SPAI = Social Phobia Anxiety Inventory (Turner, Beidel, Dancu, & Stanley, 1989); BAI = Beck Anxiety Inventory (Beck & Steer, 1993); HRSD = Hamilton Depression Rating Scale (Hamilton, 1960); STAI-S = State-Trait Anxiety Inventory, State Version (Spielberger, 1983); GAD = generalized anxiety disorder; HAM-A (Hamilton, 1959); CORE-SF = Clinical Outcomes in Routine Evaluation—Short Form (Barkham et al., 2001). Dashes indicate no relevant measures or data in the study.

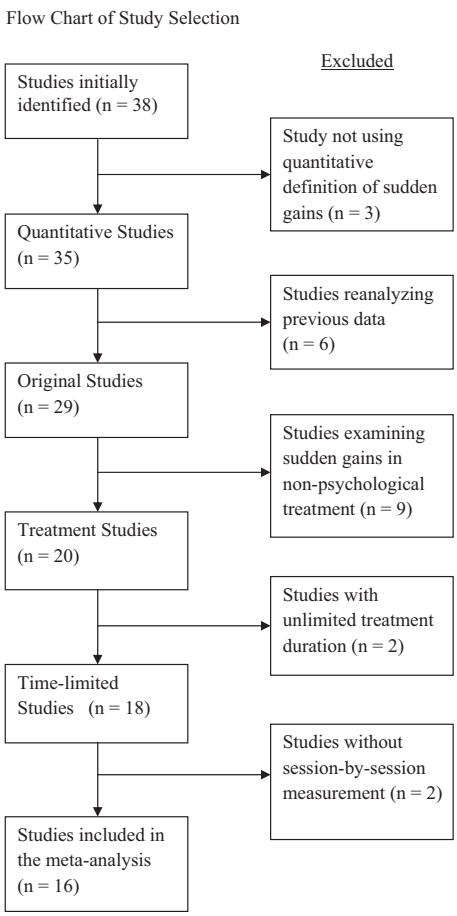

Figure 1. Flow chart of study selection.

Figure 2 presents a funnel plot of the effect sizes for primary measures at posttreatment. The fail-safe *N* was 365, indicating that it would require 365 studies with null findings in order to reduce the effect size found to zero. This fail-safe *N* is greater than Rosenthal's (1991) critical value of 90 studies. We also found no indication of publication bias according to Begg and Mazumdar's rank correlation test (Begg & Mazumdar, 1994; Kendall's τ = 0.13, z = 0.85, p = .40). The mean effect size for secondary outcome measures was in the medium range but did not significantly differ from zero (Hedges's g = 0.34, SE = 0.23, 95% CI [−0.12, 0.79], z = 1.46, p = .14, n = 4 treatment conditions), indicating that sudden gains did not result in significantly greater improvements in secondary symptoms (i.e., anxiety for treatments targeting depression and depression for treatments targeting anxiety) during treatment. Table 2 presents the effect sizes for primary and secondary outcome measures at posttreatment.

Effects at Follow-Up

Due to low sample size, it was not possible to directly compare different follow-up durations. Thus, we pooled data from all follow-up durations across studies (M = 4.44 months). At follow-up, the mean effect size of sudden gains for primary outcome measures was medium (Hedges's g = 0.56, SE = 0.10, 95% CI [0.36, 0.75], z = 5.55, $p < .001$, n = 11 treatment conditions). Figure 3 presents a funnel plot of the effect sizes for primary measures at follow-up. The fail-safe *N* was 68, indicating that it would require 68 studies with null findings in order to reduce the effect size found to zero. This fail-safe *N* is greater than Rosenthal's (1991) critical value of 65. We also found no indication of publication bias according to Begg and Mazumdar's rank correlation test (Kendall's τ = −0.18, z = 0.78, p = .44). Thus, sudden gains resulted in significantly greater improvement from pretreatment to follow-up. Only two studies reported results for secondary measures at follow-up. Effect sizes were 0.37 (SE = 0.36, 95% CI [−0.34, 1.07], z = 1.01, p = .31) in Clerkin et al. (2008) and 0.19 (SE = 0.38, 95% CI [−0.56, 0.80], z = 1.08, p = .62) in Hopko et al. (2009). Neither effect size was significant. In summary, sudden gains during treatment were related to greater improvements in primary measures but not in secondary measures at follow-up. Table 3 presents the effect sizes for primary and secondary outcome measures at follow-up.

Moderator Analyses

Effects of treatment modality on sudden gains. To examine predictors of the effects of sudden gains, we conducted moderator analyses on primary outcome measures at posttreatment. Specifically, we examined whether effect sizes for sudden gains in CBT interventions (n = 14 treatment conditions) were different from non-CBT interventions (n = four treatment conditions). One study that included diverse therapeutic interventions was not included in this analysis as it could not be reliably assigned to either condition (Stiles et al., 2003). Results indicated that the mean effect size for CBT interventions was 0.75 (SE = 0.08, 95% CI [0.53, 0.91], z = 8.91, $p < .001$), and the mean effect size for non-CBT interventions was 0.23 (SE = 0.13, 95% CI [−0.02, 0.48], z = 1.80, p = .07). The degree of heterogeneity within the groups was nonsignificant (Q_{within} = 15.68, df = 16, p = .48; I^2 = 0.0%), whereas the degree of heterogeneity between the groups was significant ($Q_{between}$ = 11.70, df = 1, $p < .001$; I^2 = 91.5%). This indicates that the effects of sudden gains in the CBT interventions were significantly greater than the effects of sudden gains in non-CBT interventions. Importantly, sudden gains were experienced at a

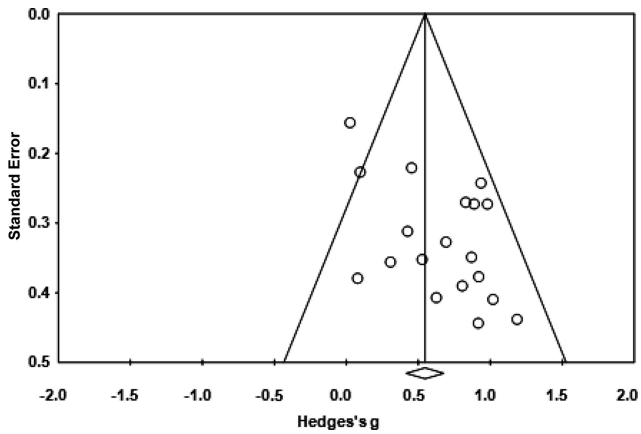

Figure 2. Funnel plot of pre-post effect sizes.

Table 2
Effect Sizes of Sudden Gains at Posttreatment

Study (condition)	Hedges's *g*	*SE*	95% CI Lower	95% CI Upper	*z*	*p*
Primary outcome measures[a]						
Busch et al., 2006	0.70	0.33	0.05	1.34	2.12*	.03
Clerkin et al., 2008	0.92	0.38	0.18	1.66	2.44*	.01
Doane et al., 2010	1.19	0.44	0.33	2.05	2.71*	.01
Gaynor et al., 2003 (CBT)	0.53	0.35	−0.16	1.22	1.50	.13
Gaynor et al., 2003 (SBFT)	0.92	0.44	0.05	1.79	2.07*	.04
Gaynor et al., 2003 (NST)	0.81	0.39	0.04	1.57	2.07*	.04
Hardy et al., 2005	0.94	0.24	0.46	1.42	3.86**	.00
Hofmann et al., 2006 (EGT)	0.43	0.31	−0.18	1.04	1.37	.17
Hofmann et al., 2006 (CBGT)	0.63	0.41	−0.17	1.43	1.54	.12
Hopko et al., 2009	1.02	0.41	0.22	1.83	2.49*	.01
Kelly et al., 2005	0.31	0.36	−0.39	1.01	0.87	.38
Kelly, Cyranowski, & Frank, 2007	0.03	0.16	−0.28	0.34	0.19	.85
Norton et al., 2010	0.89	0.27	0.36	1.43	3.26**	.00
Present et al., 2008	0.08	0.38	−0.66	0.83	0.21	.83
Stiles et al., 2003	0.10	0.23	−0.35	0.55	0.44	.66
Tang and DeRubeis, 1999	0.98	0.27	0.45	1.52	3.59**	.00
Tang et al., 2002	0.87	0.35	0.19	1.56	2.50*	.01
Tang et al., 2005	0.46	0.22	0.02	0.89	2.06*	.04
Tang et al., 2007	0.83	0.27	0.30	1.36	3.07**	.00
Secondary outcome measures[b]						
Clerkin et al., 2008	1.01	0.38	0.26	1.76	2.65*	.01
Doane et al., 2010	0.01	0.40	−0.77	0.80	0.04	.97
Hopko et al., 2009	0.47	0.39	−0.28	1.23	1.22	.22
Stiles et al., 2003	0.03	0.23	−0.42	0.48	0.13	.90

Note. CI = confidence interval; CBT = cognitive behavior treatment; SBFT = systemic behavioral family therapy; NST = nondirective supportive therapy; EGT = exposure group therapy; CBGT = bognitive behavior group therapy.
[a] Depression measures for treatments targeting major depressive disorder, and anxiety measures for treatments targeting anxiety disorders. [b] Depression measures for treatments targeting anxiety disorders, and anxiety measures for treatments targeting major depressive disorder.
* $p < .05$. ** $p < .01$.

similar frequency in CBT interventions (37.4%) compared with non-CBT interventions (37.6%), $F(1, 16) = 0.00$, $p = .98$.

In order to standardize the size of gains and compare across studies, we calculated an average size of sudden gain by dividing the average sudden gain in raw values by the standard deviation at pretreatment for individuals with sudden gains. The average size of these sudden gains was similar in the CBT interventions ($M = 1.51$, $SD = 0.35$) and in the non-CBT interventions ($M = 1.60$, $SD = 0.41$), $F(1, 12) = 0.18$, $p = .68$. Reversal rates were also similar in CBT interventions (34.0%) and non-CBT interventions (37.6%), $F(1, 16) = 0.08$, $p = .78$.

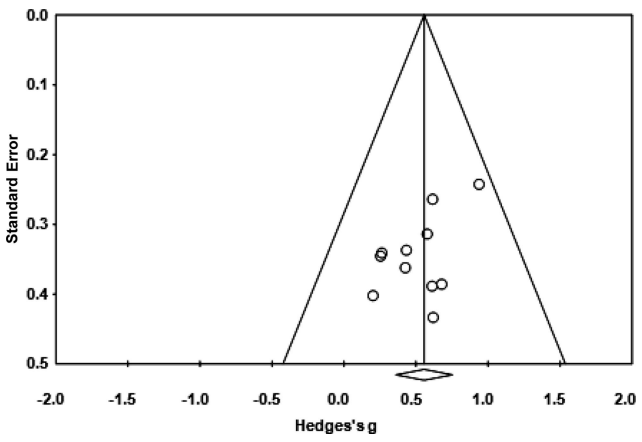

Figure 3. Funnel plot of pre-follow-up effect sizes.

Effects of diagnosis on sudden gains. We examined whether sudden gains in treatments for depression ($n = 12$ treatment conditions) were different from sudden gains in treatments for anxiety disorders ($n =$ six treatment conditions). One study that included diverse disorders was not included in this analysis (Stiles et al., 2003). Results indicated that the mean effect size in treatments for depression was 0.65 ($SE = 0.11$, 95% CI [0.43, 0.87], $z = 5.72$, $p < .001$), and the mean effect size in treatments for anxiety disorders was 0.68 ($SE = 0.18$, 95% CI [0.33, 1.03], $z = 3.84$, $p < .001$). We found significant heterogeneity within the groups ($Q_{within} = 26.80$, $df = 16$, $p = .04$; $I^2 = 40.3\%$), but not between the groups ($Q_{between} = 0.03$, $df = 1$, $p = .87$; $I^2 = 0.0\%$), suggesting that sudden gains have similar effects in treatments for anxiety disorders and depression.

Effect of including first-session gains. We examined whether the inclusion of first-session gains moderated the effect of sudden gains. Results indicated that the mean effect size in studies that included first-session gains ($n =$ five treatment conditions) was 0.46 ($SE = 0.17$, 95% CI [0.13, 0.79], $z = 2.70$, $p = .01$), and

Table 3
Effect Sizes of Sudden Gains at Follow-Up

Study (condition)	Hedges's *g*	*SE*	95% CI Lower	95% CI Upper	*z*	*p*
Primary outcome measures[a]						
Clerkin et al., 2008	0.43	0.36	−0.28	1.14	1.18	.24
Gaynor et al., 2003 (CBT)	0.26	0.35	−0.42	0.94	0.74	.46
Gaynor et al., 2003 (SBFT)	0.62	0.43	−0.23	1.47	1.43	.15
Gaynor et al., 2003 (NST)	0.68	0.39	−0.08	1.44	1.76	.08
Hardy et al., 2005	0.94	0.24	0.46	1.42	3.87**	.00
Hofmann et al., 2006 (EGT)	0.58	0.31	−0.04	1.20	1.85	.06
Hofmann et al., 2006 (CBGT)	0.21	0.40	−0.58	0.99	0.51	.61
Hopko et al., 2009	0.61	0.39	−0.15	1.38	1.58	.11
Stiles et al., 2003	0.27	0.34	−0.40	0.94	0.78	.44
Tang and DeRubeis, 1999	0.62	0.26	0.10	1.14	2.34*	.02
Tang et al., 2002	0.44	0.34	−0.23	1.10	1.29	.20
Secondary outcome measures[b]						
Clerkin et al., 2008	0.37	0.36	−0.34	1.07	1.01	.31
Hopko et al., 2009	0.19	0.38	−0.56	0.94	0.50	.62

Note. CI = confidence interval; CBT = cognitive behavior treatment; SBFT = systemic behavioral family therapy; NST = nondirective supportive therapy; EGT = exposure group therapy; CBGT = cognitive behavior group therapy.
[a] Depression measures for treatments targeting major depressive disorder, and anxiety measures for treatments targeting anxiety disorders. [b] Depression measures for treatments targeting anxiety disorders, and anxiety measures for treatments targeting major depressive disorder.
* $p < .05$. ** $p < .01$.

the mean effect size in studies that did not include first-session gains ($n = 14$ treatment conditions) was 0.67 ($SE = 0.10$, 95% CI [0.46, 0.87], $z = 6.47$, $p < .001$). The degree of heterogeneity within the groups was nonsignificant ($Q_{within} = 26.26$, $df = 17$, $p = .07$; $I^2 = 35.1\%$), and so was the degree of heterogeneity between the groups ($Q_{between} = 1.13$, $df = 1$, $p = .29$; $I^2 = 11.5\%$). Thus, no significant differences in effect size were found between studies that did and did not include first-sessions gains.

Effects of altering sudden gains criteria. Finally, we examined whether altering sudden gains criteria moderated the effect of sudden gains. Studies that used the original Tang and DeRubeis (1999) criteria ($n = 14$ treatment conditions) reported an average effect size of 0.65 ($SE = 0.11$, 95% CI [0.43, 0.86], $z = 5.84$, $p < .001$), and studies that altered the original criteria ($n =$ five treatment conditions) reported an effect size of 0.55 ($SE = 0.17$, 95% CI [0.21, 0.89], $z = 3.15$, $p < .01$). The degree of heterogeneity within the groups was significant ($Q_{within} = 29.29$, $df = 17$, $p = .03$; $I^2 = 41.9\%$), but the degree of heterogeneity between the groups was not ($Q_{between} = 0.22$, $df = 1$, $p = .64$; $I^2 = 0.0\%$), suggesting that adjustments in the sudden gains criteria did not significantly alter the effect sizes.

Additional Analyses

We examined whether the number of sessions in treatment was related to the effect of sudden gains using meta-regression. We found no such association ($\beta = -.00$, $SE = 0.03$, $z = 0.22$, $p = .82$). Similarly, publication year was unrelated to the effect of sudden gains ($\beta = -.03$, $SE = 0.02$, $z = -1.59$, $p = .11$). Randomized trials did not result in significantly greater effect sizes compared with nonrandomized trials ($Q_{between} = 1.45$, $df = 1$, $p = .23$). Timing of gains (median session of their occurrence) was not related to any moderator in the present study, nor to frequency of sudden gains or their reversal rates (all $ps > .05$). Reversal rates were unrelated to any variable in the present review except the first-session gains moderator. Studies that included first-session gains reported significantly greater reversal rates ($M = 53.4\%$) compared with studies that did not include first-session gains ($M = 31.2\%$), $F(1, 16) = 6.89$, $p < .05$.

Discussion

Sudden gains during the course of psychological treatment have been documented across diverse populations and interventions, and have been associated with better treatment outcome (Clerkin et al., 2008; Doane et al., 2010; M. A. R. Kelly et al., 2005; Tang & DeRubeis, 1999). This is the first meta-analysis synthesizing research evidence about the effects of sudden gains, as well as their magnitude, frequency, sustainability, and moderators.

Our findings indicate that individuals who experienced sudden gains evidenced greater reductions in symptoms from pre- to posttreatment, compared with individuals who did not experience sudden gains. We observed a medium between-group effect size (Hedges's $g = 0.62$) for reductions in symptoms of the disorder targeted by the treatment. Thus, sudden gains are not a transient phenomenon occurring during the course of treatment but rather represent stable and lasting changes that significantly influence treatment outcome. However, the effect size for secondary symptoms (Hedges's $g = 0.34$) was not significant, indicating that sudden gains in primary symptoms do not lead to changes in secondary symptoms. Thus, the effects of sudden gains may be circumscribed to the symptoms directly targeted by the therapeutic intervention.

Individuals who experienced sudden gains also evidenced greater reductions in symptoms from pretreatment to follow-up, compared with individuals who did not. Medium effect sizes from

pretreatment to follow-up were found for primary outcome measures (Hedges's $g = 0.56$). Although some studies reported that reductions in symptoms associated with sudden gains at posttreatment did not endure to follow-up assessments (Clerkin et al., 2008; K. A. Kelly et al., 2009; Tang et al., 2002; Vittengl et al., 2005), our quantitative review indicates that sudden gains have a lasting, long-term effect on psychiatric symptoms.

It should be noted that the present review found no differences in the effect sizes of sudden gains when comparing studies in which the original sudden gains criteria were used as described by Tang and DeRubeis (1999) with studies in which modified criteria were used. The sudden gains criteria have also been criticized for not including first-session gains, which may have significant importance (e.g., M. A. R. Kelly et al., 2005). However, we found that including first-session gains had no effect on the effect sizes of sudden gains. Studies that included first-session gains did report greater reversal rates compared with studies that did not include such gains. Thus, it is possible that first-session gains are less stable and more likely to be eroded compared with later sudden gains.

The first examinations of sudden gains were undertaken in the context of psychological treatments for depression (e.g., Hardy et al., 2005; Tang & DeRubeis, 1999; Tang et al., 2005; Tang et al., 2002). Since then, research has also documented this phenomenon across the anxiety disorders. Our findings indicate that there is no difference in the effects of sudden gains in anxiety disorders and depression, suggesting that sudden gains may represent a more general phenomenon in psychological treatments that cuts across different diagnostic groups of emotional disorders. We recommend that future researchers examine this phenomenon in other treatments and disorders, such as pain disorder and substance use disorders.

When comparing treatment modalities, we observed that the effect of sudden gains in CBT interventions is larger (Hedges's $g = 0.74$) than in non-CBT interventions (Hedges's $g = 0.23$). In other words, in CBT interventions, the difference between individuals with and without sudden gains is greater than in non-CBT interventions. Importantly, no differences in sudden gains characteristics (i.e., gain magnitude, frequency and reversal rates) were found between the treatment modalities. This suggests that the reason for the differences in effects may lie not in the gains themselves but rather in their influence on further improvements made in treatment. Tang and DeRubeis (1999) suggested that sudden gains can lead to subsequent cognitive changes, which themselves lead to further reductions in symptoms. Thus, sudden gains can spark a positive feedback loop that Tang and DeRubeis (1999) referred to as an "upward spiral."

A possible explanation for our findings is that upward spirals occur in CBT interventions more than they occur in non-CBT interventions. However, we can only speculate about the processes driving this phenomenon. Upward spirals may be the result of myriad mechanisms, including but not limited to specific cognitive techniques, behavioral techniques, the directive role of the CBT therapist, or skills acquisition. Future studies should examine the processes occurring after sudden gains in order to increase researchers' understanding of the mechanisms involved in the sudden gains phenomenon.

It is important to note that our review only included four non-CBT interventions. Future sudden gains studies should focus on non-CBT interventions in order to increase researchers' understanding of the sudden gains phenomenon in diverse treatments. In addition, future studies should focus on the processes occurring after sudden gains as they may play a significant role in the long-term effects of sudden gains. In addition, two studies included in the meta-analysis (Gaynor et al., 2003; Hofmann et al., 2006) contributed more than one effect size each. This may have impacted the average effect size reported, potentially leading to over- or underestimation.

Despite these limitations, our findings provide support for the existence of sudden gains and for their short- and long-term impact on treatment outcome. Our findings suggest that research should focus on understanding processes that follow sudden gains, in addition to processes that precede sudden gains. These ensuing processes may determine the effect of a sudden gain on treatment outcome more than the characteristics of the gain itself. Our findings suggest that CBT interventions may be especially successful in capitalizing on sudden gains and creating subsequent positive upward spirals.

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