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HÁSKÓLI ÍSLANDS

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## Tables and figures

The phenomenon of sudden gains (SG) and its association with treatment outcome was examined in this randomized controlled trial including cognitive-behavioral group therapy (CBGT) and group psychotherapy (GPT) for social anxiety disorder (SAD). This report is the first to compare SG in CBGT and GPT, with the latter therapy containing only nonspecific treatment factors. Participants were 39 college students with SAD as a primary diagnosis. Participants' symptom severity was assessed by independent assessors at baseline, post-treatment and follow-up with the Clinical Global Impression Scale (CGI) and the Liebowitz Social Anxiety Scale (LSAS). Symptom severity was assessed after each session with the Brief Fear of Negative Evaluation Scale (BFNE), the Social Interaction Anxiety Scale (SIAS) and the Social Phobia Scale (SPS). A total of 17.9% of participants experienced SG during treatment, which were not associated with significantly greater improvements at post-treatment (although close to statistical significance) or at follow-up. SG occurred in both CBGT and GPT at similar rates and magnitude, and the timing of SG was not statistically different in the two treatments.

*Sudden gains in the treatment of depression and anxiety*

In recent years, there has been increased interest in the occurrence of large improvements between two adjacent sessions in psychotherapy. Tang and DeRubeis (1999) were the first to term this phenomenon *sudden gains* (SG), which they defined by the following three criteria: (a) the gain had to be large in absolute terms, (b) the gain had to represent at least 25% reduction in symptoms and (c) the mean level of symptoms in the three sessions preceding the gain had to be significantly higher than the mean level of symptoms in the three post-gain sessions. Tang and DeRubeis (1999) found that about 40% of participants with major depressive disorder (MDD) experienced SG, which accounted for approximately 50% of their total improvement. The gainers were less depressed at post-treatment and at 18-month follow-up than participants who did not

experience SG. Since then, numerous studies of SG have been done, especially in psychotherapy for depressed patients, but also for anxiety disorders. A recent meta-analysis (Aderka, Nickerson, Bøe & Hofmann, 2012) found similar results in studies of both depression and anxiety disorders. Moreover, the effects of SG have been similar in cognitive behavioral therapy (CBT) and other types of therapies, although the treatment effect sizes for gainers have been higher for CBT than for other therapies. Aderka et al. (2012) note that relatively few studies have used interventions other than CBT and call for more studies comparing CBT to other kinds of treatments.

*Mechanisms of change and timing of sudden gains*

The factors determining SG remain largely unexplored. It has been proposed that SG result from cognitive changes made in the pre-gain session and that SG then trigger an "upward spiral" of change, leading to more improvement (Tang & DeRubeis, 1999; Tang, DeRubeis, Beberman & Pham, 2005), although the evidence for this hypothesis is mixed (see e.g. Bohn, Aderka, Schreiber, Stangier & Hofmann, 2013; Kelly, Roberts & Ciesla, 2005). Others believe that nonspecific factors of treatment, such as hope for improvement, are responsible for these sudden improvements (Ilardi & Craighead, 1994, 1999; Lambert, 2005). Vittengl, Clark and Jarrett (2005) observed that SG occurred in a pill placebo condition, which is unlikely due to theory-driven cognitive changes. Similarly, Gaynor et al. (2003) found no differences with regard to size and distribution of SG between treatments where one contained only nonspecific factors.

The timing of SG in therapy may be important in casting light on SG. Furthermore, it is important to study early gains (occurring in the beginning of therapy) and whether those gains are associated with a similar treatment outcome compared to SG occurring later in therapy. If such large improvements occur in the beginning of treatment it could indicate that nonspecific factors facilitate SG. SG commonly follows the fifth therapy

session according to previous studies (Bohn et al. 2013; Hofmann, Schulz, Meuret, Moscovitch & Suvak, 2006; Tang & DeRubeis, 1999). However, criterion c (above) in the methodology proposed by Tang & DeRubeis (1999) has been criticized by ruling out SG occurring at the outset of treatment (see e.g. Kelly et al., 2005). Assessing SG at all possible time-points, Kelly et al. (2005) observed that two-thirds of SG occurred between the first and the fifth session of a 12-session therapy. It appears that SG commonly occur quite early in treatment, which again could indicate the role of nonspecific factors in producing SG. However, early SG are often reversed later in the treatment process (Aderka et al., 2012), which may indicate that they do not reflect real changes or that the therapists or the treatment fails the client in building on initial improvements. It is imperative to study timing of SG more thoroughly than has been done to date.

*Sudden gains in the treatment of social anxiety disorder*

Only two studies have examined SG in treatment of social anxiety disorder (SAD). The first study compared cognitive-behavioral group therapy (CBGT) in accordance with Heimberg and Becker (2002) to exposure group therapy (EGT) (Hofmann et al., 2006) and found that around 19% of participants experienced SG. The criteria for SG were based on Tang and DeRubeis (1999) but slightly modified to fit measures of SAD. SG predicted increased symptom improvement at post-treatment but not at follow-up. When sudden gainers in CBGT and EGT were compared no differences in improvement emerged. The second study examined SG in individual cognitive therapy (CT) and interpersonal therapy (IPT) for SAD (Bohn et al., 2013). A total of 22.4% of participants experienced SG and had significantly lower social anxiety symptoms at post-treatment and at follow-up 12 months later than those who did not experience SG. SG were similar

in frequencies, magnitudes and timings in both treatments but sudden gainers in CT had significantly lower levels of SAD at post-treatment than sudden gainers in IPT.

The current report examined SG in a randomized controlled trial comparing CBGT (a brief form of the group CBT described by Heimberg & Becker, 2002) and group psychotherapy (GPT) for SAD among college students (Bjornsson et al., 2011). GPT was based on Yalom & Leszcz (2005) and was designed to incorporate only nonspecific treatment factors, such as an emphasis on fostering support among group members and group dynamics. No differences in outcome were found between the two treatments when controlling for pre-treatment scores, which made it the first study in the CBGT literature in which the control condition did at least as well as CBGT. This report is the first to compare SG in CBGT and a group therapy containing only nonspecific factors. We hypothesized that participants who experienced SG would experience a significantly increased symptom improvement than those without SG at the end of treatment but not at follow-up, in line with the only other study of CBGT for SAD (Hofmann et al., 2006). Furthermore, we hypothesized that SG would result in greater treatment outcome effect sizes in CBGT compared to GPT, in accordance with the finding that such effect sizes are higher, in general, in CBT compared to non-CBT treatments (Aderka et al., 2012). Finally, we will explore where in treatment SG occur and compare the timing of SG in CBGT and GPT.

## Method

### *Participants*

Methods of the study are described in more detail in Bjornsson et al. (2011). Participants were 39 students at the University of Colorado at Boulder (CU), recruited from university-based email systems and on-campus fliers. They were between 18 and

25 years old and all met DSM-IV-TR (American Psychiatric Association, 2000) criteria for social anxiety disorder as a primary diagnosis. Individuals who had received pharmacological or psychological treatment, were currently suicidal or had been diagnosed with psychotic disorder, bipolar disorder, alcohol or substance dependence or had a primary diagnosis of a different disorder were excluded from study participation. Participants who missed three or more session were excluded from the current report. The Institutional Review Board at CU approved this study.

*Measures*

*The Liebowitz Social Anxiety Scale (LSAS)*. The LSAS is a clinical interview, which assesses both the avoidance and fear of 11 social interactions and 13 performance situations on a four point Likert scale (Liebowitz, 1987). The scale has been shown to be sensitive to change following treatment (Heimberg et al., 1998) and has excellent internal consistency on different subscales (Cronbach's alpha = .81 - .92) (Heimberg et al., 1999).

*Clinical Global Impression Scale (CGI) for SAD*. The CGI is a clinical rating scale, which has been shown to be a valid measure of severity of SAD and improvement over time in a clinical population (Zaider, Heimberg, Fresco, Schneier & Liebowitz, 2003).

*The Brief Fear of Negative Evaluation Scale (BFNE)*. The BFNE is a shortened version of the Fear of Negative Evaluation Scale (Leary, 1983). It is a self-report questionnaire containing 12 items on a 5-point scale (from "Not at all" to "Extremely") that measures the cognitive components of social anxiety. The BFNE has been used to assess changes over the course of treatment (Heimberg, 1994) and has excellent interitem reliability (Cronbach's alpha = .90) and a test-retest reliability of .75 (Leary, 1983).

*Social Interaction Anxiety Scale (SIAS).* The SIAS is a self-report questionnaire of 20 items that assesses fear of social interactions on a 4-point scale, from "Not at all" to "Extremely" (Mattick & Clarke, 1998). This measure has good psychometric properties, including good interitem reliability (Cronbach's alpha = .86) (Heimberg, Mueller, Holt, Hope & Liebowitz, 1993).

*The Social Phobia Scale (SPS)*. The SPS is a self-report questionnaire of 20 items that measures the fear of being observed by others on a 4-point scale, from "Not at all" to "Extremely" (Mattick & Clarke, 1998). SPS has excellent psychometric properties, including Cronbach's alpha of .90 (Heimberg et al., 1993).

*Treatments*

Both interventions were group treatments and consisted of weekly 2-hour sessions for eight weeks. Participants were randomly assigned to either treatment with each group consisting of 5-7 participants as well as one therapist.

*Cognitive-Behavioral Group Therapy (CBGT).* The study used a shorter version of Heimberg and Becker's CBGT (2002), which involved eight 2-hour sessions instead of twelve 2.5-hour sessions. CBGT consisted primarily of psychoeducation and behavioral experiments involving integrated cognitive restructuring and exposures to feared social situations, both in-session and in vivo as homework assignments.

*Group Psychotherapy (GPT).* GPT was based on Yalom and Leszcz (2005). GPT was designed to consist only of nonspecific elements and the main focus was on fostering group dynamics; for example encouraging group members to support each other. The therapist role mostly consisted of encouraging group members to take responsibility for group discussions and to facilitate communication. Group members were asked to share their impressions of one another in a constructive way. The therapist was not allowed to

make use of specific techniques such as cognitive restructuring and group members were asked to come up with homework assignments for themselves.

*Procedure*

Potential participants were screened in a phone interview. Each participant signed an informed consent before baseline assessment was conducted. Participants who met study criteria (described above) were invited to participate in the study and randomized to either treatment. The therapists met all participants individually to discuss the treatments and address any fears or concerns of the participant. The treatment phase consisted of eight weekly two-hour sessions, as described above. At the end of each session participants rated their social anxiety on the three self-report questionnaires used in this study, BFNE, SIAS and SPS. The therapists were advanced clinical psychology graduate students who had completed at least a year of therapy supervision and training. Each therapist led one group of CBGT and one GPT group with order of group leadership randomly assigned, and were supervised throughout by licensed clinical psychologists. Independent assessors (blind to treatment assignments) conducted post-treatment assessments. They were all advanced clinical psychology graduate students with an extensive training in the interviews used. Ratings of treatment integrity revealed excellent adherence and competence for both treatments (Bjornsson et al., 2011).

*Definition of sudden gains*

We assessed SG in SAD with the BFNE, SPS and SIAS, each of which assesses different aspects of SAD. This study used the Tang and DeRubeis SG criteria (1999) with modifications according to Kelly et al. (2005). The criteria for SG were as follows:

*Criterion 1.* The gain must be large in absolute terms, at least a six point reduction on BFNE, a nine point reduction on SIAS and a nine point reduction on SPS from one week to the next. This estimate is in line with Tang and DeRubeis' (1999) seven point

reduction in BDI-II scores that represented 11% of the highest possible score on that measure.

*Criterion 2.* The gain must be relative to the previous score and represent at least 25% reduction in BFNE, SIAS or SPS scores from one week to the next.

*Criterion 3.* The gain cannot be due to normal variation in scores and must therefore represent at least a 1.5 standard deviation (*SD*) reduction in BFNE, SIAS or SPS scores from the participant's mean score over the course of study. This criterion was used to include SG at the outset of treatment, which we considered to be the baseline assessment, in which some of the nonspecific treatment factors, such as hope for improvement, are likely to have begun to take effect.

Consistent with the SG literature, a SG was considered to have reversed if any subsequent BFNE, SIAS or SPS score returned to a level that reflected giving up 50% or more of the improvement resulting from the SG before the end of therapy (Tang & DeRubeis, 1999).

*Data analyses*

Two adjacent sessions were compared to establish SG criteria. To establish the third criteria the standard deviation from the participant's mean score over the course of treatment for each scale was calculated. Standard deviations were then multiplied by 1.5. Because of the length of treatment it was considered best to divide SG into two groups depending on when they occurred during the treatment process. SG were considered *early* SG if they occurred in the first half of therapy, between the baseline assessment, individual session and group sessions 1-3. SG occurring in the second half of therapy, between sessions 4 and session 8, were considered *late* SG.

The primary outcome measures were the LSAS and CGI, which were conducted at baseline, post-treatment and at 3-month follow-up. The LSAS total score was used to

measure improvement at post-treatment and at follow-up. The CGI scale was used to assess whether patients were treatment responders (either a "1" or a "2" on the CGI improvement scale) or not. T-tests (two-tailed) and chi-squares were conducted to compare sudden gainers and non-gainers on background characteristics and other baseline variables. A series of analyses of covariance (ANCOVAs) were conducted to compare sudden gainers and non-gainers at post-treatment and follow-up and to examine whether participants with SG experienced greater treatment improvement compared to those without SG, and to explore whether SG had differential effect in the two treatments. Effect sizes (partial eta-squared & Cohen's *d*) of the improvement at post-treatment and at follow-up were calculated. Cohen's *d* is conventionally interpreted small (0.2), medium (0.5) or large (0.8) and partial eta-squared is interpreted small (0.01), medium (0.06) and large (0.14) (Cohen, 1988).

All analyses were completed using PASW-statistic 18 and Microsoft Excel.

## Results

*Occurrence of sudden gains*

A total of 11 SG were found for 7 out of 39 patients (17.9%). Three participants exhibited one SG and four exhibited two SG. Four out of the eleven SG were reversed (36.4%). Two SG occurred on BFNE, one in CBGT ($M = 17$) and one in GPT ($M = 16$). A total of five SG occurred on SIAS, three in CBGT ($M = 21.67$, $SD = 8.50$) and two in GPT ($M = 19.50$, $SD = 2.12$). A total of four SG occurred on SPS, two in CBGT ($M = 12.50$, $SD = 3.54$) and two in GPT ($M = 11.50$, $SD = 2.12$). A total of 4 out of 17 participants in CBGT (23.5%) experienced SG compared to 3 out of 22 (13.6%) participants in GPT. The difference was not statistically significant, $\chi^2(1) = .64$, $p = .425$. No differences were found between sudden gainers and non-gainers with regard to

background characteristics or symptom severity at baseline (see Table 1), except that individuals with SG had significantly lower symptoms on the SPS ($M$ = 26.83, $SD$ = 11.86) compared to those without SG ($M$ = 40.28, $SD$ = 13.60); $t(36) = 2.49$, $p = .04$.

Of participants who experienced SG, 86% were responders to either CBGT or GPT compared to 53% of non-gainers. SG accounted for 26% of total treatment responders. SG were not more likely to be associated with being a treatment responder; $\chi^2(1) = 2.52$, $p = .11$.

Table 1. Background variables and pre-treatment clinical measures

| | Full sample ($n$ = 39) | Sudden gains ($n$ = 7) | No sudden gains ($n$ = 32) | | |
|---|---|---|---|---|---|
| Variable | *N (%)* or *M (SD)* | *N (%)* or *M (SD)* | *N (%)* or *M (SD)* | Statistics | *p* |
| Gender | | | | $\chi^2(1) = .03$ | .87 |
| Men | 20 (51.3%) | 3 (42.9%) | 17 (53.1%) | | |
| Women | 19 (48.7%) | 4 (57.1%) | 15 (46.9%) | | |
| Age (years) | 19.69 (1.45) | 19.14 (1.07) | 19.81 (1.51) | $t(37) = 1.38$ | .19 |
| Race | | | | | |
| White | 34 (87.2%) | 6 (85.7%) | 28 (87.5%) | $\chi^2(2) = .02$ | .90 |
| Clinical interviews | | | | | |
| LSAS pre | 78.56 (23.22) | 73.86 (17.77) | 79.59 (24.37) | $t(37) = .72$ | .49 |
| CGI pre | 4.87 (.73) | 4.86 (.69) | 4.88 (.75) | $t(37) = .06$ | .95 |
| Self-report measures | | | | | |
| BFNE pre | 47.67 (7.21) | 43.43 (7.98) | 48.59 (6.81) | $t(37) = 1.59$ | .15 |
| SIAS pre | 55.00 (11.30) | 52.29 (12.67) | 55.59 (11.11) | $t(37) = .64$ | .54 |
| SPS pre | 38.16 (14.10) | 26.83 (11.86) | 40.28 (13.60) | $t(36) = 2.49$ | .04 |

*Comparison between sudden gainers and non-gainers*

Participants who experienced SG ($n$ = 7) improved on average by 22.29 points ($SD$ = 6.58) from baseline to post-treatment on the LSAS compared to 12.91 points ($SD$ = 18.56) for participants who did not experience SG ($n$ = 32). An ANCOVA was conducted with SG as the independent variable, pre-treatment total score on the LSAS as the covariate, and post-treatment scores on LSAS as the dependent variable. The effect of SG on symptom improvement at post-treatment is noted, although it failed to reach

statistical significance, $F(1, 36) = 2.89$, $p = .098$, partial $\eta^2 = .07$ (see Figure 1). Effect size (Cohen's *d*) for the between-group comparison (sudden gainers vs. non-gainers) in LSAS total score improvement at post-treatment was 0.67.

Participants who experienced SG improved on average by 23.20 points ($SD = 6.46$) from baseline to follow-up ($n = 5$), while those participants who did not experience SG improved on average by 19.77 points ($SD = 16.62$) from baseline to follow-up ($n = 30$). ANCOVA was conducted with SG as the independent variable, pre-treatment scores on LSAS as the covariate, and follow-up scores on LSAS as the dependent variable. The effect of SG on symptom improvement at follow-up was not statistically significant, $F(1, 32) = .87$, $p = .359$, partial $\eta^2 = .03$ (see figure 1). Effect size (Cohen's *d*) for the between-group comparison (sudden gainers vs. non-gainers) at follow-up was 0.27.

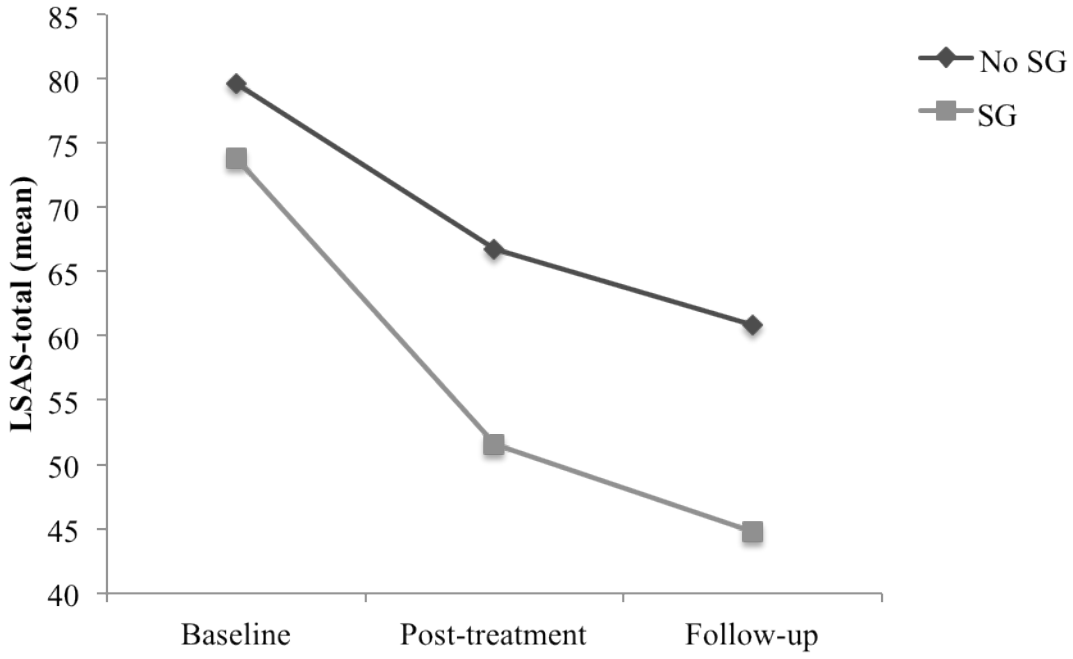


Figure 1. Mean LSAS-total scores between sudden gainers and non-gainers at baseline, post-treatment and follow-up.

*Comparison between sudden gainers and non-gainers in the two treatments*

Two ANCOVAs were conducted with SG and group (CBGT or GPT) as the independent variables, pre-treatment scores as the covariates, and either post-treatment or follow-up scores as the dependent variables. The SG X group (treatment) interaction was not statistically significant at post-treatment, $F(1, 34) = .14$, $p = .712$, partial $\eta^2 = .00$ (see Figure 2) or at follow-up, $F(1, 30) = 1.12$, $p = .299$, partial $\eta^2 = .04$ (see Figure 3). Effect sizes (Cohen's *d*) for the between-treatment SG comparisons were 0.31 at post-treatment and 1.54 at follow-up.

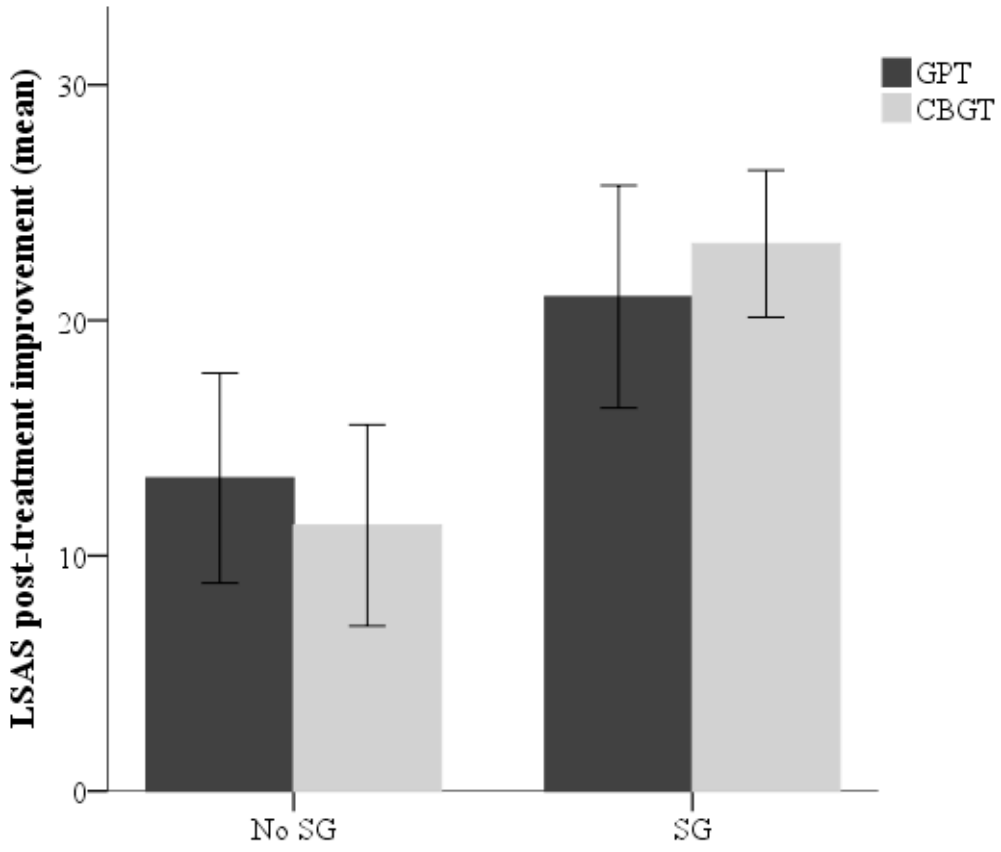


Figure 2. Mean LSAS post-treatment improvement (with error bars representing standard errors) comparison between CBGT and GPT among individuals who showed sudden gains and those who did not.

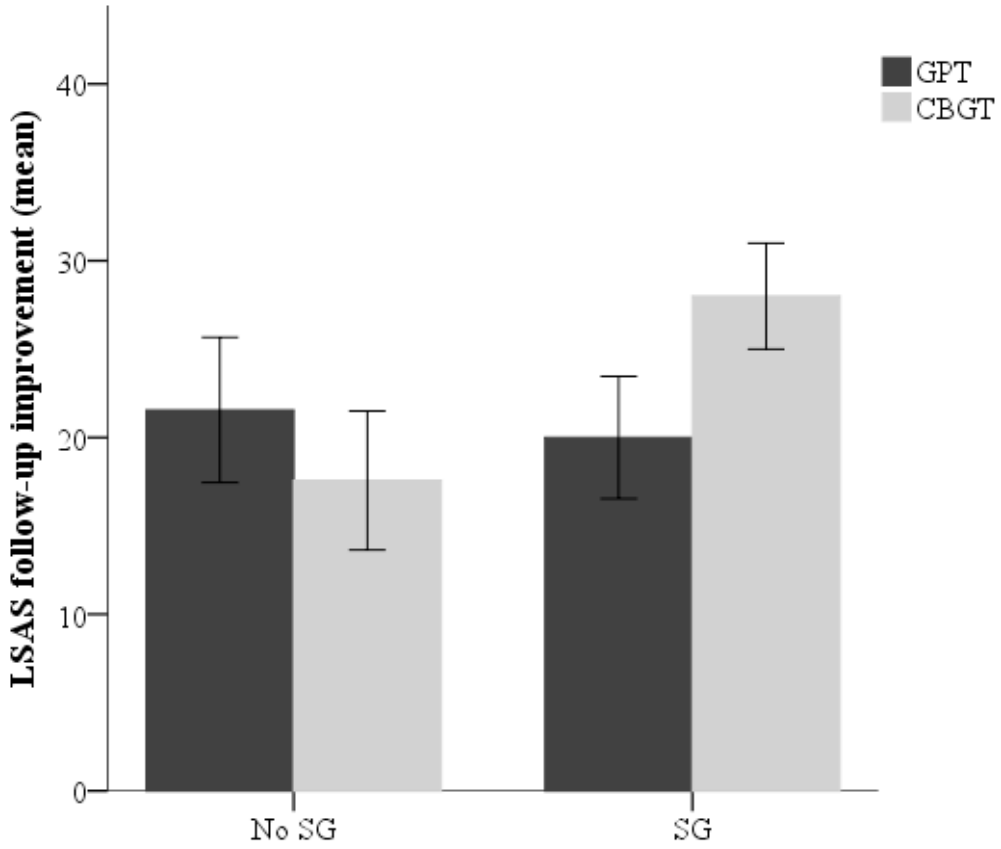


Figure 3. Mean LSAS follow-up improvement (with error bars representing standard errors) comparison between CBGT and GPT among individuals who showed sudden gains and those who did not.

*Timing of sudden gains*

Most SG occurred after baseline assessment ($n$ = 3) and the second treatment session ($n$ = 3). Two of the baseline SG occurred in CBGT, both on SPS, and one in GPT on SIAS. All baseline-SG reversed during treatment. One gain occurred after session 5 on SPS in GPT, two gains after session 6 on BFNE and SIAS (both in GPT) and two gains occurred after session 7, both on SIAS and in CBGT. No significant differences were found in the timing of SG in the two treatments, $F(1, 5) = .30$, $p = .607$, partial $\eta^2 = .06$. No difference was observed in improvement on the LSAS at post-treatment between

early sudden gainers ($M$ = 23.50, $SD$ = 6.19) and late sudden gainers ($M$ = 20.67, $SD$ = 8.08), $F(1, 4) = .11$, $p = .759$, partial $\eta^2 = .03$. Moreover, when comparing follow-up LSAS improvement between participants who experienced early SG ($M$ = 25.50, $SD$ = 7,78) and those who experienced late SG ($M$ = 21.67, $SD$ = 6.66) no difference was found, $F(1, 2) = .01$, $p = .937$, partial $\eta^2 = .00$.

## Discussion

Sudden gains are leaps in symptom improvement between two adjacent sessions and have been studied extensively in treatment of depression (Aderka et. al, 2012). Only two previous studies have examined SG in treatment for SAD and this study is the first to compare SG in CBGT for SAD and another group therapy (GPT) consisting only of nonspecific treatment factors. We used the SG criteria originally proposed by Tang and DeRubeis (1999) but with changes proposed by Kelly et al. (2005) to assess SG early in the treatment process.

This study observed SG occurring for 17.9% of participants (where four of those showed a reversal of their gain). These findings are similar to previous studies of SG in treatments for SAD, which have observed that 18.69% - 22.4% of patients experienced SG (Bohn et al., 2013; Hofmann et al., 2006). It is clear that SG occur in treatment for SAD but at a lower rate than SG in treatment for depression, which has been around 40% in most studies (Aderka et al., 2012). Participants in both CBGT and GPT experienced SG at similar rates and magnitude, which indicates that the specific factors of CBGT are not necessary to produce SG.

The difference in symptom improvement between sudden gainers and non-gainers from baseline to post-treatment was close to being statistically significant, and is likely to have reached significance in a larger sample. However, there was no difference in

improvement between the two groups from baseline to follow-up. These results replicate the findings of the only other study of SG in CBGT for SAD (Hofmann et al., 2006), which observed that SG did predict symptom improvement at post-treatment but not at follow-up. It is worth noting that out of a total of seven sudden gainers, only five showed up at the follow-up assessment, which may have impacted our results. The other pervious study of SG in psychotherapy for SAD (Bohn et al., 2013) found SG to be related to increased treatment improvement both at post-treatment and at follow-up, which is in line with recent meta-analysis of Aderka et al. (2012). It is a topic of future exploration whether there is a difference between group therapy and individual therapy in predicting SG long-term symptom improvement.

We had hypothesized that SG would result in greater treatment outcome effect sizes in CBGT compared to GPT, which was in accordance with the finding that effect sizes are higher, in general, in CBT compared to non-CBT treatments (Aderka et al., 2012). However, our results were that SG had similar effect in the two treatments, in contrast to both our hypothesis and the Bohn et al. (2013) study, which found that SG predicted better treatment outcome for individuals in CT compared to IPT. However, Hofmann et al. (2006) found that SG had similar effect in CBGT and EGT.

Previous studies have observed early SG resulting in more improvement at the end of treatment than gains occurring later in treatment (Busch, Kanter, Landes & Kohlenberg, 2006; Kelly et al., 2005). This study found no differences in improvement between those who exhibited early SG and late SG. Interestingly, every baseline-SG reversed during treatment but two thirds of the baseline-gainers experienced another SG later in treatment. Moreover, no clear pattern was found in the timing of SG, which were scattered over the whole course of treatment in both GPT and CBGT. However, lack of statistical power made it difficult to examine the effect of the timing of SG.

The study had limitations, which are important to note. First, as already mentioned, it consisted of a small sample size and therefore had limited statistical power. This fact made it more difficult to detect differences between gainers and non-gainers and differences between sudden gainers in the two treatments. It could be perceived as a limitation that three different measures (BFNE, SIAS and SPS) were used to assess SG. We do not, however, consider this a true limitation since these measures assess three different aspects of SAD (fear of negative evaluation, fear of social interactions and fear of being observed by others, respectively). Further, this study included SG that occurred between baseline and the individual session, which may be seen as existing outside of the treatment period. However, we argue that the baseline session can be considered to be the starting point of therapy, since nonspecific treatment factors such as hope for improvement become prominent then, especially after they have been assessed by trained assessors and become aware of what the treatment would entail (from reading the consent form). Finally, the sample consisted of college students with SAD as a primary diagnosis, and it may not be possible to generalize the findings to all individuals with SAD.

Future directions include replicating this study with a larger sample and to examine reversed gains further. Aderka et al. (2012) found that early SG are often reversed later in the treatment process. However, the occurrence of reversed SG does not necessarily signify that these large reductions in symptoms, though short-lived, do not signify real changes. It could be true in many cases that the therapists or the treatments simply failed these clients in maintaining improvements. We therefore consider it important to study why SG are reversed and whether preventing this reversal can become a focus of future treatment development.

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