



Sudden Gains in Psychotherapy

Auður Sjöfn Þórisdóttir

**Lokaverkefni til MS - gráðu
Sálfræðideild
Heilbrigðisvísindasvið**



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Formáli

Verkefni þetta samanstendur af fræðilegu yfirliti og rannsóknagrein til birtingar í vísindatímarit. Verkefnið fjallar um tiltekið mynstur breytinga á einkennum geðraskana í sálfræðimeðferð, sem kallast skyndiframfarir (*e. sudden gains*) og þekkist af skyndilegum og miklum breytingum á einkennum milli tveggja meðferðartíma. Í fræðilega yfirlitinu er farið yfir hvernig þetta breytingamynstur hefur verið skilgreint og skýrt og sagt frá helstu niðurstöðum rannsókna á þessu sviði. Fjallað er um annmarka í aðferðafræði rannsókna og tillögur settar fram um hvernig hana megi bæta. Í rannsóknagreininni eru skyndiframfarir metnar í tveimur hópmeðferðum við félagsfælni (*e. social anxiety disorder*) í þeim tilgangi að varpa ljósi á það hvernig og hvers vegna þetta breytingamynstur kemur fram.

Research on Sudden Gains in Psychotherapy

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Abstract

One change pattern in psychotherapy that has received considerable attention, *sudden gains*, is characterized by large improvements between adjacent treatment sessions. Some studies have found that sudden gains account for the majority of participants' total symptom improvements and that they predict better treatment outcomes. Understanding what causes them could provide important insights into the mechanisms of change in psychotherapy with implications for optimizing treatment effectiveness. However, research findings have been inconclusive on why and how sudden gains occur. In this paper, we review research on sudden gains, discuss methodological shortcomings that have impeded sudden gains research and compare sudden gains to other common change patterns; *rapid early response* and *depression spikes*.

Keywords: sudden gains, psychotherapy, mechanisms of change, rapid early response, depression spikes

Patterns of change have attracted the attention of researchers studying treatment effectiveness. Conventional comparisons between a treatment group and a control group of average symptom levels before and after treatment enable researchers to determine whether a treatment is efficacious, but fail to shed light on how and why the change occurs (Elliott, 2010; Laurenceau, Hayes & Feldman, 2007). When does symptom improvement occur during the course of treatment and what promotes the changes? Examinations of change patterns could reveal mechanisms of change in therapy, which could have implications for optimizing treatment effectiveness (Kazdin, 2007; Llewelyn & Hardy, 2001).

Psychotherapy is a complex web of common factors and specific techniques (Rosenzweig, 1936; Stevens, Hyan & Allen, 2006). Common factors are shared by different psychological interventions regardless of theoretical orientation, such as the therapeutic alliance, empathy and the client's hope for recovery. Specific techniques are based on a theory of how a given disorder or psychological problem is maintained, and are sometimes thought to be "the active" ingredient in the therapy (Jørgensen, 2004; Stevens, et al., 2006). As one example, cognitive restructuring is presumed to be the primary active ingredient in cognitive behavioral therapy (CBT) (Beck, 1976). However, many have argued against such a view, and have called for research investigating the interaction of specific and common factors over the treatment process as they relate to treatment outcome (see e.g., Bjornsson, 2011; Ilardi and Craighead, 1994).

Ilardi and Craighead (1994) analyzed average symptom severity time courses in CBT for depression and found that the majority of symptom improvements occurred very early in treatment, i.e. before the fourth treatment session and before cognitive restructuring had been administered. They called this change pattern rapid early response. These findings suggested that non-specific treatment factors, such as therapeutic alliance and hope for recovery, were

more influential in promoting changes than had previously been assumed, highlighting the need for studies on change processes and mechanisms of change in therapy (Ilardi & Craighead, 1994, 1999; Kazdin, 2007).

Tang and DeRubeis (1999a; 1999b) criticized Ilardi and Craighead for only looking at average time courses, and argued that it was important to examine individual session-to-session time courses in CBT for depression. Through their efforts, they discovered another common change pattern which they termed sudden gains. Sudden gains have received considerable attention from researchers studying change processes in therapy because the examinations of common change patterns, shared by many individuals, could reveal mechanisms of change in therapy with prospects for enhancing treatment effectiveness (Tang & DeRubeis, 1999b; Thomas & Persons, 2012).

In the present paper we review how sudden gains have been defined and assessed in the research literature. We discuss Tang and DeRubeis' theory of the mechanisms of sudden gains and critically review the evidence for it. We go on to discuss various methodological shortcomings in this literature, and we argue that it is important to assess sudden gains over the whole treatment process. We then compare sudden gains to rapid early changes and another common change pattern termed depression spikes. In conclusion, we offer suggestions for future research on sudden gains.

Sudden gains

Tang and DeRubeis (1999a; 1999b) defined sudden gains as improvements between two adjacent treatment sessions that were large in absolute terms, large relative to symptom severity before the gain and relative to symptom fluctuations before and after the gain. They described three quantitative criteria to identify sudden gains in CBT for depression. The first criterion stated that a gain, from one therapy session to the next, had to represent at least a

seven point reduction on the Beck's Depression Inventory (BDI), which was the primary outcome measure in their study. The second criterion stated that the gain had to represent at least 25% of the pre-gain session's BDI score and according to the third criterion, the mean BDI score of the three therapy sessions before the gain had to be significantly higher than the mean BDI score of the three therapy sessions after the gain. Furthermore, the sudden gains were considered to be reversed if any subsequent BDI scores returned to a level that reflected giving up 50% or more of the improvement resulting from the sudden gain (Tang & DeRubeis, 1999b).

Tang and DeRubeis (1999b) found that sudden gains occurred for about 40% of patients with major depression in CBT and that the gains accounted for a large proportion of these patients total symptom improvements. Approximately 60% of the treatment responders in CBT experienced sudden gains, indicating that sudden gains could shed light on how the majority of treatment responders improved. Furthermore, patients with sudden gains had lower levels of depression symptoms than other patients, at the end of treatment and at follow-up measures. Tang and DeRubeis (1999b) suggested that important therapeutic events occurred in the treatment sessions prior to sudden gains, i.e. that these sessions represented therapeutic breakthroughs and were "critical" for treatment outcome. Identifying sudden gains might therefore be a convenient way to identify critical sessions in CBT for depression, with important implications for enhancing treatment effectiveness (Tang and DeRubeis, 1999a; 1999b).

Tang and DeRubeis (1999b) found that patients in their study had significantly more cognitive changes in the pre-gain session compared to control sessions and therefore concluded that cognitive changes led to sudden gains, in line with the cognitive mediation hypothesis (i.e. that changing maladaptive cognitions leads to symptom improvement). They proposed a "three stage model" to explain how the sudden gains pattern occurred in CBT.

According to the model, the first sessions of CBT represent a preparation stage in which the patient and the therapist begin to form the therapeutic alliance, and the cognitive model is described and explained. The patient experience minor cognitive changes and depression symptom improvements which lay the foundation for later work. Tang and DeRubeis (1999b) suggested that the likelihood of whether a patient progresses beyond this stage, in addition to how long he remains in this stage, depended on how effective the therapist was, how strong the therapeutic alliance was and whether the patient was ready for change. Stage two involves the critical pre-gain session (i.e. the session immediately preceding the sudden gain) where presumably cognitive restructuring work leads to changes in beliefs which then lead to the sudden gain in depression symptoms. In stage three, the patient experiences an “upward spiral” such that the sudden gain leads to improved therapeutic alliance, which set the stage for further cognitive changes that maintain and lead to an even greater symptom relief, eventually leading to recovery (Tang & DeRubeis, 1999b, p. 12).

Subsequent studies have found sudden gains occurring in CBT for depression in similar frequencies as Tang and DeRubeis’s (1999b) original analysis (see for example: Busch, Kanter, Landes & Kohlberg, 2006; Kelly, Roberts & Ciesla, 2005; Tang, DeRubeis, Beberman & Pham, 2005) with some studies supporting the hypothesis that cognitive changes preceded the sudden gains (Tang et al., 2005), while other studies have not found this association (Bohn, Aderka, Schreiber, Stangier & Hofmann, 2013; Kelly et al., 2005). Further research is needed to determine whether cognitive changes are critical for generating sudden gains in treatment for depression. Andrusyna, Luborsky, Pham & Tang (2007) assessed sudden gains in supportive-expressive therapy for depression and found that therapist interpretation accuracy (but not cognitive changes) moderated the gains. Based on these findings, they suggested that different factors led to sudden gains in different treatments; cognitive changes were most critical for sudden gains in CBT and therapist interpretation

accuracy most critical in supportive-expressive therapy. Others have proposed that the influences of common treatment factors and factors outside of therapy preceding the sudden gains, such as significant life events (e.g. leaving a problematic job) and treatment adherence, have been underestimated in the generation of sudden gains (Doane, Feeny & Zoellner, 2010; Hardy et al., 2005). Supporting this notion are studies showing that sudden gains do not seem to be dependent upon the provision of treatment. Kelly, Roberts and Bottonari (2007b) found sudden gains in a non-treatment setting where self-assessment was used for measuring depression symptoms. Furthermore, Vittengl et al. (2005) observed that SG occurred in a pill placebo condition. However, more research on the role of specific and nonspecific treatment techniques in the generation of sudden gains are needed.

Sudden gains have also been found in other treatments than CBT for depression such as supportive-expressive therapy (Tang, Luborsky & Andrusyna, 2002), interpersonal psychotherapy (Kelly, Cyranowski & Frank, 2007a) and in routine clinical settings (Adler, Harmeling & Walder-Biesanz, 2013; Stiles et al., 2003). Furthermore, sudden gains are not confined to treatments of depression but have been found to occur in therapy for other disorders, such as in individual and group CBT for social anxiety disorder (Bohn et al, 2013; Hofmann, Schulz, Meuret, Moscovitch & Suvak, 2006), psychodynamic therapy for generalized anxiety disorder (Present et al., 2008), CBT for posttraumatic stress disorder (Doane et al., 2010), group CBT for panic disorder (Clerkin, Teachman & Smith-Janik, 2008), CBT for eating disorders (Cavalli & Spangler, 2013) and couples therapy (Doss, Rowe, Carhart, Madsen & Georgia, 2011).

Most research find that sudden gains are significantly related to better treatment outcomes at post-treatment (e.g. Cavalli & Spangler, 2013; Hofmann et al., 2006; Tang et al., 2005) but the findings are mixed for follow-up with some studies finding that sudden gains predict better long term outcomes (e.g. Bohn et al., 2013; Hardy et al. 2005) while others

studies do not find this association (e.g. Clerkin et al., 2008; Hofmann et al., 2006; Present et al., 2008). In most studies the majority of participants defined as treatment responders, experience sudden gains (e.g. 70-80%, Tang & DeRubeis, 1999b; Vittengl, Clark & Jarett, 2005). However, as Vittengl et al. (2005) found, participants that are both treatment responders and experience sudden gains do not necessarily have better outcomes at treatment termination than responders without sudden gains, suggesting that treatment responders without sudden gains improve more gradually. Thus, it appears that sudden gains are usually indicative of better treatment outcome post treatment (but not always at follow-up), although there are mixed findings, and the difference in treatment outcome between sudden gainers and non-gainers should be explored more fully.

Methodological concerns in sudden gains research

The mixed findings reported above and failure in identifying the mechanisms of sudden gains could be contingent, in part, on methodological shortcomings in sudden gains research.

Research has been inconsistent in the criteria used to identify sudden gains, with some studies using Tang and DeRubeis's (1999b) original criteria while others have used a modified version (Aderka et al., 2012). Tang and DeRubeis (1999b) intended the first criterion (i.e. a gain from one therapy session to the next, had to represent at least a seven point reduction on the BDI) to select only unusually large session-to-session symptom reductions, but acknowledged that the value of seven on the BDI was somewhat arbitrary (Tang & DeRubeis, 2005). Studies examining sudden gains with other measures than BDI have had to find a comparable value for the first criterion (Stiles et al., 2003). Most studies have used the reliable change index (RCI, i.e. subtracting pretreatment scores from posttreatment scores and dividing the difference with the standard error of the difference) (Jacobson & Truax, 1999), to determine whether a change might be considered a sudden gain (e.g. Bohn et al., 2013; Doane

et al., 2010; Dour, Chorpita, Lee & Weisz, 2013; Hofmann et al., 2006; Stiles et al., 2003).

The RCI could be a reasonable alternative to the first criterion because it ensures that a change is sufficiently large to exceed the margin of measurement error (Jacobson, Roberts, Berns & McGlinchey, 1999) and an RCI of 6.18 has been found for the BDI which is close to the seven point value of Tang and DeRubeis's (1999b) criterion (Stiles et al., 2003).

Tang and DeRubeis's (1999b) second criterion (i.e. a gain had to represent at least 25% of the pre-gain session's BDI score) was meant to reflect a gain that was large relative to symptom levels in the session before the gain. Nevertheless, this criterion is almost always met if a seven point reduction occurs on the BDI (Thomas & Persons, 2012) and dropping this criterion does not seem to affect the rate of sudden gains, which calls into question its usefulness in identifying them (Hardy et al., 2005; Stiles et al., 2003; Tang & DeRubeis, 2005).

The third criterion (i.e. the mean BDI score of the three therapy sessions before the gain had to be significantly higher than the mean BDI score of the three therapy sessions after the gain) has been criticized for violating assumptions of independence by using a two sample t-test for comparing mean symptom levels of the same individual before and after the gain (Kelly et al., 2007a). Even more importantly, it has been criticized for ignoring the full course of therapy, thus eliminating very early and very late sudden gains (Busch et al., 2006; Kelly et al., 2005). Tang and DeRubeis (1999b) excluded changes occurring after the first CBT session, based on the assumption that the first session differs in nature from later ones. However, as Ilardi and Craighead's (1994) analysis revealed, the majority of CBT improvements occur very early in treatment which suggests that early changes convey important information on the change processes in CBT. Kelly et al. (2005) assessed sudden gains over the full course of CBT for depression and found that early sudden gainers had significantly lower depression symptoms at post-treatment than participants with later gains

or no sudden gains. Similarly, Busch et al. (2006) found that sudden gains occurring after the first treatment session predicted better outcomes than later gains in cognitive therapy for depression. These results suggest that it is imperative for sudden gains research to include the full course of treatment in the analysis.

Another limitation in sudden gains research is lack of consensus in how sudden gains are estimated. Tang and DeRubeis (1999b) assessed changes occurring after every session in CBT in which the patients received two treatment sessions per week for four weeks and then one session per week for eight weeks. Sudden gains were thus assessed twice a week for the first third of treatment and then weekly afterwards. In contrast, Vittengl et al 2005 assessed sudden gains weekly in cognitive therapy for depression although the patients received two sessions a week. Additionally, patients sometimes miss therapy sessions which results in missing data that often results in varying periods between assessments. Such inconsistency in the estimations of sudden gains could cause various biases in interpreting findings and comparing different studies, and it is important that researchers in this field establish common criteria to assess sudden gains.

Finally, research on sudden gains is further impeded by shortcomings in research designs. Many studies have not used control groups when evaluating sudden gains (e.g Tang and DeRubeis, 1999b; Tang et al., 2005) and studies that do use control groups usually confound specific treatment techniques and common treatment factors by comparing two treatments consisting of both (see for example Tang et al., 2005). Studies are needed that systematically tease apart specific techniques and common factors, by comparing a treatment with a control group that consists only of common factors. In one of the first studies attempting to tease apart specific treatment techniques and common factors, Thorisdottir, Bjornsson, Tryggvadottir and Saevarsson (in preparation) compared sudden gains in group CBT for social anxiety disorder to group psychotherapy, designed specially to contain only

common factors of therapy (see Bjornsson et al., 2011). They found sudden gains occurring at similar rates and magnitudes across both treatments. Although more studies are needed to clarify the relative contributions of specific techniques and common factors in promoting sudden gains, these findings indicate that specific techniques do not lead to greater sudden gains than common factors.

Comparison of sudden gains to other change processes

It seems to be a prominent feature of most studies to focus on change patterns from a particular viewpoint, such as occurring in a gradual manner or discontinuously, and to employ statistical methods that increase the chance of finding these patterns. As an example if symptom changes are examined with averages from pre-treatment to post-treatment, individual symptom fluctuations will go unnoticed and the changes will seem to occur gradually (e.g. Carey, 2011; Hayes et al., 2007b; Thomas & Persons, 2012). Furthermore, by focusing on changes from a particular viewpoint, researchers sometimes overlook the full course of therapy and whether it is possible to integrate different change patterns (such as rapid early response and sudden gains) rather than considering them mutually exclusive. Tang and DeRubeis (1999b) claimed that sudden gains were qualitatively different from other changes during treatment, a view that has been generally accepted in the research literature. However, when sudden gains criteria are modified so that they allow for early gains to occur, similarities appear between sudden gains and Ilardi and Craighead's (1994) rapid early response pattern. Furthermore, in some cases it may be possible to make sense of reversed sudden gains by considering a change pattern described by Hays et al. (2007a) as depression spikes.

Sudden gains and rapid early response

Rapid early response is characterized by a marked early decrease in symptoms, which levels off as the treatment progresses (Ilardi & Craighead, 1994). In Ilardi and Craighead's (1994) review of rapid early responses in CBT for depression, the early symptom decrease occurred before the fourth treatment session and accounted for 60 - 80% of the patients total symptom improvements. Furthermore, the rapid early response predicted better overall treatment outcomes. Subsequent studies have replicated these findings in treatment for other disorders, such as in cognitive behavioral therapy for binge eating disorder (Grilo, Masheb & Terence, 2006) and in supportive-expressive therapy and cognitive therapy for mixed populations (Crits-Christoph et al., 2001).

When rapid early response is compared to sudden gains, certain similarities appear. First, both rapid early response and sudden gains occur in a brief time period, sudden gains between adjacent treatment sessions and rapid early response in the first four treatment sessions. However, when Kelly et al. (2005) examined sudden gains over the entire course of CBT for depression, they found that 10 of 15 sudden gains occurred early in treatment, before the fifth treatment session, which is approximately within the defined time course of rapid early responses. Second, both the rapid early response and the sudden gains pattern appear to be common in treatment for depression (Ilardi & Craighead, 1994; Tang & DeRubeis, 1999b). Both patterns have furthermore been found in treatment for other disorders (e.g. Doane et al., 2010; Grilo et al., 2006). Third, both rapid early response and sudden gains account for the majority of symptom improvements in treatment and predict better treatment outcomes (Ilardi & Craighead, 1994; Tang & DeRubeis, 1999b).

What distinguishes sudden gains from rapid early response is the method used for assessing them. Sudden gains are assessed in individual time courses from one therapy session to the next but rapid early response is assessed with the time courses of the average

symptom improvements. Thomas and Persons (2012) compared rapid early responses and sudden gains and found that sudden gains did not have unique predictive power over early response pattern on treatment outcome. In conclusion, it appears that these patterns are not contrary, but are rather entirely compatible with each other.

Sudden gains and depression spikes

Hayes et al. (2007a; 2007b) described depression spikes in exposure therapy for depression. Similar to anxiety spikes in exposure therapy, where anxiety increases before a decrease (Heimberg & Becker, 2002), depression spikes are characterized by sudden, large increases in depression symptoms followed by a decrease. Hayes et al. (2007a) argued that depression spikes were the “conceptual opposites of the sudden gain” (p. 414). Depression spikes were thus assumed to occur if depression symptoms increased by seven points or more, as opposite to the first criterion of sudden gains, and then leveled out by seven points or more over the course of therapy (Hayes et al., 2007a). Hayes et al. (2007a) found that depression spikes were a common phenomenon in exposure therapy for depression and predicted lower levels of depression symptoms at the end of treatment, suggesting that this change pattern was important for treatment outcome.

It may be possible to make sense of why early sudden gains are sometimes reversed by considering depression spikes. Reversed sudden gains are defined as scores returning to a level that reflects giving up 50% or more of the improvement resulting from the sudden gain. Reversed sudden gains have usually been thought to indicate instability, i.e. fluctuations in symptoms or gains that do not last (Tang & DeRubeis, 1999b). However, the same individual can have more than one sudden gain during treatment (see for example: Doane et al., 2010; Kelly et al., 2005; Tang & DeRubeis, 1999b). Early session gains should be studied in the context of the whole treatment period, and if depression spikes occur more commonly for these patients it may result in better treatment outcome. This is a completely different vision

of the treatment process compared to the view that seems to follow from the Tang and DeRubeis's criteria of some patients experiencing one early "failed" sudden gain and a later "successful" sudden gain. As an example, a sudden gain of 14 points on the BDI is reversed if scores increase again by seven points. If depression symptoms then level off again by seven points over the course of treatment then criteria for depression spike have been met.

Aderka et al. (2012) noted in their meta-analysis that sudden gains occurring after the first treatment session had greater reversal rates and were thus less stable than sudden gains occurring later in treatment. Aderka et al. (2012) noted in their meta-analysis that sudden gains occurring after the first treatment session had greater reversal rates and were thus less stable than sudden gains occurring later in treatment. However, early sudden gains have been found to predict better treatment outcomes than sudden gains occurring later in treatment (Busch et al., 2006; Kelly et al., 2005). Furthermore, in Clerkin et al.'s (2008) study, sudden gains were associated with better treatment outcomes in CBT for panic-disorder, although approximately half of the gains were reversed, suggesting that the occurrence of sudden gains is more important for treatment outcome than the stability of the gains. Most studies, however, seem to exclude reversed sudden gains from statistical analyses, following Tang and DeRubeis's (1999b) sudden gains criteria, and we need studies that compare outcomes with and without reversed sudden gains over the treatment process as a whole to examine whether reversed sudden gains influence treatment outcomes.

Conclusion and future directions

Sudden gains are a common change pattern that has intrigued researchers studying treatment effectiveness, in part because they have the potential to reveal the mechanisms of treatment and perhaps since they seem to paint a picture of treatment characterized by crucial insights and critical sessions (Aderka et al., 2012; Tang & DeRubeis, 1999b). Sudden gains predict

better treatment outcomes (i.e. lower symptom levels) at the end of therapy for various disorders and sometimes also better long-term outcomes (Aderka et al., 2012). Understanding their causes could thus be important for enhancing treatment effectiveness.

Tang and DeRubeis (1999a; 1999b) noted that cognitive changes were more prominent in the therapy session preceding the sudden gains than in other therapy sessions and thus proposed that cognitive changes, resulting from cognitive restructuring, were the primary cause for sudden gains. This theory has received some support but has been criticized for overemphasizing specific treatment techniques in generating sudden gains and for only applying to one form of therapy, that is CBT (Aderka et al., 2012; Kelly et al., 2005). Sudden gains have been found to occur in various treatments that do not use cognitive restructuring and in non-treatment settings (Aderka et al., 2012), which suggests that common treatment factors and factors outside of therapy might also be influential in generating sudden gains. Future research is needed that systematically tease apart specific techniques and common factors, to shed light on how specific techniques and common factors interact over the course of treatment to promote sudden gains.

Various shortcomings have hampered research on sudden gains and it is important to establish a consensus among researchers in this field about how sudden gains should be estimated. The criteria used to identify sudden gains have been inconsistent between studies, which makes it difficult to compare findings. The RCI could be a reasonable estimate of whether a change is large enough to be considered a sudden gain, because it ensures that a change is sufficiently large to exceed the margin of measurement error and could be used for reference across studies (Jacobson, Roberts, Berns & McGlinchey, 1999). Tang and DeRubeis's second criterion (i.e. a gain has to represent at least 25% of the pre-gain session's BDI score) should probably be eliminated since it does not seem to add much to the two other criteria (e.g. Dour et al., 2013; Hardy et al., 2005; Stiles et al., 2003). The third criterion (i.e.

the mean BDI score of the three therapy sessions before the gain has to be significantly higher than the mean BDI score of the three therapy sessions after the gain) should be altered so that it allows for the assessment of sudden gains occurring early and perhaps also late in treatment. As Ilardi and Craighead's (1994) analysis revealed, the majority of symptom improvements in CBT for depression occurs very early in treatment and early changes further predict better treatment outcomes than changes occurring later in treatment (e.g. Busch et al., 2006; Kelly et al., 2005). Early changes therefore seem to convey important information on the change mechanisms in therapy and eliminating them from analyses leaves out important information on how changes occur in treatment.

Sudden gains share many similarities with two other change patterns, rapid early response and depression spikes, although they have for the most part, been examined separately. Sudden gains and rapid early responses have in common that they occur in brief time periods, are recurrent in psychotherapies, account for the majority of symptom improvements in treatment and predict better treatment outcomes. They seem to be entirely compatible with each other but are nevertheless evaluated differently, sudden gains with individual time courses from one session to the next and rapid early response with time courses of average symptom improvements. Similarly, depression spikes have been described as conceptually opposite sudden gains although comparing the change patterns reveal similarities. According to Tang and DeRubeis's (1999b) criteria, sudden gains are considered reversed if symptoms return to a level that reflects giving up half of the gain. The same patient can experience multiple sudden gains over the course of treatment and if a pattern emerges where a patient experiences sudden gains that reverses and then levels off, the criteria for depression spikes have been met. Hayes et al. (2007) found that depression spikes predicted better treatment outcomes but research is needed that examines whether reversed sudden gains affect treatment outcomes. If sudden gains are compatible to rapid early

response and depression spikes, perhaps research might benefit from studying them together instead of separately. As an example, by studying depression spikes we could make sense of why sudden gains sometimes reverse.

In conclusion, sudden gains seem to be an important phenomenon in psychotherapy with research findings showing that they predict better outcomes at the end of treatment. However, in order to determine how and why sudden gains are important for treatment outcome, consensus needs to be established on how sudden gains are estimated in research. Research on sudden gains has potentials for identifying critical sessions in psychotherapy and how specific treatment techniques and common factors interact in creating change, with important implications for enhancing treatment effectiveness.

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Sudden gains in cognitive-behavioral group therapy and group psychotherapy for social
anxiety disorder among college students

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Abstract

The present study examined sudden gains (SG) and its association with treatment outcome in a randomized-controlled trial, comparing cognitive-behavioral group therapy (CBGT) versus group psychotherapy (GPT) for social anxiety disorder (SAD). The latter treatment was designed to incorporate only nonspecific treatment factors. The objectives of this study were to examine SG in treatment for SAD and clarify further the mechanisms of SG. Participants were 39 college students, between 18 and 25 years old, with SAD as a primary diagnosis. Independent assessors evaluated symptom severity at baseline, post-treatment and follow-up with the Clinical Global Impression Scale (CGI) and the Liebowitz Social Anxiety Scale (LSAS). Social anxiety symptoms were assessed after each treatment session with the Brief Fear of Negative Evaluation Scale (BFNE), the Social Interaction Anxiety Scale (SIAS) and the Social Phobia Scale (SPS). SG criteria was based on previous criteria by Tang and DeRubeis (1999) and modified according to Kelly, Roberts and Ciesla (2005). A total of 17.9% of participants experienced SG during treatment. SG were neither associated with significantly greater improvements at post-treatment (although they were close to reaching statistical significance) nor at follow-up in either treatment. SG appeared at similar rates and magnitudes across both treatments. These results do not support the notion that treatments such as CBGT, which capitalize on specific treatment techniques, such as cognitive restructuring, lead to greater SG compared to treatments that contain only non-specific treatment factors.

Keywords: social anxiety disorder, social phobia, sudden gains, cognitive behavioral group therapy, group psychotherapy, nonspecific treatment factors

In recent years, there has been an 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 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. Similar results have been found in subsequent studies of psychotherapy for depression and also for anxiety (Aderka, Nickerson, Bøe & Hofmann, 2012).

Mechanisms of sudden gains

Although SG appear to be common, the mechanisms that determine SG remain largely unexplored (Aderka et al., 2012). Tang and DeRubeis (1999) found that cognitive changes preceded SG in cognitive behavioral therapy (CBT) for depression. They hypothesized that cognitive changes were a causal factor for sudden gains in accordance with Beck's cognitive mediation hypothesis (Tang & DeRubeis, 1999). Tang and DeRubeis (1999) hypothesis has received some support (Tang, DeRubeis, Beberman & Pham, 2005) although other studies have not found cognitive changes preceding sudden gains in CBT for depression (Kelly, Roberts & Ciesla, 2005) or in CBT for SAD (Hofmann et al., 2006). In Andrusyna, Luborsky, Pham and Tang's (2006) study, therapist interpretation accuracy predicted sudden gains in supportive-expressive therapy, but not cognitive changes. Vittengl, Clark and Jarrett (2005) observed that SG occurred in a pill placebo condition, which cannot be attributed to theory-driven techniques. Similarly, Kelly, Roberts and Bottonari (2007) reported SG occurring in a

non-treatment setting where participants used self-evaluation to assess their depression symptoms. These findings suggest that SG are not dependent upon the provision of therapy (Kelly, Roberts & Bottonari, 2007). However, non-specific treatment factors, common to all psychotherapies, such as therapist's empathy and warmth, might have been present and caused the SG.

In order to clarify the factors generating SG or the combination of factors, there is a need for studies that systematically tease apart specific and non-specific treatment techniques. Earlier research on the factors that lead to SG have been somewhat flawed. First, many research has not used a comparison group when examining SG mechanisms (see for example Tang & DeRubeis, 1999b). Second, research that compare SG in two treatments usually confound specific and nonspecific treatment techniques by comparing two treatments which consist of both types of techniques, such as two treatments derived from cognitive-behavioral theoretical models (see for example Tang, DeRubeis, Beberman & Pham, 2005). Third, studies on SG in non-treatment settings show that SG are not dependent on specific treatment techniques but do not clarify whether the SG resulting from non-specific treatment factors differ from the SG caused by specific treatment techniques in frequency and magnitude. The current study is the first to attempt to systematically tease apart the effects of specific treatment techniques on SG, by comparing SG in a treatment containing specific theory-driven techniques (a brief form of Heimberg & Becker's (2002) cognitive-behavioral group therapy) to a treatment containing only nonspecific treatment techniques, group psychotherapy (based on Yalom & Leszcz, 2005). SG were assessed from baseline-assessments until post-treatment assessments on the premise that since nonspecific factors, such as hope for improvement, are present before treatment begins, SG can occur between baseline assessments and the first treatment session (Busch, Kanter, Landes and Kohlenberg, 2006).

Sudden Gains in the Treatment of Social Anxiety Disorder

Only two studies have examined SG in the treatment of SAD. The first study compared cognitive-behavioral group therapy (CBGT), in accordance with Heimberg and Becker (2002), to exposure group therapy (EGT) and found that around 19% of participants experienced SG (Hofmann, Schulz, Meuret, Moscovitch & Suvak, 2006). SG occurred at similar frequencies in both treatments and predicted greater symptom improvement at post-treatment but not at follow-up. When sudden gainers in CBGT and EGT were compared, no differences in improvement were found. 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. Sudden gainers had significantly lower social anxiety symptoms at post-treatment and at 12 month follow-up than those who did not experience SG. SG were similar in frequencies in the two treatments but sudden gainers in CT had significantly lower levels of SAD at post-treatment than sudden gainers in IPT.

Study hypothesis

The current study assessed SG in a randomized controlled trial comparing CBGT and group psychotherapy (GPT) for SAD among college students (Bjornsson et al., 2011). The two treatments were comparable in duration and group size. GPT was based on Yalom & Leszcz (2005) and modified to be as closely matched on non-specific factors as possible to CBGT. Non-specific treatment techniques were emphasized in GPT, such as encouraging group members to support each other, and to take responsibility for the group process by e.g. giving each other constructive but accurate feedback. The therapist was prohibited from using specific techniques such as cognitive restructuring. Ratings of treatment integrity revealed excellent adherence and competence for both treatments, including that there was no evidence of the use of specific techniques like cognitive restructuring in the GPT conditioning. There

were no differences in outcome between the two treatments at post-treatment, which made it the first study in the CBGT literature in which the control group did at least as well as CBGT (Bjornsson et al., 2011).

This study tested the following two hypotheses. First, participants with SG, in both treatments, will experience significantly greater improvements than participants without SG at the end of treatment, but not at follow-up assessment. This is in line with the only other study of SG in a group treatment for SAD (Hofmann et al., 2006). Second, SG will be associated with greater symptom improvement in CBGT than GPT. This hypothesis is in line with previous studies, which indicate that the effects of SG are smaller in non-CBT interventions (Aderka et al., 2012; Bohn et al., 2013).

Method¹

Participants and Procedure

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

¹ Study methods are described in more detail in Bjornsson et al., 2011.

more sessions were excluded from the current report. The Institutional Review Board at CU approved this study.

Measures

Clinical interviews.

Structured Clinical Interview for DSM-IV, Non-patient Version (SCID). The SCID assesses current and lifetime Axis I disorders according to the DSM-IV. It has good median interrater and test-retest reliability, with K of .80 (American Psychiatric Association, 2000; First, Spitzer & Gibbon, 1995).

The Liebowitz Social Anxiety Scale (LSAS). The LSAS assesses 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 the severity of SAD symptoms and improvement over time in a clinical population (Zaider, Heimberg, Fresco, Schneier & Liebowitz, 2003).

Self-report questionnaires.

The Brief Fear of Negative Evaluation Scale (BFNE). The BFNE is a shortened version of the Fear of Negative Evaluation Scale (Leary, 1983). BFNE contains 12 items on a 5- point scale (from "Not at all characteristic of me" to "Extremely characteristic of me") 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 inter-item reliability (Cronbach's alpha = .90) and a test-retest reliability of .75 (Leary, 1983).

Social Interaction Anxiety Scale (SIAS). The SIAS assesses fear of social interactions with 20 items on a 5-point scale (from “Not at all characteristic or true of me” to “Extremely characteristic or true of me”) (Mattick & Clarke, 1998). This measure has good psychometric properties, including good inter-item reliability, Cronbach’s $\alpha = .86$. (Heimberg, Mueller, Holt, Hope & Liebowitz, 1993).

The Social Phobia Scale (SPS). The SPS measures the fear of being observed by others by 20 items on a 5-point scale (from “Not at all characteristic or true of me” to “Extremely characteristic or true of me”) (Mattick & Clarke, 1998). SPS has excellent psychometric properties, including Cronbach’s α of .90 (Heimberg et al., 1993).

Treatments

The two group treatments 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)

A briefer version of Heimberg and Becker’s CBGT (2002) was used in this study; it involved eight 2-hour sessions instead of twelve 2.5-hour sessions. CBGT consisted primarily of psychoeducation and behavioral experiments in which cognitive restructuring was integrated with 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). It was designed to consist only of nonspecific treatment factors and to be structurally equivalent to CBGT with regard to variables such as number and length of sessions. Specific techniques, such as exposure and cognitive restructuring were proscribed. Group members were asked to take responsibility for group

discussions, and to share their impressions of each other in a constructive way. They were also asked to come up with their own homework assignments. The therapist's role was primarily to facilitate communication by encouraging group members to participate in the manner described above and to explore what they could learn about themselves from other group member's feedback.

Procedure

Potential participants were screened in a phone interview. Each participant read and signed the informed consent before baseline assessment was conducted. Participants who met study criteria (described above) were invited to participate in the study and were randomized to either treatment. The therapists met all participants individually to discuss the treatments and address any fears or concerns the participant may have had. 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 assignment) conducted post-treatment assessments. They were all advanced clinical psychology graduate students with extensive training in the interviews used

Definition of Sudden Gains

We assessed SG with the BFNE, SPS and SIAS, each of which assesses different aspects of SAD; the cognitive components of social anxiety (BFNE), the fear of being observed by others (SPS) and the fear of social interactions (SIAS). SG criteria were based on Tang and

DeRubeis (1999) criteria 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 an eight point reduction on BFNE, a nine point reduction on SIAS and an eight point reduction on SPS from one week to the next. The *reliable change index* (RCI) (Jacobson & Truax, 1991), was computed to arrive at these estimates, by dividing mean change score (post-treatment – baseline) by the standard error of change, in line with previous research (e.g. Bohn et al., 2013; Hofmann et al., 2006; Stiles et al., 2003). *Criterion 2*. The gain must be relative to the previous score and consist of 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 variations 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 treatment. This criterion was used to include SG at the outset of treatment, which we considered to be the baseline assessment (Busch et al., 2006). Consistent with the literature, SG were considered to have reversed if any subsequent BFNE, SIAS or SPS scores 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).

Statistical Analyses

SG criteria were established by comparing total scores on the BFNE, SPS and SIAS between adjacent sessions over the entire course of treatment for each participant. The primary outcome measures were the LSAS and CGI, which were conducted at baseline, post-treatment and at 3-month follow-up. 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 for comparisons between sudden gainers and non-gainers and for the effect of SG in the two treatments, in which SG or

SG and group were the independent variables, pre-treatment scores were the covariates and either post-treatment or follow-up LSAS scores were the dependent variables. Effect sizes (partial eta-squared) of the improvement at post-treatment and at follow-up were calculated.

Results

Occurrence of Sudden Gains

A total of 11 SG were found for seven out of 39 participants (17.9%). Three participants experienced one SG and four experienced two SG. Most SG occurred after baseline assessment ($n = 3$) and the second treatment session ($n = 3$). Two of the baseline SG occurred in CBGT and one in GPT. Four of the eleven SG, including the baseline SG, were reversed (36.4%) for three participants, one in CBGT and one in GPT. Sudden gainers without reversals were six (15.4%).

Two SG occurred on BFNE; one in CBGT and one in GPT. 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 ($M = 11.50$, $SD = 2.12$). Thus, the number of SG were similar across the two treatments.

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 SIAS ($M = 28.71$, $SD = 11.91$) compared to those without SG ($M = 40.28$, $SD = 13.60$); $t(37) = 2.07$, $p = .050$.

Comparison between Sudden Gainers and Non-gainers

Sudden gainers represented 26% of total treatment responders (defined as receiving either a “much improved” or “very much improved” on the CGI improvement scale) across both treatment conditions, but were not more likely to be associated with treatment response, as

measured by the CGI; $\chi^2(1) = 2.52, p = .11$. Eighty-six percent of participants with sudden gains were treatment responders compared to 53% of 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 scores 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).

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).

Comparison between Sudden Gainers in CBGT versus GPT

A total of four of 17 participants in CBGT (23.5%) experienced SG compared to three out of 22 (13.6%) participants in GPT. The difference was not statistically significant, $\chi^2(1) = .64, p = .425$. Sudden gainers in CBGT improved on average by 22.20 ($SD = 6.46$) on LSAS, compared to 16.7 ($SD = 4.16$) in GPT, from baseline to post-treatment. Only two of the four participants with SG in CBGT were present at the follow-up assessment. Their average improvement was 28 ($SD = 4.24$) on the LSAS from baseline to follow-up, compared to 19 ($SD = 6.24$) in GPT.

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 were neither statistically significant at post-treatment, $F(1, 34) = .14, p = .712$, partial $\eta^2 = .00$ (see Figure 2) nor at follow-up, $F(1, 30) = 1.12, p = .299$, partial $\eta^2 = .04$ (see Figure 3).

Discussion

Sudden gains occurred for 17.9% of participants (where four of those showed a reversal of their gain). These findings are similar to the other two previous studies of SG in treatments for SAD, where 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 in treatment for depression, which has been around 40% in most studies (Aderka et al., 2012).

We hypothesized that participants in both CBGT and GPT with SG would experience greater symptom improvements at post-treatment, but not at follow-up. This hypothesis was not supported. The difference in symptom improvement (as measured by the LSAS) 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 are similar to the findings from the only other study of SG in CBGT for SAD (Hofmann et al., 2006), in which 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 in our study, only five showed up at the follow-up assessment, which may have impacted the findings. 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 the 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. The majority of participants with SG were treatment responders, as measured by CGI but were not more likely to be treatment responders than non-gainers. However the difference between sudden gainers and non-gainers in treatment response was close to statistical significant and might have been significant in a larger sample.

We further hypothesized that SG would be associated with greater symptom improvement in CBGT than GPT. This hypothesis was not supported. We did not find a difference in the effect of SG on treatment response in the two groups, which goes against the meta-analysis of Aderka et al. (2012) which stated that the effects of SG were smaller in non-CBT interventions. It is of course entirely possible that this difference was not found in this study because of limitations to statistical power, and it is clear that the study should be replicated with a larger sample. However, this is the only study that we know of in which specific and non-specific treatment techniques were systematically teased apart. It is certainly worth exploring whether group treatments could make better use of non-specific techniques than has been done to date. Further studies are needed to clarify the relative contribution and the interaction between specific and non-specific treatment factors in generating SG in the treatment process.

The study had limitations, which are important to note. First, as already mentioned, the sample size was relatively small and the statistical power was therefore limited. This fact made it more difficult to detect differences between gainers and non-gainers and differences between sudden gainers in the two treatments. It may 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 right from the outset. Finally, the sample consisted of college students with SAD as a primary diagnosis, and the findings may not generalize to all individuals with SAD.

Summary and Future Directions

Sudden gains are leaps in symptom improvement between two adjacent treatment sessions that have been linked to better treatment outcomes at post-treatment and in some studies at follow-up measures (Aderka et al., 2012). Past research has failed to clarify the mechanisms that result in SG, in part because studies usually confound specific and nonspecific treatment techniques. This study is the first, to our knowledge, to compare SG in CBGT to a group therapy containing only nonspecific treatment factors (GPT). The main findings are that sudden gains were similar in this study as in past studies of social anxiety disorder, and seem to have similar effects on treatment response as in the only other group treatment study of SAD (Hofmann et al., 2006). We did not find differences between CBGT and GPT with regard to treatment outcome.

Future directions include replicating this study with a larger sample, which would facilitate analysis of comparisons between gainers and non-gainers, and whether there are differences between CBGT and GPT with regard to SG and their association with treatment response. We need further studies on whether certain treatment techniques (specific or not or a combination of the two) are more likely to lead to SG. Furthermore, such work should have treatment implications. As one example, Aderka et al. (2012) found that early SG are often reversed later in the treatment process. However, such reversals were not necessarily inevitable. It is possible 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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Tables and Figures

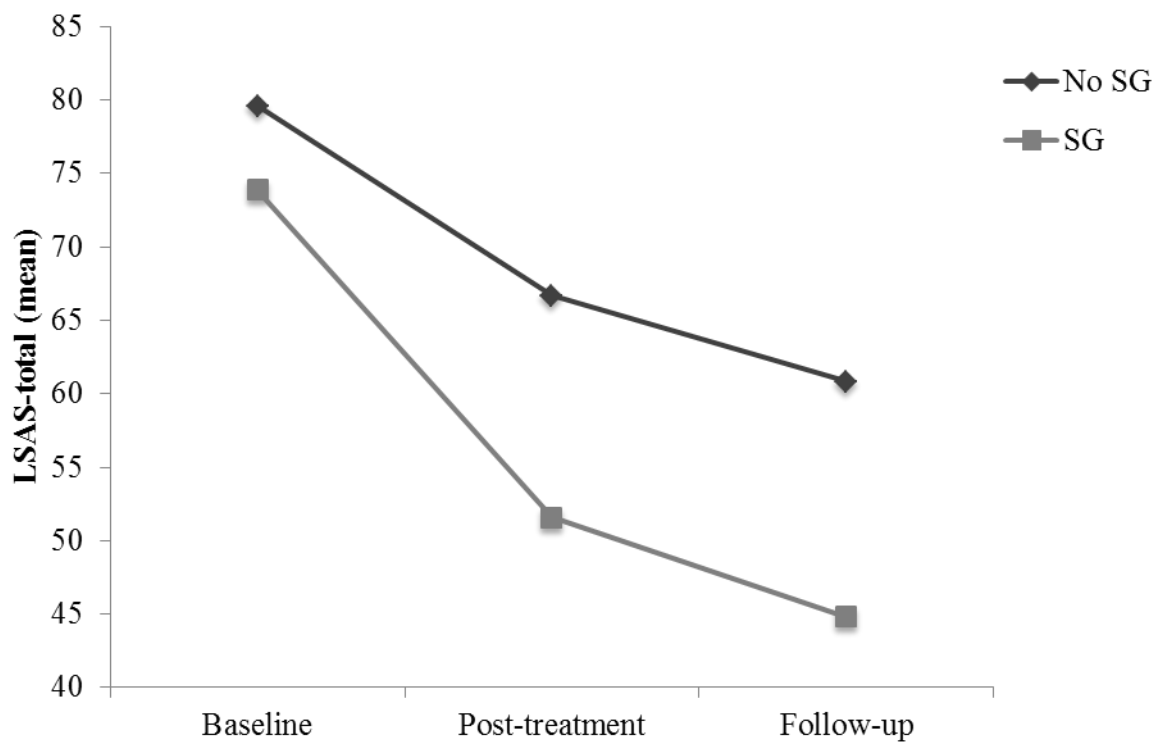


Figure 1. Mean LSAS-total scores between sudden gainers and non-gainers at baseline, post-treatment and follow-up.

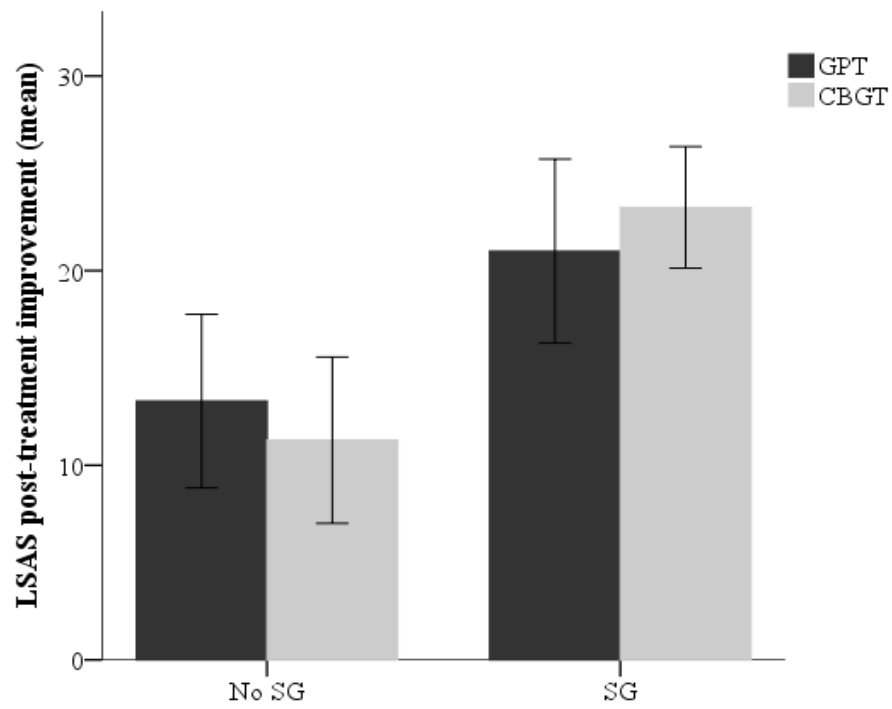


Figure 2. Mean LSAS post-treatment improvement (with error bars representing standard errors) comparison between CBGT and GPT among individuals who showed sudden gains and those who did not.

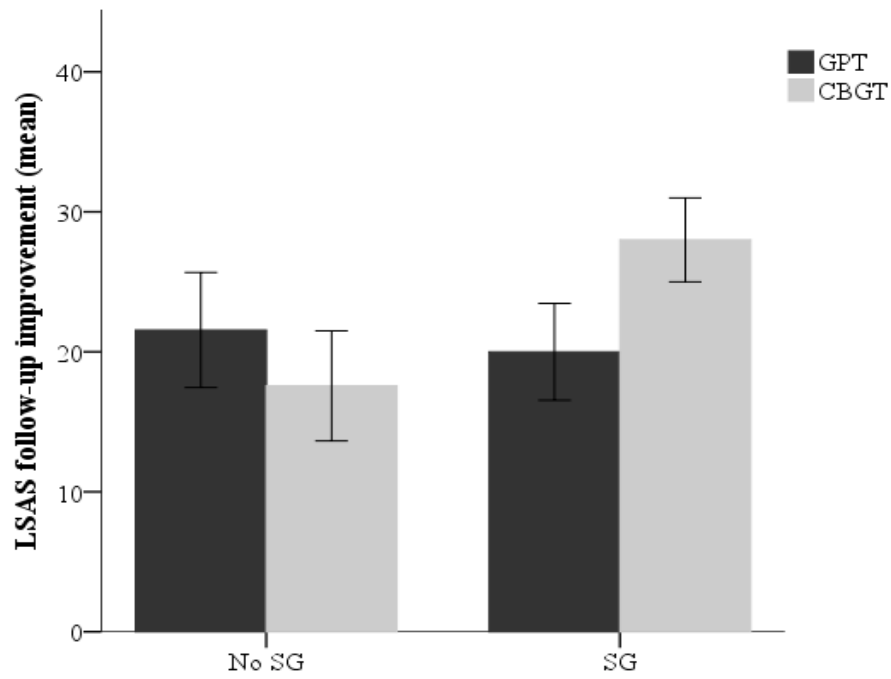


Figure 3. Mean LSAS follow-up improvement (with error bars representing standard errors) comparison between CBGT and GPT among individuals who showed sudden gains and those who did not.

Variable	Full sample (n = 39)	Sudden gains (n = 7)	No sudden gains (n = 32)	Statistics	<i>p</i>
	<i>N (%) or M (SD)</i>	<i>N (%) or M (SD)</i>	<i>N (%) or M (SD)</i>		
Gender				$\chi^2(1) = .24$.62
Men	20 (51.3%)	3 (42.9%)	17 (53.1%)		
Women	19 (48.7%)	4 (57.1%)	15 (46.9%)		
Age (years)	19.69 (1.5)	19.14 (1.1)	19.81 (1.5)	$t(37) = 1.11$.28
Ethnicity				$\chi^2(3) = .57$.90
Asian	4 (10.3%)	1 (14.3%)	3 (9.4%)		
Caucasian	34 (87.2%)	6 (85.7%)	29 (87.5%)		
Latino	1 (2.6%)	0 (0%)	1 (3.1%)		
Clinical interviews					
LSAS	78.56 (23.22)	73.86 (17.77)	79.59 (24.37)	$t(37) = .59$.56
CGI	4.87 (.73)	4.86 (.69)	4.88 (.75)	$t(37) = .06$.95
Self-report measures					
BFNE	47.67 (7.21)	43.43 (7.98)	48.59 (6.81)	$t(37) = 1.76$.09
SPS	55.00 (11.30)	52.29 (12.67)	55.59 (11.11)	$t(37) = 0.70$.49
SIAS	38.21 (13.91)	28.71 (11.91)	40.28 (13.60)	$t(37) = 2.07$.05

Table 1. Background variables and clinical characteristics at baseline