Sudden gains in Behavioural Activation for depression

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Abstract

Sudden gains have been linked to improved outcomes in cognitive behaviour therapy for depression. The relationship between sudden gains and outcome is less clear in other treatment modalities, including interpersonal psychotherapy and supportive expressive therapy, which may indicate different mechanisms of change between treatment modalities. The current study examined sudden gains in adults meeting diagnostic criteria for depression (N = 40) offered up to 12 sessions of behavioural activation treatment. Sudden gains were found in 42.5% of the sample. Sudden gains occurred early (median pre-gain session 2) and were related to outcome: those who experienced a sudden gain had significantly lower post-treatment scores on the PHQ-9. Furthermore, the proportion meeting the reliable and clinically significant change criteria at end of treatment was higher in the sudden gain group. These findings highlight the importance of understanding the mechanisms by which sudden gains relate to therapy outcome in behavioural activation.

Keywords:
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Sudden gains in therapy for depression

Tang and DeRubeis (1999) identified that for some patients a sizeable proportion of their overall response to cognitive behavioural therapy (CBT) for depression, sometimes in excess of 50%, could be attributed to a marked decrease in symptoms occurring between one session and the next. They termed these rapid, dramatic changes in symptoms 'sudden gains'. They reported that these sudden gains occur in a sizeable minority of patients (39%), that the improvements tended to be maintained, and that those people who made a sudden gain tended to have lower scores at post-treatment and follow-up than those who had not. A number of subsequent studies of CBT for depression have broadly corroborated these initial findings (Hardy et al., 2005; Tang, DeRubeis, Beberman & Pham, 2005; Tang, DeRubeis, Hollon, Amsterdam & Shelton, 2007). Research into sudden gains has expanded to problems other than depression, such as panic disorder (Clerkin, Teachman & Smith-Janik, 2008) and PTSD (Doane, Feeny & Zoellner, 2010) and to therapies other than CBT (e.g. interpersonal psychotherapy; Kelly, Cyranowski & Frank, 2007). A recent meta-analysis (Aderka, Nickerson, Bøe & Hofmann, 2012) concluded that individuals who experience sudden gains during therapy had significantly greater improvement at end of treatment and follow-up than those who did not.

Tang and DeRubeis (1999) have argued that sudden gains are caused by cognitive changes, in line with Beck's model (Beck, Rush, Shaw & Emery, 1979). This conclusion is debated by Ilardi and Craighead (1999) who argue that the cause of these sudden improvements in symptoms relates to non-specific therapy effects. Of relevance to this argument is the timing of sudden gains, which tend to occur early in therapy (e.g. median pre-gain session 5, Tang & DeRubeis,1999) although differences in the timing of gains have been reported (e.g. Busch, Kanter, Landes & Kohlenberg, 2006). The importance of understanding the mechanisms of change in CBT and other psychological treatments has led to considerable interest in and investigation of the sudden gain phenomena (e.g. Hardy et al., 2005; Stiles et al., 2003; Tang et al., 2005; Tang et al., 2007); however, only a few studies have investigated therapies other than CBT.

Kelly, Cyranowski and Frank (2007) point out that the research available raises an intriguing possibility that the relationship between sudden gains and outcome may differ between therapeutic modalities. Tang, Luborsky and Andrusyna (2002) found that sudden gains occur in supportive expressive therapy, but they tended to be less stable than those in CBT: those who experienced a sudden gain had better outcomes post-treatment, but there was no difference between the groups at 6 month follow up. Kelly et al. found that sudden gains occur in interpersonal therapy but there was no link between the occurrence of a sudden gain and outcome measured at post-treatment or follow-up. The meta-analysis of Aderka et al. (2012) found similar rates of sudden gains in non-CBT and CBT treatments, but while the presence of a sudden gain appeared to predict improvement at post-treatment in CBT, the relationship appeared less clear for other therapies. These results could indicate, as Kelly et al. argue, that the mechanisms of change are different across different treatment modalities.

Few studies have examined the role of sudden gains in behavioural activation (BA) treatments for depression. Behavioural activation is based on operant conditioning principles and suggests that depression results from a change in environmental context that alters the person's access to sources of positive reinforcement. The first published study of the sudden gain phenomena in the BA treatment of depression used a sample of patients with cancer (Hopko, Robertson & Carvalho, 2009). Hopko et al. compared two behavioural approaches and found similar rates of sudden gain (50%) in both treatments, and that the sudden gain patients had significantly higher remission rates at end of therapy. A subsequent study identified the occurrence of sudden gains in BA treatment of depression in a community sample (Hunnicutt-Ferguson, Hoxha & Gollan, 2012). Hunnicutt-Ferguson et al. found 35.7% of their sample experienced a sudden gain and that these patients had significantly lower self-reported depression at the end of treatment compared with those who did not make a sudden gain.

The aim of the current study is to add to the small but growing literature on sudden gains in behavioural activation treatments for depression. While there is a consistent relationship between sudden gains and improved outcome in CBT treatments, the relationship is less consistent in non-CBT treatments. There is some preliminary

evidence that in BA, as in CBT, sudden gains are linked to improved outcomes, but further studies are required to establish whether the relationship is as consistent as it is in CBT. The current study aimed to establish whether there is a relationship between sudden gains and outcome in a brief BA treatment, delivered in a British primary care setting.

Method

Participants

We selected the sample from a 'phase II' randomised controlled trial of behavioural activation delivered by generic mental health workers compared to usual care for adults with depression (Ekers, 2011). Participants were aged 18 or over and were recruited from either general practice directly or primary care mental health services. A computer-based assessment, the Clinical Interview Schedule - Revised, was used to confirm ICD 10 diagnosis of depression. Exclusion criteria included suicidal risk, psychotic symptoms, diagnosis of bipolar disorder, organic brain disease or the use of alcohol/non-prescription drugs requiring clinical intervention.

Measures

CIS-R. The Clinical Interview Schedule - Revised is a structured interview which covers 14 symptom clusters (Lewis, 1992). Additional questions allow for the diagnoses of ICD-10 disorders. The CIS-R has acceptable psychometric properties (Lewis, 1992).

PHQ-9. The PHQ-9 is a nine-item self-report measure of depression (Kroenke, 2001). Each item is rated on a 0-3 scale based on the frequency of depressive symptoms over the last two weeks, and is summed to give a total score (range 0-27), with high scores indicating more severe depression. We defined improvement on this measure using the reliable and clinically significant change criteria reported in McMillan, Richards and Gilbody (2010). Reliable improvement was estimated as an improvement in scores of ≥5 points from pre- to post-treatment and clinically significant change required a move from a clinical range (≥10) at pre-treatment to a

post-treatment score in the non-clinical range (≤9). For a participant to be classified as improved they had to meet both of these criteria.

Procedure

Participants were randomised, with stratification for baseline depression severity, to either behavioural activation (N = 24) or usual care (N = 23). Participants randomised to the control condition were assigned to the care of their GP or primary care mental health worker and if necessary offered interventions in line with normal practice. At the end of the main treatment phase, these participants were then offered behavioural activation based on the manual used in the intervention arm. The behavioural activation intervention was based on two previously developed behavioural approaches and is described in more detail below (Hopko, 2003;Martell, 2001).

For the purpose of the analyses reported here, the treatments received by the two groups are analysed together. We excluded two participants in the usual care arm who were no longer in the clinical range (≥10) on the PHQ-9 at the start of their treatment from the analysis and five participants in the usual care arm who did not start treatment. The final sample, therefore, consisted of 40 participants (original behavioural activation arm: N = 24; usual care followed by behavioural activation: N = 16). There were no significant differences between these two groups in terms of gender (treatment: 65.2% female; usual care: 58.8% female; Fisher's Exact Test, p = 0.75), age (treatment: M = 46.4, sd = 10.4; usual care: 44.6, sd = 10.2; t = 0.56, df = 0.38, p = 0.58) or number of completed sessions (treatment: M = 8.3, sd = 4.1; usual care: M = 9.2, sd = 3.7; t = -0.77, df = 38, p = 0.44). Although the difference between the two groups in terms of pre-treatment PHQ-9 score was not significant (t = 1.30, df = 38, p = 0.20), the usual care group (M = 17.6, sd = 4.5) scored approximately half a standard deviation lower than the treatment group (M = 19.5, sd = 4.3) on the PHQ-9 at pre-treatment. This may reflect the improvement that the usual care group experienced during the period in which they received usual care before behavioural activation.

The PHQ-9 was completed at the start of each treatment session and is therefore used as the basis of the assessment of sudden gains in depressive symptoms. Further details of the procedure can be found in Ekers et al. (2011).

Treatment

Behavioural activation consisted of up to 12 one-hour face-to-face sessions. The aim of the treatment was to increase contact with stable and diverse sources of positive reinforcement through the scheduling of activities and to reduce the frequency of negatively reinforced avoidant behaviours. Sessions included the development of a shared formulation, self-monitoring, identifying 'depressed behaviours', developing alternative goal orientated behaviours, and activity scheduling. Sessions also covered the role of avoidance and rumination through functional analysis of these behaviours and the development of alternative responses. The treatment manual is available from author DE on request. The treatment was delivered by two qualified mental health nurses with no previous formal training in the delivery of psychological treatment.

Audio-recordings of 20% of sessions were rated to establish adherence to the behavioural activation manual. Sessions were rated by independent Cognitive Behaviour Therapists with experience of both cognitive and behavioural treatments for depression. Raters assessed session and homework content against the behavioural activation protocol. All of the assessed sessions were rated as behavioural activation dominant in relation to session and homework content; none were rated as having other therapeutic modalities dominant. All rated sessions were rated overall as examples of behavioural activation. Further details are provided in Ekers et al. (2011).

Data analysis

Definition of a sudden gain

Tang and DeRubeis (1999) defined a sudden gain as a between-session improvement in symptoms that met three criteria. First, the gain has to be large in absolute terms, which for the BDI they operationalised as an improvement of 7

points or more. For studies of sudden gains that have not used the BDI, the reliable change index (Jacobson & Truax, 1991) for the measure has been used in all instances. We used the reliable improvement criterion of ≥5 points for the PHQ-9 for this criterion (McMillan, Richards & Gilbody, 2010). Secondly, the improvement has to be large relative to symptom severity before the gain, which Tang and DeRubeis operationalised as an improvement that was at least 25% of the score in the pre-gain session. We adopted this criterion unchanged.

The third criterion, that the improvement must be large relative to symptom fluctuation before and after the gain, has proved the most difficult to operationalise. The original definition used a t-test to establish that the three scores before the sudden gain were significantly higher than the three scores after it. However, this has been criticised because it prevents the examination of very early or very late gains. Subsequent definitions have required only two sessions before or after the gain to allow the examination of early and late gains. The use of a t-test for this criterion has also been questioned on statistical grounds because the comparison is based on repeated measurement of the same person over time, which violates the assumption of independence of errors. Subsequent definitions have instead required that the mean score of the three sessions before the sudden gain (or two for early sudden gains) is higher than the mean of the three sessions after the sudden gain (or two for late sudden gains), where higher is defined as at least 2.78 times the pooled standard deviation (with 2.78 rather than the usual 1.95 selected because of the small n). We used this re-worked form of Tang and DeRubeis' stability criterion in the current study.

Although the requirement of two rather than three sessions allows an examination of earlier and later sudden gains than the earlier definition, as Kelly et al. (2005) point out this still does not allow the examination of gains occurring after the first session or penultimate session. Kelly et al. (2005) proposed an alternative version of the stability criterion, which states that the improvement must be at least 1.5 standard deviations of the mean of the person's session-by-session scores. Therefore we adopted the definition of Tang and DeRubeis to examine sudden gains occurring from between the second and third sessions onwards, but to permit the exploration of very early sudden gains used the definition of Kelly et al. (2005) for gains

occurring between the first and second session. This method has also been used by Hopko et al. (2009) and Clerkin et al. (2008).

A reversal in a sudden gain was defined as occurring when a participant's PHQ-9 score increased by 50% or more of the sudden gain improvement (this is the definition used by Tang & DeRubeis and has been consistently adopted unchanged in the literature).

Statistical analysis.

The sudden gain and no-gain groups were compared using t-test for continuous outcomes and Fisher's Exact test for dichotomous ones. Baseline differences between the groups were controlled for using a multiple regression.

Results

Sudden gain characteristics

42.5% (17/40) of the sample experienced at least one sudden gain (see Table 1). Of the 17 participants who experienced a sudden gain, 3 experienced more than one gain during treatment. A reversal of the sudden gain occurred for 2 out of the 17 participants. The median pre-gain session was session 2 (interquartile range = 1 – 4). Only two of the first sudden gains occurred after session 4 (gain between sessions 5-6 and 8-9). Of the three people who experienced more than one sudden gain during treatment, two experienced the second gain after session four (second gain sessions 5-6 and 10-11).

The mean improvement in PHQ-9 scores for the sudden gain was 7.4 (sd = 2.2). For the sudden gain group, the mean overall improvement from pre- to post-treatment was 13.9 (sd = 5.2); therefore, the sudden gain improvement represents approximately 50% of the overall improvement in PHQ-9 scores for those who made a sudden gain.

Sudden gains group vs. no-gain group characteristics

Table 2 summarises the pre-treatment and other characteristics of the sudden gain (N = 17) and no-gain groups (N = 23). There were no significant differences between the two groups in terms of age (sudden gain group: M = 45.5, sd = 10.6; no-gain group: M = 45.8, sd = 10.2; t = 0.09, df = 38, p = 0.93). For gender, the two groups were broadly comparable: the proportion female was not significantly different (sudden gain group = 58.8% female; no-gain group = 65.2%; Fisher's Exact, p = 0.75).

There was no significant difference in pre-treatment PHQ-9 scores (sudden gain group: M = 19.4, sd = 3.9; no-gain group: M = 18.2, sd = 4.8; t = -0.80, df = 38, p = 0.43). However, the difference in the number of treatment sessions did approach significance (sudden gain group: M=9.88, sd=3.2; no-gain group: M = 7.9, sd = 4.1; t = -1.73, df = 38, p = 0.092).

Sudden gains and post-treatment outcomes

Those who made a sudden gain were more likely to have improved at post-treatment. At post-treatment the sudden gain group had a mean score of 5.3 (sd = 3.6) on the PHQ-9, which was significantly lower than that of the no-gain group (10.2, sd = 6.8) (t = 2.92, df = 35.0, p = 0.006). 82.4% of the sudden gain group met criteria for reliable and clinically significant change, which was significantly higher than the no-gain group (47.8%) (Fisher's Exact: p = 0.046).

Although there were no significant differences on pre-treatment and other descriptive variables between the sudden gain and no-gain group, the difference in the number of treatment sessions did approach significance (t = -1.73, df = 38, p = 0.092). A multiple regression was run to examine whether sudden gain status predicted post-treatment PHQ-9 scores after controlling for this potential confound. Both variables (number of sessions, sudden gain group) were entered together in a single step. Whilst the number of sessions approached significance in the regression (β = - 0.28, p = 0.073), sudden gain group status continued to predict variation in post-treatment scores after controlling for differences in number of sessions (β = -0.32, p = 0.037).

Discussion

Sudden gain characteristics

We found that 42.5% of participants receiving behavioural activation experienced sudden gains and that those experiencing sudden gains had better outcomes post-treatment than those who did not. The rates of sudden gains in our study are similar to those found in the samples of Hopko et al. (2009; 50%) and Hunnicutt-Ferguson et al. (2012; 35.7%). These rates are similar to those originally reported for CBT for depression by Tang and DeRubeis (1999; 39%) despite the difference in the length of therapy offered (up to 12 sessions in the current study cf. up to 20 in the studies analysed by Tang & DeRubeis).

Our results support the evidence suggesting that sudden gains in behavioural treatments occur earlier than in CBT: the median session prior to the sudden gain in the current study was session 2. Hopko, Robertson and Carvalho (2009) report that 10 of the 13 sudden gains in their sample had occurred by session 4 and Hunnicutt-Ferguson et al. (2012) report session 1 as the median pre-gain session. In contrast, a median pre-gain session of 5 is reported in several of the CBT studies (Hardy et al., 2005; Tang & DeRubeis, 1999; Tang et al., 2007) and as late as session 10 in one study (Busch, Kanter, Landes & Kohlenberg, 2006). However, as described below, it is possible that the observed difference in the timing of sudden gains between CBT and BA is an artefact of different definitions of these gains.

Impact of sudden gains

Our findings contribute to the growing evidence that sudden gains in behavioural activation are linked to improved outcomes at post-treatment. In the current study, those who experienced a sudden gain had significantly lower post-treatment scores on the PHQ-9 than those who had not experienced a sudden gain. Furthermore, the proportion meeting the reliable and clinically significant change criteria was higher in the sudden gain group. Sudden gains in this sample contribute around 50% of the overall improvement in PHQ-9 scores. Whilst a sudden large improvement in symptoms obviously will contribute to an individual's response to treatment overall, the research indicates that not all sudden gains are related to an improved outcome:

the relationship between sudden symptom improvement and eventual outcome is not established in non-CBT treatments (Aderka et al. 2012).

Limitations, future directions and conclusion

There are a number of limitations of the current study. The current study examines post-treatment outcome, but does not establish whether sudden gains are linked to improved outcome at follow-up. Whilst Hopko et al.'s (2009) study showed that those participants with sudden gains had improved outcomes at 3 months follow up, neither Hunnicutt-Ferguson et al. (2012) nor the current study include follow up data, so this finding needs to be replicated and extended. The stability of the relationship between gain and outcome has been shown at follow up after CBT for depression (Hardy et al., 2005) but is less clear in the longer term (Tang et al., 2002). Future research should evaluate follow up at 6-12 months and attempt to establish whether there is a difference in relapse rate for the sudden gains group.

The differences in the timing of sudden gains in the BA versus CBT research should be interpreted with some caution: it is possible that differences in the reported characteristics of sudden gains across treatments are due to differences in the definition used. The definition of sudden gains in the current study differed from one of the previous studies of the impact of sudden gains in BA treatment: Hunnicutt-Ferguson et al. (2012) used Busch et al.'s (2006) adaptation of Tang and DeRubeis's definition. As Hunnicutt-Ferguson et al. note, modifying the criteria for sudden gains or using different measures may lead to differing results, including the timing of the sudden gains. Future research is needed to examine whether different treatments do show differences in the timing of the sudden gains when the definition of a gain is the same across treatments.

If sudden gains are found to occur earlier in BA than CBT when a consistent definition is used across treatments, it would be useful to examine hypotheses about this in future research. Hunnicutt-Ferguson et al. point out that early BA sessions focus on an increase in rewarding activities, which should lead to an early decrease in depressive symptoms. In support, a recent review of the current evidence supporting BA treatments for depression (Dimidjian, Barrera, Martell, Munoz & Lewinsohn, 2011) concludes that there is evidence to support the relationship

between activity level and mood, although acknowledges that more work is needed to establish the temporal relationship between these. Given that CBT treatment of depression also usually starts with activity scheduling, this may not explain any differences in the timing of sudden gains. One possibility is that the characteristics of the BA formulation may contribute to an increased likelihood of early sudden gains. Formulations of a person's difficulties are typically presented early on in both BA and CBT, but the simplicity of the BA formulation with a focus on what behavioural changes are required may be sufficient to lead to alterations in a person's behaviour even before formal between-session tasks, such as activity scheduling, are agreed.

To examine this possibility, future research is required to examine what happens within and between sessions immediately either side of a sudden gain. Both of the previous studies of sudden gains in BA have concluded that engagement with behaviour change may be responsible for the marked between-session changes in mood during BA treatment (Hopko et al.,2009; Hunnicutt-Ferguson et al., 2012). Hunnicutt-Ferguson et al. (2012) argue that the gains coincide with engagement with goal-driven activity whilst Hopko et al. (2009) suggest that sudden gains could reflect "self-activation in the absence of therapist guidance" (p. 353). There is as yet no evidence to support this, but future research should examine the level of activation of patients who make sudden gains.

Current research has established a link between sudden gains and improved outcome for CBT, a link that does not appear to hold for some other therapeutic modalities. This study adds to the evidence that in BA sudden gains also appear to be related to an improved outcome. It seems relevant that sudden gains which are predictive of good outcome are usually found early on in the course of therapy (Stiles et al., 2003), as sudden gains in BA may occur earlier in therapy. There is a need for future research to focus on the mechanisms by which sudden gains occur in CBT and BA. Specifically, it will be important to establish whether the mechanisms responsible for the sudden gain differ between BA and CBT.

This would be a timely direction for future research into sudden gains in BA: Dimidjian et al.'s (2011) review of the current state of the research into BA calls for increased research into the process of change in BA in order to optimise treatment outcome. The sudden gain phenomenon provides a useful opportunity to test

hypotheses regarding the mediators of change. Examination of the sessions immediately before and after the sudden gain could offer an important insight into how BA works. Furthermore, given the relationship to outcome in both CBT and BA, understanding the mechanisms by which a sudden gain is linked to outcome could help improve treatment efficacy across both therapies.

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Table 1: Sudden gain characteristics of sample

Sudden gain characteristic	Result
At least one sudden gain	42.5% (17/40)
More than one sudden gain	17.6% (3/17)
Sudden gain reversed	11.8% (2/17)
Median (interquartile range) of pre-gain session for first gain	2 (1-4)
Mean (standard deviation) sudden gain improvement on PHQ-9	M = 7.4 (sd = 2.2)
Mean (standard deviation) overall improvement on PHQ-9 for sudden gain group	M = 13.9 (sd = 5.2)
Sudden gain as a % of overall improvement	53.0%

Table 2: Characteristics of the sudden gain and no-gain groups

Characteristic	Sudden gain group (N = 17)	No-gain group (N = 23)	Statistic
Age	M = 45.5, sd = 10.6	M = 45.8, sd = 10.2	t = 0.09, df = 38, p = 0.93
Gender	% female = 58.8%	% female = 65.2%	Fisher's Exact, p = 0.75
Pre-treatment PHQ-9 score	M = 19.4, sd = 3.9	M = 18.2, sd = 4.8	t = -0.80, df = 38, p = 0.43
No. of treatment sessions	M = 9.88, sd = 3.2	M = 7.9, sd = 4.1	t = -1.73, df = 38, p = 0.092

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