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# 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.



## 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, 2007); however, only a few studies have investigated therapies other than CBT.

Kelly et al. (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

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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, Richards, McMillan, Bland, & Gilbody, 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, Pelosi, Araya, & Dunn, 1992). Additional questions allow for the diagnoses of ICD-10 disorders. The CIS-R has acceptable psychometric properties (Lewis et al., 1992).

#### PHQ-9

The PHQ-9 is a nine-item self-report measure of depression (Kroenke, Spitzer, & Williams, 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 $\geq$5 points from pre- to post-treatment and clinically significant change required a move from a clinical range ($\geq$10) at pre-treatment to a post-treatment score in the non-clinical range ($\leq$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 ($\geq$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