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Accelerated Outpatient Individual Cognitive Behavioural Therapy for Social Anxiety Disorder: A Preliminary Pilot Study

Bethany M. Wootton*

Discipline of Clinical Psychology, Graduate School of Health, University of Technology Sydney, Ultimo, 2007, Australia and Department of Medicine (Psychology), University of Tasmania, Locked Bag 30, Hobart, TAS, 7001, Australia

Alexandra Hunn*

Department of Medicine (Psychology), University of Tasmania, Locked Bag 30, Hobart, TAS, 7001, Australia

Annabelle Moody

Department of Medicine (Psychology), University of Tasmania, Locked Bag 30, Hobart, TAS, 7001, Australia

Bethany R. Lusk

Department of Medicine (Psychology), University of Tasmania, Locked Bag 30, Hobart, TAS, 7001, Australia

Valerie A. Ranson

Department of Medicine (Psychology), University of Tasmania, Locked Bag 30, Hobart, TAS, 7001, Australia

Kim L. Felmingham

School of Psychological Sciences, University of Melbourne, Parkville, VIC, 3010, Australia

Background: Social anxiety disorder (SAD) is a common and chronic mental health condition. Given the significant prevalence and impairment caused by SAD, it is important to investigate

Correspondence to Bethany M. Wootton, Discipline of Clinical Psychology, Graduate School of Health, University of Technology Sydney, Ultimo, NSW 2007, Australia. E-mail: bethany.wootton@uts.edu.au
* Co-first authors.

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novel ways to improve the efficacy of cognitive behavioural therapy (CBT) for SAD. One approach may be to provide CBT in an accelerated fashion, which involves multiple sessions per week. Such accelerated treatments have been shown to be effective in other anxiety disorders, but in SAD this accelerated treatment has only been studied in a group treatment format. **Aims:** The aim of this study was to provide a preliminary investigation of the efficacy of individual accelerated CBT (aCBT) in the treatment of SAD. **Method:** The studied utilized an open trial design. Seventeen participants commenced the treatment, which consisted of 12 sessions delivered over 4 weeks. **Results:** The results indicated that participants obtained moderate to large effect sizes on measures of SAD at post-treatment (range $d = 0.76$–0.92) and 3-month follow-up (range $d = 1.31$–1.79). In addition, at post-treatment, 59% of participants no longer met criteria for SAD, and this number increased to 71% at 3-month follow-up. **Conclusions:** The results provide preliminary evidence to suggest that individual aCBT may be an important treatment option for individuals with SAD.

Keywords: social anxiety disorder, treatment outcome, accelerated, intensive, individual cognitive behavioural therapy

Introduction

Social anxiety disorder (SAD) is characterized by a fear of negative evaluation and resultant avoidance of social or performance situations (American Psychiatric Association, 2013). It is a common disorder, with a 12-month prevalence rate of approximately 7% (Kessler et al., 2012). The course of the disorder is chronic, and is associated with significant disability (Keller, 2006). Fortunately, effective treatments are available for SAD and include both psychological and pharmacological approaches (Mayo-Wilson et al., 2014). The most evidence-based psychological treatment for SAD is cognitive behavioural therapy (CBT). Typically, CBT for SAD is focused on weekly individual or group treatment. While group CBT demonstrates large between-group effect sizes ($g = 0.84$) when compared with a wait-list control group (Barkowski et al., 2016), there is some research to suggest that individual CBT out-performs group-based CBT (Stangier et al., 2003). While the relative efficacy of group versus individual treatment requires further investigation, based on current evidence some clinical guidelines recommend individual treatment over group-based treatment for SAD as the first-line treatment (e.g. National Institute for Health and Clinical Excellence, 2013).

There is now considerable evidence from controlled trials to support the efficacy of weekly individual CBT approaches for SAD, with effect sizes at post-treatment ranging from 1.17 to 2.14 (Clark et al., 2003; Mörtberg et al., 2007; Stangier et al., 2003). However, despite these findings, it has been suggested that as many as 40% of patients do not report clinically significant change after treatment (Mörtberg et al., 2007). Given that SAD is a common, chronic and impairing condition that has a high non-response rate despite the availability of effective treatment, it is important to understand how to deliver CBT interventions to this population in the most clinically and cost-effective way. A potential avenue for improving outcomes in individual CBT is to administer the treatment in an accelerated format.

In accelerated CBT (aCBT) the patient attends treatment on an outpatient basis for multiple sessions per week. Due to the shorter time frame between treatment sessions, there is the potential for enhanced learning, perhaps making aCBT more potent than standard weekly CBT. Accelerated CBT is generally under-utilized in the treatment of internalizing disorders (Jónsson et al., 2015). Currently there are a small number of studies that demonstrate the

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efficacy of out-patient aCBT for a number of anxiety and related disorders including obsessive compulsive disorder (Foa et al., 2005; Storch et al., 2007), post-traumatic stress disorder (Ehlers et al., 2014), agoraphobia (Knuts et al., 2015) and panic disorder (Chase et al., 2012; Teng et al., 2015; Wootton and MacGregor, 2016). Results from these preliminary studies indicate that accelerated treatments are efficacious and acceptable to patients (Bevan et al., 2010).

To date, aCBT has only been studied in SAD in a group format and has been studied in both child (Donovan et al., 2014) and adult (Chaker et al., 2010; Mörtberg et al., 2005, 2007) populations. In the only study investigating aCBT in a paediatric population, Donovan et al. (2014) randomly assigned 40 children to either group-based aCBT or wait-ist control. The aCBT protocol in this study consisted of three weekend treatment sessions across 15 days (week 1: 3 h on Saturday and 3 h on Sunday; week 2: 3 h on Saturday; and week 3: 3 h on Saturday). The study resulted in promising outcomes with 52.4% of children in the treatment group being diagnosis free (compared with 15.8% in the control condition) at post-treatment (Donovan et al., 2014). However, between-group effect sizes [calculated based on means and standard deviations (*SD*) provided in the manuscript] on self-report measures of social anxiety were small and non-significant.

Thus far there have been three trials investigating the efficacy of group aCBT for SAD in adult samples. In an initial open trial, Mörtberg et al. (2005) tested a 3-week aCBT protocol consisting of: week 1: 5 h Monday to Thursday and 3 h on Friday; week 2: no treatment; week 3: 5 h Monday to Wednesday and 3 h on Thursday with 37 individuals with SAD. The results of the study indicated reasonable outcomes with a moderate effect size on the Liebowitz Social Anxiety Scale (Liebowitz, 1987) at post-treatment ($d = 0.75$) and a large effect at 3-month follow-up ($d = 1.18$) and 12-month follow-up ($d = 1.35$).

Chaker et al. (2010) investigated an aCBT group program that consisted of approximately 13 h of treatment over 2 days for individuals with SAD with a primary fear of blushing. On the Social Phobia Diagnostic Questionnaire (Newman et al., 2003), a moderate effect size was found at post-treatment ($d = .74$) and a large effect size was found at 6-month follow-up ($d = .96$; Chaker et al., 2010). On the main outcome measure, the Blushing, Trembling and Sweating Questionnaire (Blushing Version) (Bögels and Reith, 1999), a large effect was found at post-treatment ($d = 1.14$) and 6-month follow-up ($d = 1.76$; Chaker et al., 2010).

In the largest and most comprehensive study conducted to date, Mörtberg et al. (2007) randomly assigned 100 participants to either (1) individual cognitive therapy consisting of 16 weekly sessions; (2) intensive group cognitive therapy (IGCT) consisting of 16 sessions over 3 weeks (week 1: 9 sessions; week 2: no sessions; week 3: 7 sessions), or (3) treatment as usual (medication management). Based on a social phobia symptom composite score devised by the authors, patients receiving the individual treatment reported improved outcomes over those in the IGCT condition, and those in the IGCT condition did not differ significantly from the treatment as usual condition (Mörtberg et al., 2007).

While to our knowledge no studies to date have examined the efficacy of individually administered aCBT when compared with standard (weekly) individual treatment for SAD, this has been examined in other diagnostic groups. For example, Ehlers et al. (2014) examined the efficacy of accelerated treatment and standard treatment in patients with post-traumatic stress disorder and found that both treatments were equally efficacious. Similarly, Storch et al. (2007) compared accelerated and standard treatment approaches for paediatric obsessive-compulsive disorder. While results were equivalent between groups on some measures, other outcomes including remission status favoured the intensive treatment group (Storch et al., 2007).

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A recent meta-analysis of brief, intensive and accelerated CBT for childhood anxiety disorders demonstrated that treatment intensity significantly moderated outcomes, whereby more intensive treatments led to enhanced outcomes (Öst and Ollendick, 2017). This study also found that accelerated treatment produced a larger between-group effect size ($g = 0.93$) than brief ($g = 0.29$) and intensive ($g = 0.84$) treatments, and demonstrated that accelerated treatments may also demonstrate other benefits, such as reduced patient attrition when compared with standard CBT (Öst and Ollendick, 2017). Thus, while the literature is small at this stage there is evidence to suggest that accelerated treatments may out-perform standard treatments, at least for some diagnostic groups.

Across all the intensive treatments provided for SAD to date, there is considerable variability in the treatment delivery format and also in the number of sessions provided. For example, some treatment protocols contained several days or weeks where patients did not meet with a therapist at all (e.g. Donovan et al., 2014, Mortberg et al., 2005), which may limit some of the benefits of the accelerated treatment approach as patients may be unlikely to practise the required skills during these large breaks from treatment. In addition, the number of sessions (and hours involved in treatment) varies considerably across studies. For example, the treatment delivered in the Chaker et al. (2010) study involved only 13 h of treatment, while the treatment delivered in the Mörtberg et al. (2005) study involved over 40 h of treatment. Examining total treatment time in accelerated treatments is important as previous research has indicated that shorter treatments produce faster rates of change (Stulz et al., 2013).

There are several limitations to the current aCBT literature. Firstly, there is a great deal of variability in the treatment delivery format. Secondly, all of the studies to date have used a group treatment approach. This is particularly problematic because some research suggests that individual CBT out-performs group-based CBT (Stangier et al., 2003). Finally, the delivery of aCBT in existing treatments for SAD may not allow for optimal learning to take place as all treatments contain several days (or weeks) where participants are not meeting with the therapist at all, and are thus potentially are not having their treatment skills reinforced during this time. Therefore, the aim of the current study is to extend the literature by evaluating the efficacy of aCBT for SAD when delivered on an individual, outpatient basis with multiple (three) sessions across each week.

Method

Design and participants

The present study employed an open trial design comparing pre-treatment with post-treatment, pre-treatment with 3-month follow-up, and post-treatment with 3-month follow-up. Participants were recruited from the University of Tasmania psychology clinic and via advertisements placed in community newspapers and flyers on community noticeboards. To be eligible for the study, participants were required to be aged 14–65 years and have a primary diagnosis of SAD. Participants were excluded if they (1) did not have the time to attend three times per week for treatment; (2) reported prior non-responsiveness to adequate CBT (defined as at least weekly sessions with in-session exposure and homework tasks); or (3) displayed moderate to severe suicide risk based on the Columbia Suicide Severity Rating Scale (Posner et al., 2011). Seventeen participants (mean age = 39.25 years, $SD = 17.68$; 71% female; 88% Anglo) with a primary diagnosis of SAD provided informed consent and

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commenced the study, and 13/17 (76%) completed the treatment (attended at least eight of the 12 treatment sessions). The study participant flow is given in Fig. 1. The study was approved by the institutional Human Research Ethics Committee and the trial was registered with Australian New Zealand Clinical Trials Registry as ACTRN12615000929505.

Measures

Diagnostic status was assessed in a face-to-face interview by either a provisionally registered psychologist (under the supervision of a registered clinical psychologist) or by a clinical psychology registrar using The Diagnostic Interview for Anxiety, Mood, and OCD and Related Neuropsychiatric Disorders (DIAMOND; Tolin et al., 2018). The DIAMOND is a clinician-administered diagnostic interview, which demonstrates adequate reliability (inter-rater reliability: .70; test–retest reliability: .86; Tolin et al., 2018) for the SAD diagnosis.

The primary outcome measure was the Social Phobia Inventory (SPIN; Connor et al., 2000), a 17-item measure of social anxiety symptoms. The SPIN is a widely used measure of social anxiety symptoms and demonstrates good reliability and validity in previous studies (Antony et al., 2006; Connor et al., 2000). The scale has been demonstrated to be sensitive to the effects of treatment (Antony et al., 2006) and a cut-off score of 19 can be used to discriminate between those with clinical levels of symptomatology and controls (Connor et al., 2000). In the current study Cronbach's alpha was .85.

Other measures of social anxiety symptoms included the Social Interaction Anxiety Scale (SIAS) and Social Phobia Scale (SPS; Mattick and Clarke, 1998). The SIAS and SPS demonstrate excellent reliability and validity in previous studies (Mattick and Clarke, 1998; Osman et al., 1998). In the current study Cronbach's alpha was .74 for the SIAS and .92 for the SPS.

Depressive symptoms were measured with the depression subscale of the Depression Anxiety Stress Scales-21 Item (DASS-21; Lovibond and Lovibond, 1995). The DASS-21 is a widely used measure of psychological distress and the depression subscale demonstrates excellent reliability and validity in previous studies (Osman et al., 2012; Sinclair et al., 2012). Cronbach's alpha in the current study for the depression subscale was .90.

Patient acceptability was measured with a 4-item questionnaire, which has been used extensively in previous research (e.g. Wootton et al., 2014). The scale asks participants to rate their overall satisfaction with the treatment on a 5-point Likert scale, as well as how logical the treatment was, and whether the treatment was worth their time. Participants are also asked to indicate how likely they would be to recommend the treatment to a friend.

The DIAMOND was administered in full at baseline, while only the SAD section was delivered at post-treatment and 3-month follow-up. All self-report measures were administered at pre-treatment, post-treatment and 3-month follow-up, with the exception of the acceptability questionnaire, which was administered at post-treatment only.

Treatment

Treatment was based on an accelerated CBT treatment manual which was created by the first two authors specifically for the purpose of this study. The treatment was devised based on the theoretical model of SAD devised by Rapee and Heimberg (1997). Treatment involved twelve 50-minute treatment sessions delivered over 4 weeks (three sessions per week: Monday,

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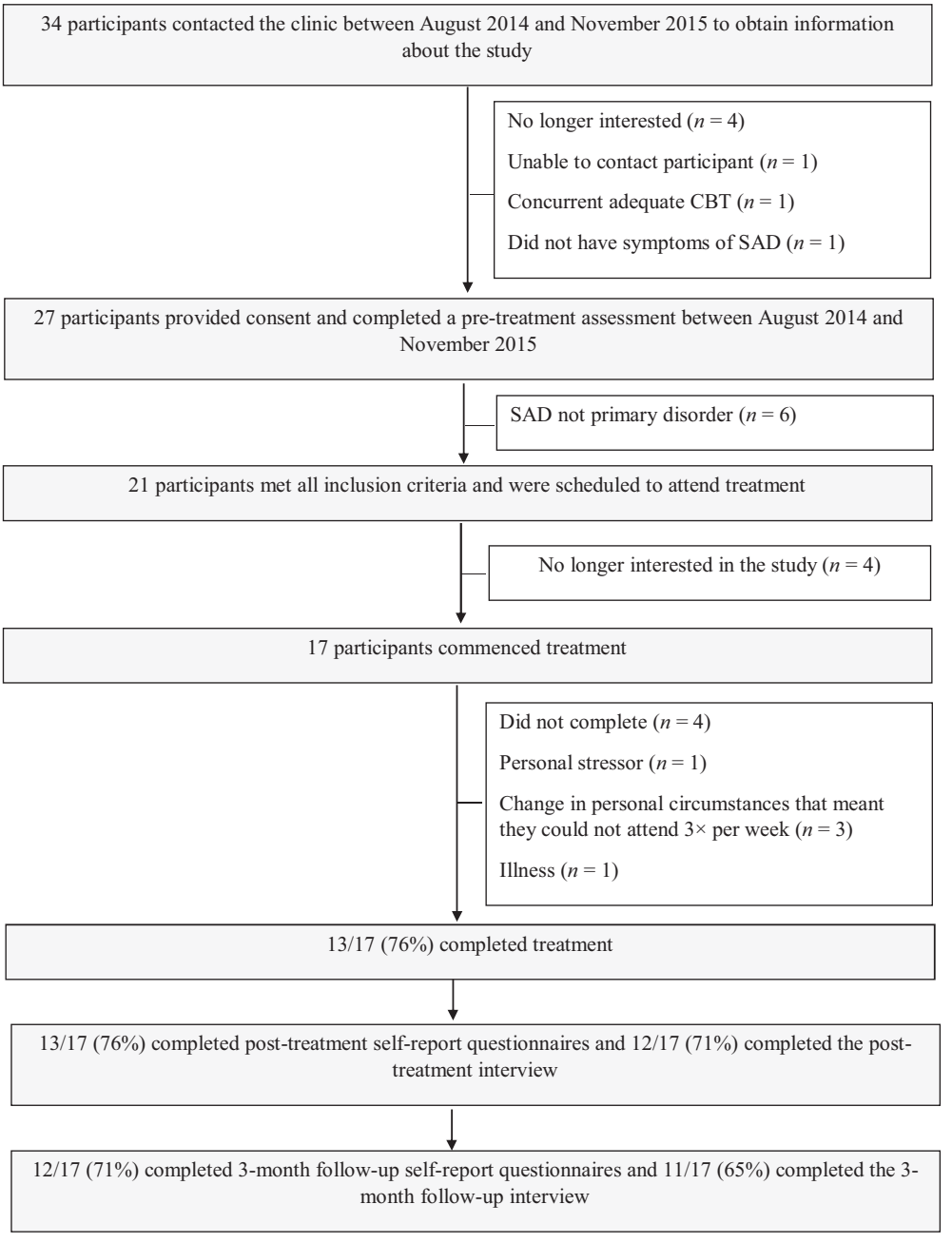

Figure 1. Participant flow

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Wednesday and Friday). Participants were also required to complete homework following each session. Session 1 included psycho-education, sessions 2 and 3 involved challenging automatic thoughts, session 4 involved challenging core beliefs, sessions 5 to 11 involved graded *in vivo* exposure, and session 12 focused on relapse prevention. The treatment was provided by either a provisional psychologist (under the supervision of a registered clinical psychologist) or by a clinical psychology registrar. Treatment fidelity was assessed informally during weekly supervision sessions.

Data analysis

Differences between those who completed treatment and those who did not (as well as those who were no longer interested in the study and those who completed) were analysed using independent samples *t*-tests for continuous variables and chi-square tests for categorical variables. Preliminary analysis of the dependent variables indicated that all variables and residuals were normally distributed (all *p*-values $> .01$) using the Shapiro–Wilk test. Skewness and kurtosis were also within acceptable limits for all variables. Homogeneity of variance was seen across all dependent variables using Levene's test (all *p*-values $>.01$).

Pre-treatment to post-treatment, pre-treatment to 3-month follow-up, and post-treatment to 3-month follow-up changes in self-report measures were analysed with mixed linear models (MLM) for both the intention to treat (ITT) and completer samples separately. The analyses used modelled change over time after controlling for the individual's baseline by means of specifying a random intercept and a random slope. The last observation carried forward (LOCF) method was used in the ITT sample where there was missing data. Effect sizes (Cohen's *d*) with 95% confidence intervals (CI) were calculated for within-group changes according to the following formula: $d = \frac{X_1 - X_2}{SD_{\text{pooled}}}$, where X_1 is the pre-treatment score and X_2 is the post-treatment (or follow-up) score. The SD_{pooled} was calculated with the following formula: $\sqrt{\frac{(N_1 - 1) \times {SD_1}^2 + (N_2 - 1) \times {SD_2}^2}{N_1 + N_2 - 2}}$, where N_1 is the sample size at pre-treatment, N_2 is the sample size at post-treatment (or follow-up), SD_1 is the standard deviation at pre-treatment, and SD_2 is the standard deviation of the post-treatment (or follow-up). Power calculations indicated that a sample size of 12 participants was sufficient to detect a pre-treatment to post-treatment within-group effect size difference in symptoms of SAD of 0.80 with alpha of 0.05 and power of 0.80.

Three criteria of clinical significance were employed. Firstly, changes in diagnostic status based on the DIAMOND were calculated. Secondly, reliable change (improvement or deterioration) on the SPIN was measured using the Jacobson and Truax (1991) reliable change index. Thirdly, clinically significant change was calculated based on those who met reliable change index criteria and also scored below the clinically significant cut-off score on the SPIN (a score of 19) (Connor et al., 2000) at post-treatment and 3-month follow-up. In the analyses of clinical significance the LOCF was used where there was missing data. Acceptability of the treatment was assessed using descriptive statistics only. All analyses were conducted using SPSS version 22 (IBM Inc., USA).

Results

Demographic information

Seventeen participants (mean age = 39.24 years, *SD* = 17.68; 71% female; 88% Anglo) with a primary diagnosis of SAD commenced the study and 13/17 (76%) completed the

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treatment. On average, participants had a mean of 1.47 ($SD = 1.28$) diagnoses and co-morbid conditions included major depressive disorder (29.4%), generalized anxiety disorder (29.4%), persistent depressive disorder (17.6%), bipolar II disorder (17.6%), agoraphobia (11.8%), specific phobia (5.9%), body dysmorphic disorder (5.9%), obsessive-compulsive disorder (5.9%), and premenstrual dysphoric disorder (5.9%). At baseline 6/17 (35%) were medicated on psychiatric medications. The participants received on average 9.11 sessions ($SD = 4.48$) (range = 1–12) of CBT during the treatment period.

Attrition

At pre-treatment all participants completed the self-report measures. At post-treatment 13/17 (76%) participants completed the self-report measures and 12/17 (71%) completed the 3-month follow-up self-report measures. All participants completed the diagnostic interview at pre-treatment. Twelve out of the 17 participants (71%) completed the post-treatment diagnostic assessment and 11/17 (65%) completed the 3-month follow-up diagnostic assessment. Those who completed at least eight of the treatment sessions were deemed to have completed treatment and 13/17 (76%) met this threshold. The amount of missing data ranged from 0% (session 1) to 35% (session 11) across each weekly administration of measures. The mean percentage of missing data across weekly measures was 24.02 ($SD = 9.20$).

A significant difference was not found between those who completed treatment ($n = 13$) and those who did not ($n = 4$) on age ($t_{15} = –.16$, $p > .05$), sex (χ^2 (1, $N = 17$) = .05, $p >$ .05), medication status (χ^2 (1, $N = 17$) = 0.24, $p > .05$), baseline severity on the outcome measures (SPIN: $t_{14} = –1.61$, $p > .05$; SIAS: $t_{15} = –.32$, $p > .05$; SPS: $t_{15} = –1.16$, $p > .05$: DASS-D: $t_{15} = –.74$, $p > .05$), or number of co-morbid diagnoses ($t_{15} = .05$, $p > .05$). We also compared those who commenced treatment ($n = 17$) and those who did not ($n = 4$) and found no difference on age ($t_{19} = .10$, $p > .05$), sex (χ^2 (1, $N = 21$) = 0.03, $p > .05$), medication status (χ^2 (1, $N = 21$) = 0.30, $p > .05$), baseline severity on the outcome measures (SPIN: $t_{17} = –.89$, $p > .05$; SIAS: $t_{19} = –.68$, $p > .05$; SPS: $t_{19} = –1.45$, $p > .05$: DASS-D: $t_{19} = –.27$, $p >$.05), or number of co-morbid diagnoses ($t_{19} = .60$, $p >$.05).

Outcomes

The means, standard deviations and effects sizes for the completer sample on each self-report outcome measure are presented in Table 1. These results were analysed separately for those who completed post-treatment measures ($n = 13$ for the SIAS, SPS and DASS-D and $n = 12$ for the SPIN) and those who completed follow-up measures ($n = 12$ for the SIAS, SPS and DASS-D and $n = 11$ for the SPIN). For the completer sample, the results of the MLM indicated a statistically significant effect for Time for the primary outcome measure at post-treatment (SPIN; $F_{1,11} = 22.60$, $p = .001$) and 3-month follow up (SPIN; $F_{2,19.15} = 63.06$, $p < .001$). A large effect size was seen at both post-treatment and 3-month follow-up. Bonferroni-corrected pairwise comparisons revealed that there was a significant reduction from pre-treatment to post-treatment ($p = .001$) and from pre-treatment to 3-month follow-up ($p < .001$). There was also a significant reduction from post-treatment to 3-month follow up ($p < .01$).

There was a statistically significant effect for Time with large effect sizes on the other measures of SAD symptomatology including the SIAS (post: $F_{1,11.99} = 13.62$, $p < .01$; 3-month follow-up: $F_{2,10.74} = 54.01$, $p <.001$) and SPS (post: $F_{1,12.00} = 12.07$, $p < .01$;

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Table 1. Estimated means, standard deviations and effect sizes (Cohen's *d*) for the outcome measures for the completer sample

Measure	Pre-treatment	*n*	Post-treatment	*n*	3-month follow-up	*n*	Effect size pre-treatment to post-treatment	Effect size pre-treatment to 3-month follow-up	Effect size post-treatment to 3-month follow-up
Completer sample (pre-treatment to post-treatment)									
SPIN	47.75 (9.56)	12	32.58 (13.68)	12	–	–	1.29 (0.37 –2.11)	–	–
SIAS	57.39 (10.06)	13	44.23 (15.00)	13	–	–	1.03 (0.18–1.81)	–	–
SPS	46.07 (15.68)	13	29.62 (16.30)	13	–	–	1.03 (0.18–1.81)	–	–
DASS-D	21.23 (12.15)	13	10.77 (7.10)	13	–	–	1.05 (0.20–1.84)	–	–
Completer sample (pre-treatment to 3-month follow-up)									
SPIN	48.09 (11.74)	11	32.91 (13.63)	11	21.36 (11.71)	11	1.19 (0.25–2.05)	2.28 (1.14–3.25)	0.91 (0.00–1.75)
SIAS	57.50 (12.08)	12	44.75 (13.30)	12	29.25 (8.81)	12	1.00 (0.12–1.81)	2.67 (1.50–3.66)	1.37 (0.44–2.21)
SPS	45.92 (15.52)	12	30.33 (15.96)	12	18.42 (10.37)	12	0.99 (0.11–1.80)	2.08 (1.03–2.99)	0.88 (0.02–1.69)
DASS-D	22.33 (11.18)	12	11.00 (8.31)	12	12.33 (8.31)	12	1.15 (0.25–1.97)	1.02 (0.13–1.83)	–0.16 (–0.96–0.65)

SPIN, Social Phobia Inventory; SIAS, Social Interaction Anxiety Scale; SPS, Social Phobia Scale; DASS-D, Depression subscale of the Depression Anxiety Stress Scales.

3-month follow-up: $F_{2,14.96} = 41.79, p < .001$). Pairwise comparisons indicated that, on all of the SAD symptom measures, scores reduced significantly from pre-treatment to post-treatment (all p-values $< .05$) and from pre-treatment to 3-month follow-up (all p-values $< .05$). There was also a significant reduction from post-treatment to 3-month follow-up on the SIAS and SPS ($p = < .01$).

Finally, there was a significant effect for Time on the measure of depression (post: DASS-D; $F_{1,12.00} = 12.62$, $p < .01$; 3-month follow-up: DASS-D; $F_{2,10.62} = 8.14$, $p < .01$), with Bonferroni-corrected pairwise comparisons revealing a significant reduction from pre-treatment to post-treatment ($p < .01$) and pre-treatment to 3-month follow-up ($p = .02$). There was no significant change from post-treatment to 3-month follow-up on the DASS-D ($p > .05$).

The means, standard deviations and effects sizes for the ITT sample ($n = 17$) for each of the self-report outcome measure are presented in Table 2. There was a statistically significant effect for Time for the SPIN ($F_{2,20.15} = 20.42$, $p < .001$) with a large effect. A statistically significant effect for Time (with medium to large effect sizes) was also seen on the other measures of SAD symptomatology (SIAS: $F_{2,18.00} = 21.97$, $p < .001$; SPS: $F_{2,20.75} = 17.47$, $p < .001$). Pairwise comparisons indicated that on all of the SAD symptom measures, scores reduced significantly from pre-treatment to post-treatment (all p-values $<.05$) and from pre-treatment to 3-month follow-up (all p-values $< .05$). Scores on all SAD symptom measures also reduced significantly from post-treatment to 3-month follow-up (p-values $= < .01$). There was a significant effect for Time on the DASS-D ($F_{2,16.21} = 6.15, p = .01$), with Bonferroni-corrected pairwise comparisons revealing a significant reduction from pre-treatment to post-treatment ($p < .01$) and pre-treatment to 3-month follow-up ($p = .02$). There was no significant change from post-treatment to 3-month follow-up on the DASS-D ($p > .05$).

Clinical significance

At pre-treatment all participants met criteria for SAD on the DIAMOND. Using conservative ITT criteria, at post-treatment 10/17 (59%) participants no longer met criteria for SAD on the DIAMOND and at 3-month follow-up 12/17 (71%) no longer met criteria for SAD. Pre-treatment data on the SPIN was available for only 16 participants. Of these 16 participants, 8/16 (50%) met criteria for reliable improvement at post-treatment and 11/16 (67%) at 3-month follow-up. No participants met criteria for reliable deterioration. Two of the 17 participants (12%) met the conservative criteria for clinically significant change at post-treatment and 7/16 (44%) met criteria for clinically significant change at 3-month follow-up.

Acceptability

Acceptability was assessed at post-treatment only and was completed by 12/17 participants (71%). When asked to provide a rating from 1 (*not at all*) to 5 (*extremely*), participants reported high levels of treatment satisfaction (mean = 4.58, $SD = 0.67$). Participants also found the treatment logical (*how logical was the treatment?*; mean = 4.67, $SD = 0.49$) and thought it was worth their time (*was the treatment worth your time?*; mean = 4.75, $SD = 0.45$). All 12 participants who completed the treatment (100%) indicated that they would *recommend the treatment to a friend.*

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Table 2. Estimated marginal means, standard deviations and effect sizes (Cohen's *d*) for the outcome measures for the ITT sample

Measure	Pre-treatment	*n*	Post-treatment	*n*	3-month follow-up	*n*	Effect size pre-treatment to post-treatment	Effect size pre-treatment to 3-month follow-up	Effect size post-treatment to 3-month follow-up
SPIN	44.82 (13.55)	16	33.12 (11.75)	16	25.59 (12.59)	16	0.92 (0.17–1.63)	1.47 (0.66–2.21)	0.62 (-0.11–1.31)
SIAS	57.00 (9.68)	17	46.94 (12.54)	17	36.00 (13.52)	17	0.90 (0.17–1.58)	1.79 (0.95–2.53)	0.84 (0.12–1.52)
SPS	43.59 (18.05)	17	31.00 (15.17)	17	22.59 (13.70)	17	0.76 (0.04–1.43)	1.31 (0.54–2.02)	0.58 (-0.12–1.25)
DASS-D	20.12 (10.08)	17	12.12 (7.71)	17	13.06 (7.53)	17	0.89 (0.17–1.57)	0.79 (0.08–1.47)	–0.12 (–0.79–0.55)

SPIN, Social Phobia Inventory; SIAS, Social Interaction Anxiety Scale; SPS, Social Phobia Scale; DASS-D, Depression subscale of the Depression Anxiety Stress Scales.

Discussion

The aim of this study was to evaluate the efficacy of aCBT for SAD when delivered on an individual, out-patient basis with multiple (three) sessions across each week. Treatment consisted of a total of 12 h across 4 weeks, and results indicated that participants experienced a significant reduction in SAD symptoms. When measured at post-treatment, large effect sizes were reported on the SPIN ($d = 0.92$) and SIAS ($d = 0.90$), and a medium effect size was found on the SPS ($d = 0.76$). Larger effect sizes were found at the follow-up assessment on the SPIN ($d = 1.47$), SPS ($d = 1.31$) and SIAS ($d = 1.79$), indicating that symptoms continued to reduce in the 3 months following treatment cessation. Furthermore, 67% of the ITT sample demonstrated a reliable decrease in their SAD symptoms, as measured on the SPIN, and 44% of the participants' SAD symptoms could be conservatively deemed to have made clinically significant change. Participants' perception of the treatment at post-treatment was positive, indicating that aCBT is likely to be well-received by patients. In addition, treatment appeared to influence self-reported depression symptoms, with a large treatment effect on the DASS-21 depression subscale at post-treatment ($d = 0.89$) and moderate effect at follow-up ($d = 0.79$).

In comparison with the existing literature on aCBT for adults with SAD, the current study demonstrates preliminary, yet promising, results. For example, two studies (Mörtberg et al., 2005, 2007) reported moderate to large effects following 41 h of treatment administered over 3 weeks ($d = 0.75$ in Mörtberg et al., 2005; $d = 0.79$ in Mörtberg et al., 2007). These results are comparatively smaller than the post-treatment effects reported in the current study ($d = 0.76$–0.92). As also seen in the current study, Mörtberg et al. (2005) reported a further decrease in SAD symptoms in the 3 months succeeding treatment cessation, resulting in a large effect size ($d = 1.18$). However, this was a comparatively smaller effect than those reported in our study at the 3-month follow-up time point ($d = 1.31$–1.79). The treatment employed by Mörtberg et al. (2005, 2007) involved a shorter time period (3 weeks) than the current study (4 weeks). However, the number of treatment hours required for participants was greater; 41 hours (Mörtberg et al., 2005, 2007), in comparison with 12 h in the currrent study. Comparatively, aCBT as delivered in our study may offer a larger reduction in SAD symptoms in fewer treatment hours, albeit over a longer time period, than the group treatment in the studies by Mörtberg et al. (2005, 2007).

The study by Chaker et al. (2010) also reported a large effect size at post-treatment ($d = 1.14$), which is generally comparative to the effect reported in the current study. Again, follow-up assessment revealed a further reduction in symptoms ($d = 1.76$ at 6-month follow-up; Chaker et al., 2010), which is consistent with the results of our study ($d = 1.31$–1.79) at 3-month follow-up. Whilst the treatment in the study of Chaker et al. was delivered in approximately the same number of hours (12 h and 45 min) as our study (12 h), it also offers the benefits of receiving treatment in only 2 days, in comparison with 4 weeks in the current study.

In comparison with the post-treatment effect sizes reported in research on weekly individual CBT for SAD (e.g. $d = 1.17$–2.14; Clark et al., 2003; Mörtberg et al., 2007; Stangier et al., 2003), our study reports comparable effects at post-treatment ($d = 0.76$–0.92). This may indicate that while there may be no additional benefit in terms of symptom reduction achieved by accelerating treatment, clinicians and clients may take advantage from a shorter overall treatment period, which may be advantageous in terms of treatment planning and resource distribution. However, it is important that the acceptability and efficacy of aCBT be studied in comparison with standard weekly treatment within a randomized controlled trial in the future.

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In sum, aCBT for SAD when delivered three times a week across 4 weeks offers a brief and convenient treatment for SAD that may be more effective than group aCBT and as effective as standard weekly CBT for SAD. The results of this study indicate that clinicians are not at risk of decreasing treatment efficacy if choosing to accelerate treatment. However, as an accelerated protocol may not improve outcomes over standard weekly treatment, further research is required. Accelerated treatment may nevertheless be more attractive to some patients or treatment providers who may wish to reduce the time frame of treatment. Furthermore, aCBT may be advantageous for individuals with severe and impairing SAD symptoms who wish to experience a rapid reduction in symptoms, and for those in a rural or remote setting who need to travel long distances to treatment.

Despite promising results, the current study presents with several limitations. Firstly, the sample was relatively small and largely homogenous. While a power analysis indicated that the study was sufficiently powered to obtain meaningful results these results should be interpreted with caution given the small sample size and wide confidence intervals. Secondly, while the results compare favourably with other studies, a lack of a comparison group limits the conclusions that may be confidently drawn from the current study. The inclusion of a wait-list control group and/or weekly CBT treatment group in future research would strengthen the validity of results, as would an examination of treatment fidelity. Thirdly, while participants rated the intervention as acceptable, acceptability was only rated at post-treatment, thus those participants who did not reach this stage of treatment may have indicated lower acceptability ratings. Future research should measure acceptability throughout treatment and the acceptability in this study should be interpreted with caution. Fourthly, minimal demographic and concurrent treatment information was collected in this pilot study, and future studies should obtain further information on concurrent pharmacological and non-pharmacological treatment. Finally, the uncontrolled design used cannot account for natural fluctuations in SAD symptoms. Future studies investigating aCBT delivered in this format should use a controlled design and investigate the dose–response relationship in a controlled fashion. Furthermore, future studies should include both treatment-naive and treatment-resistant cases.

In conclusion, the current study provides preliminary evidence that aCBT is effective in the treatment of SAD in adults. Twelve hours of treatment administered in 50-minute sessions three times weekly, over 4 weeks, appears to produce significant reductions in SAD symptoms. The severity of symptoms is further reduced when assessed 3 months following treatment cessation. The results of the current study compare well with other available options for the treatment of SAD with CBT. Despite promising results, the current study is hindered by a small sample size and the lack of comparison group, and thus further research is needed to confirm the efficacy of aCBT for SAD.

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