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A randomised controlled trial of cognitive behavioural treatment for obsessive compulsive disorder in children and adolescents

Tim I. Williams · Paul M. Salkovskis · Liz Forrester · Sam Turner · Hilary White · Mark A. Allsopp

Abstract  Cognitive behaviour therapy (CBT) for young people with obsessive compulsive disorder (OCD) has become the treatment of first choice. However, the literature is largely based on studies emphasising exposure and response prevention. In this study, we report on a randomised controlled trial of CBT for young people carried out in typical outpatient clinic conditions which focused on cognitions. A randomised controlled trial compares 10 sessions of manualised cognitive behavioural treatment with a 12-week waiting list for adolescents and children with OCD. Assessors were blind to treatment allocation. 21 consecutive patients with OCD aged between 9 and 18 years were recruited. The group who received treatment improved more than a comparison group who waited for 3 months. The second group was treated subsequently using the same protocol and made similar gains. In conclusion, CBT can be delivered effectively to young people with OCD in typical outpatient settings.

Keywords  Obsessive compulsive disorder · Cognitive behaviour therapy · Randomised controlled trial · Treatment · Young people

Introduction

Obsessive compulsive disorder (OCD) is particularly a severe and disabling psychological disorder. Recent epidemiological studies suggest that it affects around 1.9–3.2% of the adult population and may have a 1-year prevalence rate of up to 4% in late adolescence [13]. Follow-up studies have shown that it has a chronic relapsing course such that 50% of adult patients report their first symptoms in childhood or adolescence and 50% of patients with OCD in adolescence will continue to suffer disabling effects from OCD in adulthood [2, 8, 35]. An effective treatment for OCD in adolescence could offer significant savings for the health service and improved quality of life for many patients.

For adults, pharmacological treatments (selective serotonin re-uptake inhibitors, SSRIs) have been shown to reduce the level of symptomatology, but in up to 90% of patients these gains are lost within 7 weeks of stopping treatment [34]. The benefits of SSRIs for young people with OCD have also been demonstrated (see [17] for a meta-analysis), but there are concerns about the safety of medication in young people on the grounds of both physiology and risk taking behaviour. Although controversial, these concerns impact on the acceptability of pharmacotherapy. In adults, it is known that SSRIs do not enhance short-term adherence to psychological treatment, and in the long term their use may impair the efficacy of psychological treatments [15].

Recent expert consensus guidelines suggest that cognitive behavioural treatments are the first choice treatments for children and adolescents with OCD [21, 26] although a Cochrane review concluded only that “behavioural or cognitive behaviour therapy (CBT) appears to be a promising treatment for OCD in children...
and adolescents” [27]. Until recently, there have been few trials which examine the effectiveness of cognitive behavioural therapy for young people, based on the assumption that adult-based research would generalise. In general, controlled trials have demonstrated that CBT techniques used for adults have generalised well [4, 6, 9, 11, 22]. The largest of these trials [22] demonstrated that both medication and cognitive behavioural therapy were effective treatments for OCD. However, there was a significant site x treatment interaction such that in one site CBT was significantly more effective than medication whereas in the other site medication was found to be more effective. Other controlled trials of CBT for young people have found similar beneficial effects of CBT compared with medication [4, 11].

The components of CBT for children have often been poorly specified. Two components have been used by most studies—exposure and response prevention (E/RP) and anxiety management [4, 6, 9, 11, 22]. Bolton and Perrin [9] have demonstrated that E/RP alone (with minimal anxiety management) is sufficient to achieve significant benefits. De Haan et al. [11] investigated the use of targeted cognitive techniques including manipulating responsibility cognitions demonstrating comparable effects for CBT and medication. Two studies have delivered CBT in group format [4, 6], without compromising the effect of CBT. Furthermore, Barrett et al. [6] involved parents in treatment with beneficial effects.

An alternative to managing anxiety around exposure and response prevention is to target cognitions specific to OCD. A number of candidate cognitive processes have been suggested and a systematic review of cognitions related to OCD symptoms in young people [28] suggested that three models have some support: increased responsibility, meta-cognitions, and thought-action fusion. These models are difficult to distinguish empirically, because the processes overlap. An appraisal of responsibility for intrusive thoughts is a form of meta-cognition (thinking about thoughts) and the appraisal that one’s thoughts might result in actions or events occurring is of no significance unless one appraises oneself as responsible for the thought. Evidence for responsibility cognitions being specific to OCD in adults has been provided by Salkovskis and his colleagues [29] have developed a package of cognitive behavioural treatment methods which concentrates on responsibility cognitions. The components that seem to be particularly important are:

1. normalising the nature of unpleasant intrusive cognitions;
2. identifying the nature of the link between the thoughts and the feelings of discomfort/anxiety and subsequent neutralising rituals;
3. examining the logical nature of the links, and putting them to the test by means of behavioural experiments;
4. comparing real danger with the worry about causing harm, i.e. discriminating between thinking about and acting upon;
5. helping the patient to identify that the effect of attempting to control the thoughts leads to recurrence of the thoughts or images;
6. helping the patient to consider alternative non-threatening accounts of their obsessive problems.

The therapy also includes psychoeducation about anxiety and an explanation of how trying to carry out exposure and response prevention might seem difficult. Although some exposure work was undertaken, it was always explained in terms of finding out what happens to cognitions and emotions. The therapists did not insist on waiting until the uncomfortable feelings had subsided as is required by exposure and response prevention. A pilot study [36] of this form of CBT with young people suggested that the changes in symptoms were paralleled by the changes in the responsibility beliefs which initiate and maintain compulsive behaviours. The techniques also affect meta-cognition in that they alter the beliefs about the nature and meaning of cognitions associated with OCD. This form of CBT differs from that used in the POTS trial [22] in its concentration on cognitions. The treatment does not aim to teach the children to resist the impulse to carry out compulsions, but rather to identify and change the misconceptions underlying the motivation to carry out compulsions. A much larger study would be needed to test this form of CBT against other forms of CBT or indeed a purely behavioural treatment such as that in Bolton and Perrin [9].
Methods

Young people were recruited to the trial with a primary diagnosis of OCD based on a semi-structured interview for mental health problems (ADIS-C) [33]. In total, 21 young people aged 9–18 years (mean age 13 years 7 months; 13 boys, 8 girls) were recruited from 22 cases referred by Child and Adolescent Mental Health Teams or Family Doctors for the trial (see Fig. 1 for CONSORT diagram). Children were included if OCD was the major problem, and it had been present for at least 6 months. Children were excluded if they were unable to speak and understand English fluently, if they had co-occurring psychosis or autism spectrum disorder. At initial assessments, 11 presented with no other clinical diagnoses, while 4 received diagnoses of generalised anxiety disorder, 4 specific phobia, 4 separation anxiety, 2 ADHD, 2 social phobia, and 1 dysthymia. (The numbers do not add up to 10 because only 2 had one additional diagnosis, the others having up to three additional diagnoses.) Seven of the participants were taking medication throughout the trial and had been taking the same dose for 12 weeks prior to the trial (two on paroxetine 5 mg/day, three on fluoxetine 20 mg/day, one on fluvoxamine 50 mg/day, one on clomipramine 50 mg/day). There was no significant multivariate difference between the treated group and the waiting list group at initial assessment \[ F(6, 13) = 0.58, \text{n.s.} \]. The therapists for the trial (TW, HW, ST) were clinical psychologists employed by the National Health Service (NHS) in England to work in community child and adolescent mental health clinics. All treatment took place in NHS clinics and was recorded on audiotape if the young person consented.

Fig. 1 CONSORT diagram

Assessed for eligibility (n=22)

Excluded (n= 1)
Not meeting inclusion criteria due to
ASD diagnosis (n= 1)

randomisation

Allocated to CRT (n = 11)
Received allocated intervention (n = 11)
Did not receive allocated intervention (n = 0)

Allocated to list (n = 10)
Received allocated intervention (n = 10)
Did not receive allocated intervention (n = 0)

Lost to follow-up (n= 1)
Refused CRT part way through
course – no longer willing to engage with therapist

Follow-Up

Unable to tolerate waiting list,
sought treatment elsewhere

Lost to follow-up (n= 1)

Analysis

Analyzed (n = 11)
Excluded from analysis (n = 0)

Analyzed (n = 10)
Excluded from analysis (n = 0)
Procedure

Once consent was received for taking part in the trial, the participants were allocated to either immediate treatment (ten 1-h sessions) or a 12-week waiting list. Allocation was carried out on a predetermined random number schedule with no replacements by the trial administrator. Participants in the waiting list condition were informed that they would have to wait 12 weeks for therapy, but they were given the phone number of the lead clinician (TW) to contact in the case of a significant deterioration in OCD. Assessments were conducted at the beginning and end of treatment (and/or waiting list period), and 12 weeks after the end of treatment. All assessments were completed in the child’s home unless specifically requested to be elsewhere. Assessors were blind to the allocation of the participants, and the participants were instructed not to reveal whether they had received treatment. The assessors completed two semi-structured interviews: Children’s Yale-Brown Obsessive Compulsive Scale (C-YBOCS) [32] to assess OCD symptoms, and the ADIS-C [33], a semi-structured interview to assess the presence of other comorbid disorders. Participant completed measures included Child Depression Inventory [18], Obsessions and Compulsions Inventory (OCI) [14] modified for children, Multidimensional Anxiety Scale for Children [20], the Children’s Responsibility Attributions Scale (CRAS), the Children’s Responsibility Interpretations questionnaire (CRIQ). Both the CRIQ and the CRAS are instruments modified from the adult measures of responsibility cognitions published in Salkovskis et al. [31]. The CRAS is scored such that increasing score indicates a decreasing level of responsibility attributions, whereas the other self-report scales are scored such that increasing score indicates increasing difficulties. An unpublished study by the first author found that the internal reliability of the CRAS was good in a large normative sample of 13–14-year olds (Cronbach’s α = 0.85). The internal reliability in a sample of young people with mental health problems for the other scales was also high (OCI: Cronbach’s α = 0.93; RIQ frequency: Cronbach’s α = 0.86; RIQ belief: Cronbach’s α = 0.85). Copies of these measures are available on http://psychology.iop.kcl.ac.uk/ocdkids/questionnaires/questionnaires.aspx.

Treatments

Cognitive behaviour therapy was based on the principles outlined by Salkovskis [29]. Participants worked with their therapists to understand the cognitive distortions which maintain their OCD. The aim of treatment is to alter responsibility cognitions, primarily by doing experiments both in session and at home. For instance, one common belief encountered in OCD is that the sufferer is uniquely responsible for harm occurring if certain rituals are not carried out satisfactorily. The therapist and the participant would agree during the session to carry out an experiment designed to see what happens if responsibility is shared. Another common task during therapy is to attempt to elicit the worrying intrusive thoughts and examine whether they have real meaning or are just thoughts. Treatment fidelity was ensured through clinic notes, regular meetings of the therapist team and audiotapes of treatment sessions.

Participants were allocated by the trial administrator to the two groups using a table of random numbers. Only the trial administrator was aware which participants were in which group.

Results

Plan of analysis

The primary measure of OCD symptoms is the CYBOCS. We first report an analysis of covariance of the CYBOCS score at 3-month post-baseline assessment (with baseline CYBOCS as the covariate), at which stage only one of the two groups will have received an active treatment. This analysis tests the hypothesis that there will be a group effect at 3 months. We will then repeat the analysis of covariance of the CYBOCS scores at 6-month post-baseline when both groups will have received treatment. This analysis tests the hypothesis that there will be a period by group effect on the analysis of the CYBOCS scores at 3 and 6 months. Finally, we will analyse the secondary measures (i.e. self-report data) in the same way using initial values of the measure as the covariate.

Two clients dropped out of the study during the first phase (one from each condition). In the following analyses, the last observation was carried forward from the last available observation. This is, therefore, an intention to treat analysis.

Analysis of primary measure

Figure 2 shows the mean CYBOCS scores at baseline, 3 and 6 months. The analysis of covariance (baseline CYBOCS as covariate) of the CYBOCS at 3 months showed a significant group effect [F(1) = 7.07, p = 0.016]. The figure shows that this is due to a much improved CYBOCS score for the group treated with CBT first. The group that was allocated to the waiting list showed little or no improvement over the same period. Cohen’s effect size (d) for the difference between the two groups divided by the mean standard deviation was calculated as 1.07, which is considered to be a large treatment effect.
At 6 months, the analysis of covariance of CYBOCS (using baseline CYBOCS as covariate) showed no group effect as expected since both groups had received treatment, although there was a trend towards a significant effect of the covariate \[F(1, 19) = 3.70, p = 0.07\] suggesting that the severity of OCD at the beginning of the trial influences the outcome. As can be seen from Fig. 2, both groups had improved significantly and the group that was treated first showed continuing slight improvement.

Analysis of secondary measures

Analysis of the secondary data (the self-report questionnaires OCI, CRAS, CDI, CRIQ frequency, CRIQ belief; see Table 1) used the same procedures, i.e., analysis of covariance using the baseline value of the measure as the covariate. There were no statistically significant group effects at 3 months [OCI: \(F(1, 19) = 0.29, p = 0.59\); CDI: \(F(1, 19) = 0.49, p = 0.49\); CRIQ frequency: \(F(1, 19) = 2.07, p = 0.17\); CRIQ belief: \(F(1, 19) = 0.90, p = 0.36\); CRAS: \(F(1, 19) = 0.41, p = 0.53\); MASC: \(F(1, 19) = 0.008, p = 0.929\)] but there were significant effects of the covariate for each measure [OCI: \(F(1, 19) = 11.19, p = 0.004\); CDI: \(F(1, 19) = 12.20, p = 0.003\); CRIQ frequency: \(F(1, 19) = 13.06, p = 0.002\); CRIQ belief: \(F(1, 19) = 17.26, p = 0.001\); CRAS: \(F(1, 19) = 27.12, p < 0.001\), except the MASC \[F(1, 19) = 0.13, p = 0.72\].

Inspection of Table 2 shows that the groups were improving on all measures at the 3-month point.

A second analysis of covariance was carried out to determine if the treatment was effective for both groups using data from baseline and 6 months later after both groups had received CBT. The results showed that there was only a significant time effect \[F(1, 19) = 53.14, p < 0.001\] and no group \[F(1, 19) = 0.06, p = 0.82\] or interaction effect \[F(1, 19) = 0.73, p = 0.40\]. As shown in Fig. 1, the changes in CYBOCS are largest when the groups are receiving treatment. Overall, the CYBOCS scores of the participants reduced from 22.12 (SE = 1.08) to 9.64 (SE = 1.79). Analysis of the secondary data also showed significant changes from baseline to 6 months [OCI: \(F(1, 19) = 18.35, p < 0.001\); CDI: \(F = 23.16, p < 0.001\); RIQB: \(F(1, 19) = 32.8, p < 0.001\); RIQF: \(F(1, 19) = 25.15, p < 0.001\); RAS: \(F(1, 19) = 10.75, p = 0.004\); MASC: \(F(1, 19) = 22.81, p < 0.001\), but no group effects [OCI: \(F(1, 19) = 0.45, p = 0.51\); CDI: \(F(1, 19) = 0.53, p = 0.48\); RIQB: \(F(1, 19) = 0.30, p = 0.59\); RIQF: \(F(1, 19) = 0.003, p = 0.38\); RAS: \(F(1, 19) = 0.89, p = 0.36\); MASC: \(F(1, 19) = 0.30, p = 0.60\)] or interaction effects [OCI: \(F(1, 19) = 1.41, p = 0.25\); CDI: \(F(1, 19) = 0.42, p = 0.53\); RIQB: \(F(1, 19) = 1.03, p = 0.32\); RIQF: \(F(1, 19) = 0.83, p = 0.38\); RAS: \(F(1, 19) = 2.08, p = 0.17\); MASC: \(F(1, 19) = 0.27, p = 0.61\)]. Table 1 shows the means and standard deviations for all the measures.

We followed the recommendations of Morris and DeShon (2002 cited in [1]) to calculate the effect size as the difference between the immediately pre-treatment CYBOCS and the post-treatment CYBOCS divided by the pre-treatment standard deviation (effect size = 2.62) which also allows for the calculation of the effect size of the waiting list. This effect size was compared with those from other published trials which included a control group (see Table 2). The results from this study fall within the range of values reported heretofore.

![Fig. 2 Mean CYBOCS scores for both groups at baseline, 3 and 6 months. Error bars represent standard errors of the mean at each time point](image)

Discussion

Cognitive behaviour therapy largely based on the use of experiments to tackle the cognitive biases in OCD produced a significantly greater reduction in OCD symptoms of the participants than a waiting list condition. Being placed on the waiting list first did not affect the power of the subsequent treatment. The self-report measures showed a statistically significant reduction in self-reported symptoms over the two time periods but did not demonstrate an effect of treatment. The study also shows only small changes in symptoms for the young people placed on the waiting list (cf. [1]), therefore confirming the chronic nature of OCD in young people. This adds to the body of evidence in favour of CBT for OCD in young people. In
view of the potential problems associated with medication, the results of this study support the view that young people with OCD should, therefore, be offered CBT as the first-line treatment by child and adolescent mental health services.

This is the first randomised controlled study of a CBT approach based on responsibility cognitions (following [29, 30]) with young people. Although the numbers treated in this trial were small, the demonstration that the treatment was as effective for the waiting list group demonstrates that waiting for treatment had no effect on eventual outcome and increases the power of the study. The 3-month follow-up of the group that was treated first suggests that, at least at first, the participants continued to improve after cessation of treatment.

The failure of the self-report measures to demonstrate a difference between the groups at the 3-month point is difficult to explain. Although a failure to find changes in depression symptoms is not unusual in trials of CBT for OCD [25], we had predicted a change in OCD symptoms paralleling responsibility cognitions as was found in a pilot study [36]. One possibility is that as found by Anholt et al. [3] the changes in behaviour rather than the changes in cognitions were the most significant feature of treatment. However, it is also possible that the hope that someone else was going to help with the problem improved the subjective feelings tapped by self-report measures including the appraisals of responsibility or affected the responsibility cognitions directly (e.g. someone else is taking charge of the OCD for me), but did not alter the assessor-rated severity. If that is so, it might indicate that changing cognitions alone are not sufficient to alter behaviour. Indeed, the individual formulations of OCD used in this treatment method use multiple maintenance cycles which are described for each affected person. Yet another possibility is that the self-report measures are not valid in this population. Further research on the validity and reliability of self-report measures would be helpful.

Other trials of CBT with young people have tended to concentrate on managing the anxiety or discomfort experienced when undertaking exposure and response prevention, e.g. [4, 6, 22]. The effect size observed in this trial is somewhat less than those seen in previous trials of CBT for young people with OCD (mean effect size 1.98, Table 3 in [1]), although the differences in the methods of effect size calculation make the comparison somewhat problematic.

Differences in the treatment offered may also be important in determining the effectiveness of the intervention. There are two lines of evidence: intra-trial site differences and inter-trial differences. The POTS trial compared CBT using E/RP and management of the distress associated with OCD with a placebo medication condition but found considerable site differences for both the CBT and a third medication-only condition [22]. (Unfortunately because the site data are only reported as Hedge’s $g$ effect sizes, we were unable to compare effect sizes using the Morris and DeShon method [25] and calculated Hedge’s $g$ effect sizes in the following text.) The Hedge’s $g$ effect size for the POTS trial form of CBT varied from 0.51 in Chapel Hill to 1.6 in Pennsylvania (a statistically significant

<table>
<thead>
<tr>
<th>Measure</th>
<th>Baseline CBT</th>
<th>Baseline WL</th>
<th>3 months CBT</th>
<th>3 months WL</th>
<th>6 months CBT</th>
<th>6 months WL</th>
</tr>
</thead>
<tbody>
<tr>
<td>CYBOCS</td>
<td>23.09 (1.22)</td>
<td>21.05 (1.84)</td>
<td>12.09 (2.25)</td>
<td>19.60 (2.03)</td>
<td>9.23 (2.45)</td>
<td>10.10 (2.74)</td>
</tr>
<tr>
<td>OCI</td>
<td>59.30 (8.28)</td>
<td>73.55 (8.26)</td>
<td>45.00 (8.30)</td>
<td>60.30 (9.62)</td>
<td>37.10 (8.73)</td>
<td>34.30 (5.51)</td>
</tr>
<tr>
<td>CDI</td>
<td>17.85 (2.76)</td>
<td>14.67 (1.82)</td>
<td>12.9 (2.62)</td>
<td>12.78 (2.92)</td>
<td>10.50 (2.41)</td>
<td>9.06 (2.56)</td>
</tr>
<tr>
<td>MASC</td>
<td>59.8 (6.87)</td>
<td>66.3 (6.74)</td>
<td>49.7 (5.90)</td>
<td>56.6 (6.69)</td>
<td>41.1 (4.40)</td>
<td>43.0 (7.28)</td>
</tr>
<tr>
<td>CRAS</td>
<td>49.00 (6.00)</td>
<td>51.00 (8.10)</td>
<td>58.00 (7.54)</td>
<td>63.63 (6.15)</td>
<td>58.00 (8.03)</td>
<td>74.13 (8.31)</td>
</tr>
<tr>
<td>CRIQ belief</td>
<td>634.38 (103.66)</td>
<td>781.11 (152.50)</td>
<td>563.13 (99.26)</td>
<td>537.78 (150.76)</td>
<td>320.63 (86.94)</td>
<td>332.22 (102.34)</td>
</tr>
<tr>
<td>CRIQ frequency</td>
<td>29.25 (4.31)</td>
<td>31.44 (5.14)</td>
<td>25.38 (3.90)</td>
<td>19.33 (5.45)</td>
<td>16.75 (4.36)</td>
<td>13.89 (4.35)</td>
</tr>
</tbody>
</table>

Table 2 Effect sizes on CYBOCS calculated according to Morris and DeShon [25] for controlled studies of CBT in young people

<table>
<thead>
<tr>
<th>Study</th>
<th>Individual CBT effect size</th>
<th>Waiting list or placebo control effect size</th>
</tr>
</thead>
<tbody>
<tr>
<td>This study</td>
<td>-2.61792</td>
<td>-0.24914</td>
</tr>
<tr>
<td>March et al. [22]</td>
<td>-2.6087</td>
<td>-0.81818</td>
</tr>
<tr>
<td>De Haan et al. [11]</td>
<td>-2.10169</td>
<td>N/A</td>
</tr>
<tr>
<td>Barrett et al. [6]</td>
<td>-3.55349</td>
<td>0.198543</td>
</tr>
</tbody>
</table>

Negative numbers indicate improved CYBOCS scores. N/A indicates that the study did not include a waiting list control
difference). The Hedge’s g effect size in this trial (1.07) falls within that range. Bolton and Perrin [9] demonstrated the benefits of E/RP alone compared with a wait list (Hedge’s g effect size 1.23).

Our study does not provide information on the relative merits of different forms of CBT. The difficulty with attempting such a comparison is that the effect sizes of different models of CBT appear to be comparable (see previous paragraph). Of more interest in clinical practice are issues such as the use of combinations of medication and CBT and the involvement of the family. Studies with adults have begun to show that offering medication first diminishes the subsequent effectiveness of CBT [15].

Most of the studies on CBT with young people have included the parents in treatment sessions, although Bolton and Perrin [9] only provided feedback at the end of each session. Recent work by Barrett et al. [6] showed that very substantial reductions in OCD could be obtained by the use of family management components in the treatment package. The families of young people with OCD differ in some important respects from the families of young people with anxiety disorders [5, 12]. Barrett’s study [5] demonstrated that the parents were less confident in their child’s ability, less rewarding of independence, and were less likely to use positive problem solving than the parents of children with anxiety disorders. Derisley et al. [12] found that parents tended to use avoidant coping techniques as well as having more symptoms of mental health problems. The NICE guidelines too [26] suggest that the contribution of the family to the treatment of young people with OCD needs further investigation. However, the involvement of families in the treatment of other emotional disorders in young people has not always resulted in more improvement than a less explicit component of family change [10]. Nevertheless, it would be worth investigating whether a family component focused on problem-solving strategies and developing confidence in the child’s abilities (based on Barrett’s studies [5, 6]), and would be helpful in increasing the effect of the CBT used in this study as has been partially demonstrated by Barrett et al. [6].

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