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Comorbidity and treatment response in pediatric OCD: A pilot study of group cognitive-behavioural treatment

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Abstract

This pilot study evaluated the effectiveness of group CBT on treatment outcomes for children and adolescents who presented with OCD and complex comorbid conditions, including depression, attention deficit/hyperactivity disorder and pervasive developmental disorders. Specifically, the impact of comorbidity on treatment response rates and remission rates was examined. Forty-three youth (aged 7 - 17) with OCD participated in group family based CBT. Assessments were conducted at pre and post-treatment and 6 months. Eighty six percent of youth presented with a secondary psychiatric disorder, and 74% presented with a tertiary psychiatric condition. Contrary to expected, comorbidity was not associated with poorer treatment outcomes at post assessment. At longer term follow up (6 months) however treatment outcomes were poorer for youth with multiple comorbid conditions and for those with attention deficit/hyperactivity disorder. The finding that group CBT is largely effective for youth with comorbid conditions is of clinical and practical significance. Group delivery of CBT provides an efficient and cost-effective approach, and alleviates strain on services and service providers. Continued efforts are needed to improve long term outcomes for youth with multiple comorbid conditions and attention deficit/hyperactivity disorder. Examining treatment response as function of comorbidity with larger clinical samples is important to extend this research.

Key Words: OCD, children, youth, treatment response, group CBT, comorbidity
1. Introduction

Pediatric obsessive-compulsive disorder (OCD) is a debilitating neurobehavioral anxiety disorder affecting between 1% to 4% of children and youth (Douglass et al., 1995; Valleni-Basile et al., 1995; Shaffer et al., 1996; Zohar, 1999). During childhood, the condition is associated with impairment and dysfunction across multiple domains, including family relationships and household routines (Cooper, 1996; Barrett et al., 2001), school functioning (Toro et al., 1992; Piacentini et al., 2003) and peer relationships (Allsopp and Verduyn, 1990; Storch et al., 2006), leading to lifelong suffering if left untreated (Stewart et al., 2004). The high rate of comorbidity associated with paediatric OCD is one reason why this disorder is so debilitating and why it is so often described as a complex psychiatric disorder and often times difficult to treat (Geller et al., 2003; Masi et al., 2004; Sukhodolsky et al., 2005; Masi et al., 2006; Termine et al., 2006; Storch et al., 2008; Krebs and Heyman, 2010). In fact, comorbidity is the norm in this clinical sample, with up to 80% of children affected having at least one comorbid diagnosis (Swedo et al., 1989; Geller et al., 1996; Storch et al., 2008; Lewin et al., 2010), and as many as 50% to 60% of youth experiencing two or more other mental disorders during their lifetime (Rasmussen and Eisen, 1990).

Some of the most commonly co-occurring psychiatric conditions associated with pediatric OCD include other anxiety disorders (affecting 26 – 70% of children with OCD), depression (10 – 73% of children with OCD), tics and Tourette’s Syndrome (17 – 59% of children with OCD), attention deficit/hyperactivity disorders (10 – 50% of children with OCD), disruptive behavioural disorders (10 – 57%), and pervasive developmental disorders (e.g., Flament et al. 1990; Swedo et al., 1992; Thomsen, 1994; Geller et al., 1996; 2001a, 2001b; Ivarsson et al., 2008). In addition to these more frequently co-occurring conditions, youth with OCD may also present with comorbid eating disorders, body dysmorphia and
trichotillomania (King et al., 1995; Geller et al., 2001a; Phillips et al., 2005). Studies have suggested that certain comorbid psychiatric disorders associated with OCD (for example disruptive behavioural disorders, depression, attention deficit disorder) not only impact upon the severity of a child’s OCD but also have a negative effect on children’s psychosocial functioning and response to treatment (see Storch et al., 2008; Storch et al., 2010). Understanding the unique correlates of specific comorbid conditions and the impact of such on OCD treatment outcomes is important in both the assessment of OCD and the prescription of individualized treatments.

Cognitive-behavioural treatment (CBT), including exposure and response prevention (ERP), has been designated as probably efficacious for pediatric OCD based on a recent systematic review of the psychosocial treatment literature (see Barrett et al., 2008). Combined with the recommendations included in the OCD Expert Consensus guidelines (March et al., 1997) and other recent meta-analyses (e.g., O’Kearney et al., 2010), the general consensus in the literature and among experts in the field is that CBT combined with exposure and response prevention (ERP), either alone or in combination with a serotonin reuptake inhibiting (SRI) medication is both an effective and acceptable treatment for children and youth with OCD (see Barrett et al., 2004; Abramowitz et al., 2005; Barrett et al., 2008; O’Kearney et al., 2010).

Despite the fact that CBT produces impressive treatment effect sizes (i.e., between group effect sizes of 0.99 to 2.84; Barrett et al., 2008) and that the majority of children and adolescents with OCD experience clinically significant reduction in OCD symptoms following CBT, the outcomes in terms of actual remission rates provide less than optimal results. Across published studies and across sites within studies (e.g., POTS, 2004), remission rates vary. However, based on results from the largest multi-site RCT to date (Pediatric OCD Treatment Study (POTS), 2004), as many as 60% of children receiving CBT alone, 50%
receiving combined CBT and serotonergic medication, and almost 80% receiving serotonergic medication failed to fully remit following treatment. These findings suggest that one in two treatment-seeking children and youth will continue to suffer clinically significant OCD even after combined CBT and SRI treatment. There is therefore a pressing need to understand the predictors and moderators of treatment response in paediatric OCD, in order to determine which children will respond optimally to current generation treatments, and to identify ways to augment or refine these treatments for those who do not. Based on a recent review of predictors and moderators of treatment response (Farrell et al., in press) a consensus is emerging in regards to specific comorbid conditions that might attenuate treatment response for children with OCD.

To date, severity of OCD at pre-treatment, family dysfunction (Ginsburg et al., 2008; Garcia et al., 2010) and family accommodation (Garcia et al., 2010) have all been shown to be associated with poorer response to CBT. Conversely, in medication alone studies, comorbid tics have been associated with poorer outcome (Ginsburg et al., 2008), whilst across treatment modalities, externalising disorders have been found to be associated with poorer response (Garcia et al., 2010). Gender, age and duration of illness do not appear to differentially predict treatment outcome (Ginsburg et al., 2008; Garcia et al., 2010).

In a recent study, Storch and colleagues (2008) specifically examined the impact of comorbidity on response to CBT in a sample of 96 youth with a primary diagnosis of OCD. In this study, it was found that having one or more comorbid conditions was associated with a poorer response to CBT outcome, and that the number of comorbid conditions was negatively related to outcome. Moreover, and consistent with both Garcia et al. (2010) and Ginsburg et al. (2008), Storch and colleagues found that the presence of comorbid externalising disorders (i.e., attention deficit / hyperactivity disorder, oppositional defiant disorder, and conduct disorder) was associated with a poorer treatment response, and that both externalising
disorders and depressive disorders were associated with lower treatment remission rates. The authors of this study did not find evidence to suggest that comorbid anxiety disorders or comorbid tic disorders were associated with a poorer response to CBT, even though these co-occurring disorders were seen in a number of the youth.

Few studies have examined the important issue of moderators of treatment response in paediatric OCD. March and colleagues (2007) reported on the impact of comorbid tic disorder on outcomes in the POTS trial (2004), examining treatment response for the 15 percent of the POTS sample (n=17 of 112) who had a comorbid tic disorder. In patients without tic disorders, outcomes were consistent with the entire intent-to-treat sample (POTS, 2004) with combined treatment (CBT+ sertraline) being superior to CBT alone, which was superior to sertraline alone, which was superior to the placebo condition (POTS, 2004). However, for the sample with comorbid tic disorders, sertraline alone did not differ significantly from the placebo condition, whilst combined treatment (CBT + sertraline) remained superior to CBT, and CBT remained superior to PBO. This finding, consistent with Ginsburg et al. (2008), provides strong evidence that children with comorbid OCD and tic disorders respond differentially to medication alone versus cognitive-behavioural treatments. Based on this finding, March and colleagues (2007) recommend that children with OCD and comorbid tic disorder should begin treatment with CBT alone or a combined treatment of CBT and SRI, given that medication alone does not provide benefit over a placebo pill.

The issue of treatment response as a function of comorbidity in paediatric OCD warrants further consideration in terms of: (a) an examination across a broader array of comorbid diagnoses, and (b) an examination of treatment response to group-based CBT – which to date has not been systematically examined. Whilst our understanding about specific comorbidities is advancing, we still know very little about the impact of other commonly co-occurring psychiatric conditions Pervasive Developmental Disorders (PDD), including
Autism Spectrum Disorders (ASD), given that these disorders are frequently excluded from randomised controlled treatment trials and to date have not been systematically examined as potential predictors of treatment response. In particular, CBT might be more difficult to deliver with children who have comorbid PDD and/or ASD, due to poor emotional understanding and cognitive rigidity characteristic of these disorders (Krebs and Heyman, 2010). Furthermore, whilst group-based CBT has been established as possibly efficacious and provides an alternative to individual CBT for children and youth with OCD (see Barrett et al., 2008) we do not yet know what impact comorbidity may have on group treatment outcomes.

Group CBT is a favourable modality of therapy, with evidence so far providing comparable outcomes to individual CBT in a randomised controlled trial at post-treatment and at 18 months follow-up (Barrett et al., 2004; Barrett et al., 2005). In fact, group CBT offers an efficient and economical alternative, which improves both access to treatment and reduces treatment costs and therapist time. Moreover, group therapy arguably provides additional benefits for children and families beyond the technical aspects of CBT, through providing peer normalisation and peer support in a positive group setting. However, to date little is known about the impact of comorbidity on CBT outcomes for children treated in groups.

Given that certain comorbid disorders would likely have a negative impact on the group therapy process – for example disruptive behavioural disorders or ASD, it is important to establish the impact of such on group CBT outcomes for pediatric OCD.

The present study aims to (1) evaluate the effectiveness of group CBT in an open pilot trial design, to specifically examine the impact of comorbidity on outcome, using a highly comorbid sample of children with OCD up to 6 months following treatment; and (2) to examine the effect of specific comorbidity’s on treatment response and treatment remission in a children and youth with OCD (aged 7 – 17 years). Based on studies involving signal detection analysis to identify optimal cut-offs on the Yale-Brown Obsessive-Compulsive
Scale (YBOCS; Goodman et al., 1989; Child YBOCS; Scahill et al., 1997) for predicting treatment response and clinical remission (Tolin et al., 2005; Storch et al., 2010), this study uses criteria of at least 25% reduction in CYBOCS scores for determining treatment response, and a reduction of 50% on the CYBOCS combined with a post-treatment CYBOCS score of <14 for determining treatment remission (Storch et al., 2010).

Given that comorbidity with other anxiety disorders has not previously been identified as a significant predictor of response to treatment, nor have tics or Tourette's in the case of CBT, this study aims to examine other arguably more complex comorbidity – that is, comorbid conditions that have previously been indicated to possibly attenuate treatment response or to date have not yet been adequately studied, including (a) depression (DEP), (b) attention deficit / hyperactivity disorders (ADHD) and (c) pervasive developmental disorders (PDD), including ASD. This paper examines the impact of comorbidity on group treatment response and remission at post-treatment and at 6 months follow-up following a standardised group cognitive-behavioural treatment. Based on previous research (e.g., Storch et al., 2008; Storch, Lewin et al., 2010), it was hypothesised (a) based on pre-treatment comparisons, the presence of comorbid conditions (i.e., DEP, ADHD, PDD) would be associated with significantly worse OCD and higher functional impairment at baseline relative to children without these comorbid conditions (i.e., no comorbid DEP, ADHD, or PDD); (b) group treatment would be effective for the overall sample; however, (c) specific comorbidity (i.e., DEP, ADHD, PDD) would be associated with poorer treatment response and treatment remission following group-based CBT, relative to children without comorbidity.

2. Method

2.1. Participants
Participants were 43 children and adolescents (aged 7 – 17 years), with a mean age of 11.09 years (SD = 2.52), comprised of 30 males and 13 females, who were consecutively referred for treatment of OCD to Griffith University and whom completed assessment and participated in treatment. There were a further four participants who were referred to the program, who were eligible for participation, but whom withdrew prior to completion of assessment or prior to treatment commencing and were therefore excluded for these reasons. Participants were selected into this study on the basis of a Diagnostic and Statistical Manual (DSM-IV; American Psychiatric Association, 1994) diagnosis of OCD. Exclusion criteria included psychosis, intellectual disability, mental retardation, or currently receiving psychotherapy. There were no referrals to the project during this time that met exclusion criteria. All children were offered treatment following their assessment.

Based on ADIS-P diagnostic interview (ADIS-P; Silverman and Albano, 1996), this sample was deemed typical of paediatric OCD, consisting of high comorbidity, with 86% presenting with a secondary psychiatric diagnosis and 74% presenting with a tertiary diagnosis. Eighty-six percent of the sample presented with primary OCD, whereas the remainder (n=6) had OCD as either secondary or tertiary. Table 1 presents diagnostic information for the sample, including principal, secondary and tertiary diagnoses. On assessment, the mean CY-BOCS (Scahill et al., 1997) rating was 21.36 (SD = 6.63) indicating the sample was overall within the upper range of moderate severity. Sixty-seven percent of the sample were stabilised on an SRI medication prior to enrolment into this study, and they did not alter their medication during this trial.

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In regards to the comorbidity groups of interest in the current study, Figure 1 displays the frequency of each comorbidity group, including also the proportion of the current sample who did not present with any of the three comorbid conditions of interest (i.e., no comorbid n = 23) within the PDD group, 60% (n = 9) were diagnosed PDD Not Otherwise Specified, and 40% (n = 6) were diagnosed with Asperger’s Syndrome.

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2.2. Measures
2.2.1. Interview Measures
2.2.1.1. The Anxiety Disorders Interview Schedule for Children – Parent version (ADIS-P; Silverman and Albano, 1996). The ADIS-P was developed specifically to diagnose anxiety disorders in children (Silverman and Eisen, 1992), and possesses good inter-rater and retest reliability. The ADIS-C/P has demonstrated good sensitivity to treatment effects in both childhood anxiety (Kendall, 1994; Barrett et al., 1996; Ollendick et al., 2009) and childhood OCD research (Albano et al., 1996; Waters et al., 2001; Barrett et al., 2004). This interview was administered to the child’s parent/s to ascertain an OCD diagnosis and confirm secondary and tertiary comorbid diagnoses, including other anxiety disorders, mood disorders (including major depressive disorder [MDD] and dysthymia), externalizing disorders (including attention deficit / hyperactivity disorder [AD/HD] and oppositional defiant disorder [ODD]) and to screen for Pervasive Developmental Disorders (PDD). Each diagnosis receives a Clinician Severity Rating (CSR) based on clinician judgment, scored 0 – 8, with a score of 4 indicating a clinically significant diagnosis.
Any child who scored positive on the ADIS-P screen for a PDD diagnosis and who also had a confirmed diagnosis of PDD or an ASD (including Asperger’s Syndrome) by a community pediatrician or child psychiatrist was deemed to have a diagnosis of PDD in the current study. For tics and Tourette’s syndrome, as the ADIS-P does not screen specifically for these disorders, a diagnosis was based on a brief structured clinical interview devised by the first author for this purpose. Forty percent \((n = 17)\) of the sample had a tic disorder at pre-treatment. Inter-rater reliability has been conducted across 20% of the video-taped diagnostic interviews by an independent rater, with results indicating excellent reliability (primary diagnosis \(\kappa = 1.0\); secondary diagnosis \(\kappa = 0.84\); tertiary diagnosis \(\kappa = 0.83\)).

2.2.1.2. National Institute of Mental Health Global Obsessive-Compulsive Scale (NIMH–GOCS; Insel et al., 1983). This clinician-rated device consists of a single item measuring global diagnostic severity on a scale from 1 (minimal symptoms, within normal range) to 15 (very severe). The GOCS also provides a scale of clinical global improvement (CGI), ranging from 1 (very much improved) through to 7 (very much worse), with 4 indicating no change. The GOCS has demonstrated good to excellent retest reliability (Kim et al., 1992; Kim et al., 1993), and adequate to good convergent validity with the SCL-90 OC scale and the CY-BOCS (see Taylor, 1998).

2.2.1.3. Children’s Yale-Brown Obsessive-Compulsive Scale (CY-BOCS; Scahill et al., 1997). The CY-BOCS is a widely used, clinician-rated, semi-structured interview, assessing severity of OCD symptomatology. The CY-BOCS rates severity of obsessions and compulsions across five scales: (a) time occupied by symptoms, (b) interference, (c) distress, (d) resistance, and (e) degree of control over symptoms, and also provides a total severity score. The CY-BOCS shows reasonable reliability and validity, with good to excellent inter-
rater agreement and high internal consistency for total score (Scahill et. al., 1997). This interview was administered to children (including parents for the younger sample, 7 to 11 years) to assess overall OCD symptom severity.

2.2.2. Self-Report Measures

2.2.2.1. Child OCD Impact Scale – Child/Adolescent Report and Parent Report (COIS – C/P; Piacentini et al., 2001). The Child OCD Impact Scale assesses the impact of OCD on psychosocial functioning from child and parent report. This measure includes 20 items each across three domains, rated on 4-point likert-scales. The COIS-C/P assesses three domains of impairment including school, social, and family/home. Four additional items assess the global impact of OCD on school/work, home, social, and going out. Studies using the child COIS have shown excellent internal consistencies for the three subscales and the total score (range $r = 0.78$ to 0.85; Piacentini et al., 2001), and good convergent validity between the COIS total score and the CY-BOCS ($r = 0.46$; Piacentini et al., 2001).

2.2.2.2. Multidimensional Anxiety Scale for Children (MASC; March, 1997). This self-report measure assesses anxiety symptoms in children across a number of scales, including physical symptoms, harm avoidance, social anxiety and separation/panic. The MASC is comprised of 39 items assessing frequency of anxiety symptoms/concerns, with items being scored 0 (not at all) to 3 (often), and provides a total anxiety score. Research has indicated that the MASC has good internal reliability and convergent validity (March, 1997; March et al., 1997).

2.2.2.3. Children’s Depression Inventory (CDI; Kovacs, 1992). The CDI is comprised of 27 items assessing symptoms of depression, scored 0 (absence of symptom), 1 (mild symptom), or 2 (definite symptom), with higher scores indicating increasing severity and a total score that ranges from 0 to 54. The extensive use of the CDI in clinical and experimental research has provided ample evidence to support its reliability and validity (see Kovacs, 1992).
2.2.2.4. Family Accommodation Scale (FAS; Calvocoressi et al., 1995). This measure assesses the frequency and severity of parental accommodation to OCD, and provides a total by summing 8 of the 12 items, which are scored as 0 (never/no accommodation) to 4 (daily/extreme accommodation). There is an additional item that rates the parents distress associated with accommodation, and a further three items which assess the consequences of not participating in accommodation to their child’s OCD behaviors. A recent psychometric evaluation of the FAS used with a child sample of OCD patients (n=96) provides evidence for good internal consistency, as well as good convergent and divergent validity of the measure (Flessner et al., 2011).

2.3. Procedure

All procedures and protocols used in this study received institution review board approval through the university human research ethics committee. Following referral into this study, participants were screened via a brief parent interview assessing for obsessive-compulsive symptomatology. If eligible, families attended an assessment at the university psychology clinic, conducted by the first author and postgraduate clinically trained clinicians. On attending this interview, the research aims were explained to all participants and written informed consent was gained from parents. Initial assessment interviews involved ADIS-P interviews with parents and the CY-BOCS interview with children (including parents for younger children 7-11 years). Interviewers were trained in diagnostic interviews and CY-BOCS interviews through observation of the first author, followed by supervision of their interviewing skills by the first author. Assessment also included the completion of a number of self-report questionnaires, including the OCD Impact Scale (child and parent version). Complete assessments were conducted at pre and post-treatment in the clinic, and brief
versions of these assessments were conducted at 6 months follow-up over the telephone (including the ADIS-P – OCD section only, the interviewer also rated NIM-GOCS, and the child CY-BOCS interview at this time). When a sufficient number of youth (within a similar age range) had entered the study and been assessed, a treatment group was formed.

2.3.1. Treatment Protocol: The treatment program entitled OCD Busters (Child and Adolescent Versions) (Farrell and Waters, 2008) was a manualised family-based CBT treatment protocol, based on March and colleagues' individual CBT protocol (“How I ran OCD off My Land”; March et al.,1994; March and Mulle, 1998). Children aged 7 – 12 years participated in the child version of the program and those aged 13 – 17 years completed the adolescent program. The treatment involved 13 weekly child group sessions and 2 booster sessions (1 month and 3 months post-treatment), each running for approximately 1.5 hours, with each session including a brief parental involvement/family review of progress at the end of the child group session (15 minutes). In addition to the child group sessions, there were 3 structured parent group sessions (1 hour each), and two individual family review sessions, each 1 hour in duration scheduled at week 5 and week 10 of the program (including the child and his/her parents and siblings if desired). Individual family review sessions allowed for the therapist to engage the child in therapist-assisted ERP, and address family accommodation and any issues that were not being addressed in the group sessions (i.e., family conflict).

There were six trained therapists who delivered the program at the clinic in group format with two therapists implementing each group program under the supervision of the first author. Therapists were all postgraduate level clinicians with previous experience in CBT treatment of child anxiety disorders, but not specifically OCD. At least one therapist for each group however had some previous experience in delivering CBT treatment for child OCD. All clinicians received formal weekly supervision wherein clinicians reported on client progress, adherence to the treatment protocol, and provided an opportunity to ask questions/problem...
solve treatment difficulties or group process issues. Treatment fidelity was assessed by an independent rater, who viewed 20% of group sessions (random selection), and rated each session on a likert scale from 0 – 5 (0 = very poor fidelity – 5 = excellent fidelity). This approach is consistent with other treatment studies (e.g., Tolin et al., 2005; Storch et al., 2010); results in the current study found excellent adherence to the treatment protocol ($m = 4.76; sd = 0.44$).

The child group sessions focused on psychoeducation, cognitive training, anxiety management training, developing stimulus hierarchies, graded exposure and response prevention (including in session group ERP exercises, and establishing homework ERP), building buffer zones with support networks, and relapse prevention. Parent group sessions were conducted by one therapist from the child group following the child sessions at week 2, 5 and 10, and focused on psychoeducation, problem-solving skills, strategies to reduce parental involvement in the child’s symptoms, along with encouraging family support of home-based exposure and response prevention trials. At least one parent from each family was required to attend each parent session. In the majority of cases (87% of the sample) mothers attended the parent session. The program emphasized that the coping strategies taught needed to be practiced as a family on a daily basis. Children could miss up to two sessions provided that missed sessions were caught up with the therapist before the group session. No family missed more than two sessions. The two booster sessions provided additional opportunities for children to gain assistance in generalizing the skills learnt in previous sessions. There were at least four children in every group with a maximum of seven children per group.

3.0 Results
All statistical analyses were conducted using SPSS version 19.0. Initial data analyses compared baseline demographics and clinical characteristics across two groups – those children with a comorbid diagnosis of interest (i.e., DEP, ADHD, PDD: n=20) and those children without any of the three comorbid conditions of interest (n= 23). Independent samples t-tests ($t$) were computed for continuous variables and chi-square ($\chi^2$) analyses were computed for categorical data. Treatment outcome was examined at post-treatment and 6-month follow-up across the comorbid versus non-comorbid groups by way of repeated measures mixed-factorial ANOVAs, including a 2 group (comorbid vs non-comorbid) X 3 time point design (pre, post, follow-up).

As noted earlier, treatment response was defined as at least a 25% reduction in CYBOCS scores at post-treatment and 6 month follow-up, whereas treatment remission was defined as a reduction of 50% on the CYBOCS combined with a post-treatment / follow-up CYBOCS score of <14 (see Tolin et al., 2005; Storch et al., 2010). Chi-square analyses examined treatment response and treatment remission at post-treatment and 6 month-follow-up across the separate comorbid conditions of interest (i.e., DEP, ADHD, PDD) relative to children without each of the comorbid conditions. Analyses were conducted in two ways – firstly, comparing children who had one of the three comorbid conditions of interest, versus children who did not have that comorbid condition (but may have had another comorbid condition); as well as examining children with each of the comorbid conditions relative to children without any of the three comorbid conditions of interest. The results of both sets of analyses did not differ; hence the first approach is presented here (as these analyses allowed for a larger $n$ in the non-comorbid group).

3.1. Pre-treatment Comparisons. Baseline demographics (including age, gender, and combined family income) and clinical characteristics (diagnostic severity, OC severity,
anxiety, depression, functional impairment, family accommodation) were examined across the two groups – those children with a comorbid diagnosis of interest (i.e., DEP, ADHD, PDD: n=20) and those children without any of the three comorbid conditions of interest (n=23). On demographic variables, there were no significant group differences on age or income; however, the comorbid group was comprised of significantly less females (i.e., 23%) than the non-comorbid group (77%) $\chi^2 = 4.11 (1); p < 0.05$.

On clinical characteristics, the comorbid group did not differ significantly from the non-comorbid group on most severity ratings of OCD (measured by the NIMH GOCS, NIMH CGI and CYBOCS scores), self-reported anxiety (MASC), or child rated functional impairment (COIS-P/C). The comorbid group was however significantly higher on self-report ratings of depression (CDI) $t = 3.37 (33); p < 0.005$, parent ratings of functional impairment (COIS-P) $t = 2.74 (34); p < 0.01$ and on parent rated family accommodation $t = 2.67 (34); p < 0.01$. Given the comorbid group included children with a diagnosis of depression (n=5), group differences on self-reported depression were also examined excluding the children with a diagnosis of MDD or dysthymia. Results demonstrated the comorbid group continued to be significantly higher on self-reported depression even after excluding the children with a diagnosis (CDI) $t = 2.59 (28); p < 0.02$. Table 2 displays the baseline demographics and clinical characteristics data. With an increase in the number of comorbid diagnoses present at pre-treatment (i.e., one to three comorbid conditions were recorded), there were significant positive correlations observed with diagnostic severity (CSR; $r = 0.32$, p<0.05), family accommodation (FAS; $r = 0.35$, p<0.05), depression (CDI; $r = 0.45$, p<0.01) and anxiety (MASC; $r = 0.37$, p<0.05).

3.2 Group Treatment Outcome. The effectiveness of group CBT was evaluated by way of repeated measures, mixed factorial ANOVAs – with a time (pre, post, 6 months follow-up) X
group (comorbid versus non-comorbid group) design across primary outcome measures of OCD diagnostic severity (ADIS-P CSR; NIMG GOCS; CGI), OCD symptom severity (CY-BOCS) and OCD functional impairment (COIS-C/P). The multivariate Time x Group interaction was not significant; however, there were significant Time main effects across each variable. The same repeated measures ANOVAs were conducted also across each of the specific diagnostic comorbidities (i.e., DEP versus no-DEP; ADHD versus no-ADHD; PDD versus no-PDD) and there were also no significant time X group interactions.

Post-hoc analyses of the significant multivariate time main effects demonstrated that group CBT was effective regardless of comorbidity, with significant reductions for the entire sample from pre- to post-treatment on the ADIS-P CSR $t(36)=10.937, p < .001$, on the NIMH GOCS $t(36)= 9.843, p < .000$, on the CY-BOCS $t(37)= 5.93, p < .001$, on COIS-P $t(36)= 4.766, p < .001$, and on the child COIS $t(36)= 3.056, p < .005$, with all outcomes associated with moderate (i.e., COIS-C) to large effect sizes in the range of $d $0.69 – 1.65.

Gains were maintained to 6 months follow-up, with no significant differences from post-treatment to 6 months follow-up across primary outcome measures. Table 3 presents the means, standard deviations and t-tests for the time main effects from pre- to post-treatment (including means at follow-up).

### 3.3 Treatment Response as a Function of Comorbidity

At post-treatment, there was an overall mean reduction in CY-BOCS ratings of 45% ($m$ reduction CYBOCS = 8.08; $sd$ = 8.39). Furthermore, at post-treatment 60.5% of the entire sample was classified as responders (i.e., > 25% reduction in CYBOCS). This response was largely maintained to 6-month follow-up with 56% of the sample classified as responders at follow-up. Figure 2 presents the percent of responders for each comorbidity group at post-treatment and follow-up.
At post-treatment, there was no significant difference in the percent of children classified as responders between the comorbid versus non-comorbid groups. Furthermore, across each of the comorbidity groups, there were no significant differences in the percentage of children classified as responders, relative to children without that comorbid condition. At 6-month follow-up, there was likewise no significant difference in the percent of children classified as responders between the comorbid versus non-comorbid groups. There was a significant difference in the percent of children classified as responders for children with ADHD (43% responders) versus children without ADHD (81% responders) $\chi^2 = 3.99$ (1); $p = 0.06$. There were however no differences across DEP or PDD groups relative to children without comorbidity.

The impact of the number of comorbid conditions (i.e., one to three comorbid conditions) on outcome was examined by way of a correlation with percent reduction on CYBOCS scores from pre to post-treatment, and pre- to 6-month follow-up. Correlations amongst the frequency of comorbid diagnoses and CYBOCS reduction was significant at 6-months follow-up only, with a moderate negative correlations ($r = -.37$; $p< .05$).

3.4 Treatment Remission as a Function of Comorbidity. Remission was defined as a score of less than 14 on the CYBOCS combined with at least a 50% reduction on the CYBOCS. At post-treatment, 47% of the entire sample was classified as achieving treatment remission. This response was largely maintained to 6-month follow-up with 44% of the sample classified as in remission at follow-up. Figure 3 presents the percent of children classified as treatment remitters for each comorbidity group at post-treatment and follow-up.
At post-treatment, there was no significant difference in the percent of children classified as being in remission of their OCD between the comorbid versus non-comorbid groups. Furthermore, across each of the comorbidity groups, there were no significant differences in the percentage of children classified as remitters, relative to children without that comorbid condition. At 6-month follow-up, there was likewise no significant difference in the percent of children classified as remitters between the comorbid versus non-comorbid groups. There was however a significant difference in the percent of children classified as being treatment remitters at 6 months follow-up for children with ADHD (25% as remitters) versus children without ADHD (65% as remitters) $\chi^2 = 4.05$ (1); $p < 0.05$. There were no differences across DEP or PDD groups relative to children without comorbidity.

4.0 Discussion

This pilot study evaluated the effectiveness of group CBT on outcomes for children and youth with OCD who presented with complex comorbidity, including depression (DEP), attention deficit/hyperactivity disorder (ADHD) and pervasive developmental disorders (PDD, including ASD). Specifically, this study examined the correlates of complex comorbidity at baseline (including severity, and impairment), as well as group treatment outcomes at post-treatment and 6 month follow-up, by way of remission rates and treatment response as a function of comorbidity. It was expected that (a) the presence of comorbidity (i.e., DEP, ADHD, PDD) would be associated with significantly worse OCD and higher functional impairment at baseline; (b) group CBT would be effective for children with OCD; however, (c) comorbidity (i.e., DEP, ADHD, PDD) would be associated with poorer treatment
response and treatment remission following group-based CBT relative to children without this comorbidity.

This sample was a highly comorbid sample, with comorbidity rates above those reported in most previous studies (e.g., Rasmussen and Eisen, 1990; Storch et al., 2008) – with 86% of the sample presenting with a secondary psychiatric disorder, and 74% presenting with a tertiary psychiatric condition. This sample was also uncharacteristically predominantly male, with 79% of the sample male – differing from past pediatric treatment samples, which usually report equal (or close to equal) gender ratios (e.g., Barrett et al., 2004; POTS, 2004; Storch et al., 2008). The high rates of comorbidity and the male predominance in this sample may be attributable to the relatively high rates of comorbid ADHD (21%) and PDD (35%) within the current sample. In regards to baseline characteristics of the children classified as having one of the three comorbid conditions, relative to children without one of these comorbid conditions, the comorbid group were significantly higher on self-report ratings of depression and on ratings of family accommodation to their OCD symptoms providing evidence in line with previous investigations (e.g., Storch et al., 2008), which tends to support higher impairment associated with higher rates of comorbidity. Furthermore, the comorbid children were consistently more severe across measures of diagnostic severity and OC severity, as well as parental report of functional impairment – although not statistically significant, which may have been a function of limited power in the current study.

In regards to treatment outcome following group CBT, the current study demonstrated that there were no significant time X comorbid condition interactions, suggesting that overall children did not respond differentially to group-based CBT as a function of their comorbidity. Importantly, group CBT involving children with a range of comorbid conditions, including comorbid PDD, produced favourable outcomes across all primary outcome variables comparable to those reported in the POTS trial (2004) – the largest, most rigorous multi-site
RCT to date. In fact, the current study of group CBT demonstrated an overall mean reduction of 47% on the CY-BOCS – which is largely equivalent to POTS outcomes (i.e., 46% mean reductions for CBT and 53% for combined CBT+SRI). In terms of the overall response to group CBT, the current study found 47% remission rate, which is also comparable to that reported in the POTS trial (i.e., 39% remission defined by CYBOCS <10). These outcomes are very promising and suggest that overall group based CBT including parental involvement, with a highly comorbid sample was largely effective – for all children.

Whilst the current findings provide support for group CBT in treating OCD with complex comorbidity, the results also suggest that the number of comorbid conditions was negatively associated with outcomes at longer term follow-up. That is, the most complicated cases, including more than one comorbid condition, was associated with a significant decline in treatment response over time. The data presented in the current study did not provide evidence for differential effects on treatment response or treatment remission rates for children who had comorbid depression or pervasive developmental disorders; however, the results did suggest that comorbid attention deficit/hyperactivity disorder was associated with a significantly poorer response and remission rate by 6-month follow-up. This finding is consistent with previous research that has found that externalising disorders predict a poorer response to CBT (Storch et al., 2008; Garcia et al., 2010).

Overall, the current study provides promise for the treatment of OCD, including complex comorbidity, within group-based delivery of CBT. Contrary to hypotheses, the presence of depression and pervasive developmental disorders (including ASD) was not associated with a poorer response to treatment up to 6 months follow-up. Furthermore, the overall quality of outcomes for children involved in this group treatment study did not appear to be eroded by having more complex cases present within the group. Moreover, given that none of specific comorbidity’s had a negative effect on outcomes at post-treatment, the
results of the current study are suggestive that group CBT may actually offer additional benefits for arguably more complex cases, characterised by high comorbidity. Group based CBT provides normalisation of OCD, as well as provides peer support, and arguably increased compliance with treatment and motivation for change, driven by in-group norms that underlie group therapy. In our clinical experience of running groups, we found that children who were finding exposure and response prevention difficult, did actually make very positive progress (even if slightly delayed), once they witnessed others in their peer-group making significant change and being rewarded for such. The finding that group CBT is largely effective children with comorbid conditions is of clinical and practical significance given the recent push in health and medical research to enhance patient access to evidence-based treatments. Group delivery of CBT provides an efficient and cost-effective means of providing treatment to patients, and alleviates strain on services and service providers. The results do suggest however, that outcomes for children with multiple comorbid conditions, and for children with comorbid attention deficit/hyperactivity disorder, were attenuated at 6 months follow-up. This finding highlights the need to determine if outcomes might be improved for these more complex (i.e., numerous comorbid conditions) or “difficult-to-treat” cases through increased booster sessions following treatment, and/or by tailoring treatments to address comorbid psychopathology. For example, one approach might be a modular treatment program for children with comorbid OCD and externalising disorders, which differentially targets OCD symptoms, as well as behaviour management, emotion regulation and parental contingency management of child behaviour, and is delivered on a flexible, individualised basis.

There are a number of limitations to this study that require highlighting. Firstly, this study was an open clinical pilot study, hence it was not possible to quantify the relative response to CBT versus a control condition. Furthermore, the examination of multiple
comorbid conditions within this clinical sample resulted in relatively small samples for each group. Further research with larger clinical sub-samples across each comorbid group is necessary to confirm outcomes reported in this pilot trial with greater statistical confidence. This was particularly evident in the depression group; hence, analyses are likely to be affected by limited power, potentially leading to type 2 errors and therefore require replication with larger samples. Additionally, this study did not use structured, psychometrically validated interviews for diagnoses of Pervasive Developmental Disorders. This may have implications for the validity of these diagnoses in the current study; however, this study did attempt to address this limitation by developing semi-structured clinical interview questions, and sought confirmation of PDD diagnoses by external pediatric specialists. Reliance on parent only ADIS interviews is a limitation in the current study; however past research demonstrates good convergence between parents and clinicians, which is frequently higher than with child report (Grills and Ollendick, 2003). Future research investigating the impact of comorbidity on treatment response with large samples, examining multiple treatments, and including children with complex comorbid conditions, is warranted to advance our understanding about moderators of treatment response in pediatric OCD.

In sum, this study provides an evaluation of group-based CBT up to 6 months following treatment for children with OCD and co-occurring psychiatric conditions, including clinical depression, attention deficit/hyperactivity disorder and pervasive developmental disorders. Group-based CBT which incorporates individual parental involvement, individual family review sessions, in-session group exposure exercises and booster sessions at 1 month and 3 months follow-up, appears both feasible and effective for children with arguably more “difficult-to-treat” OCD, defined by a range of complex comorbid conditions. Treatment outcomes are attenuated at follow-up for children with two or more comorbid conditions and for children with attention deficit/hyperactivity disorder.
Efforts to improve treatment for these children are warranted through innovation to current CBT approaches.
Note:

1 PDD was used as a broad category for children with a diagnosis of PDD, autistic spectrum disorder or Asperger’s syndrome that was confirmed by a community psychiatrist or paediatrician – which is the standard requirement for national health fund rebates and school ascertainment in QLD, Australia. Full diagnostic interviews were not conducted for ASD and/or PDD given the training required and length of time required for these standard diagnostic assessment tools, and given that it was outside the scope of the current study. If a child was positive to the ADIS-P screen and didn’t yet have a diagnosis by a community paediatrician or psychiatrist, the referral was made for this purpose. All families followed up on this referral when this was the case (n=2) and all received a PDD diagnosis.
References


Goodman, W.K., Price, L.H., Rasmussen, S.A., Mazure, C., Fleischmann, R.L., Hill, C.L.,
Krebs, G., Heyman, I., 2010. Treatment-resistant obsessive-compulsive disorder in young


Toro, J., Cervera, M., Osejo, E., Salamero, M., 1992. Obsessive compulsive disorder in


Table 1. Participant Diagnoses at Pre-Treatment based on ADIS-P Interviews

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Principal Diagnosis</th>
<th>Secondary Diagnosis</th>
<th>Third Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>N</td>
<td></td>
</tr>
<tr>
<td>OCD</td>
<td>37</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>86%</td>
<td>9%</td>
<td>5%</td>
</tr>
<tr>
<td>GAD</td>
<td>1</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>2%</td>
<td>21%</td>
<td>14%</td>
</tr>
<tr>
<td>SAD</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>2%</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>SoPH</td>
<td>0</td>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>12%</td>
<td>7%</td>
</tr>
<tr>
<td>SpPH</td>
<td>1</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>2%</td>
<td>16%</td>
<td>7%</td>
</tr>
<tr>
<td>MDD</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>2%</td>
<td>5%</td>
</tr>
<tr>
<td>DYS</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>2%</td>
<td>2%</td>
</tr>
<tr>
<td>ADHD</td>
<td>1</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>2%</td>
<td>2%</td>
<td>14%</td>
</tr>
<tr>
<td>ODD</td>
<td>0</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>%</td>
<td>2%</td>
<td>%</td>
</tr>
<tr>
<td>PDD</td>
<td>2</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>5%</td>
<td>14%</td>
<td>16%</td>
</tr>
<tr>
<td>TOTAL</td>
<td>43</td>
<td>37</td>
<td>32</td>
</tr>
<tr>
<td></td>
<td>100%</td>
<td>86%</td>
<td>74%</td>
</tr>
</tbody>
</table>

Abbreviations: GAD = generalised anxiety disorder, SAD = separation anxiety disorder, SoPH = social phobia, SpPH = specific phobia, MDD = major depressive disorder, DYS = Dysthymic Disorder, ADHD = attention deficit / hyperactivity disorder, ODD = oppositional defiant disorder, PDD = pervasive developmental disorder.
Table 2. Baseline Demographic and Clinical Characteristics across Comorbid versus Non-Comorbid Groups

<table>
<thead>
<tr>
<th></th>
<th>Comorbidity</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>t or $\chi^2$</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Comorbid</td>
<td>11.95</td>
<td>2.86</td>
<td>1.91</td>
<td>0.07</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>10.48</td>
<td>2.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender (female) %</td>
<td>Comorbid</td>
<td>23%</td>
<td>-</td>
<td>4.11</td>
<td>0.04*</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>77%</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined Family Income</td>
<td>Comorbid</td>
<td>61-70K</td>
<td>3.57</td>
<td>-1.02</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>71-80K</td>
<td>2.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADIS-P CSR</td>
<td>Comorbid</td>
<td>6.35</td>
<td>0.81</td>
<td>2.33</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>5.55</td>
<td>1.37</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIMH GOCS</td>
<td>Comorbid</td>
<td>9.42</td>
<td>2.06</td>
<td>1.12</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>8.73</td>
<td>1.88</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NIMH CGI</td>
<td>Comorbid</td>
<td>4.44</td>
<td>1.15</td>
<td>-.717</td>
<td>0.49</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>4.68</td>
<td>0.95</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total CYBOCS</td>
<td>Comorbid</td>
<td>22.00</td>
<td>5.96</td>
<td>0.65</td>
<td>0.52</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>20.65</td>
<td>7.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Accommodation</td>
<td>Comorbid</td>
<td>25.44</td>
<td>12.63</td>
<td>2.67</td>
<td>0.01**</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>15.38</td>
<td>4.84</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CDI Total</td>
<td>Comorbid</td>
<td>15.00</td>
<td>8.93</td>
<td>3.37</td>
<td>0.002***</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>7.13</td>
<td>4.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MASC Total</td>
<td>Comorbid</td>
<td>63.87</td>
<td>16.18</td>
<td>1.65</td>
<td>1.04</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>54.08</td>
<td>17.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COIS-P</td>
<td>Comorbid</td>
<td>38.93</td>
<td>24.91</td>
<td>2.74</td>
<td>0.009**</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>32.00</td>
<td>27.26</td>
<td></td>
<td></td>
</tr>
<tr>
<td>COIS-C</td>
<td>Comorbid</td>
<td>46.44</td>
<td>20.90</td>
<td>0.76</td>
<td>0.45</td>
</tr>
<tr>
<td></td>
<td>Non-comorbid</td>
<td>28.95</td>
<td>18.28</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: * p < 0.05; **p<0.010; ***p<0.005
Table 3. Treatment Outcome Main Effects of Time across Primary Outcome Measures.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Pre Mean (SD)</th>
<th>Post Mean (SD)</th>
<th>F-UP Mean (SD)</th>
<th>Cohen’s d (pre - post)</th>
<th>Significance (Pre-to Post-Treatment)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIMH GOCS</td>
<td>8.89 (1.92)</td>
<td>4.83 (2.44)</td>
<td>4.66 (2.24)</td>
<td>1.65</td>
<td>t (36)= 9.843 ***</td>
</tr>
<tr>
<td>ADIS-P CSR</td>
<td>5.89 (1.92)</td>
<td>2.08 (2.28)</td>
<td>2.54 (2.33)</td>
<td>1.61</td>
<td>t (36)= 10.937 ***</td>
</tr>
<tr>
<td>CYBOCS</td>
<td>20.45 (6.82)</td>
<td>12.43 (8.18)</td>
<td>10.32 (7.37)</td>
<td>0.92</td>
<td>t (37)= 5.93 ***</td>
</tr>
<tr>
<td>COIS-P</td>
<td>35.32 (21.07)</td>
<td>21.36 (20.19)</td>
<td>-</td>
<td>0.90</td>
<td>t (36)= 4.766 ***</td>
</tr>
<tr>
<td>COIS-C</td>
<td>29.00 (17.07)</td>
<td>16.17 (19.41)</td>
<td>-</td>
<td>0.69</td>
<td>t (36)= 3.056 **</td>
</tr>
</tbody>
</table>

Note: *** p < 0.001; ** p < 0.005


Cohen’s $d$ is calculated correcting for dependence between means (associated with repeated measures design) using Morris & DeShon’s (2002) procedure (equation 8).
Figure 1. Percentage of the total sample within each of the comorbid groups.
Figure 2. Percent classified as responders at post-treatment and follow-up.
Figure 3. Percent classified as remitters at post-treatment and follow-up.