CBT for Anxious Children with Comorbid ADHD

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Abstract

Attention-deficit/hyperactivity disorder (ADHD) and anxiety are the most common psychiatric disorders of childhood and are highly comorbid. While cognitive behavioural therapy (CBT) has demonstrated general efficacy in treating paediatric anxiety, it is not clear whether ADHD comorbidity impacts treatment response. While some previous studies have found a negative impact of comorbidity, others have found no difference, and the role of ADHD subtype has never been assessed. We examined ADHD diagnosis as a predictor of treatment response and remission in a study of 842 children and adolescents aged 6 to 18 years undergoing group-based CBT for primary anxiety. A subsample of 94 children met criteria for comorbid, mild-to-moderate ADHD, mostly comprising Predominantly Inattentive (n = 61) and Combined (n = 27) subtypes. Neither ADHD diagnosis nor subtype predicted response or remission rates for children's primary anxiety disorders. Children with ADHD also showed modest yet significant improvements in ADHD symptoms after CBT treatment for anxiety. Our findings strongly support the suitability of manualised group-based CBT for anxiety treatment in children with non-primary ADHD. Further research should examine whether the positive outcomes reported can be extended to children with primary or severe ADHD.

Declaration of Originality

The works found within this thesis are original and have not been submitted for publication, written by another person, nor submitted for a higher degree to any other university or institution.

Karen Gould

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CBT for Anxious Children with Comorbid ADHD

Anxiety and attention-deficit/hyperactivity disorder (ADHD) are the two most commonly occurring psychiatric disorders in children and adolescents (Lawrence et al. 2015; Tannock, 2009). Both are associated with significant distress and impairment in social and academic functioning that can persist to adulthood (Langley, Bergman, McCracken, & Piacentini, 2004; Loe & Feldman, 2007; Rapee, Schniering, & Hudson, 2009; Wehmeier, Schacht, & Barkley, 2010). Around five percent of children experience anxiety disorders at any given time, and lifetime prevalence in studies of children and adolescents is estimated at 15-20 percent (Beesdo, Knappe, & Pine, 2009; Rapee et al., 2009). Where anxiety is excessive and impairing in children, several different disorders may be diagnosed, including separation anxiety disorder (SAD), specific phobias (SP), social anxiety disorder (SoAD) and generalised anxiety disorder (GAD); common to all is excessive fear and anticipation of imminent and future threat and related behaviour disturbances (The Diagnostic and Statistical Manual of Mental Disorders,5th ed. [DSM-5]; American Psychiatric Association [APA], 2013). While prevalence figures for ADHD vary widely, it is estimated that around 5 percent of children experience the disorder worldwide (Polanczyk, Silva de Lima, Horta, Biederman, & Rohde, 2007). ADHD is characterised by persistent impairment due to symptoms on one or both of two dimensions – inattention and hyperactivity-impulsivity (DSM-5). Depending on the distribution of symptoms, subtype diagnosis of ADHD – Predominantly Inattentive Type (ADHD-I), ADHD- Predominantly Hyperactive-Impulsive Type (ADHD-H) or ADHD – Combined Type (ADHD-C) is indicated.

Comorbidity of these disorders is common, and, if untreated, results in greater functional impairment than either disorder alone (Manassis, 2007). While pharmacological treatments are generally more effective in treating ADHD, cognitive behavioural therapy (CBT) is considered the gold standard in treatment of anxiety in children (James, James,

Cowdrey, Soler, & Choke, 2015; Van der Oord, Prins, Oosterlaan, & Emmelkamp, 2008). The present study focuses on the treatment of anxiety disorders in this comorbid group, aiming to examine the efficacy of CBT for children with ADHD diagnosis in addition to a primary anxiety diagnosis. If treatment is less successful in this group, it will be important to investigate modifications and accommodations to typical CBT approaches. On the other hand, evidence of efficacy may encourage families to seek anxiety treatment for children with ADHD and provide an evidence base for therapists wishing to extend CBT treatments to comorbid children. CBT treatment for primary disorders has sometimes been shown to have incidental effects on the comorbid condition (Borkovec, Abel, & Newman, 1995), so a secondary question examined is whether CBT for anxiety leads to improvements in comorbid ADHD. We begin with a review of research on the comorbid presentation, including aspects of its cognitive and behavioural profile that may impact treatment response.

Understanding Comorbid ADHD and Anxiety

Prevalence and Consequence.

Prevalence of ADHD is elevated amongst those with anxiety disorders, affecting around 25 percent of anxious children (Souza, Pinheiro, & Mattos, 2005). Correspondingly, population studies estimate that around a quarter of all children with ADHD also experience anxiety disorders, a rate that is roughly 2-4 times greater than that seen in the general population (Jarrett, Wolff, Davis, Cowart, & Ollendick, 2012; Pliszka, 2014). Rates of comorbidity are usually even higher amongst children referred to paediatric or psychiatric clinics, with anxious comorbidity reaching 40 percent amongst those treated for ADHD (Tannock, 2009). While the prevalence of ADHD declines over the course of development, anxiety rates are higher amongst adults, and for children who retain their ADHD diagnosis

into adulthood, the ADHD comorbidity rate increases to around 50 percent (Adler, Barkley, Newcorn, Spencer, & Weiss, 2007).

Children with comorbid anxiety and ADHD are a highly impaired group. The comorbidity is associated with greater rates of attentional problems and school fears and lower levels of social competence compared to both single diagnoses (Bowen, Chavira, Bailey, Stein, & Stein, 2008). Compared to children with ADHD alone, those who also have internalising symptoms (including anxiety) tend to have worse self-esteem, giving poorer evaluations of their own behaviour, academic performance, and social popularity (Bussing, Zima, & Perwien, 2000). Amongst clinically referred children with ADHD, lower academic achievement scores have also been noted in those with comorbid anxiety (Jensen et al., 2001). On the other hand, the presence of anxiety does not appear to increase behaviour problems compared to "straight" ADHD, and rates of conduct disorder are actually lower in the anxious subgroup (Manassis, 2007).

The long term sequelae of comorbidity in these children offer cause for concern, as children with both diagnoses in middle childhood report greater social problems and withdrawal in late adolescence (Newcorn et al., 2004). Although little research has been performed on the impact of comorbidity in adulthood, the increasing rates of anxiety amongst those with ADHD suggest continued impairment is likely amongst this group (Adler et al., 2007). And although around half of children with ADHD will no longer meet criteria for the disorder by adulthood, elevated rates of anxiety have been noted even in adults whose ADHD symptoms drop to sub-threshold levels (Biederman et al., 1994; Hechtman, 2000).

Reasons for High Comorbidity.

The unique challenges of children with ADHD-anxiety and the higher-than-chance rates of occurrence of the comorbidity have given rise to several attempts to explain its aetiology. Researchers have questioned whether the comorbidity consists of two separate coinciding disorders, or a distinct disorder with features of both anxiety and ADHD; whether anxiety might be an epiphenomenon of primary ADHD processes, or conversely, whether ADHD might arise from primary anxiety (Tannock, 2009). It is easy to imagine how the repeated failures experienced by children with ADHD, due to inattentive mistakes or impulsive missteps, might naturally lead to a state of heightened vigilance and worry consistent with anxiety disorders. Similarly, the rumination, distractibility and restlessness of children with anxiety disorders could establish an inattentive or hyperactive-impulsive behaviour profile.

High rates of comorbidity may point to underlying processes which, when delineated, may be key to improvements in both anxiety and ADHD symptoms. However, there is also the possibility that comorbidity rates are merely artefactual – simply related to overlapping methods of diagnosis and classification rather than co-occurrence of two disorders (Jarrett & Ollendick, 2008). Anxiety disorders and ADHD may present with some similar symptoms, for example, restlessness and poor concentration are symptoms of both ADHD and generalised anxiety disorder (GAD). For this reason, Milberger, Biederman, Faraone, Murphy, and Tsuang (1995) tested whether re-diagnosing comorbid adults and children without inclusion of overlapping symptoms would lead to lower rates of comorbid diagnosis. In adults with comorbid ADHD and GAD, 75-88 percent retained both diagnoses after common symptoms were discounted, suggesting that these high comorbidity rates are not solely artefactual of such overlaps. Using the diagnostic criteria of the time (DSM-III-R), ADHD symptoms overlapped with GAD symptoms only in adults, therefore overlap of

anxiety and ADHD symptoms was not assessed in children. Jarret and Ollendick (2008) also point out that this study's findings may be of less relevance to today's ADHD-I — the then-current DSM-III-R criteria for ADHD diagnosis contained only a single dimension, making it more equivalent to a DSM-5 ADHD-C diagnosis. The question of whether and to what degree comorbidity rates may be inflated by symptom overlap, therefore, cannot be entirely dismissed, and warrants further investigation using contemporary diagnostic criteria in children and adults.

Genetic Roots of Comorbidity.

If the comorbidity is not purely artefactual, several other explanations are possible. Some studies have sought to understand whether underlying genetic mechanisms lead to both anxiety and ADHD symptoms. Family studies offer the opportunity to examine whether comorbid traits tend to be passed down together – known as co-segregation – which might suggest such mechanisms, or even the existence of anxious ADHD as a discrete disorder distinct from the two individual conditions. If a distinct subtype of ADHD linked to anxiety disorders could be distinguished, its aetiology, underlying physiology and course could provide targets for unique and potentially more efficacious treatments for this group (Braaten et al., 2003). The findings of family studies to date present a complex picture. In one, higher rates of anxiety disorders were found in first-degree relatives of all children with ADHD (even non-anxious ADHD) than in typically developing populations, suggesting common genetic risk for the two conditions (Biederman, Faraone, Keenan, Steingard, & Tsuang, 1991). Also, relatives of ADHD probands who themselves had ADHD were also more likely to have anxiety than those relatives who did not, suggesting that the two conditions are transmitted together in families, a process known as cosegregation. On the other hand, the same study found that relatives of children with comorbid ADHD and anxiety had double the risk of anxiety disorders than relatives of non-anxious children with ADHD, suggesting

independent transmission of anxiety risk. On the question of whether ADHD is an epiphenomenon secondary to anxiety in the comorbidity, the researchers argued that this was unlikely on the basis that familial risk of ADHD was the same in comorbid ADHD-anxiety as in relatives of children with "pure" ADHD.

These initial findings, however, were only partially consistent with subsequent investigations. For example, Braaten et al. (2003) found that the risk for anxiety disorders in relatives was significantly higher if ADHD probands had comorbid anxiety as opposed to "pure" ADHD, while the risk of ADHD was the same for these two probands, suggesting separate transmission of anxiety risk rather than a common risk factor that might manifest as either condition. Amongst relatives of comorbid probands, rates of ADHD were not significantly higher in relatives who had anxiety than those who did not, suggesting a lack of cosegregation that would result if a combined ADHD-anxiety subtype were being passed on as a distinct disorder. A follow-up study by Biederman et al. (1992) also found that ADHD and anxiety did not cosegregate among relatives of probands with comorbid ADHD-anxiety.

Taken together, these findings suggest that the two conditions of ADHD and anxiety are most likely independently transmitted (Tannock, 2009). If a genetically distinct "anxious-ADHD" subtype were driving the comorbidity, stronger evidence of cosegregation would be expected. If the comorbidity were due to a common underlying risk factor that could be expressed as either ADHD or anxiety symptoms, similar levels of anxiety would be expected in probands with comorbid ADHD-anxiety and those with "pure" ADHD, as opposed to the higher rates of anxiety seen in relatives of comorbid probands in all three of these studies. However, these findings do not preclude the possibility that multiple, possibly contradictory processes are at work in the genetic aetiology of ADHD-anxiety – such heterogeneity may be difficult to detect when looking at overall patterns of transmission in families. Multivariate twin studies would be useful in teasing apart common and independent sources of genetic

variance, and would also help in disentangling the role of family environments in heredity (Jarrett & Ollendick, 2008).

Influences of Cognition and Temperament.

The difficulties faced by those with comorbid ADHD and anxiety may stem in part from cognitive differences associated with risk of both conditions. Whether the result of genetic or environmental processes, these could be of importance in understanding the frequent occurrence of this comorbidity, as well as its sequelae and treatment response. In studies comparing children with ADHD alone and those with comorbid anxiety, working memory and effortful processing have been found to be impaired in both groups, but more so for comorbid children (Jarrett et al., 2012; Tannock, 2009). On the other hand, children with comorbid anxiety perform better on tests of sustained attention, selective attention and response inhibition compared to children with ADHD alone (Bloemsma et al., 2012; Tannock, 2009).

These findings largely align with attentional control theories of anxiety, in which anxiety (including worry about performance in cognitive tests) is proposed to increase motivation, improving some aspects of performance, while simultaneously overtaxing the processing and storage capacities of working memory, resulting in worsened performance on tasks that make heavy demands on those functions (Eysenck, Derakshan, Santos, & Calvo, 2007; Tannock, 2009). While attentional control theory predicts that inhibition may be impaired in the presence of anxiety, increased effort due to performance anxiety is proposed to drive recruitment of alternative mechanisms that can compensate for such difficulties, provided the overall task demands are not too great (Eysenck et al., 2007).

Findings of better response inhibition in more anxious children with ADHD compared to children with ADHD alone (Bloemsma et al., 2012) also align with Quay's (1988)

proposal that the Behavioral Inhibition System (a set of neuropsychological processes driving avoidance of situations associated with negative consequences; Gray (1991)) is underactive in ADHD but overactive in anxiety. Opposing mechanisms underlying the two disorders may result in more balanced inhibition in comorbid ADHD-anxiety (Bloemsma et al., 2012). It is not yet clear, however, whether the improved response inhibition demonstrated on laboratory measures translates to improvements in controlling inappropriate responses in real life situations. One study using a parent-report measure of "every day" executive function found that children with anxiety and ADHD demonstrated more problems with inhibition than children with either diagnosis alone (Sørensen, Plessen, Nicholas, & Lundervold, 2011), while consistent with Quay's theory, children with anxiety alone were rated more positively on inhibition than those with ADHD or even normal controls.

It may be that difficulties in coping with the simultaneous demands of real-life situations overwhelm compensatory responses elicited by higher motivation in anxious children with ADHD (Eysenck et al., 2007), or that the emotional content of real life scenarios results in different effects to those seen in the laboratory (Jarrett & Ollendick, 2008). While findings are preliminary, social cognition deficits may underlie poor cognitive performance in emotionally loaded situations. Children with comorbid ADHD-anxiety have been found to show reduced auditory perception of anger compared with either single diagnosis (Manassis, Tannock, Young, & Francis-John, 2007). On the other hand, a reduced prevalence of comorbid conduct disorder amongst anxious children with ADHD suggests some improvements in "real world" behaviour regulation relative to "pure" ADHD (Manassis, 2007).

Multiple Subtypes, Multiple Pathways.

Heterogeneity in variables such as temperament, cognitive function and symptom presentation may be important in understanding comorbid ADHD and its response to treatment. DSM-5 specifies ten distinct anxiety disorders and three ADHD subtypes. Therefore, children with comorbid ADHD-anxiety may present with thirty combinations of disorders, before one even considers the possibility of multiple comorbid anxiety disorders. In line with genetic evidence of independent transmission of anxiety and ADHD conditions, researchers have not found evidence for particular individual anxiety disorders tending to present more often with comorbid ADHD (Tannock, 2009), that is, the relative likelihood of a child having SAD, for example, versus GAD, remains the same irrespective of ADHD diagnosis, although overall rates of anxiety are higher in children with ADHD.

Similarly, consistent differences have not emerged in the prevalence of anxiety symptoms or disorders experienced by children with one subtype of ADHD versus another. While early research found higher rates of internalising symptoms in inattentive children without hyperactivity, these findings have not been supported by more recent studies (Power, Costigan, Eiraldi, & Leff, 2004). Power et al. (2004) suggested that the differences in these findings may be due to changes in diagnosis of the predominantly inattentive group over the years, suggesting that still further subtyping may identify a group of children more predisposed to anxiety. Some evidence indicates that children with so-called sluggish cognitive tempo (SCT), characterised by inconsistent alertness and slower thinking, have higher rates of anxiety, and were more likely to be identified in pre-DSM-IV definitions of ADHD without hyperactivity than on DSM-IV/DSM-5 ADHD-I type (Bernad, Servera, Becker, & Burns, 2015). The SCT construct (possibly a distinct attention-related disorder) is still being delineated by researchers (Barkley, 2016), however it may be important in future studies investigating comorbid ADHD-anxiety.

Despite a lack of evidence for clear-cut patterns linking anxiety symptoms and ADHD subtypes, the bi-dimensional nature of ADHD remains important to recent theories which aim to explain the development of comorbid ADHD-anxiety (and ADHD more broadly) in terms of multiple pathways, incorporating bidirectional influences between ADHD and anxiety symptoms throughout development. Drawing on evidence from genetics, cognition and temperament research in ADHD, Nigg, Goldsmith, and Sachek (2004) proposed six speculative pathways to ADHD, each resulting in a distinct pattern of outcomes, comorbidities and importantly, treatment susceptibility. Of these pathways, anxiety symptoms were central to two. In the "primary ADHD-C with anxiety" trajectory, weak regulatory control manifesting from infancy or toddler years is described along with low hostility and high negative withdrawal (anxiety). Evidence cited for this proposed pathway include findings of cognitive tests in which some anxious individuals tend to show fast, impulsive responding, specifically under conditions designed to activate the BIS, such as when feedback on failure was given (Wallace, Newman, & Bachorowski, 1991). While high BIS activation is generally described as promoting more thoughtful, reflective behaviour than that seen in typically impulsive individuals, situation-specific pressure to respond may lead to high levels of general arousal, overwhelming cognitive control and promoting impulsivity and careless errors in some anxious individuals (Newman & Wallace, 1993). Nigg et al. (2004) point out that a small subgroup of children with ADHD show cortical overarousal on scalp electrode recordings, (Barry, Johnstone, & Clark, 2003), providing a potential pathophysiological marker of this subtype. The ultimate clinical presentation of a child following this trajectory would be dominated by high anxiety and anxious impulsivity, executive deficits, and possibly general cortical overarousal, but few comorbid oppositional symptoms as seen in other ADHD trajectories.

The second proposed pathway incorporating anxiety symptoms is the "Primary or secondary ADHD-I" trajectory. In these children, anxiety might be the primary presentation and temperamental regulatory control is less prominent. Cognitive regulation is disrupted in relation to high anxiety-intrusive thoughts, leading to evident inattentiveness on certain cognitive tests such as sustained attention, but otherwise more intact executive function. This a pathway is speculative, but might explain the development of anxious ADHD-I, in which impulsivity is not prominent and behavioural regulation is normal (Power et al., 2004). Others amongst the proposed developmental pathways to ADHD involve abnormally low levels of anxiety, leading to higher rates of oppositional behaviour, suggesting that presentations, outcomes, and even effective treatments for ADHD in children with comorbid anxiety may be quite distinct from those of their non-anxious peers. Similarly, heterogeneity even amongst children with comorbid ADHD-anxiety is illustrated by the two hypothesised pathways to the comorbidity. This may mean some comorbid children are more similar to children with "pure anxiety" in their cognitive profile and temperament, while others are more distinct, perhaps requiring different treatments to account for differences in regulatory control or executive function.

In summary, research on comorbid ADHD-anxiety indicates that these children are likely to be a heterogeneous group. Evidence from family studies does not support the transmission of a distinct, homogenous anxious-ADHD subtype, and the full range of different anxiety disorders and ADHD subtypes are present amongst the comorbid group. Theoretical models propose multiple pathways to the comorbidity, which may have different implications for treatment, although these have not yet been empirically evaluated. On average, however, the comorbid group does appear to present with impairments in academic achievement and social competence greater than those seen in either disorder alone, and are more likely to show deficits in certain areas of executive function, such as working memory.

Children with ADHD and comorbid anxiety show poorer academic and social outcomes than those with ADHD alone, emphasising the importance of treating anxiety in this group. On the other hand, given their academic, cognitive and social deficits relative to other children with anxiety, it is important to examine whether CBT treatments are as effective in this group as in their less impaired peers.

Treating Anxiety in Children with Comorbid ADHD

While children with comorbid ADHD-anxiety may seek treatment for either or both of their disorders, the present paper focuses on treatment of anxiety, with a focus on the "gold standard" treatment of cognitive behavioural therapy. CBT interventions aim to reduce anxiety by targeting both thoughts and behaviours that may cause or perpetuate disorders (Clark & Fairburn, 1997). CBT involves a collaborative approach between the therapist and client, and can be delivered either individually or in a group setting (Lovelock, Matthews, & Murphy, 2010). Several manualised treatment approaches have been developed specifically for children, using age-appropriate vocabulary and stimulus materials (Kendall et al., 1997; Lyneham, Abbott, Wignall, & Rapee, 2003). Key elements generally include psychoeducation, cognitive restructuring and gradual exposure to feared situations. A systematic review of randomised controlled trials found that CBT is an effective treatment for paediatric anxiety, with average remission rates of around 60 percent (James, James, Cowdrey, Soler, & Choke, 2015).

Possible Barriers to Treatment Success.

The cognitive elements of CBT – such as reflecting on one's own thoughts and formulating hypotheses about behaviour change – seem likely to present a challenge for children with the executive function difficulties we have described in anxious ADHD. Even the behavioural elements of treatment, such as exposure, may be less successful in this group

if lack of sustained attention makes children unable to habituate to anxiety during such exercises (Halldorsdottir & Ollendick, 2016; Storch et al., 2008). While use of medication to treat ADHD may reduce overactivity in the therapy setting, cognitive differences may still be evident between medicated children with ADHD and their typically developing peers (Manassis, 2007). The effect of stimulant medication is stronger on cognitive tasks without an executive function component (e.g. complex reaction time, delayed matching to sample) than on those with demands on executive functions (e.g. inhibition, working memory, strategy formation, planning, and set-shifting), and overall, stimulant use does not appear to completely correct the cognitive deficits of ADHD (Swanson, Baler, & Volkow, 2011). Furthermore, anxious children with ADHD may be even less likely to show improvements in working memory after stimulant medication use than non-comorbid children with ADHD, although their hyperactivity symptoms may be just as effectively reduced (Bedard & Tannock, 2008; Tannock, Ickowicz, & Schachar, 1995). While empirical research on the treatment impact of cognitive deficits is scarce, executive dysfunction has been linked to poorer CBT response in PTSD and OCD (D'Alcante et al., 2012; Flessner et al., 2010; Nijdam, de Vries, Gersons, & Olff, 2015), along with poorer response in the treatment of other non-anxiety related psychological disorders (Kundermann et al., 2015; Wessels et al., 2015).

Along with executive function deficits, less well-studied differences related to emotion processing may also impact the ability of children with ADHD to engage with CBT for anxiety. Manassis, Tannock and Masellis (1996) found that children with ADHD made more false-alarm errors and had lower sensitivity on an auditory emotion recognition task than children with anxiety, indicating a lack of caution when interpreting and responding to emotional tones of voice. This finding was later replicated in anxious-ADHD comorbid children, who showed lower sensitivity to anger in recordings of voices (but not pictures of

faces) than children with anxiety or ADHD alone (Manassis et al., 2007). Other modest deficits in theory of mind and facial emotion recognition have also been reported in "pure" ADHD (Bora & Pantelis, 2016). While the role of social cognition in CBT response has not been well investigated, it may be that abilities in perspective taking or emotion recognition are important to treatment success (Lickel, MacLean, Blakeley-Smith, & Hepburn, 2012).

If ADHD symptoms do present a barrier to treatment response, treatment modifications may be necessary to better accommodate the cognitive and behavioural profile of children with this comorbidity. While modified CBT programs are available for children with other neurodevelopmental disorders, such as high-functioning autism (Attwood & Scarpa, 2013), manualised adaptations specifically targeting children with comorbid ADHD and anxiety have not yet been developed. Manassis (2007) suggests that modular approaches may be helpful in treating comorbid children, enabling therapists to modify the pace of therapy or simplify content (Chorpita, Daleiden, & Weisz, 2005). However, empirical demonstration of an improved treatment response is required before ADHD-specific treatment modifications can be recommended – it may be that they are no more effective than standard CBT for anxiety, despite theoretical appeal.

Further, while thus far we have considered the cognitive deficits associated with ADHD-anxiety as potential barriers to treatment engagement, the relationship could also function in the opposite direction, with CBT leading to even greater improvements in these children. If CBT is able to generalise and remediate deficits in executive function, children whose clinical symptoms are linked to pre-treatment executive dysfunction may respond particularly well to therapy as their abilities improve. For example, practice of metacognition, guidance in problem solving, and training in emotion recognition are common elements of CBT programs for children. *Enhanced* treatment response might be seen if these activities alleviate cognitive deficits that perpetuate or worsen anxiety in comorbid children. Mohlman

(2013) reported some initial evidence for this therapeutic mechanism in a different population, reporting that anxious older adults whose executive function improved during CBT also benefited from a greater reduction in worry post-treatment.

Empirical Investigations of Treatment Response.

Having considered theoretical arguments for an impact of ADHD comorbidity on treatment response, we now turn to an examination of empirical studies that have tested for such effects in practice. A qualitative review by Halldorsdottir and Ollendick (2014) reported contrasting findings in research to date, with some studies finding poorer response in children with ADHD, while others found no difference. In evaluating these findings, several differences in methodologies should be considered. Many studies have reported on ADHD not as a distinct disorder, but rather as part of a group of disruptive behaviour disorders (DBDs; also including oppositional-defiant disorder and conduct disorder). These conditions have different presentations and aetiology, which seem likely to have distinct clinical implications (Jarrett et al., 2012). Given the heterogeneity even within ADHD as already discussed, the introduction of further heterogeneity by confounding ADHD with DBBs is unfortunate. None of the five analyses reviewed by Halldorsdottir and Ollendick (2014) which considered all DBBs together found a significant predictive effect on CBT response. One, however, found that externalising symptoms (but not diagnoses), predicted poorer treatment response in OCD (Garcia et al., 2010). A subsequent, large, combined study by Hudson et al. (2015) found that treatment response was negatively impacted by the presence of diagnosed DBBs, although differences in remission were non-significant. A study by

Rapee et al. (2013) found no significant difference in response or remission for children with DBBs[1].

Studies reporting specifically on ADHD symptoms or diagnosis, rather than externalising as a whole, have found mixed results. As the lack of consistency in the literature does not support broad conclusions, we will briefly review noteworthy findings and limitations of some of the ADHD-specific studies individually before attempting to summarise the current evidence base for its comorbidity as a predictor of treatment response. We will begin with studies targeting specific individual anxiety disorders, before reviewing studies of trans-diagnostic anxiety.

In a recent study of specific phobia treatment, Halldorsdottir and Ollendick (2016) found that children aged 6 to 15 years with higher scores on the Attention Problems subscale of the CBCL responded less favourably to single-session CBT than those with lower scores. A strength of this study was its use of other scales on the CBCL to control for non-ADHD conduct problems, and inclusion of longitudinal assessments. Persistently poorer outcomes were demonstrated from post-treatment to four-year follow-up. Only 11 of the 83 participants met criteria for diagnosis of ADHD, however, and results were based on scale scores across the sample rather than diagnostic categories. While the Attention Problems subscale of the CBCL taps symptoms of both inattention and hyperactivity, it does not allow separate examination of hyperactive and impulsive symptom dimensions.

In another disorder-specific study, this time using the Anxiety Disorders Interview Schedule for DSM-IV, Parent Version (ADIS-IV-P; Silverman & Albano, 1996) to assign clinical diagnoses, impact of ADHD comorbidity on response to treatment for obsessive-

[1] Remission rates were different in an overall chi-square analysis comparing mood, anxiety and externalising comorbidity groups with non-comorbid children, but there was no significant difference for the externalising group, based on subsequent analysis of percentage deviation and standardised residuals by the present authors.

compulsive disorder (OCD) was examined in 7 to 19-year-old children (Storch et al., 2008). Of the total sample of 96, 25 children met criteria for ADHD. Average response rates for those with comorbid ADHD were lower at 60 percent, versus 75 percent across the whole sample, with marginal significance (p = .04, uncorrected for multiple testing). Analyses were also performed with remission of OCD as an outcome variable; there was no significant difference between children with and without ADHD diagnoses. The researchers also reported on medication use, noting no difference in treatment response for children taking medication for their ADHD symptoms, and a significantly lower rate of remission for children taking ADHD medication (p = .02).

Turning to trans-diagnostic anxiety research, Halldorsdottir et al. (2015) reported on ADHD comorbidity in a subsample of 488 children aged 7 to 17 who were treated by either CBT or medication for GAD, SAD or SoAD. Children were assessed for anxiety and other comorbid disorders using the ADIS-IV, Child and Parent versions (ADIS-IV-C/P; Silverman & Albano, 1996), and ADHD was considered separately from other DBBs. While the overall study sample was large, only 12 children in the CBT treatment group met diagnostic criteria for ADHD. Also, the authors did not correct for multiple hypothesis testing due to the exploratory nature of the analyses, and therefore urged caution in the interpretation of their findings. ADHD comorbidity had a significant effect on CBT response immediately post-treatment (odds ratio = .175, 95% CI [.044, .688], p= .01 indicating lower likelihood of treatment response), however the difference in response was no longer significant six months after the end of treatment. Remission analyses found a large and significant effect of ADHD comorbidity indicating lower rates of remission (odds ratio = 15.70, 95% CI [1.89, 130.75], p = .01). By six months, the overall interaction between treatment and comorbidity on remission was no longer significant; lower rates of remission were noted for the ADHD group, although the difference was not reported. Given the small sub-sample size and

reduced significance of effects at follow-up, a robust effect of ADHD on treatment response is not evident from this study alone.

In an earlier trans-diagnostic study of CBT for anxiety, Manassis et al. (2002) divided 78 children aged 8-12 years with GAD, SAD, SP, SoAD and PD into high and low scorers on the Hyperactivity Index of the Conners Parent Rating Scale (Conners, 1989). After a 12-week CBT program, no differences in treatment response were identified in children scoring above or below the median on hyperactivity. This study may have lost power by dichotomising the scale measure, however (Dawson & Weiss, 2012), and its use of a hyperactivity-focused scale makes its findings less applicable to children with ADHD-I.

Another trans-diagnostic study by Southam-Gerow, Kendall, and Weersing (2001), however, used scales tapping both hyperactive and inattentive dimensions in 135 children aged 8-14 years with diagnoses of either overanxious disorder, GAD, SAD, SoAD or avoidant disorder. Scores on the Attention Problems subscales of the CBCL and the Teacher Report Form (TRF; Achenbach, 2001b) did not predict remission at post-treatment or one-year follow-up. This study only assessed treatment response as a dichotomous variable requiring absence of all anxiety disorders, however, so it is possible that effects on reductions in severity were missed. Again, separate analyses on the inattentive and hyperactive symptom dimensions of ADHD were not possible. The authors also noted a caveat of limited variability in externalising symptoms in the sample, although 14 percent of participants met criteria for a diagnosis of ADHD according to ADIS-IV-P interviews.

Taken together, these five studies present a mixed picture of the impact of ADHD on treatment response. Treatment effects were not always found, and when they were, they did not always persist to follow-up. There is no clear pattern across studies using anxiety remission versus response as an outcome variable, with one study finding a more robust

predictive effect for remission than response (Halldorsdottir et al., 2015), while another found the opposite pattern (Storch et al., 2008). A substantial limitation of most studies was the availability of very small sample sizes, mostly because the ADHD comorbid children formed a small subsample of children from anxiety-focused treatment studies. While the majority of the studies cited used clinical interviews to assign anxiety diagnoses, ADHD diagnoses based on clinical interviews were not always available or could not be used as predictor variables due to the small samples. In the two studies using ADIS-IV-C/P diagnoses as predictor variables, significant effects were found at some time points. It is possible that effects on treatment are larger in clinically diagnosable cases of ADHD, perhaps due to their inclusion of children with ADHD-I and ADHD-H subtypes, whose parents or teachers are likely to endorse fewer total items on unidimensional checklist measures than children with ADHD-C type.

Separate consideration of these ADHD subtypes, which was not possible in any of the treatment studies we have reviewed, would likely be helpful in capturing heterogeneity in treatment response, given that the subtypes display differences in impairment profiles in academic and social settings (Willcutt et al., 2012). A single-case design study on combined ADHD and anxiety therapy for twelve comorbid children (Jarrett & Ollendick, 2012) did offer some indication that symptom dimensions could be important in treatment response, reporting that improvements in anxiety were more closely linked to improvements in hyperactivity than inattention. While research on subtype differences is at times contradictory (Nigg, Tannock, & Rohde, 2010), inattentive symptoms appear to have a more detrimental impact on academic outcomes, while the hyperactivity dimension is more closely associated with peer rejection, aggression and physical injury (Marshall, Hynd, Handwerk, & Hall, 1997; Willcutt et al., 2012). Laboratory tests of attention and memory have found greater impairments associated with the inattentive symptom dimension (Carr, Henderson, &

Nigg, 2010; Huang, Shang, & Gau, 2012), which may lead to poorer learning during therapy. On the other hand, disruptive behaviour during therapy sessions and poor peer relations during group sessions could reduce treatment efficacy in children with more prominent hyperactive-impulsive symptoms. For similar reasons, it seems important to control in future studies for the potential confounding effect of additional ODD and CD diagnoses, accounting for the different cognitive and behavioural profile of children who also have DBD comorbidity (Jarrett et al., 2012; Jensen et al., 2001). Larger studies that better account for heterogeneity may offer a clearer picture of the efficacy of CBT treatment for anxiety in children with comorbid ADHD.

Effect of Anxiety Treatment on ADHD Symptoms.

While improvement in anxiety symptoms was of primary interest in the studies reviewed above, there is some evidence that children may also experience improvement in their ADHD symptoms after CBT treatment, even when these are not targeted. Kendall, Brady and Verduin (2001) reported that rates of ADHD in a group of 173 children treated for anxiety dropped significantly from 15% to 4.7% following treatment, remaining at 3.6% at follow-up. Those children whose anxiety symptoms successfully responded to treatment were significantly more likely to show concurrent improvement in comorbid disorders (although this finding was reported across several anxiety and non-anxiety comorbidities, and does not specifically relate to the improvements in ADHD symptoms).

The finding of substantial remission of ADHD following CBT may seem surprising, given that ADHD was not targeted, and the relatively modest effectiveness of psychosocial approaches in general treatment of ADHD in children (Antshel & Barkley, 2008; Van der Oord et al., 2008). On the other hand, children with comorbid anxiety have demonstrated better response to behavioural treatment for ADHD than non-anxious children (The MTA

Cooperative Group, 1999), so may be differentially susceptible. Kendall et al. (2001) offered three possible explanations for the improvement in non-targeted ADHD symptoms: application of skills learned in therapy to non-anxiety specific problems, alleviation of overlapping symptoms, and amelioration of underlying psychosocial processes common to the two disorders that result in both overlapping and disorder-specific symptoms. Of these three hypotheses, the second was considered unlikely, due to the size of the effect – as previously discussed, symptoms of the disorders do not overlap to such an extent that the removal of symptoms from one comorbidity would greatly diminish diagnosis rates of the other (Milberger et al., 1995).

Consistent with the first and third of these hypotheses, Jarrett and Ollendick (2008) proposed that response of ADHD symptoms to psychosocial treatments might be best understood with reference to the underlying heterogeneity in the disorder. For example, in Nigg et al.'s (2004) hypothetical ADHD-C/anxiety pathway, regulatory difficulties are a primary mechanism leading to both ADHD and anxiety symptoms, whereas in the ADHD-I/anxiety pathway, inattention symptoms are proposed to occur when otherwise-intact regulatory processes are disrupted by high levels of anxiety. Children following this second developmental trajectory may gain more benefit from psychosocial treatments, particularly those targeting anxiety. Alleviation of anxiety symptoms in these children may free up executive functions, leading to broad-based improvements in functioning and allowing for greater engagement with cognitive therapies. Therefore, when considering impact of CBT on ADHD symptoms, as with effects on anxiety symptoms, it appears important that researchers attend as closely as possible to heterogeneity within the ADHD-anxiety comorbid population.

The Present Study

Access to over a decade of data from children receiving anxiety treatment at the Centre for Emotional Health enabled us to examine ADHD comorbidity in a far larger sample than those previously studied. An important consideration in the interpretation of our findings, however, is that all participants in the present study were assessed as having a *primary* anxiety disorder – that is, their ADHD symptoms were deemed to be causing less impairment at pre-treatment than an anxiety disorder. Children were also excluded if symptoms of ADHD were very severe (see Method).

Our key research questions were: a) does ADHD diagnosis predict response to CBT for anxiety?; and b) do ADHD symptoms improve during the treatment? The second of these questions is more exploratory and intended to provide a basis for further research – as no control group was tested for ADHD symptom improvement we cannot attribute causality of any changes to the treatment. In order to account for possible confounds, we aimed to examine and control for any differences associated with ADHD diagnostic status, including additional DBD diagnoses such as ODD. While neurocognitive assessments and more recent diagnostic specifiers such as SCT were not available, we aimed to account for some of the heterogeneity within the ADHD group by treating the three subtypes separately in answering both key questions. While our sample contained very few participants with ADHD-H, we nevertheless reported our findings on these children separately, as an exploratory approach to this under-studied subgroup.

Externalising comorbidity when treated as a whole was not found to predict treatment response in a previous study using this dataset (Rapee et al., 2013), however ADHD comorbidity appears to be more likely to impact treatment response when examined separately from DBDs (Halldorsdottir & Ollendick, 2014). It is also possible that differential

effects amongst the subtypes of ADHD have been obscured in previous analyses – the size of this dataset offers a rare opportunity to test for such differences.

Although findings of previous research have been mixed, we hypothesised that ADHD comorbidity would negatively impact anxiety treatment response when diagnoses rather than scale measures were used, as previously reported by Halldorsdottir and Ollendick (2015~~5~~4). Based on the findings of Kendall et al. (2001), we hypothesised that ADHD symptoms would improve after CBT treatment for anxiety. For both anxiety and ADHD outcomes, we tentatively hypothesised that ADHD-I and ADHD-C subtype diagnoses would predict poorer treatment response, due to association of the inattentive symptom dimension with poorer learning outcomes in academic settings (Willcutt et al., 2012). While hyperactivity is associated with greater disruptive behaviour and peer rejection (Willcutt et al., 2012), we speculated that the high levels of group supervision and parental involvement described in the present study may have limited its impact on treatment response.

Method

Participants

Participants were 842 children and adolescents, aged between 6 and 18 years (M_{age} = 10.21, SD = 2.57), along with their parents, who participated in a manualised CBT treatment program at the Centre for Emotional Health (CEH) at Macquarie University, Sydney, Australia, between the years of 2000 and 2011. All participants met DSM-IV (APA, 1994) criteria for a primary anxiety disorder diagnosis, assigned at the CEH after a semi-structured diagnostic interview (ADIS-IV-C/P; Silverman & Albano, 1996; described in detail below). The most common primary disorder was GAD (*n* = 425, 50.5%), followed by SoAD (n = 179, 21.3%), SAD (n = 116, 13.8%), OCD (n = 49, 5.8%), SP (n = 57, 6.8%), PTSD (n = 3, 0.4%), PD (n = 11, 1.3%) and anxiety disorder – not otherwise specified (*n* = 2, .2%).

All participants received 9-12 sessions of group-based family treatment in the CEH's Cool Kids program, with around 65% participating in clinical trials as follows: *n* = 58 (Rapee, Abbott, & Lyneham, 2006); *n* = 82 (Hudson et al., 2009); *n* = 198 (Hudson, Newall, et al., 2014); *n* = 213 – currently unpublished randomised clinical trials. A further 291 participants received treatment at the CEH without participating in a clinical trial, however screening, assessment and group-based Cool Kids treatment were the same for these participants, and all participants were told they were receiving treatment at a research clinic. Due to different study protocols, 54 participants also received an integrated depression management program and 99 parents received five additional parent anxiety management sessions (however, no additional benefits were reported for children of parents undergoing these additional sessions; Hudson, Newall, et al., 2014).

Participants with additional comorbid mood and externalising diagnoses were eligible for treatment trials, provided that these diagnoses were less interfering than a primary anxiety diagnosis, and that severity of externalising disorders (ADHD and ODD) did not exceed a clinical severity rating cut-off of 6 (further description of ADIS-IV diagnoses and CSRs in Measures). Children whose ratings exceeded this cut-off were excluded on the grounds that their behaviour may be too disruptive in group settings. Forty-three children were excluded from treatment trials because ADHD (not anxiety) was primary, and 11 children were excluded due to ADHD severity exceeding the cut-off.

Ninety-four included participants met criteria for a diagnosis of ADHD (M_{CSR} = 4.78, SD = 0.78), with the majority of these classified as ADHD-I (*n* = 61, 64.89%), followed by ADHD-C (*n* = 27, 28.72%), and a much smaller number of children with ADHD-H (*n* = 5, 5.32%). Subtype information was lost on one participant with ADHD due to archival damage. A total of 75 participants had ODD, which was the only DBD diagnosed in the

sample. Table 1 presents a comparison of demographic and clinical variables among the groups.

Participants were excluded if they presented with psychoses, severe suicidal ideation, or intellectual disability, and if they were undertaking concurrent psychological treatment. Participants using anxiety or mood medication were included provided that medication use was stable. Participants taking medication for ADHD were also included. In some cases, parents reported beginning ADHD medication after the start of the trial ($n_{ADHD\text{-}I}$ = 7, $n_{ADHD\text{-}C}$ = 4). We performed sensitivity analyses to determine whether inclusion of these participants affected the outcomes. As a result, these participants were excluded in all analyses of change in ADHD symptoms over time, but retained in all analyses of response to anxiety treatment, where their inclusion had no impact on any outcome. Use of ADHD medication at any time was treated as a covariate in these analyses.

Measurements

Diagnoses. All diagnoses were assigned according the ADIS-IV-C/P (Silverman & Albano, 1996), a semi-structured clinical interview administered to both parents and children. During ADIS-IV-C/P assessment, parents and children are asked to indicate whether symptoms of anxiety disorders and other common disorders of childhood are present or absent, with the interviewer prompting for examples and clarification where presence or absence is unclear. If the number of symptoms endorsed is sufficient to meet DSM-IV criteria, parents and children then indicate to what degree these symptoms cause distress and impairment, using a nine-point scale "feelings thermometer" (Silverman, Saavedra, & Pina, 2001). To qualify for diagnosis, these ratings had to exceed four, indicating a moderate level of impairment.

Graduate clinical psychology students or qualified clinical psychologists assigned composite diagnoses based on the "or" rule – that is, diagnosis could be assigned if symptoms reported by either parent or child met criteria. Clinicians also assigned a Clinician Severity Rating (CSR) on a scale of 0 to 8 for each diagnosis, representing the interviewer's clinical judgement of the distress and disability associated with each primary and secondary disorder. Again, ratings of 4 or more (indicating symptoms are "definitely disturbing/disabling", Figure 1) were necessary for diagnosis. Where multiple diagnoses are assigned, the diagnosis with the highest CSR is considered primary.

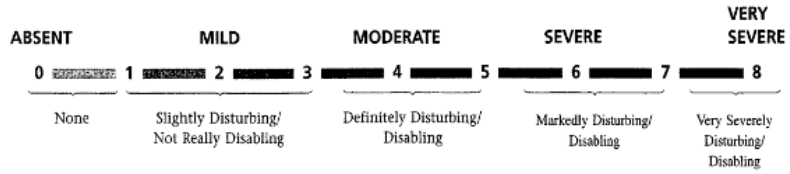

Figure 1. ADIS-IV-C/P Clinician Severity Ratings (Silverman & Albano, 1996)

The ADIS-IV-C/P has demonstrated strong concurrent validity (Wood, Piacentini, Bergman, McCracken, & Barrios, 2002) and good-to-excellent test-retest reliability (Silverman et al., 2001), as well as sensitivity to clinical change (Silverman & Ollendick, 2005). Reliability assessments for the anxiety disorder sections performed at the Centre for Emotional Health (including data from some of the trials included in the present study) have demonstrated inter-rater agreement of $\varkappa = 1.00$ for diagnosis of an anxiety disorder and $\varkappa =$ 0.68 to 0.93 for agreement on the specific anxiety disorder.

Along with anxiety disorder sections, an ADHD module of the ADIS was completed if parents or children answered yes to either of two screening questions: "1. Does your child often make little mistakes, get distracted, have trouble completing tasks or have trouble listening much more than other children his/her age?" or; "2. Does he/she have difficulty

staying seated, seem constantly restless, or struggle to play quietly much more than other children his/her age?". Clinicians were specifically instructed to request clarification that these problems were experienced greatly in excess of those experienced by the child's peers, as typical children would be expected to display these behaviours from time to time.

The ADHD module contains subsections with symptom checklists for inattention and hyperactivity/impulsivity dimensions. More than six symptoms on the relevant dimension must be present for a diagnosis of either ADHD-I or ADHD-H. If more than six symptoms from both dimensions are met, the child may be diagnosed with ADHD-C. For each of these diagnoses, symptoms must have been present prior to the age of seven, and must be manifest in two or more environments (at home, at school or with peers). As with anxiety diagnoses, parents and children assign an interference score, and clinicians assign a CSR which must exceed 4 for a diagnosable condition. Considering the inclusion criteria for group treatment of ADHD CSR≤6, therefore, children with ADHD who remained in the study could be considered to have "definitely disturbing/disabling" or "markedly disturbing/disabling" ADHD symptoms, but those with "very severely disturbing/disabling symptoms" were excluded. While clinicians record whether children are currently using medication, the CSRs are assigned based on the child's current presenting level of severity, irrespective of medication use.

The ADHD module of the ADIS-IV-C/P has demonstrated good convergent validity with parent ratings on the Child Behavior Checklist (CBCL; Achenbach, 2001a) and teacher ratings on the Teacher's Report Form (TRF; Achenbach, 2001b), as well as acceptable inter-rater agreement (Jarrett, Wolff, & Ollendick, 2006). Lyneham, Abbott and Rapee (2007) have previously reported good interrater agreement for ADHD ($\varkappa$ = .77). We completed additional interrater agreement analyses for the present dataset based on double-coding of video-recorded interviews by the author, a graduate psychology student blind to the original

diagnoses. Diagnostic agreement was excellent at $\varkappa$ = .91 for diagnosis (requiring agreement on presence of ADHD and subtype) and ICC = .98 for CSR.

Anxiety symptoms. While ADIS CSRs were the primary outcome measure in the present study, participants also completed parent- and child-report versions of the Spence Children's Anxiety Scale (SCAS; Spence, 1998). The 38 items in the SCAS are designed to measure specific anxiety subtypes, but also map onto a single, higher-order factor, with total scores from a possible range of 0-114 reported in the present study. The SCAS has good internal consistency and test-retest reliability and adequate convergent and discriminant validity (Nauta et al., 2004; Spence, 1998). Because of higher levels of missing data in the paternal reports, only maternal reports are presented in Table 1.

Externalising symptoms. While CSR for ADHD diagnosis was our main outcome variable for ADHD symptoms, we also report externalising scale data at pre-treatment in Table 1 to provide further description of this sample. Because two different externalising scales were used across the decade during which these data were collected, a standardised score was computed for maternal report of externalising symptoms from the available scale. Around 24% of mothers completed the Child Behavior Checklist (CBCL; Achenbach, 2001a; externalising subscale), while the remainder completed the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001; Conduct Problems, Hyperactivity, and Peer Problems subscales). Both scales are widely used as measures of externalising symptoms and have good psychometric properties (Goodman & Scott, 1999; Hawes & Dadds, 2004)

Parental symptoms. Parents completed the Depression Anxiety Stress Scales (DASS; Lovibond & Lovibond, 1995), a 21 item scale assessing depression, anxiety, and stress symptoms experienced over the past week, with excellent internal consistency, temporal stability, and convergent and discriminant validity (Brown, Chorpita, Korotitsch, &

Barlow, 1997). The three DASS subscales were summed to create an overall measure of parental psychopathology.

Treatment

Cool Kids. The Cool Kids treatment is a ten-session, CBT-based, manualised program delivered in groups for children with anxiety disorders and their parents. The program is broad-based rather than diagnosis-specific and includes affect recognition, cognitive restructuring, social skills training, assertiveness, gradual exposure exercises and child management techniques for parents (with a focus on decreasing overprotection). These major elements of the program have remained consistent across the years in which the present data were collected, although minor revisions to workbooks were published in 2003 and 2006 (Rapee et al., 2013).

At least one parent attends all sessions with each child, and parents are also tasked with overseeing in vivo exposure exercises between sessions, which are collaboratively planned by clinicians and families. Groups attended by families in the present study comprised four to eight children who may have had different anxiety disorders but were selected to be close in age (usually within two years) and balanced in gender. Two therapists were usually assigned to each group, often working with parents separately to their children. Therapists were either clinical psychologists or supervised clinical psychology graduate students. During randomised clinical trials, adherence to treatment protocol was assessed via audio recordings.

Management of ADHD behaviours. Diagnosis of ADHD was not considered when assigning groups, i.e., no special ADHD-specific groups were formed, nor were children deliberately spread across groups. However, given the proportion of children with ADHD in

the sample (94 out of 842 children), groups were unlikely to include more than one child meeting criteria for diagnosis.

Therapists were not given specific training on dealing with behaviour issues in children with comorbid ADHD, however general behaviour management techniques formed part of Cool Kids therapist training and manualised procedures. For example, while rewards and token systems were not mandated, therapists were encouraged to use them where helpful to keep children on task, and a second therapist was available to provide additional help managing individual children's behaviour. Homework completion was reviewed at the start of each session, and where children or parents had forgotten to complete homework, therapists would actively problem solve with the family to ensure future task completion. Depending on individual therapists' approaches and the needs of children in a group, strategies to improve homework compliance could include using memory cues, scheduling homework time and reducing complexity of tasks.

Procedure

Families were recruited to trials or received between-trials treatment after their parents contacted the CEH seeking help with their children's anxiety. The majority of parents were made aware of the Centre by word of mouth or media coverage, or after referral by school counsellors or medical practitioners. After a brief telephone screening, parents provided written consent to participate in research and children provided verbal assent (under age 14) or written consent (over age 14).

During trials, clinicians completing assessments were blind to treatment condition. Between trials, clinicians were aware that the children were assigned to Cool Kids treatment, as this was the only treatment being offered at the CEH during these periods. After completion of diagnostic interviews and questionnaires, families completed Cool Kids

treatment. Post-treatment measures were usually taken one week after completion, and follow-up measures were taken at time points ranging from three months to twelve months post-completion, according to different trial protocols. All pre-treatment measures were repeated at post-treatment and follow-up assessments. Families were encouraged to wait until after the follow-up assessment before seeking additional treatment. Pre-treatment diagnoses were considered remitted if CSR lower than four was assigned at these time points. Where a diagnosis of ADHD had been assigned at pre-treatment assessment, the ADHD module of the ADIS-IV-C/P was always administered at post-treatment and follow-up assessments (rather than asking the ADHD screening questions before proceeding to the module).

Statistical analyses

Preliminary analyses were descriptive comparisons of children with and without comorbid ADHD diagnoses. Chi-square tests were used to compare proportions for categorical variables (e.g. gender, primary diagnosis), whereas ANOVAs with Bonferroni-corrected post-hoc comparisons were used to test for differences in means (e.g. age, externalising scores). These analyses were performed using SPSS version 22.

To test for effects of predictors on outcomes and incidental response of ADHD to anxiety treatment, mixed models were fitted using maximum likelihood estimation in Stata version 14. Mixed model longitudinal analyses do not require participants to have complete data across all time points, and allow for different participants' data being collected at different time points (e.g., one participant's follow up assessment may be taken at three months post-treatment, while another's could be taken at six months post-treatment). While in some repeated measures analyses, cases are deleted list-wise if data are incomplete across time points, in mixed models, each time point at which a participant provided data is included in the model and contributes to model variance and parameter estimates (Schlomer, Bauman,

& Card; 2010). This allows increased power when using datasets like that of the present study, in which trial follow-up protocols varied and some participants dropped out over time. It also avoids bias that can occur when using only complete cases, due to the fact that such cases may not be representative of the population (Gibbons, Hedeker, & DuToit, 2010).

These models can also allow for correlation of measures taken from the same participant that tends to occur in longitudinal studies, by modelling an effect of each individual on outcome as a random intercept which increases or decreases their score on the outcome variable by a constant amount relative to other individuals. Individuals usually also differ in the rate at which they change over time. In these cases, a random slope for time can also be fitted (Gibbons et al., 2010). For each analysis, we tested the significance of linear and quadratic fixed effects of time, as well as a random slope for time, in "unconditional models," before adding predictors (Hoffman, 2015).

Our two research questions relate to outcomes in two different disorders – anxiety and ADHD. For each, we aimed to examine two measures of outcome – response and remission. Therefore, a total of four models were fitted: anxiety response, anxiety remission, ADHD response and ADHD remission. Our first research question was whether ADHD diagnosis predicted anxiety outcomes. The *response* anxiety outcomes analysis used a linear mixed model with CSR of the child's primary anxiety disorder as the dependent variable. Fixed effects of time in the unconditional model, where significant, indicate change in the outcome variable post-baseline. A significant interaction of ADHD diagnosis with time in the predictor model would indicate that it is a predictor of treatment response. Given a negative effect of time (reduction in symptom severity), positive β values of the interaction with time indicate poorer response to treatment (a smaller reduction in CSR over time). Negative values predict better treatment response.

The *remission* analysis used a logistic mixed model with presence or absence of the primary anxiety disorder as the dependent variable. In this model, significant interactions of predictors with time indicate that these variables are associated with differences in the likelihood of remission after treatment. Odds ratios greater than one indicate remission of the primary disorder is more likely, whereas odds ratios less than one predict lower likelihood of remission.

The next two models were used to examine questions about ADHD outcomes for response and remission. The response model used ADHD CSR as the dependent variable. A significant effect for time in the unconditional model would indicate overall change in ADHD symptoms over time. While previous studies have established that anxiety improves, on average, after Cool Kids treatment, this study is the first to examine change in ADHD symptoms during the treatment. Thus changes over time were examined in greater detail in ADHD outcome analyses – slopes and mean contrasts are also reported. A remission analysis was also performed using presence or absence of ADHD diagnosis as the outcome variable. As described in Model Set 1, significant interactions of predictor variables with time in these models indicate that they are associated with differences in response or remission. In the ADHD models, the interaction of ADHD subtype with time was tested to determine whether response or remission differed by subtype. To test whether any improvements in ADHD are dependent on treatment response to anxiety, we also tested the significance of an interaction between change in anxiety severity and time in the ADHD outcome models. To control for multiple testing (four models), we considered predictors and slopes significant at a Bonferroni-corrected a level of $p < 0.0125$ (0.05/4). Bonferroni corrections were also applied in post-hoc comparisons of marginal means.

Table 1

Comparison on demographic and clinical variables among ADHD diagnosis groups.

Factor	Non-ADHD (n = 748)	ADHD Combined Type (n = 27)	ADHD Predominantly Inattentive Type (n = 61)	ADHD Predominantly Hyperactive Type (n = 5)	Comparison Statistic
Age (mo.), mean (SD)	127.59 (31.57)	114.60 (18.68)	128.47 (29.27)	138.20 (20.44)	$F(3) = 1.64$ ($p = .177$)
Gender (% boys)	49.7	68.0	76.3	40.0	$\chi^2(3) = 18.30$ ($p < .001$)
Taking ADHD medication (%)	7.6	48.0	22.4	20.0	$\chi^2(3) = 55.83$ ($p < .001$) [#]
Ethnicity (% Australian)	74.2	76.5	74.5	60.0	$\chi^2(30) = 24.53$ ($p = .747$)
Family type (% two-original)	85.0	80.0	88.1	80.0	$\chi^2(9) = 7.57$ ($p = .577$)
Low income (%)	19.7	18.7	19.4	0.0	$\chi^2(36) = 24.09$ ($p = .935$)
Parental psychopathology, mean (SD)	24.27 (13.22)	26.52 (11.66)	24.46 (11.97)	32.60 (19.86)	$F(3) = 0.89$ ($p = .446$)
Primary diagnosis					$\chi^2(12) = 11.07$ ($p = .523$)
(% GAD)	49.9	64.0	57.6	60.0	
(% SoAD)	20.8	12.0	25.4	20.0	
(% SAD)	13.9	16.0	10.2	0	
(% SP)	7.4	0	1.7	20.0	
(% Other)	8.0	8.0	5.1	0	
Anxiety comorbidity (% present)	89.1	96.0	94.9	80.0	$\chi^2(3) = 3.63$ ($p = .305$)
Mood comorbidity (% present)	18.4	4.0	6.8	0.0	$\chi^2(3) = 4.55$ ($p = .208$)
DBD comorbidity (% present)	8.4	24.0	5.1	40.0	$\chi^2(3) = 14.15$ ($p = .003$) [#]
Externalising scale standardised score, mean (SD)	0.15 (0.96)	1.72 (0.66)	0.95 (0.97)	1.06 (0.87)	$F(3) = 33.95$ ($p < .001$) [#]

Note. One participant for whom subtype data were lost was not included in these descriptive statistics. DBD = Disruptive Behaviour Disorders (all DBD diagnoses were Oppositional Defiant Disorder), GAD = Generalised Anxiety Disorder, SoAD = Social Anxiety Disorder, SAD = Separation Anxiety Disorder, SP = Specific Phobia, Other = Other anxiety disorder including obsessive compulsive disorder, post-traumatic stress disorder and panic disorder. [#] Indicates significant differences amongst ADHD subtypes (i.e., comparison statistic remains significant if non-ADHD group is not included in the comparison)

Results

Missing Data and Assumption Testing

A total of 664 participants (78.9%) completed treatment (defined as eight or more sessions). The percentage of missing data for primary diagnostic and outcome measures was 0% at pre-treatment, 12.7% at post-treatment and 30.3% at follow-up (defined as all follow-up sessions missing – individual *n* for follow up time points are presented in Table 1.)

We examined whether there were any differences in the likelihood of missing data at post and follow-up across ADHD versus non-ADHD diagnoses. Maximum likelihood estimation as used in our models can handle missing data on the outcome variable that is predicted by other variables within the dataset (data "missing at random" (MAR); Little & Rubin, 1987), however completion and drop-out statistics for participants with ADHD may be of interest in their own right, and are therefore reported. There was no significant difference in the presence of missing data (indicating drop-out) between children with diagnoses of ADHD and those with no ADHD diagnoses at post χ^2 (1, N = 842) = .692, p = .406 or at follow-up (considering all follow-up time points together), χ^2 (1, N = 842) = .075, p = .784. We also checked for differences in missingness by ADHD diagnosis at each of the three follow-up time points individually, all of which were non-significant (p >.05). Likewise, there were no significant differences in treatment completion rates between ADHD and non-ADHD diagnosed children, χ^2 (1, N = 735) = 1.62, p > .05). These analyses were repeated with ADHD subtype as a variable, and no differences were found amongst the different subtypes, or between any subtype group and the non-ADHD group (p > .05 for all analyses).

Missingness on predictor variables, on the other hand, is not handled by maximum likelihood estimation in mixed models in Stata – the cases which are not complete on all predictors are dropped from the model (Hoffman, 2015). Missingness on these variables

cannot be ignored without risk of biasing the model, unless it is very low, or the observations are missing completely at random. Fortunately, the majority of predictor variables assessed in these analyses were collected as part of the initial, pre-treatment ADIS, and are thus complete for all cases. The exception is the ADHD medication variable, which was missing for eight out of the 842 participants (0.9%), including two of the 94 ADHD participants, a level of missingness that is unlikely to bias results even with listwise deletion.

Assumptions of the models were checked by visual inspection of plots of model residuals for fixed effects and residuals from best linear unbiased predictions (BLUPs) for the random effects. Some heteroscedasticity was noted, therefore a robust variance estimator was used in all linear models. In anxiety analyses, a random intercept for study was initially included after it showed significance in an empty means model, indicating very small but significant differences across trials ($\chi 2 = 36.79$, $p < .001$, ICC = .04). However, during assumption testing of predictor models, residuals from BLUPs for the study intercept were found to violate normality assumptions. In these models, inclusion of the random intercept for study also produced worse model fit. Therefore, the third level of nesting for study was not retained in final models.

Preliminary Analyses

Table 1 presents descriptive comparisons by ADHD diagnosis on demographic and clinical variables. When children with no ADHD diagnosis and those with the three ADHD subtypes were compared, significant differences emerged for sex, use of ADHD medication, DBB comorbidity rates, and externalizing scale scores. Adjusted residuals exceeding 2.0 indicated that the proportion of boys was significantly higher in the ADHD-I group relative to the other groups, and significantly lower in the non-ADHD group, relative to the other groups. ADHD medication use was higher in the ADHD-I and ADHD-C groups and lower in

the non-ADHD group. DBB comorbidity was higher in the ADHD-C and ADHD-H groups. Post-hoc tests with Bonferroni correction indicated that externalising scale scores were higher (indicating more externalising problems) in ADHD-I and ADHD-C than in the non-ADHD group ($p < .001$), and higher in ADHD-C than in ADHD-I ($p = .004$).

As our planned ADHD outcomes models would only include the children with ADHD, we also repeated these group comparisons without the non-ADHD children. When only children with ADHD were included, significant differences emerged among the subtypes on use of ADHD medication ($\chi^2(3) = 7.38$, $p = .025$), DBB comorbidity rates ($\chi^2(3) = 11.71$, $p = .003$), and externalising scale scores ($F(3) = 5.91$, $p = .004$). Standardised residuals exceeding 2.0 indicated that in these comparisons, children with ADHD-C had relatively higher rates of ADHD medication use and ADHD-I showed relatively lower rates. DBB comorbidity was significantly higher in ADHD-C and significantly lower in ADHD-I. Post-hoc comparisons with Bonferroni correction indicated that the ADHD-I group showed significantly lower externalising scores than the ADHD-C group ($p = .01$).

Anxiety Outcomes

Table 2 presents observed mean CSRs and remission rates for the primary anxiety disorder in the ADHD and non-ADHD diagnostic groups at each time point. As not all participants were assessed at all time points, the included *n* is noted for each. The significance of observed changes (and their differences by diagnosis) is examined in the mixed model analyses that follow.

Table 2

Observed mean clinical severity rating (CSR) and remission rate of primary anxiety disorder for ADHD diagnostic groups at each time point.

	Mean Anxiety CSR (SD)					Remission Rate (%)				
Time Point	No ADHD	Any ADHD	ADHD-C	ADHD-I	ADHD-H	No ADHD	Any ADHD	ADHD-C	ADHD-I	ADHD-H
Pre-treatment (*n* = 842)	6.51 (0.83)	6.48 (0.70)	6.59 (0.69)	6.41 (0.67)	6.80 (1.10)	—	—	—	—	—
Post-treatment (*n* = 735)	3.48 (1.90)	3.65 (1.95)	3.24 (2.11)	3.88 (1.92)	3.20 (1.30)	50.5	50.0	48.0	50.0	60.0
3-month follow-up (*n* = 132)	2.44 (2.02)	2.53 (2.34)	2.63 (2.77)	2.50 (2.22)	2.00 (—[#])	69.0	63.2	62.5	60.0	100.0
6-month follow-up (*n* = 385)	3.20 (1.95)	3.33 (1.80)	3.40 (1.84)	3.47 (1.78)	1.67 (1.53)	58.8	53.5	50.0	50.0	100.0

Note: No ADHD = No Attention-Deficit Hyperactivity Disorder (ADHD) diagnosis; Any ADHD = any ADHD subtype diagnosis; ADHD-C = ADHD, Combined Type; ADHD-I = ADHD, Predominantly Inattentive Type; ADHD-H = ADHD, Predominantly Hyperactive Type. For CSRs, 4 is the clinical cut-off. [#]Only one ADHD-H participant at this time point.

Response Analysis. Mixed linear growth models were fitted with occasions (i.e. assessment time points) nested by individual and severity of primary anxiety disorder as the outcome variable. An unconditional model indicated that a fixed, linear effect of time and its random variance were each significant ($p < .001$), indicating a significant decrease in severity over time across participants, and individual differences therein. A fixed quadratic effect for time was also significant ($p < .001$), indicating deceleration in the rate of symptom improvement. Thus, the final predictor model included random intercepts for individual, fixed effects of time and time-squared, and a random slope for time. Covariates were included for all variables on which the diagnostic groups differed in Table 1, with the exception of externalising scale scores – this difference is considered core to the ADHD diagnosis and not to be "partialled out".

Coefficients of fixed effects are presented in Table 3. ADHD diagnosis, including any subtype, did not predict treatment response (overall significance of interaction between time and diagnosis, $p = .34$). Figure 1 shows the estimated marginal means for ADHD severity by each subtype. Comparisons at each time point indicated no significant differences in slope between the diagnostic groups ($p > .05$, Bonferroni adjusted).

Table 3

Results of mixed models examining response (severity of primary diagnosis is outcome) and remission (loss of diagnosis is outcome)

Parameters	Response[a]		Remission[b]	
	β	(95% CI)	O.R	(95% CI)
Predictors (interactions with time)				
ADHD diagnosis				
No ADHD	—[c]	—[c]	—[c]	—[c]
ADHD-C	-0.12	(-0.51-0.27)	0.52	(0.08-3.23)
ADHD-I	0.11	(-0.12-0.35)	0.40	(0.13-1.29)
ADHD-H	-0.72	(-1.40-0.03)	15.60[d]	(-675.66-706.83)[d]
DBB comorbidity	0.16	(-0.04-0.38)	0.58	(0.21-1.63)
Medication use	0.10	(-0.10-0.30)	1.58	(0.52-4.86)
Sex	0.06	(-0.06-0.19)	0.74	(0.40-1.37)
Main effects				
ADHD diagnosis				
No ADHD	—[c]	—[c]	—[c]	—[c]
ADHD-C	0.02	(-0.33-0.38)	1.72	(0.18-16.63)
ADHD-I	0.10	(-0.14-0.32)	2.41	(0.57-10.24)
ADHD-H	0.22	(-0.52-0.96)	-14.73[d]	(-705.99-676.52)[d]
DBB comorbidity	0.33*	(0.12-0.54)	0.87	(0.23-3.27)
Medication use	-0.09	(-0.30-0.12)	0.62	(0.16-2.42)
Sex	0.14	(0.01-0.29)	1.21	(0.56-2.59)
Time				(381.90-
	-3.97*	(-4.16--3.76)	62126.15*	.117685.30)
$Time^2$	0.93*	(0.87-0.99)	0.13*	(0.06-0.29)

Note: All models included random intercepts of individual and random slope for time. Parameters under the subhead Predictors are interactions of the named variable with time. [a] Linear mixed model. [b]Logistic mixed model. [c] Reference category. [d] β coefficients reported as Stata could not estimate ORs for this parameter due to small group size of ADHD-H.*p<.0125.

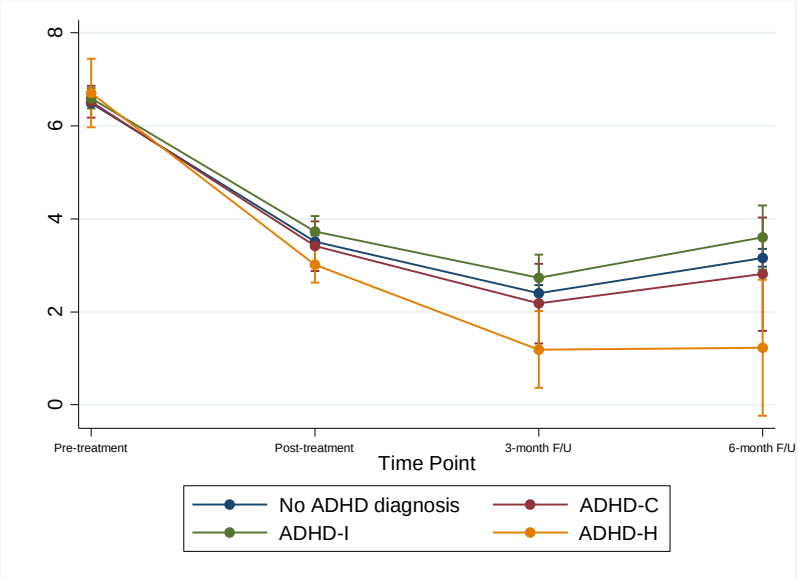

Figure 1. Model predicted margins for clinical severity rating (CSR) of primary anxiety disorder for children with ADHD Combined Type (ADHD-C), ADHD Predominantly Inattentive Type (ADHD-I) and ADHD Predominantly Hyperactive Type (ADHD-H), at each time point, with 95% confidence intervals.

Remission Analysis. Logistic mixed models (with occasion nested by individual) used remission of the primary anxiety disorder as a binary outcome variable. An unconditional model indicated that a fixed, linear effect of time and its random variance were each significant along with a fixed quadratic effect of time ($p < .001$), indicating a significant increase in remission rates over time that slowed in its rate of increase. Thus the final predictor model included random intercepts for individual, fixed effects of time and time-squared, and a random slope for time. As in the response analysis, covariates were included to account for differences identified between the diagnosis groups.

Results are presented in Table 3. ADHD diagnosis, including any subtype, did not predict remission of the primary anxiety disorder (overall significance of interaction between time and diagnosis, $p = .47$).

Sensitivity Analyses. Two sensitivity analyses were performed for the anxiety outcome analyses. Firstly, we noted that 56 children in the "non-ADHD" category ($n = 748$) reported taking ADHD medication. Presumably, these children have previously been diagnosed with ADHD, but due to good symptom control, did not meet criteria for diagnosis at assessment. To test whether the presence of these children in our "non-ADHD" group may be confounding our results, we re-ran the anxiety outcome analyses with these children excluded. The pattern of significance did not change.

Secondly, to test whether lack of predictive effect of ADHD may have been due to loss of power in modelling the three subtypes of ADHD as values of our categorical variable, we repeated the anxiety outcome analyses with a binary variable indicating presence or absence of any ADHD subtype. This variable did not predict either response or remission.

ADHD Outcomes

Table 4 presents mean ADHD CSRs and remission rates by subtype at each time point. As not all participants were assessed at all time points, the included *n* is noted for each. The significance of observed changes (and their differences by diagnosis) is examined in the mixed model analyses that follow.

Table 4

Observed mean clinical severity rating (CSR) and remission rate of ADHD for ADHD diagnostic groups at each time point.

	Mean ADHD CSR (SD)				Remission Rate (%)			
Time Point	Any ADHD	ADHD-C	ADHD-I	ADHD-H	Any ADHD	ADHD-C	ADHD-I	ADHD-H
Pre-treatment (*n* = 83[a])	4.80 (0.79)	5.00 (0.85)	4.70 (0.77)	5.00 (0.71)	—	—	—	—
Post-treatment (*n* = 74)	3.64 (1.87)	4.33 (1.74)	3.46 (1.89)	2.60 (1.67)	36.5	19.0	39.6	80.0
3-month follow-up (*n* = 16)	3.18 (2.42)	4.86 (1.34)	2.13 (2.36)	0.00 (—[b])	50	14.3	75.0	100.0
6-month follow-up (*n* = 38)	3.55 (2.00)	4.33 (1.41)	3.31 (2.07)	3.33 (3.06)	36.8	22.2	42.3	33.3

Note: No ADHD = No Attention-Deficit Hyperactivity Disorder (ADHD) diagnosis; Any ADHD = any ADHD subtype diagnosis; ADHD-C = ADHD, Combined Type; ADHD-I = ADHD, Predominantly Inattentive Type; ADHD-H = ADHD, Predominantly Hyperactive Type. For CSRs, 4 is the clinical cut-off. [a] Participants who changed ADHD medication status are excluded from ADHD outcome analyses (see Method). [b] Only one ADHD-H participant at this time point.

Response Analysis. ADHD outcome models only included participants with ADHD diagnoses. An unconditional model indicated that a fixed, linear effect of time and its random variance were each significant ($p < .001$), indicating a significant decrease in severity of ADHD symptoms over time across participants, and individual differences therein. A fixed quadratic effect for time was also significant ($p < .001$), indicating deceleration in the rate of symptom improvement. Thus predictor models included random intercept for individual, fixed effects of time and time-squared, and a random slope for time. Covariates were included to control for differences amongst subtype groups as indicated in Table 1.

Coefficients of fixed effects are presented in Table 5. ADHD subtype did not predict treatment response (overall significance of interaction between time and diagnosis, $p = 0.18$). Individual predictor parameters for ADHD-I and ADHD-H were non-significant after correction for multiple testing (respectively $p = .148$ and $p = .047$ unadjusted, ADHD-C as reference category). Comparisons at each time point indicated no significant differences in slope between the subtype groups ($p > .05$, Bonferroni adjusted). Figure 2 shows the estimated marginal means for ADHD severity by each subtype at each time point.

Table 5

Results of mixed models examining response (severity of ADHD is outcome) and remission (loss of ADHD diagnosis is outcome)

Parameters	Response[a]		Remission[b]	
	β	(95% CI)	O.R	(95% CI)
Predictors (interactions with time)				
ADHD subtype				
ADHD-C	—[c]	—[c]	—[c]	—[c]
ADHD-I	-0.29	(-.69-.10)	0.90	(0.25-3.16)
ADHD-H	-0.79	(-1.57--.01)	0.57	(0.10-3.31)
DBB comorbidity	0.23	(-0.51-0.97)	0.38	(0.06-2.56)
Medication use	0.34	(-0.06-0.74)	0.62	(0.21-1.84)
Main effects				
ADHD subtype				
ADHD-C	—[c]	—[c]	—[c]	—[c]
ADHD-I	-0.45	(-0.90--0.01)	4.73	(0.40-56.06)
ADHD-H	-0.30	(-1.02-0.42)	21.20	(0.73-614.78)
DBB comorbidity	-0.50	(-1.16-0.16)	10.15	(0.53-196.04)
Medication use	0.33	(-0.06-0.74)	0.88	(0.12-6.44)
Time	-1.52*	(-2.25--0.79)	1051.13*	(30.60-36110.39)
Time^2	0.40*	(0.18-0.64)	0.19*	(0.08-0.45)

Note: All models included random intercepts of individual and random slope for time. Parameters under the subhead Predictors are interactions of the named variable with time. [a] Linear mixed model. [b]Logistic mixed model. [c] Reference category. * p<.0125.

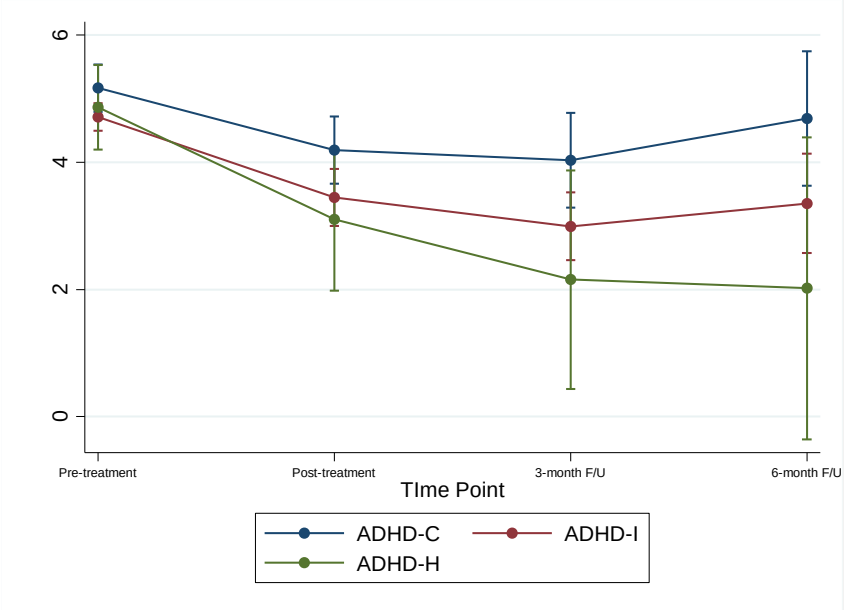

Figure 2. Model predicted margins for clinical severity rating (CSR) of ADHD Combined Type (ADHD-C), ADHD Predominantly Inattentive Type (ADHD-I) and ADHD Predominantly Hyperactive Type (ADHD-H), at each time point, with 95% confidence intervals.

An anxiety change score (calculated for each participant by averaging residualised anxiety change scores across post and follow-up timepoints) did not predict change in ADHD symptoms, whether added to the unconditional (time only) model or the final model, indicating that change in ADHD symptoms was not predicted by change in anxiety symptoms ($p > .05$).

To describe change in ADHD severity over time across children of all subtypes, mean slopes are presented in Table 6, along with contrasts of marginal means compared to pre-treatment CSR. Significantly negative slopes at pre- and post-treatment indicated decline in symptom severity ($p < .001$). At 3-months follow-up, slope was flat ($p > .05$) and at 6-

months follow-up, a positive slope indicated some worsening of severity (p =.046), which was not significant after adjustment for multiple testing. Significant contrasts with pre-treatment severity indicate that on average, children were significantly better off in terms of ADHD severity at all post-treatment time points, compared to baseline.

Table 6

Mean Slopes and Contrasts (Versus Pre-treatment CSR) at Each Time Point across All Children for ADHD Response

Time Point	Slope	(95% CI)	Contrast vs Pre-Treatment CSR	(95% Bonferroni CI)
Pre-treatment	-1.62*	(-2.24--1.01)	—[a]	—[a]
Post-treatment	-0.81*	(-1.04--0.58)	-1.22*	(-1.76--0.67)
3-month F/U	0.00	(-0.37-0.38)	-1.62*	(-2.23--1.00)
6-month F/U	0.82	(0.02-1.62)	-1.20*	(-2.06--0.36)

Note: [a] Reference time point. * p<.0125.

Remission Analysis. Logistic mixed models (with occasion nested by individual) used remission of the primary anxiety disorder as a binary outcome variable. An unconditional model indicated that a fixed, linear effect of time and its random variance were each significant along with a fixed quadratic effect of time (p < .001), indicating a significant increase in remission rates over time that slowed in its rate of increase. Thus the final predictor model included random intercepts for individual, fixed effects of time and time-squared, and a random slope for time. As in the response analysis, covariates were included to account for differences identified between the diagnosis groups.

Results are presented in Table 5. ADHD subtype did not predict remission of ADHD (overall significance of interaction between time and subtype, p = .80). Change in anxiety did not predict remission if added to the unconditional (time only) model, or the predictor model (p > .05), indicating that anxiety improvement was not associated with remission of ADHD.

Discussion

Key Findings

Our first research question was whether ADHD diagnosis predicts response to CBT treatment for anxiety. Contrary to our hypothesis, results indicate that amongst children with mild-to-moderate, non-primary ADHD, group-based CBT treatment for anxiety is just as successful as in children unaffected by ADHD comorbidity. No subtype of ADHD significantly predicted poorer or better anxiety outcomes, in either symptom response or remission of the primary anxiety disorder. This result was obtained while controlling for differences between the ADHD and non-ADHD groups on DBD comorbidity, sex and medication use.

Our second research question was whether ADHD symptoms improved after treatment of comorbid anxiety. Results indicate that, as hypothesised, children's ADHD symptoms did improve significantly after CBT for anxiety, with the majority of gains retained by six months' follow up. Improvements were fairly modest, with the average participant improving by around 1 to 1.5 points on the ADIS-IV-C/P severity scale, however in our mild-to-moderate ADHD sample, this was sufficient for around a third of children to drop below the cut-off for a diagnosable disorder. Contrary to our hypothesis, improvement in ADHD did not appear to be linked to improvement in anxiety, as anxiety severity change scores did not predict ADHD outcomes. ADHD symptom response and disorder remission rates did not significantly differ by ADHD subtype.

Comparisons with Previous Research

Anxiety Outcomes. Our finding of no impact of ADHD on anxiety treatment response is consistent with findings of some previous studies, but not with others. The Child Adolescent Multimodal Study (CAMS; Halldorsdottir et al., 2015) was similar to the current

study in that multiple anxiety disorders were included, and children with primary ADHD were excluded. In contrast to the current study, however, children with ADIS-IV-C/P diagnosed ADHD were found to show worse CBT response and remission rates immediately post-treatment, although these effects failed to reach significance at follow-up.

Differences in the CAMS design may account for the opposing findings. Firstly, the CAMS trial used individual treatment rather than group based CBT sessions, as used in the present study, and parents in the CAMS trial attended only two sessions, as compared to all sessions in the Cool Kids program (Compton et al., 2010). Secondly, the Halldorsdottir et al. (2015) analysis used ratings on the Clinical Global Impression Improvement Scale (Guy, 1976) to create a binary variable for treatment response, requiring a rating of "very much improved" or "much improved", whereas in the present study, CSRs were used as a continuous measure, sensitive to improvements of any size. Thus, it may be that measurement differences could account for the different findings. On the other hand, the same measure of remission, ADIS-IV-C/P diagnosis, was used in both studies. A more substantive difference between the two studies is sample size. Our sample included 94 children with ADHD, whereas in the CAMS study, which included multiple treatment types, only 12 children with ADHD were assigned to the CBT condition. Significance values were not corrected for multiple testing in the latter paper, so it may be that the finding was due to chance in this smaller, exploratory analysis.

The Manassis et al. (2002) trans-diagnostic study aligned with the current study in finding no effect of ADHD symptoms on treatment response, using a different measurement approach. Children were not assigned ADHD diagnoses, but rather a median split on a hyperactivity index was used to check for differences in treatment response between children with high or low levels of these symptoms. We noted in our Introduction that the use of a hyperactivity index may have made this finding less applicable to children with ADHD-I. In

the present study, however, the ADHD-I group was well represented, and our findings still align with those of Manassis et al. (2002) – while the ADHD-I children showed less improvement relative to the non-ADHD group and other subtypes (Figure 1), the difference was not statistically significant. A further notable finding from the Manassis et al. (2002) study was that higher hyperactivity was not associated with differential response to group versus individual CBT treatment, both of which were examined in their design. This gives some suggestion that our results, too, may have remained the same if individual treatment had been used.

Our null findings for predictive effect also align with those of the third previous trans-diagnostic ADHD-anxiety study we reviewed, in which Southam-Gerow et al. (2001) found no impact of higher CBCL/TRF Attention Problems scores on response to CBT for anxiety. As in the CAMS trial (Halldorsdottir et al., 2015), treatment was individual- rather than group-based, and few sessions involved parents (Kendall et al., 1997). A strength of this study relative to our own was its use of teacher-report (see Limitations, below).

It is difficult to compare our findings with those of the two anxiety-disorder specific studies reviewed (Halldorsdottir & Ollendick, 2016; Storch et al., 2008), as our sample contained few children with these disorders (specific phobia and OCD), especially in the ADHD subgroups. Halldorsdottir and Ollendick (2016) found that children with specific phobia who had higher CBCL Attention Problems scores showed poorer treatment response, an effect that persisted to follow-up. While the other studies reviewed and the present study have all used treatments of around 10-14 weeks in duration, this specific phobia study was unique in employing a single, three-hour treatment (albeit with similar components to typical CBT programs, such as psychoeducation, exposure and cognitive restructuring). It may be that such brief treatments are less suitable for children with ADHD, or that the predictive

effect of ADHD is specific to SP. Again, the use of scale scores rather than ADHD diagnosis as the predictor variable complicates comparison of this study with our own.

Storch et al. (2008) found an impact of ADHD on treatment response (p = .04), but not remission for children with OCD, while warning of the possibility of Type I error as no statistical correction was used. A similarity with the present study is that parents were involved in all sessions, although these were individual- rather than group-based. Again, however, there were many methodological differences in this study that could account for findings that contrast with ours, including the use of intensive treatment in some participants and weekly treatment in others (impact of ADHD symptoms was not assessed separately). As we have suggested in reference to the Halldorsdottir and Ollendick study of specific phobia (2016), it is possible that more intensive treatments are less effective in children with ADHD than those delivered on a weekly basis, perhaps because revision and reinforcement of concepts over a longer period (multiple weeks) supports more effective learning in children with ADHD. Distributed practice (or spaced practice) is often recommended for children with ADHD (e.g. Wells, 2010), and one study found increased improvements during working memory training if distributed practice of cognitive remediation tasks was applied, versus less "spread out" sessions (Wang, Zhou, & Shah, 2014). The longer individual sessions involved in intensive treatments may also pose a challenge to children with greater difficulty in maintaining focus.

In summary, our findings contribute to a literature of mixed findings on ADHD as a treatment response predictor, however with the addition of our larger study's null result, the case for a robust effect appears weak. Amongst previous studies examining multiple anxiety disorders, only one found that ADHD had a negative effect on treatment response. This study's sample size was considerably smaller than our own, and significance of effects did not persist to follow-up. Based on research to date, it is difficult to argue that predictive

effects of ADHD diagnosis are likely to be specific to treatment modes (such as individual versus group treatment) or measurement approaches (such as use of scale or diagnostic measures), as neither have been consistently associated with poorer treatment response. It may be, however, that more robust predictive effects apply in certain anxiety disorders that were not well represented in our sample, such as specific phobia and OCD, or that treatment response is poorer for children with ADHD when intensive interventions are employed.

ADHD Outcomes. While few studies of anxiety treatment have reported on outcomes in non-anxious comorbid disorders, our finding of improvements in ADHD is consistent with that of Kendall et al. (2001), who reported a significant ADHD remission rate of 69 percent at post CBT treatment, maintained to follow-up. This study was similar to ours in that ADIS-P interviews were used for ADHD diagnosis. Our remission rates are considerably lower than those reported by Kendall et al. (2001), a difference that could be accounted for by their smaller sample size (n = 26 in the ADHD group), or differences in methodology.

While the present study found that anxiety response did not predict ADHD response, Kendall et al. (2001) reported that those who showed remission of primary anxiety were more likely to show remission of comorbidity, linking improvement in the primary disorder to improvement in the comorbid disorder. Their analysis, however, included all comorbid disorders, including anxiety comorbidities, making the link to ADHD comorbidity questionable. Anxiety disorders were the predominant comorbidity in the sample, so any association between secondary and primary anxiety disorder improvement would be likely to present as an overall association between primary and comorbid disorder response, even if improvements in other disorders such as ADHD were not linked to improvements in primary anxiety. Further research is required to establish whether changes in ADHD symptoms are

linked to improvements in anxiety, and whether this link is specific to certain children or treatment conditions.

Strengths of the Present Study

An important strength of this study was its large sample size. Previous studies of treatment response in comorbid ADHD-anxiety have included between 11 and 26 participants with diagnosed ADHD, compared with our subsample of 94. This in turn has enabled the present study to provide the first examination of separate treatment response trajectories for subtype groups.

Our use of clinical interviews to assign diagnosis and clinical severity ratings also offers certain advantages over studies using scale measures. In such studies, symptoms of inattention and hyperactivity are typically combined into a single measure, meaning children with ADHD-C would generally present with greater symptom severity. A child with purely inattentive or purely hyperactive symptoms may experience just as much life interference as a child with ADHD-C, but not receive a high score on a combined measure. Thus, some previous studies may have included these children in the typically developing control group, potentially masking or exacerbating effects of ADHD diagnosis on treatment response. On the other hand, by using a clinical interview based on DSM-IV criteria, we were able to better ensure that children with ADHD-I or ADHD-H/I were appropriately identified. Clinicians were also trained to verify that symptoms being reported were greatly excess of those typical of children their own age, which may have somewhat reduced bias due to some parents tending to over-endorse comorbid disorders when one disorder is present (Hartung et al., 2009).

Use of a clinical interview that assessed comorbid disorders as the primary anxiety assessment offered two further key advantages. Firstly, information on severity or remission

of the comorbid disorder was available at every time point, allowing us to also report on change in the comorbid condition. Secondly, we were able to control for comorbid DBDs, which are common in children with ADHD and may influence response to anxiety symptoms (Halldorsdottir & Ollendick, 2016). Assessments and treatments were all conducted at a single clinic using the same manualised, group-based treatment (albeit with minor modifications over the duration of data collection). This consistency reduces noise that might result from inclusion of different treatment modalities and locations in the same study. Our measures also allowed us to test for prediction of both change in severity and end-point diagnosis, which could be differentially affected by comorbidity (Rapee et al., 2013).

Limitations of the Present Study

The most important limitation of this study was its exclusion of children with primary ADHD, or ADHD in excess of severity cut-offs (although only 11 children were excluded on the severity criterion). Manassis (2007) notes that exclusion of children with primary ADHD is typical in studies of anxiety treatment for comorbid children, and that included children may thus fail to resemble the comorbid children most commonly seen in the community. However, only a third of children with ADHD presenting for anxiety treatment at our clinic were excluded on these two criterion, suggesting reasonable generalisability for our findings, at least to similar anxiety treatment settings.

We also note that children with comorbid ADHD made up a small proportion of our overall sample, meaning treatment groups were unlikely to include more than one child with this comorbidity, possibly buffering negative impacts due to positive peer modelling. Our findings do not generalise to groups dominated by children with ADHD, where group dynamics could lead to greater disruption and poorer outcomes. Individual therapy settings may also have produced a different outcome (although Southam-Gerow et al. (2001) and

Manassis et al. (2002) also found no effect of comorbidity with individual therapy). While the consistency of our single-clinic, manualised protocol dataset is in some respects an advantage, it may also reduce generalisation of these findings to other CBT-based approaches. High levels of parental involvement in Cool Kids treatment may have influenced our results (see Clinical Implications).

Further, we note that our sample included very few children with ADHD-H (proportions of the other two subtypes are relatively representative in this age group; , e.g. see Willcutt, 2012). Subtype data were not retained on excluded children, so we cannot discern whether the low numbers of Predominantly Hyperactive children are due to their exclusion from the study, or due to lower rates of such children presenting for anxiety treatment. The ADHD-H group was not represented in Power et al.'s (2004) examination of anxiety prevalence by subtype. Trends towards better response in children with ADHD-H were apparent on visual inspection of data, but were non-significant given the small sample size and may be due to chance. Similarly, our sample included few comorbid ADHD participants with certain of the anxiety disorders, especially SP, OCD, PTSD and PD. Therefore, our findings should be considered most relevant to children whose ADHD is comorbid with primary GAD, SoAD or SAD.

In considering our finding of symptom improvement in ADHD after CBT-based anxiety treatment, it is crucial to consider that in the studies of anxiety treatment that comprised our dataset, there was no control group for ADHD outcomes, therefore causality of ADHD improvements cannot be attributed to the treatment. Further, we did not include teacher-reports in any of our ADHD measures, which form an important part of valid clinical ADHD assessment (Barkley, 2014). Parents, for example, tend to under-identify problems at school, but are better placed than teachers to report on problems in the home setting (Mitsis,

McKay, Schulz, Newcorn, & Halperin, 2000). Our follow-up period of six months is also relatively brief, so the long term stability of improvements could not be assessed.

Clinical and Theoretical Implications

With these caveats in mind, we believe this study presents clear and positive implications for clinicians treating this highly impaired and vulnerable group of children. Previous researchers have speculated that children with comorbid ADHD may require augmented or extended treatments, or that ADHD symptoms may need to be treated prior or concurrently in order for CBT to produce benefits similar to those of children without ADHD (Halldorsdottir & Ollendick, 2016; Storch et al., 2008). Our findings do not support the necessity of adapted approaches for children with mild-moderate non-primary ADHD, given similar clinical conditions to those we have described.

Treating primary anxiety in ADHD-comorbid children with a widely available, manualised CBT approach such as the Cool Kids program is likely to be both practical and beneficial. In terms of treatment modality recommendations, our findings add some weight to the emerging suggestion that CBT treatments incorporating parental involvement are beneficial for children with ADHD. A recent study by Maric, Steensel, and van Bögels (2015) found that for high ADHD symptom children, parental involvement led to significantly better anxiety outcomes at one year follow-up, whereas for children with low ADHD symptoms, family-based CBT offered no advantage over child-only therapy. It may be that parental involvement, such as was required in the Cool Kids program during the study period, buffers any negative impact of ADHD symptoms on treatment response. Parental supervision during therapy and homework activities could compensate for children's forgetfulness, distractibility or restlessness (on the other hand, Storch et al. (2008) reported negative impact of ADHD comorbidity for treatment with a family-based CBT program).

We also note that use of rewards for behaviour management at therapists' discretion and well-supervised treatments in a university teaching clinic may have contributed to good outcomes for comorbid children.

Our finding of no difference in CBT response in children with and without ADHD comorbidity has interesting implications for theories of therapeutic processes and ADHD-anxiety comorbidity. In introducing this comorbidity, we reviewed a number of key cognitive and behaviour differences that distinguish this group from typically developing children and from those children with either condition alone. Certain neurocognitive differences, particularly executive dysfunction deficits and their associated behaviours, have been hypothesised to make children with ADHD less susceptible to treatments such as CBT (Halldorsdottir & Ollendick, 2014; Maric et al., 2015; Storch et al., 2008).

Researchers have speculated that these children may engage less successfully with key mechanisms of therapy such as habituation to anxiety during exposure exercises, and indeed it seems logical that inattentive, hyperactive or impulsive children would have difficulty concentrating fully on such tasks. Likewise, the deficits in working memory associated with this comorbidity would seem likely to make tasks such as cognitive restructuring highly challenging. Our findings do not preclude these difficulties being experienced by some children with comorbid ADHD, but they do indicate that overall, resulting impairment is not substantial enough to impact treatment success for the group as a whole. Sources of heterogeneity in treatment response may be at play that were untapped in our study, with subtype diagnosis insufficient to distinguish between treatment responders and non-responders.

Another possibility is that improvements in areas of cognitive dysfunction during therapy offset any disadvantages they initially presented – if a child's executive dysfunction

is alleviated through practice of metacognitive activities, training in problem solving/goal setting and parental coaching, these gains could subsequently produce compensatory improvements in anxiety not seen in typically developing children who begin therapy at a higher cognitive baseline. A single-case design investigation of simultaneous psychosocial treatment of executive dysfunction-related difficulties and anxiety conducted by Jarrett and Ollendick (2012) offers a preliminary exploration of this possibility. During a combination treatment incorporating the Cool Kids anxiety program and parent management training for ADHD, comorbid children showed clinically significant improvements in both disorders. Improvements were concurrent, with no clear evidence for changes in one disorder potentiating subsequent changes in the other (Jarrett, 2013). Changes in neurocognitive abilities (including working memory) were highly heterogeneous in the $n = 8$ sample, with no significance at the group level. Further research with larger samples is required to establish whether changes in cognitive function may mediate treatment response in some children with this comorbidity.

The modest yet significant improvements seen in ADHD severity in the present study are encouraging, yet prior to replication of this finding in randomised controlled trials, we cannot portray such changes as a reliable outcome of CBT-based anxiety treatment. Observed ADHD improvement is nevertheless interesting in light of similar findings by Kendall et al. (2001) and the MTA Cooperative Group's (1999) report of heightened susceptibility to psychosocial treatment for ADHD in children with comorbid anxiety. The intensive behavioural treatment in the MTA study, however, is not equivalent to standard CBT, and the lack of control group for ADHD outcomes in Kendall et al.'s (2001) study and our own makes it difficult to determine a causal role of CBT-based anxiety treatment in observed ADHD symptom improvement.

Reduced ADHD severity ratings could be due to a "halo effect", in which parents or children tend to report improvements in all disorders addressed by the post-treatment assessments as a generalisation of response to improvements in anxiety symptoms. Similarly, the improvements could be due to overlapping symptoms (such as distractibility or restlessness) being addressed by anxiety treatment. If either of these explanations were responsible, however, one would expect children who showed greatest improvement in anxiety to also show greatest improvement in ADHD. The lack of association between degree of ADHD improvement and degree of anxiety improvement in our study, however, suggests halo effects and overlapping symptoms are unlikely to fully account for the effect.

In their multiple pathways model of ADHD development, Nigg et al. (2004) described a hypothetical group in which ADHD may develop secondary to an anxiety disorder in which state anxiety tends to momentarily overwhelm otherwise intact regulatory functions. Jarret and Ollendick (2008) subsequently proposed that ADHD symptoms of such children might be easier to treat through psychosocial approaches that relieve anxiety, allowing executive capacities to function more normally. Although in Nigg et al.'s (2004) speculative example, the group hypothesised as having intact regulatory function was described with a primarily inattentive presentation, ADHD-I was not linked to better ADHD symptom response in the present study. It could be that developmental pathways to ADHD in which anxiety is primary and executive functions are spared may result in either Inattentive or Hyperactive/Impulsive symptom profiles. The role of executive functions in treatment response across subtypes should be examined in future studies.

Future research

Involving children with severe or primary ADHD in future trials of comorbid anxiety treatment would address the most prominent limitation of our own study and most similar

research to date. Further, despite our finding of unimpaired treatment response in our mild-to-moderate ADHD group, future research may also support the development and evaluation of adapted CBT approaches to provide still better outcomes for these children. Typical 60 percent remission rates for anxiety-focused CBT leave room for improvement, and the unique cognitive and behavioural differences of children with comorbid ADHD may provide targets for synergistic improvements in broader domains of functioning.

Development of enhanced treatments may be aided by identification of sources of heterogeneity in comorbid children. Specific cognitive deficits may impact treatment response, for example, Manassis (2007) has suggested that imagery-based CBT might be more effective in children who have impaired verbal working memory. However, although certain deficits are common in ADHD, they show high levels of variance amongst individuals (Doyle, Biederman, Seidman, Weber, & Faraone, 2000). Therefore, a "one size fits all" version of CBT for ADHD-anxiety is unlikely to show improved efficacy in all children. Research testing cognitive predictors of response to different treatments would be informative. Social cognition, temperament or personality variables could be equally important, and the role of SCT in treatment response also remains to be explored (Bernad et al., 2015; Manassis et al., 2007; Nigg et al., 2004).

Although ADHD subtype differences were not significant in our anxiety outcomes analysis and only nominally significant in our ADHD response analysis, it is interesting to note that the ADHD-H group showed the largest observed improvements on both measures. A follow-up analysis of Jarrett and Ollendick's (2012) combination therapy study revealed that changes in hyperactivity symptoms were more closely linked to changes in anxiety symptoms than inattentive symptoms. Jarrett (2013) also notes that in the MTA study, parent-reported anxiety moderated treatment outcomes for hyperactivity/impulsivity but not inattention. Future studies should include larger numbers of children with ADHD-H than

were available in the present study to assess the possibility of a special link between anxiety and hyperactivity.

Higher numbers of children with ADHD-H would likely be found in studies of anxiety treatment in younger children (Willcutt, 2012), which would also shed light on the earliest developmental stages of this comorbidity. While CBT as a general treatment for ADHD in children has not shown great efficacy, evidence is mounting that it is beneficial for adult ADHD (Philipsen, 2012). Given even higher levels of comorbid anxiety in this group (Adler et al., 2007), CBT treatments targeting anxious comorbidity in adults seems like a promising subject of research.

Anxiety response aside, we believe the present findings and those of Kendall et al. (2001) indicating improvement in *ADHD* symptoms after treatment of comorbid anxiety justify further experimental research. If well-designed RCTs can demonstrate that improvements in ADHD are stable and indeed due to treatment, the scope of potential improvements will need to be assessed, in terms of: (i) which children benefit (only those whose anxiety is primary?) ; (ii) how much improvement is possible (especially relative to currently available treatments), and; (iii) whether combination therapy with stimulant medication offers additional benefits. We recommend the use of item-level data and analyses to indicate which particular symptoms of ADHD are alleviated by CBT for anxiety. If the symptoms impacted are common to both anxiety and ADHD, they may shed light on common mechanisms underlying the comorbidity.

Future research could also explore why children with comorbid anxiety appear to be more susceptible to psychosocial ADHD treatment. Jarrett et al. (2006) have speculated that increased awareness of personal shortcomings in some children leads to both comorbid internalizing symptoms and willingness to engage with therapies that might lead to self-

improvement. Measures of self-appraisal would thus be a worthwhile inclusion in treatment trials. Another avenue for understanding the mechanisms of CBT effects on ADHD would be to compare treatments emphasising different components. CBT treatments incorporate many elements, from psychoeducation to behavioural experiments to cognitive restructuring, any of which may be more or less important. It is even possible that improvements are due to incidental outcomes, such as improved parent-child relationships developed through joint attendance at therapy.

In CBT treatments for adult ADHD, therapy focuses on training in organisational and attentional skills, while addressing the failure experiences, negative self-schemas and demand-related distress that contribute to avoidance of such adaptive behaviours (Safren, Sprich, Chulvick, & Otto, 2004). Existing anxiety treatments may incidentally target these processes, but adapting them to explicitly emphasise these elements could produce enhanced outcomes.

Conclusion

In our enthusiasm for discovering differences between diagnostic groups, researchers should be cautious not to make limiting assumptions about the diverse capabilities of children with neurodevelopmental disorders. Children with mild-to-moderate ADHD of any subtype are likely to obtain similar benefits from typical, manualised, group-based cognitive behavioural therapy for primary anxiety compared to those of other anxious children without ADHD. These children are also likely to benefit from a small reduction in their ADHD symptoms, maintained for at least six months post-treatment. Comorbid anxiety increases distress and impairment in children with ADHD, and parents and clinicians should be aware of the availability and appropriateness of effective treatments. Future research should aim to extend these benefits to children with more severe comorbid ADHD, and investigate

predictors of differential response of symptoms of both disorders during CBT treatment. Understanding which elements of therapies work best for whom should promote improved outcomes for a more diverse group of children.

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