Pre-treatment Predictors of Treatment Outcome in Childhood Anxiety Disorders: A Systematic Review

Ashleigh Knight

Bachelor of Arts – Psychology (Honours)

Centre for Emotional Health
Department of Psychology
Faculty of Human Sciences
Macquarie University

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Table of Contents

List of Tables

List of Figures

Statement by Candidate

I certify that the work in this thesis entitled “Pre –treatment predictors of treatment outcome for childhood anxiety disorders: A systematic review” has not previously been submitted for a degree nor has it been submitted as part of requirements for a degree to any other university or institution other than Macquarie University. All information sources and literature used are indicated in the thesis. I also certify that the thesis is an original piece of research and it has been written by me. Professor Jennifer Hudson, Professor Mike Jones and Dr Lauren McLellan gave supervision and support.

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Ashleigh Knight

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Date:

Summary

As current strategies for improving the treatment of childhood anxiety disorders have yet to markedly improve outcomes, the focus of research has shifted to determining what variables can predict treatment outcome. At the same time, other research (e.g. Rapee et al., 2013) has postulated that the way we define treatment outcome; either categorically (endpoint) or continuously (rate of change) can have an impact on the results found. As such, this study aimed to conduct a broader systematic review of the literature, examining all researched predictors of treatment outcome for childhood anxiety disorders.

The final sample included 47 articles and four dissertations. Article were included if the children were diagnosed with a primary anxiety disorder, and were under the age of 19 at the time of treatment. All articles must have had a sample size larger than 50, and reported pre –treatment variables as predictors of treatment outcome. Predictors researched by more than three articles were reviewed systematically. All other predictors were included in a summary table as no conclusions could be drawn at this time. Predictors were grouped into three main categories – child demographic, child diagnostic and parental psychopathology; and then broken down within these categories. Within each category, results separated by the type of outcome used (endpoint and rate of change) and then by the person who completed the outcome measure (diagnostic; clinician, parent and child).

Results determined that all child demographic variables were not significant predictors of outcome, irrespective of outcome definition. There was some evidence to suggest that comorbid depression and externalizing disorders were associated with decreased treatment outcome for endpoint measures only. Similarly, higher symptom severity was associated with worse treatment outcome for endpoint measures only; however these results were not consistently found., When examining possible parental predictors, maternal anxiety was

associated with treatment outcome using the endpoint measure only, although the direction of this relationship is currently unclear. The current study also found that the weight of the evidence shows that parental and paternal anxiety and, maternal and paternal depression was not associated with treatment outcome. However there was tentative evidence that measures of parental psychopathology was associated with poorer outcomes. The inconclusiveness of this evidence suggests that further research is required to confirm the specific relationships between parental psychological conditions and treatment outcomes.

The current study also highlighted that significant results were most common found when using a diagnostic or clinician reported measure of outcome (particularly with endpoint) and for the child diagnostic and parental psychopathology predictors, a diagnostic or clinician reported measure was also more likely to find a significant result. These reports also highlight the importance of continuing to use these measures in research. The findings of the current study emphasize some of the challenge of this field of research and provide suggests for future research and clinical applications. These suggestions include the development of a measure to evaluate the quality of research into predictors, the examination of the interaction between possible predictors and focusing on understanding in more detail the characteristics of a child and their family before beginning treatment to develop a more individualized treatment rather that the current treatment manuals being used.

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Pre-treatment Predictors of Outcome for Childhood Anxiety Disorders

Approximately one in five children[1] will struggle with an anxiety disorder before they reach adulthood (Rapee, Schniering, & Hudson, 2009). Further, research has consistently shown the negative and detrimental impact that anxiety disorders have on development and socio – economic outcomes (Rapee et al., 2009). Cognitive behavioural therapy (CBT) has consistently been found to be an effective treatment for childhood anxiety disorders (Chorpita et al., 2011) with remission rates of up to 60% both post treatment (Cartwright-Hatton, Roberts, Chitsabesan, Fothergill, & Harrington, 2004) and in long-term follow-up (In-Albon & Schneider, 2006). Consequently, these remission rates suggest that treating children with CBT at a young age is an effective strategy for avoiding these negative impacts. Despite this, the variability in success rates suggests that current forms of treatment for childhood anxiety disorders are not consistently effective in treating every child with this disorder. As a result, two new avenues of research have developed. First, researchers have amended treatment programs provided through inclusion of parents (e.g. Barrett, Dadds, & Rapee, 1996) addition or changes in the skills taught (e.g. Rice, 2009) and duration of treatment (e.g. Ollendick et al., 2009). However, two recent systematic reviews have found no change in the overall remission rate, irrespective of the amendments made (Cartwright-Hatton et al., 2004; In-Albon & Schneider, 2006). The second avenue of research has focused on understanding why treatment is effective (Garcia et al., 2010; Paul, 1967). Specially, researchers have focused on examining possible mediators and moderators (or predictors) of treatment outcome. In the case of predictors of treatment outcome, this variability could be due to differences in statistical analyses, small sample sizes or the variables examined. To address these challenges, and allow for researchers to draw stronger conclusions regarding these predictors, a systematic review of the literature is required. As the variability of the data prevents a meta –

[1] Although a variety of terms have been used in the literature to describe children and adolescents, in the current study the term children is used to describe any individual under the age of 18.

analysis from being conducted, the aim of the current study is to conduct a systematic review of the literature in regards to pre-treatment predictors of outcome for childhood anxiety disorders.

The study will begin with a review of the literature summarizing our current understanding of childhood anxiety disorders and current strategies to measure and treat it, and why this is not always effective before completing the systematic review and finishing with a discussion of the findings and their applications.

Childhood Anxiety Disorders

Fear, or anxiety, is an evolutionarily derived response to detecting and responding to threats for survival (Bateson, Brilot, & Nettle, 2011), comprising of biological, physiological and behavioral components (Albano & Kendall, 2002), that becomes maladaptive when it interferes with the general functioning of an individual (Beesdo, Knappe, & Pine, 2009). Anxiety is described as a 'future – oriented' emotion, whereby individuals fear the future to be uncontrollable and unpredictable, and as such avoid or experience excessive worry about the future (Barlow, 2002; Barlow, Allen, & Choate, 2004; Silverman & Ollendick, 2005). Individuals struggling with an anxiety disorder report feeling increased physiological symptoms (for example sweaty palms and increased heart rate), and a tendency to avoid the anxious situation/object or to feel extremely distressed when facing it (Beesdo et al., 2009).

Mental illnesses are generally diagnosed using the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013) which is currently in its 5th version. The most common anxiety disorders in childhood are separation anxiety disorder (SAD; 4.9%; Bittner et al., 2007) generalized anxiety disorder (GAD; 5.8%; Bittner et al., 2007), social phobia (SP; 1.5%; Bittner et al., 2007) and specific phobias (SpPh; 6%;Bittner et al., 2007).

Table 1

Description of Anxiety Disorders as per the DSM – IV – TR

DSM - IV - TR		
Code	Disorder	Clinical Features
300.02	Generalized Anxiety Disorder	Excessive worry or anxiety, occurring more days than not, about future events or activities that is difficult to control.
309.21	Separation Anxiety Disorder	Excessive anxiety when separated from home or from attachment figures.
300.23	Social Phobia	Marked, persistent fear of social or performance situations in which embarrassment may occur.
300.29	Specific Phobia	Marked, persistent fear of clearly described objects or situations that invokes immediate anxiety and is either avoided or experienced with extreme distress.
300.01	Panic Disorder without Agoraphobia	Recurrent, unexpected panic attacks and a persistent anxiety regarding possible future attacks.
300.21	Panic Disorder with Agoraphobia	Recurrent, unexpected panic attacks and a persistent anxiety regarding possible future attacks, combined with the presence of Agoraphobia
309.81	Post Traumatic Stress Disorder	Exposure to an extreme traumatic event leading to the development of persistent, distressing recollections of the event, avoidance of stimuli associated with the event and evidence of increased arousal.
300.30	Obsessive Compulsive Disorder	Recurrent obsessions and compulsions that are severe enough to be time consuming or cause marked distress or significant impairment.
	Adjustment Disorder with mixed Depression and Anxiety	Development of clinically significant emotional or behaviour symptoms in response to a psychosocial stressor with persistent symptoms of depression and anxiety.
300.22	Agoraphobia without Panic Disorder	Anxiety regarding particular places or situations in which escape is not possible, or embarrassing, or which help may be difficult if a panic attack was to occur.
300.00	Anxiety Disorder Not Otherwise Specified	Presence of an excessive anxiety or worry that does not meet criteria for any other anxiety or adjustment disorder.

Note: DSM = Diagnostic and Statistical Manual;

Ehringer, Rhee, Young, Corley, and Hewitt (2006) in examining the prevalence rates and genetic contribution of various childhood internalizing disorders found that, for both GAD and SAD, the prevalence increased with age, and that both disorders were most commonly found in females; with often a 2:1 or 3:1 ratio of females to males (Beesdo et al., 2009; see also Bittner et al., 2007). Table 1 outlines the various anxiety disorders as per the DSM – IV – TR.

Measuring and Diagnosing Anxiety Disorders

One method to diagnose children with an anxiety disorder is a structured or semi – structured interview with the parent and/ or child (Silverman & Ollendick, 2005) as they allow for a better separation of clinical and subclinical symptoms (Beesdo et al., 2009). Interviews with parents are often conducted to supplement the information provided by the children since younger children often experience difficulties in identifying and communicating their thoughts and feelings (Beesdo et al., 2009). The most common diagnostic interview is the Anxiety Disorder Interview Schedule (ADIS-IV-C/P; Silverman, Saavedra, & Pina, 2001) a semi -structured interview designed to assess a child's anxiety and other related disorders through separate interviews with the child (ADIS – IV - C) and the parent (ADIS – IV - P), based on DSM IV criteria (Silverman et al., 2001). The ADIS – IV – C/P is used by a clinician to diagnoses a variety of anxiety and related disorders, as well as assigning severity ratings (known as Clinician Severity Ratings [CSR]) for each diagnosis (Silverman & Ollendick, 2005; Silverman et al., 2001). A CSR of 4 or higher is considered clinical diagnosis, while diagnoses less than 4 are classified as subclinical. The primary diagnosis is generally the disorder the clinician assesses as the most severe and the disorder having the greatest impact on a child's life (Silverman & Ollendick, 2005). The ADIS – IV – C/P has strong inter-rater reliability and convergent validity in various clinical samples (see for a review (see Silverman & Ollendick, 2005 for a review). However, the ADIS – IV – C/P

can be a time consuming measure, as a full diagnostic interview with both the child and parent can take up to 3 or 4 hours, making it a thorough measure but resource intensive.

Alternative semi – structured diagnostic measures include Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (K - SADS- E; Kaufman et al., 1997); and the Children's Yale – Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997). The K-SADS-E is similar to the ADIS in that it identifies and diagnoses multiple psychological conditions, while the CY-BOCS is a measure designed to diagnose OCD specifically. As such, the same strengths and limitations apply to all three measures.

Analyses of clinical measures of outcome are predominately determined using two definitions of outcome; remission of primary/ any/ all anxiety disorders and CSR change score. Remission is defined as a clinician reporting a CSR of 3 or less on the relevant anxiety disorder post treatment, allowing for a categorical analysis comparing those who recovered to those who are not (an endpoint measure); while a CSR change score is a continuous measure that shows the decrease in severity of symptoms over time (rate of change; Hudson, Rapee, et al., 2009). Some researchers have attempted to combine the two to account for children who show a significant decrease in severity rating, however their primary diagnosis is still clinical post treatment, for example, for example Berman, Weems, Silverman, and Kurtines (2000) defined remission as a CSR less than 3, or a decrease in CSR score of 4 points or more. Most researchers have included both measures separately as the remission measure does not control the severity of initial symptoms. Instead, this severity is controlled for by the CSR change score, which enables researchers to examine both the number of children who respond, and the number that are diagnosis free following treatment.

Assessment of the change in the child's symptom severity can also be augmented through the use of measures designed to assess the functioning of the child. For example, the

Children's Global Assessment Scale (CGAS; Shaffer et al., 1983) is designed to assess a child's lowest level of functioning at a specified time point and is often used conjunctively with the ADIS and the CSR. Schorre and Vandvik (2004) conducted a review of the CGAS providing evidence of the measure's adequate reliability and validity of these measures. In addition, Schorre and Vandvik (2004) reported that the CGAS is sensitive to treatment changes.

Anxiety symptoms are also commonly measured through questionnaires given to the child, parent, and occasionally clinicians and teachers. A variety of questionnaires have been developed, some of which are designed to identify symptoms or behaviours related to a child's fear or anxiety (e.g. Schniering & Rapee, 2002). Most measures have been found to have strong internal consistency, test – retest reliability and concurrent validity with other measures and diagnostic interviews, particularly when examining total scores (Silverman & Ollendick, 2005) however, some of these questionnaires are often developed based on adult measures of anxiety and as such may not control for the developmental aspects of a child's anxiety (Beesdo et al., 2009). Generally speaking, children will be asked to complete multiple measures before and after treatment assessing various aspects of a child's anxiety and related (e.g. Kendall, 1994) However, ratings given to the level of symptomology is arbitrary, and does not provide a context or real world understanding of the child's anxiety (Silverman & Ollendick, 2005), although there has been some attempts to develop norms and conduct population studies (Rapee et al., 2009). These measures can also be biased due to the individual completing the measure (De Los Reyes & Kazdin, 2005). A child, for example, may choose not to endorse a particular item or minimize their level of anxiety. Similarly, a parent may not be aware, or misconstrue, a child's anxiety. These measures are designed to have high internal reliability and validity, and are a convenient, effective strategy for researchers to gather information in a time effective manner.

Due to the strengths and limitations of both the diagnostic interviews and the self-report measure, the use of both in combination as part of a multi model assessment is generally considered best practice (De Los Reyes & Kazdin, 2005). A common issue when using multiple sources of information such as diagnostic interviews, self reports and parental questionnaires, is the disagreement in the outcomes (Silverman & Ollendick, 2005). Consistently, research has found limited agreement in the types of diagnoses given, when comparing parent and child report on the ADIS – IV – C/P as well as order of diagnoses given (Kendall & Flannery-Schroeder, 1998). Research into questionnaires however finds more agreement at the symptom level, particularly for observable behaviors (Kendall & Flannery-Schroeder, 1998). Even though there are inconsistencies, both parent and children provide meaningful information about different symptoms that is relevant for assessing treatment effectiveness and whether there is a reduction of symptoms over time.

Treatment of Childhood Anxiety Disorders

CBT is a psychological treatment based on the theory that mental illnesses, such as anxiety, are maintained through maladaptive cognitions about the world, self and other (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). Treatment therefore involves addressing these cognitions and helping the individual to develop new strategies and behaviours to control and manage the cognitions (Hofmann et al., 2012). In the case of childhood anxiety, treatment programs generally consist of 5 distinct techniques – psychoeducation, somatic management skills training, cognitive restructuring, exposure methods and relapse prevention planning (Albano & Kendall, 2002). Psychoeducation involves the therapist teaching the child and/or the parent about the nature of anxiety and the role of avoidance behavior while cognitive restructuring teaches the child how to address and challenge the maladaptive cognitions (Albano & Kendall, 2002). Both somatic management and exposure techniques involve the child addressing the behavioural responses to the anxiety-provoking situations;

somatic management techniques aim to address the physiological responses and often involve relaxation techniques, while exposure is a graduated, systematic and controlled introduction to the feared stimuli. Lastly, relapse prevention involves a discussion of strategies to consolidate and generalize the treatment gains (Albano & Kendall, 2002). Treatment generally involves sessions with a psychologist, followed by the setting of homework (such as continued exposure to the feared stimuli in order to reinforce the techniques learnt in sessions) to complete prior to the next session. CBT has classified as an 'effective treatment' for childhood anxiety disorders, with outcomes often more beneficial than pharmacological methods (Hofmann et al., 2012).

The 'Coping Cat' treatment program, developed by Kendall (1994) was the initial treatment program designed to address childhood anxiety disorders. Consisting of 14 – 18, 60-minute individual sessions with the child; following treatment, 66% of the children were classified as in remission of the primary diagnosis; with similar results at a 2 – 5 year follow up (Kendall, Safford, Flannery-Schroeder, & Webb, 2004). Since then, treatment programs have been adjusted to include group structures, shortening the duration of treatment, including additional techniques and tested on a variety of populations (e.g. Pina, Silverman, Fuentes, Kurtines, & Weems, 2003). However, as often noted by researchers, there has been limited to no research determining what components of therapy (i.e. psychoeducation, somatic management techniques, cognitive restructuring, exposure methods and relapse prevention) are effective in treating anxiety, and instead research has focused on enhancing treatment to address the approximately 40% of children who currently do not improve from treatment (Creswell & Cartwright-Hatton, 2007).

In line with this, a strong focus of research has been the addition of parents in treatment. It has been proposed that by educating the parents on the development and maintenance of anxiety, they can reinforce the cognitive and behavioural strategies their child

has been taught, ideally improving treatment outcome. However, a recent meta – analysis did not support this hypothesis, with findings suggest that CBT with an additional family component (CBT + FAM) have equal outcomes to CBT only (In-Albon & Schneider, 2006). Possible explanations as to why this occurs have been proposed, with suggestions varying from the impact of marital discord, parental psychopathology or psychological stressors on the parental involvement (Albano & Kendall, 2002) to differences in treatment delivery, parental factors targeted, a lack of a clear theoretical model and definition of outcome (Breinholst, Esbjorn, Reinholdt-Dunne, & Stallard, 2012). Although research has begun to examine these possible explanations, no definitive result have been found, and as such, researchers have begun to focus on why possible explanations of why the current treatment programs are not consistently effective.

Understanding Why Treatment Is Not Always Effective

Two main strands of thinking have prevailed in defining why CBT treatment is not always successful for children with anxiety disorders. The first suggests that methodological issues in the analyses conducted impact the findings (Albano & Kendall, 2002), while the other proposes that there are predictors and mediators influencing treatment outcome (Wolitzky-Taylor, Arch, Rosenfield, & Craske, 2012). Some studies only examine the results for participants who complete the entire treatment program (known as treatment completers) and do not include participants who began treatment but dropped out of treatment (the 'intent – to – treat' sample). The exclusion of treatment dropouts can have a huge impact on the findings as other research has found significant differences in the characteristics of treatment completers to treatment dropouts (Kendall & Sugarman, 1997), however as the focus is on determining if treatment is effective for those who receive the entire treatment, some researchers have opted to focus on treatment completers only. Similarly, as mentioned previously, the definition of treatment outcome and the self-report scales themselves can

impact results. For example, Rapee et al. (2013) examined whether comorbid diagnoses can impact treatment outcome. Although they found significant effects when treatment outcome was defined categorically based on whether the primary diagnosis was in remission or not, these effects were not replicated using the self- report measures, which defined treatment outcome as change in scores over time. Similar effects have been found when defining treatment outcome as remission of primary diagnosis to remission of all diagnoses (e.g. Hudson, Lester, et al., 2013) or comparing between different self or parent report measures (e.g. Crawford & Manassis, 2001). This limits the generalizability of the results, as the result itself is dependent on the measure used or the definition of outcome, and impacts the development of theory by preventing consistent results from being found. Additionally factors such as sample size, distribution of diagnoses and type of analysis conducted can have an impact on the findings. A smaller sample, or smaller subsample may not have the statistical power necessary to find a significant effect, and while it has historically been assumed that treatment is effective for all anxiety disorders, more recent analyses have found different outcomes depending on the primary diagnosis (e.g. Barmish, 2009). Although it would be of benefit to develop some consistency between researchers regarding the aforementioned issues, it is unlikely that controlling for these methodological and statistical variations will result in better outcomes for the children themselves, but will instead make replication and generalization more effective, and allow for researchers in the future to complete more complex statistical analyses such as meta – analyses. Similarly, as amending aspects of the treatment program itself, such as including parents in the program, has yet to significantly improve the outcome for children, it is the examination of predictors and mediators of outcome that is becoming vibrant area of research.

Mediators and Predictors of Treatment Outcome.

A ‘moderator’, or predictor variable is a variable that affects the relationship between an independent variable and the dependent variable, while a ‘mediator’ variable explains the relationship between the independent and dependent variable (Baron & Kenny, 1986; March & Curry, 1998). In the context of research into the treatment outcome of anxiety disorders, it can be said that the treatment itself is the independent variable, while the outcome is the dependent variable. Therefore, the predictor is a factor that influences this relationship; either in a positive manner (such as improving treatment outcome), or in a negative manner (such as decreasing treatment outcome), while a mediator explains the relationship; identifying the specific mechanisms that improve or decrease treatment outcome (Kraemer, Wilson, Fairburn, & Agras, 2002). As such, predictors are generally stable traits or characteristics such as age or gender that can be determined pre treatment, while mediators often are within treatment effects such as the therapist child relationship (Kraemer et al., 2002). For example a response to treatment may be moderated or depend on a child’s age, while it may also be mediated or explained by the relationship between the therapist and child. Mediators and predictors are often used in regression analyses, as either a covariate to be controlled for, or as part of an interaction with the independent variable (Kraemer et al., 2002; March & Curry, 1998). As it is possible for hundreds of variables to be involved in the relationship between treatment and its outcome, it is therefore important to use a theoretical basis to determine which variables are most likely to significantly interact with treatment and focus on those. Researchers have therefore focused on using the developmental model of anxiety to determine possible predictors to measure.

The Aetiological Model of Anxiety Disorders

Research has consistently shown that the development of an anxiety disorder is due to a combination of genetic, behavioural, familial and environmental factors (Albano & Kendall,

2002)[2]. Familial aspects of anxiety can be broken down into three part, genetics, familial environment and individual aspects (Hettema, Neale, & Kendler, 2001) Up to 50% of the overlap between parental and child anxious behaviors is due to genetic transmission (Bögels & Brechman-Toussaint, 2006; Hudson & Rapee, 2009), particularly traits such as behavioural inhibition, anxiety sensitivity and fear of negative evaluation. However, twin studies have shown the genetic heredity of an anxiety disorder is only 30% (Bögels & Brechman-Toussaint, 2006; Hettema et al., 2001), indicating that a variety of parental behaviours and other environments can have a strong influence on the development of an anxiety disorder. These behaviours include the parenting style, parental psychopathology, and the familial relationship.

Parenting style has been identified as significant yet small risk factor for the development of anxiety disorder (McLeod, Wood, & Weisz, 2007) with research consistently highlighting the importance of parental over-control (or over – protection) and rejection (or negativity; Bögels & Brechman-Toussaint, 2006). Parental over – control is defined as a stable pattern of behavior whereby parents excessively manage their child's activities, display high levels of vigilance and discourage problem solving (Bögels & Brechman-Toussaint, 2006). Conceptually, this behaviour teaches a child that the world is unsafe and dangerous, helping to contribute to the development of an anxiety disorder (Hudson & Rapee, 2009). Hudson, Doyle, and Gar (2009) examined the role of maternal over – protection and found that mothers displayed significantly higher protective behaviors when playing with a clinically anxious child as opposed to when playing with a non – anxious child, suggesting that the child's anxious behavior encourages the over- involvement, resulting to a bidirectional relationship.

[2] A complete review of the literature is beyond the scope of this thesis. For further information, please see Hudson and Rapee (2004).

Parental negativity, on the other hand, is described as an absence of parental warmth and acceptance, affecting a child's beliefs and cognitions (Bögels & Brechman-Toussaint, 2006). A review by Hudson and Rapee (2009) concluded that while the evidence for parental over – control as a risk factor for the development of an anxiety disorder is strong, research into the role of negativity is more limited. Retrospective reports from adults with an anxiety disorder consistently endorse higher levels of negativity from their parents than adults without an anxiety disorder (Hudson & Rapee, 2009) while a recent meta-analysis by McLeod et al. (2007) found a stronger effect for parental over - control than parental rejection. They note, however, that parenting only accounted for 4% of the variance in anxiety, highlighting the importance of other factors.

Similar to parenting style, maternal and paternal anxiety has consistently been shown as a risk factor (Beesdo et al., 2009), due to both the genetic contribution and the anxious behaviours (otherwise known as modelled) displayed by the parents (Beesdo et al., 2009). Children with parents who have at least one anxiety disorder are at a higher risk of having their own disorder (Beesdo et al., 2009) and parental anxiety is the strongest risk factor for a child developing an anxiety disorder (Bögels & Brechman-Toussaint, 2006).

Various aspects of the familial structure, such as interparental conflict, negative family environments and martial conflict are also strong risk factors for the development of psychopathology and internalising problems (Hudson & Rapee, 2009). For example, a longitudinal study conducted by Spence, Najman, Bor, O'Callaghan, and Williams (2002) found that children under 5 who experienced either their parents distressed martial relationship or break up were at higher risk of anxiety and depression at 14 years old. Family related traumas such as parental death and sexual abuse have also been found to be risk factors (Hudson & Rapee, 2009). Jekielek (1998) determined that both parental conflict and martial disruption negatively affect a child's emotional well-being; with parental conflict

predicting higher levels of anxiety and depression, while Volbrecht and Goldsmith (2010) determined that early negative family affect and family stress predicted anxiety symptoms.

In addition to the influence of genetics and the family, research has highlighted that a child's age, gender, temperament and socio-economic status can contribute to the development of an anxiety disorder. Females have consistently been found to be at a higher risk of developing anxiety than males (Beesdo et al., 2009). Moreover, older children are more likely to report having a single, or multiple, anxiety disorders (Rapee et al., 2009). Behavioural Inhibition (BI) a temperamental style characterized by caution, avoidance and shyness in new and unfamiliar situations (Hudson, Dodd, & Bovopoulos, 2011) and has consistently been found to predict the development of anxiety (Volbrecht & Goldsmith, 2010). However since research (e.g. Prior, Smart, Sanson, & Oberklaid, 2000) has determined that not all children with BI will develop an anxiety disorder, it has been suggested that it's the interplay between BI and other factors that is important (Bögels & Brechman-Toussaint, 2006; Hudson et al., 2011). Lastly, epidemiological studies have found higher rates of anxiety disorders among lower socio – economic (SES) classes, and that a lower household income is associated with having anxiety, but to date, there is no clarity as to whether SES is a risk factor for anxiety, or a consequence of having anxiety (Beesdo et al., 2009). Overall however, it can be concluded that the development of an anxiety disorder is a consequence of a variety of factors and that it varies for each individual child.

Pre – treatment Predictors of Outcome

Due to the complexity of the aetiological model, research has predominately focused on examining each predictor separately. To date, the results have been varied. For example, when examining age; some papers (e.g. Bodden et al., 2008) found better outcomes for younger children compared to older, while other studies (e.g. Alfano et al., 2009)) found the no effect.

To address these difficulties, Nilsen, Eisemann, and Kvernmo (2013) conducted a systematic review of the literature, examining possible predictors and moderators of treatment outcome for children diagnosed with anxiety and depression. Using a sample of 32 articles, their findings suggested that neither demographic nor clinical factors moderate treatment outcome for childhood anxiety, an unexpected result considering the nature of an anxiety disorder and its development. Specifically, they examined whether age, gender, ethnicity, IQ, duration of symptoms, diagnosis, symptom severity and comorbidity predicted or moderated treatment outcome, drawing conclusions by comparing the number of studies that found significant results to the number of studies that found non – significant results; irrespective of the type of outcome or analysis conducted. This type of analysis is limiting, as an effect may be significant when using a categorical definition of outcome, only to be non-significant using a continuous measure (Rapee et al., 2013). As such, it is possible that controlling for outcome definition and type of analysis (univariate or multivariate) may have an impact on the findings. Similarly, the sample source may have limited or influenced the results - the study only included articles published in peer-reviewed journals. Although this does ensure that only studies of high quality are included, it is apparent that journals prefer to publish statistically significant results, unintentionally providing a bias (McLeod & Weisz, 2004). To address this bias without compromising on the quality of the research, McLeod and Weisz (2004) have argued the importance of using dissertations in examining clinical trials suggesting that the nature of a dissertation process provides a similar review process to that of a peer – reviewed journal. Additionally, as dissertations are not as limited by factors such as page limit and word count, they have the opportunity to provide further details about the study and analyses that are often omitted from peer reviewed journals. Lastly, the study aimed to produce a homologous sample, and as such excluded studies that focused on PTSD and OCD. Although the latest version of the DSM has opted to separate PTSD and OCD from the other anxiety

disorders, previous versions have included them, and studies have found the same CBT programs to be effective at treating both the more common types of anxiety and OCD/ PTSD (Rapee et al., 2009). As such, the removal of such studies may account for the non-significant results. The current study aims to address these concerns to provide a clearer understanding of possible predictors of outcome.

Current Study Aim and Hypotheses

This study aims to conduct a systematic review of the literature examining pre – treatment predictors of outcome for childhood anxiety disorders. It aims to address both methodological issues identified in the definition of outcome by comparing and contrasting the results from a variety of sources and determined through a variety of measures; and also the limited nature of previous analyses by including both OCD and PTSD as types of diagnosis. Lastly, it aims to identify areas that require further research by highlighting possible predictors that have only been examined once or twice.

Method

Inclusion and exclusion criteria

This systematic review investigated pre-treatment predictors of outcome for child and adolescent anxiety disorders. A paper was included in the review if (a) the child or adolescent was under the age of 19 at the time of treatment; (b) participants had been diagnosed with a primary anxiety disorder using DSM – III - R or IV (American Psychiatric Association, 1987, 1994) criteria regardless of comorbid/secondary conditions (note: studies that included subclinical anxiety diagnoses e.g. Bernstein, Layne, Egan, & Tennison, 2005 were excluded); (c) psychological treatment was offered for an anxiety disorder (note: studies that included both psychological and psychotropic treatment e.g Compton et al., 2007 were excluded); (d) the study reported and used pre-treatment variables as predictors or moderators of treatment outcome (note: studies that used change scores of pre - treatment variables as mediators of treatment outcome e.g. Kendall & Treadwell, 2007 were excluded); (e) the study used diagnostic, clinician, parent and/or child report measures of anxiety as outcome variables; (f) the study had been published in a journal or was available as a dissertation; (g) it was written in English; (h) it was published post 1994; (i) it was based on either a randomized controlled trial (RCT) or controlled study, or used the same treatment manual as used in RCTs; and (j) was based on an overall sample size greater than 50. A sample size of 50 was specified given quality assessment strategies that use this cut off to represent adequate sample size (Moncrieff, Churchill, Drummond, & McGuire, 2001). Further, as the majority of studies did not conduct power analyses, this cut off was used in an attempt to only examine studies whose outcome was likely to be reflective of a ‘true’ finding rather than the consequence of a Type 1 error rate. Further, in order to accurately reflect the sample of children currently receiving these treatment programs, the current study included all forms of primary anxiety disorders including OCD and PTSD. Although this differs from the procedure used by Nilsen et al.

(2013); it more accurately reflects the idea that all anxiety disorders have the same core components and can therefore be treated using the same techniques.

Research strategy

The first author searched Cochrane's Database of Systematic Reviews, PsycINFO, PubMed, ProQuest, EMBASE, and MedLine databases using the following keywords: anxiety, phobia, children, youth, intervention, outcome and treatment. In addition, the reference lists of retrieved articles and of review papers were also examined for relevant studies.

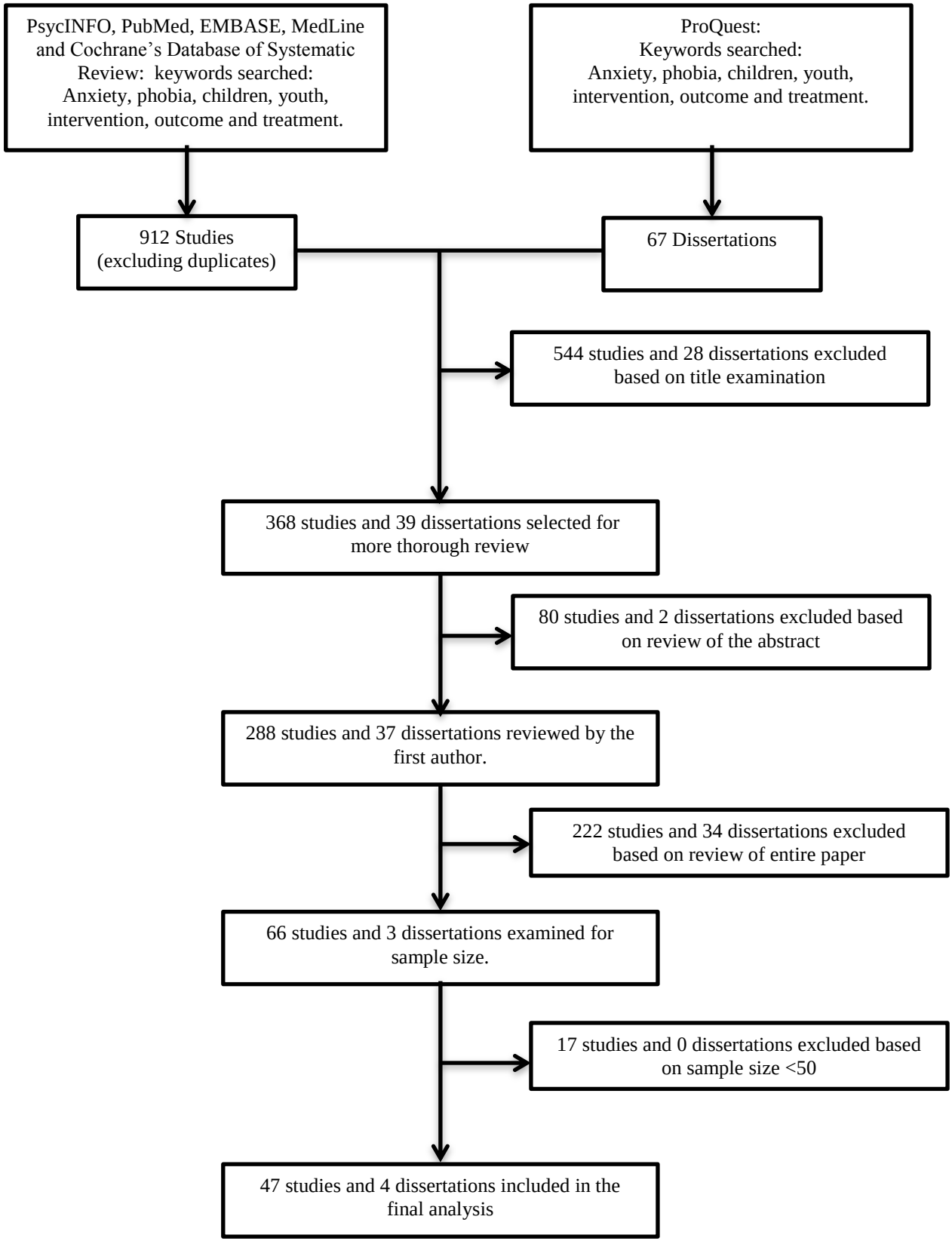

Figure 1: Flow chart of manuscript selection process

Search results

Initially, over 2,000 articles were located through the database search. After excluding duplicated, the first author reviewed 912 articles and 67 dissertations. After various stages of review a total of 47 articles and four dissertations were included in the current review (see Figure 1). Descriptive information regarding each paper can be seen in Table 2.

Analysis Strategy

The final sample of papers used a variety of outcome definitions and pre – treatment predictors that did not provide enough overlapping information to conduct a meta-analysis and gain effect sizes. Instead, the literature was reviewed systematically. In the current study, predictors investigated by more than two studies are reported by category. Identified predictors include child demographic variables and diagnostic features, and parental psychopathology. However, interactions between treatment format/type and predictors of outcome are not reported in this review. Instead, this review focused purely on pre-treatment predictors of outcome. Predictors that were examined less than three papers are briefly and separately discussed at the end of the review (and can be seen in Table 7) because the preliminary stage of research investigating the predictor limited the conclusions that could be drawn. Interactions between treatment format/type and predictors of outcome are not reported in this review.

Information about predictors of outcome will be reported separately according to the method of measuring outcome. Previous research conducted by Rapee et al. (2013) suggests that results can be influenced by the type of outcome measurement. As such, outcome was first classified as either endpoint score, whereby the analysis examined a child's level of anxiety at a specific point in time after treatment; or a rate of change score, whereby the analysis examined the *change* in a child's anxiety over assessment time points (e.g. pre treatment to post treatment, or pre treatment to follow up). Furthermore, within each method

of measuring outcome results were reported separately based on the type and rater of outcome. Specifically, endpoint analyses are reported separately for diagnostic measures such as remission of primary diagnosis or clinician rated endpoint such as the Clinician Global Improvement (CGI) score, as well as parent and child endpoint reports. Rate of change analyses are reported separately according to the rater (clinician, parent, child). One paper Beidel, Turner, and Morris (2000) determined response to treatment according to child and clinician report, so this measure of outcome was reported under clinician endpoint. Another paper, Rapee (2000) created a composite measure of outcome using parent and child reported anxiety measures. Results from this paper are included under parent-reported outcome. Some studies also used a measure of reliable change either based on clinician, parent or child report (Liber, van Widenfelt, Goedhart, et al., 2008; Liber et al., 2010). As the reliable change score is an assessment of whether or not the child has improved based on set criteria, it was categorized for the purpose of the review as an endpoint measurement of outcome. Lastly, to prevent overrepresentation of a result, samples which were used by multiple papers to examine the same predictor were only included once. It should be noted that due to the nature of the research conducted, often multiple studies used the same original sample; however only identifical analyses or analyses that appear to use the majority of the same sample are omitted.

Table 2

Descriptive information of included studies

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/ Individual	Participants	
Alfano et al. (2009)	Beidel et al. (2000); Beidel et al. (2007)	88 children, (45 boys; 7 – 17 years)	Social Phobia	- Group (12, 150 minute sessions)	- Child	CSR SPAI-C C-GAS
Angelosante (2007)	Kendall, Hudson, Gosch, Flannery-Schroeder, and Suveg (2008)	98 children (7 – 13 years)	GAD, SAD, Social Phobia	- Individual (16, 50 – 65 minute sessions)	- Child; Parent (2 sessions) (n=35) - Parent/Child (n=63)	ADIS IV-P/C C-GAS CSR
Baker and Hudson (2013)		116 children, (60 boys; 7 – 13 years old)	GAD, Social Phobia, Specific Phobia, SAD, OCD	- Group (10, 120 minutes sessions)	- Parent/Child	ADIS IV-P/C SCAS SCAS-P
Barmish (2009)	Kendall et al. (2008)	101 children, (59 boys; 7 – 14 years)	GAD, Social Phobia, SAD	- Individual (16, 60 minute sessions)	- Child; Parent (2 sessions)(n= 48) - Parent/Child (n=53)	ADIS IV-P/C CSR MASC CBCL Int.
Barrett et al. (1996)		79 children, (45 boys; 7 – 14 years old)	OAD, SAD, Social Phobia	- Individual (12, 60 – 80 minute sessions)	- Child (n=28) - Parent/Child (n=25) - Treated waitlist (n=26)	ADIS III-P/C
Barrett, Duffy, Dadds, and Rapee (2001)	Barrett et al. (1996)	52 children, (28 boys; 13 – 21 years)	OAD, SAD, Social Phobia	- Individual (12, 60 – 80 minute sessions)	- Child - Parent/Child	ADIS IV-C Clinician rated improvement RCMAS FSSC-R CBCL Int.

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/ Individual	Participants	
Barrington, Prior, Richardson, and Allen (2005)		54 children (19 boys; 7 – 14 years)	PD, SAD, Social Phobia, Specific Phobia, GAD, OCD, PTSD, ADNOS, Adjustment Disorder with Mixed Anxiety and Depression	- Individual CBT (n 28; 12 sessions) - Individual TAU (n=28; 12 sessions)	- Child - Parent - Parent/Child	ADIS IV-C/P SCAS SCAS-P BASC-P/T RCMAS
Beidel et al. (2000)		50 children (20 boys; 8 – 12 years)	Social Phobia	- Group (12, 60-90 minute sessions) - Individual (12, 60 minute sessions)	- Child	SPAI-C C-GAS
Berman et al. (2000)	Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al. (1999); Silverman, Kurtines, Ginsburg, Weems, Rabian, et al. (1999)	106 children (56 boys; 6 – 17 years)	Simple Phobia, OAD, GAD, Social Phobia, AG	- Individual (10, 80 minute sessions) (n=65) - Group (12, 55 minute sessions) (n=41)	- Parent/Child	ADIS III/IV-P/C CSR
Bodden et al. (2008)		128 children (52 boys; 8 – 17 years old)	Social Phobia, SAD, GAD, Specific Phobia, PD	- Individual (13, 60 – 90 minute sessions)	- Child (n= 62) - Parent/Child (n=52)	ADIS III/IV-P/C SCARED-P/C STAI-P/C CBCL-Int

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/ Individual	Participants	
Cobham, Dadds, and Spence (1998)		67 children (34 boys; 7 – 14 years)	SAD, OAD, GAD, Simple Phobia, Social Phobia, AG	- Group (14, 60 – 80 minutes sessions)	- Child (n=35) - Parent/Child (n=32)	ADIS III/IV-P/C RCMAS STAIC
Cobham, Dadds, Spence, and McDermott (2010)	Cobham et al. (1998)	60 children (31 boys; 10 – 17 years)	SAD, OAD, GAD, Simple Phobia, Social Phobia, AG	- Group (10, 60 – 80 minute sessions)	- Child - Parent/Child	ADIS IV-P/C CGI I RCMAS CBCL Int.
Cooper, Gallop, Willetts, and Creswell (2008)		55 children (25 boys; 6-15 years)	GAD, Social Phobia, SAD, Specific Phobia	- Individual - Group - Other	- Child - Parent	ADIS IV-P/C PSWQ-C SASC STAIC
Crawford and Manassis (2001)		61 children (34 boys; 8 – 12 years)	GAD, SAD, Simple Phobia, Social Phobia, PD and other[a]	- Individual (12 sessions) - Group (12 sessions)	- Parent/Child	RCMAS-P/C C-GAS
Crawley, Beidas, Benjamin, Martin, and Kendall (2008)		166 children (95 boys; 7 – 17 years)	GAD, Social Phobia, SAD	- Individual	- Parent/Child or Child and/or parent	ADIS IV-P/C
Eley et al. (2012)		359 children (181 boys; 6 – 13 years)	SAD, Social Phobia, GAD, Specific Phobia, PD, AG, OCD, PTSD, ADNOS	- Group (10-12 sessions) -Individual (4-10 sessions)	-Parent/Child -Parent	ADIS IV-P/C

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Ferguson (2002)		71 children, (37 boys, 6 – 17 years)	Social Phobia, Specific Phobia	- Individual (10, 60 minute sessions)	- Parent/Child	ADIS III-P/C FSSC-R CASI RCMAS-PC STAI-C
Festen et al. (2013)		145 children, (63 boys; 8 – 18 years old)	SAD, Social Phobia, GAD, Specific Phobia, PD	- Individual (12 sessions)	- Child (12 sessions); Parent (2 sessions)	RCADS
Hedtke (2007)	Kendall et al. (2008)	87 children (50 boys; 7 – 14 years)	GAD, Social Phobia, SAD	- Individual (16, 60 minute sessions)	- Child; Parent (2 sessions) - Parent/Child	CSR MASC-C C-GAS CBCL Int.
Hirshfeld-Becker et al. (2010)		64 children (30; 4 – 7 years)	GAD, SAD, Social Phobia, AG, Specific Phobia, PD, OCD	- Individual (20 sessions)	- Parent (7 sessions); Parent/Child (8 to 13 sessions).	ADIS IV-P/C CGI-I
Hudson, Newall, et al. (2013)		209 children, (6 – 13 years old)	GAD, SAD, PD, OCD, Specific Phobia, Social Phobia, PTSD, ADNOS	- Group CBT (n=100; 12, 120 minutes sessions) - Group CBT + BPAM (n=102; 5, 45 minute sessions)	- Parent/Child.	ADIS IV-P/C
Kendall, Brady, and Verduin (2001)	Kendall (1994); Kendall et al. (1997)	173 children (107 boys; 8 - 13 years)	SAD, OAD, GAD, Avoidant Disorder, Social Phobia	- Individual (16 – 20, 60 minute sessions)	- Child; Parent (2 sessions).	ADIS IV-P/C RCMAS STAI-C CBCL TRF

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Kendall et al. (1997)		94 children (58 boys; 9 – 13 years)	OAD, SAD, Avoidant Disorder	- Individual (16 – 20, 60 minute sessions)	- Child; Parent (2 sessions)	RCMAS STAI-C FSSC-R CQ-C/P NASSQ CBCL Int.
Kendall et al. (2008)		161 children (90 boys; 7 – 14 years)	GAD, SAD, Social Phobia	- Individual (16, 60 minute sessions)	- Child (n=55) - Parent/Child (n=103)	ADIS IV-P/C CSR CBCL-Int MASC
Kendall et al. (2004)	Kendall et al. (1997)	86 children (9-13 years)	GAD, SAD, Social Phobia	- Individual (16 – 20 , 60 minute sessions)	- Child; Parent (2 sessions)	ADIS IV-P/C RCMAS CBCL Int. STAI-C-P
Kerns, Read, Klugman, and Kendall (2013)	Kendall et al. (1997)	91 children (58 boys; 8 – 14 years)	GAD, Social Phobia, SAD	- Individual (16 – 20, 60 minute sessions)	- Child; Parent (2 sessions)	CSR
Kley, Heinrichs, Bender, and Tuschen-Caffier (2012)		75 children (32 boys; 8 – 12 years)	Social Phobia	- Group (12, 90 minute sessions)	- Child	SPAI-C CBCL Anx.
Legerstee et al. (2008)		178 children (93 boys; 8 – 16 years)	GAD, SAD, Social Phobia, Specific Phobia	-Individual (n=65) -Group (n=62)	-Child (10 sessions); Parent (4 sessions)	ADIS IV-P/C

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Legerstee et al. (2010)	Legerstee et al. (2008); Liber, van Widenfelt, Utens, et al. (2008)	91 children (8 – 16 years)	GAD, SAD, Social Phobia, Specific Phobia	Phase 1: - ICBT (14 sessions) - GCBT (14 sessions) Phase 2: - ICBT: (10 sessions)	Phase 1: - Child (10 sessions); Parent (4 sessions) Phase 2: - Parent/Child	ADIS IV-P/C
Legerstee et al. (2009)	Legerstee et al. (2008); Liber, van Widenfelt, Utens, et al. (2008)	131 (65 boys; 8-16 years)	GAD, SAD, Social Phobia, Specific Phobia	- Individual (n=90) Group (n=41)	- Child	ADIS IV-P/C
Lester et al. (2012)		374 children (188 boys; 6 – 13 years)	SAD, Social Phobia, GAD, Specific Phobia, PD, AG, OCD, PTSD, ADNOS	- Group (10-12 sessions) -Individual (4-10 sessions)	- Parent/Child -Parent	ADIS IV-P/C
Liber, van Widenfelt, Goedhart, et al. (2008)	Liber, van Widenfelt, Utens, et al. (2008)	124 children (75 boys; 8 – 12 years)	SAD, GAD, Social Phobia, Specific Phobia.	- Individual (n=65; 14 sessions) - Group (n=59; 14 sessions)	- Child (10 sessions); Parent (4 sessions)	ADIS IV-P/C CBCL Int. MASC-C
Liber et al. (2010)	Liber, van Widenfelt, Utens, et al. (2008)	124 children (75 boys; 8 – 12 years)	SAD, GAD, Social Phobia, Specific Phobia.	- Individual (n=65; 14 sessions) - Group (n=59; 14 sessions)	- Child (10 sessions); Parent (4 sessions)	ADIS IV-P/C CBCL Int. MASC-C

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Manassis et al. (2002)		78 children (42 boys; 8 – 12 years)	GAD, SAD, Simple Phobia, Social Phobia, PD	- Individual (n=41; 12, 90 minute sessions) - Group (n=37; 12 90 minute sessions)	- Parent/Child	MASC-P/C C-GAS
Mitchell, Newall, Broeren, and Hudson (2013)	Hudson, Newall, et al. (2013)	67 children (33 boys; 6 – 13 years)	GAD; Social Phobia, SAD, OCD, Specific Phobia	- Group CBT (n= 36; 10, 120 minute sessions) - Group CBT (n=28; 10, 120 minute sessions) + Parent Anxiety Management sessions (5, 45 minute sessions)	- Parent/Child	CSR SCAS SCAS-P
Nauta, Scholing, Emmelkamp, and Minderaa (2003)		79 children (39 boys; 7 – 18 years)	SAD, Social Phobia, GAD, PD with/without AG	- Individual (12, unspecified sessions)	- Child; Parent/Child (7 sessions)	CBCL Int. SCAS SCAS-P FSSC-R
Ollendick, Ost, Reuterskiold, and Costa (2010)	Ollendick et al. (2009)	100 children (58 boys; M= 10.21 years, SD= 2.26 years)	Specific Phobia	- Individual (1 session, 3 hours)	- Child	CSR

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Ost, Svensson, Hellstrom, and Lindwall (2001)		60 children (23 boys; 7- 17 years)	Specific Phobia	- Individual (1 session, 3 hours)	- Child - Parent/Child	CSR BAT child reported anxiety level
Panichelli-Mindel, Flannery-Schroeder, Kendall, and Angelosante (2005)	Kendall (1994); Kendall et al. (1997)	171 children (104 boys; 8-14 years)	SAD, GAD, OAD, Social Phobia, Avoidant Disorder	- Individual (20, 60 minute sessions)	- Child; Parent (2 sessions).	ADIS III –P/C RCMAS STAI-C STAI-C-P CQ-C/P FSSC-R
Pina et al. (2003)	Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al. (1999); Silverman, Kurtines, Ginsburg, Weems, Rabian, et al. (1999)	131 children (71 boys; 6 – 16 years)	GAD, OAD, SAD, Social Phobia, Specific Phobia	- Individual (10 – 12 sessions) - Group (10 – 12 sessions)	- Parent/Child	ADIS IV-P/C RCMAS FSSC-R
Puleo and Kendall (2011)	Kendall et al. (2008)	50 children (28 boys; 7 – 14 years)	SAD, GAD, Social Phobia	- Individual (16, 60 minute sessions)	- Parent/Child (n=26) - Child; Parent (2 sessions) (n=24)	ADIS IV-P/C CSR
Rapee (2000)		95 children (39 boys; 7 – 16 years)	SAD, GAD, Social Phobia, other anxiety disorder	- Groups (9, 90 minute sessions).	- Parent/Child	CBCL Int. RCMAS FSSC-R

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Rapee (2003)		165 children (73 boys; 7 – 16 years)	SAD, GAD, OAD, Social Phobia, Avoidant disorder	- Groups (11, 90 – 120 minute sessions)	- Parent/Child	CBCL Int. RCMAS FSSC-R
Rapee et al. (2013)		750 children (394 boys; 6 - 18 years)	GAD, Social Phobia, SAD, Specific Phobia, OCD, and other anxiety disorders	- Group (10, 120 minute sessions)	- Parent/Child	ADIS IV-P/C CSR SCAS P/C
Saavedra (2005)	Silverman, Kurtines, Ginsburg, Weems, Lumpkin, et al. (1999); Silverman, Kurtines, Ginsburg, Weems, Rabian, et al. (1999)	67 children (35 boys; 6 -17 years)	Social Phobia, Overanxious disorder, GAD	- Individual (10, 80 minute sessions) - Group (10, 80 minute sessions)	- Parent/Child	CBCL Int. YASR RCMAS MAS

Paper	Sample Source	Sample Size	Primary Anxiety Disorder	Treatment Program		Outcome Measures
				Group/Individual	Participants	
Scully (2011)	Mendlowitz (2010)	82 children (36 boys; 8 – 17 years)	OCD	- Individual (n = 35; 12 sessions) - Group (n= 47; 12 sessions)	- Parent/Child	CY-BOCS
Shortt, Barrett, and Fox (2001)		71 children (29 boys; 6 – 10 years)	GAD, SAD, Social Phobia	- Group (10, 90 minute sessions)	- Parent/Child	ADIS IV-P/C RCMAS CBCL Int.
Southam-Gerow, Kendall, and Weersing (2001)	Kendall (1994); Kendall et al. (1997)	135 children (82 boys; 8 – 15 years)	SAD, GAD, OAD, Social Phobia, Avoidant Disorder	- Individual (12 sessions)	- Child; Parent (2 sessions)	ADIS IV-P/C
Storch, Merlo, Larson, Geffken, et al. (2008)	Storch et al. (2007)	96 children (53 boys; 7 – 19 years)	OCD	- Individual (14, 90 minute session) - Individual intensive (14 sessions)	- Parent/Child	CGI-I CY-BOCS ADIS IV-P/C
Storch, Merlo, Larson, Bloss, et al. (2008)	Storch et al. (2007)	92 children (49 boys; 7 – 19 years)	OCD	- Individual weekly (n =48; 14 sessions) - Individual intensive (n = 42; 14 sessions)	- Parent/Child	CGI-I CGI-S CY-BOCS
Treadwell, Flannery-Schroeder, and Kendall (1995)	Kendall (1994)	151 children (86 boys; 8 – 13 years).	OAD, SAD, Avoidant Disorder	- Individual (16, 50-60 minute sessions)	- Child; Parent (2 sessions)	FSSC-R RCMAS STAI-C STAIC-C-P NASSQ CBCL

Note: ADNOS= Anxiety Disorder Not Otherwise Specified; AG= Agoraphobia; GAD= Generalized Anxiety Disorder; OAD= Overanxious Disorder; OCD = Obsessive Compulsive Disorder; PD= Panic Disorder; PTSD= Post-Traumatic Stress Disorder; SAD= Separation Anxiety Disorder; CBT= Cognitive Behavioural Therapy; TAU= Treatment As Usual; P= Parent report; C= Child report;
ADIS-III/IV= Anxiety Disorder Interview Schedule III/IV; BASC= Behaviour Assessment System for Children; BAT= Behavioral Assessment Task – Performance; CASI= Child Anxiety Sensitivity Index; CBCL-Anx.= Child Behaviour Checklist Anxiety subscale; CBCL – Int.= Child Behaviour Checklist – Internalizing subscale; C-GAS= Children's Global Assessment Scale; CGI-I= Children Global Inventory – Improvement Scale; CGI-S= Children Global Inventory – Severity Scale; CQ= Coping Questionnaire; CSR= Clinician Severity Rating; CY-BOCS= Children's Yale-Brown Obsessive Compulsive Scale; FSSC-R= Fear Survey Schedule for Children – Revised; MAS= Taylor Manifest Anxiety Scale; MASC= Multidimensional Anxiety Scale – Children; NASSQ= Children's Negative Affectivity Self Statement Questionnaire; PSWQ-C= Penn State Worry Questionnaire for Children; RCADS= Revised Children Anxiety and Depression Scale; RCMAS = Revised Children's Manifest Anxiety Scale; SAS-C= Social Anxiety Scale for Children; SCARED = Screen for Child Anxiety Related Emotional Disorders; SCAS= Spence Children Anxiety Scale; SPAI-C= Social Phobia and Anxiety Inventory for Children; STAI-C= State Trait Anxiety Inventory for Children; TRF= Teacher Report Form; YASR= Young Adult Self Report
a= such as trichotillomania and selective mutism

Results

Descriptive Information

The current analysis included a total of 51studies. Of the CBT studies[3], there were a variety of different treatment formats used. These included individual (e.g., Kendall et al., 1997) or group programs (e.g.,Himle, Fischer, van Etten, Janeck, & Hanna, 2003); child only (e.g., Alfano et al., 2009) parent and child combined (e.g., Cobham et al., 1998);parent and child separate (e.g., Cooper et al., 2008) or parent only (e.g., Hirshfeld-Becker et al., 2010). There were not enough studies to group each pre-treatment predictor by treatment type. Also, results of other research suggest the format of CBT for childhood anxiety does not impact on outcome (In-Albon & Schneider, 2006; James, James, Cowdrey, Soler, & Choke, 2013). Thus these studies were grouped together.

At pre – treatment, children's ages ranged from 4 – 19[4] years old**.** All studies included both boys and girls. Participants were included in original treatment studies based on anxiety diagnoses using the Anxiety Disorder Inventory Schedule - Child and Parent Versions III/ IV (ADIS - C/P Silverman, 1987; Silverman & Albano, 1996; Silverman & Nelles, 1988); the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version(K - SADS- E; Kaufman et al., 1997); and the Children's Yale – Brown Obsessive Compulsive Scale (CY-BOCS; Scahill et al., 1997). These diagnostic interviews were used as a diagnostic endpoint outcome variable in analyses reported this review. It should be noted that in the current study, only the ADIS was used by researchers as a diagnostic endpoint measure. Anxiety symptoms were measured using seven different clinician report measure, nine parent report measure and 12 child report measures. Only one clinician observation measure of anxiety symptoms was used, and this was an observation

[3] Of the 51 studies, most used a form of CBT, however one study included 'treatment as usual' (TAU) as a form of therapy and some used education, support and attention treatment (ESA) .
[4] The 19 year old participants came from a group of papers that used Storch et al. (2007) as their sample source.

measure of child approach towards a feared stimulus (Ost et al., 2001). Table 3 provides a summary of the current findings.

Table 3:

Number and percentage of significant findings for each predictor.

	Endpoint								**Rate of Change**					
	Diagnostic		**Clinician**		**Parent**		**Child**		**Clinician**		**Parent**		**Child**	
	Sig/NS	**(%)**	**Sig/NS**	**(%)**	**Sig/NS**	**(%)**	**Sig/NS**	**(%)**	**Sig/NS**	**(%)**	**Sig/NS**	**(%)**	**Sig/NS**	**(%)**
Age	2/11	18	0/5	0	0/1	0	0/3	0	0/3	0	0/6	0	0/8	0
Gender	1/11	9	1/5	20	0/1	0	0/3	0	0/3	0	0/5	0	0/5	0
Ethnicity	0/6	0	0/2	0	0/1	0	0/1	0			0/2	0	0/3	0
SES/Income	0/7	0	0/2	0	0/1	0	0/2	0			0/1	0	0/1	0
Primary Diagnosis	2/7	29	1/1	100			0/1	0	2/3	67	2/2	100	0/2	0
Comorbid Anxiety	0/3	0	0/1	0	0/1	0	0/1	0	0/1	0	0/1	0	0/1	0
Comorbid Depression	2/6	33	0/1	0			1/1	100	0/3	0	0/2	0	1/4	25
Comorbid Externalizing Disorders	1/5	20	1/1	100			0/1	0	0/1	0	1/4	25	1/4	25
Presence of a Comorbid Disorder	3/11	27	1/5	20	0/2	0	1/4	25	0/4	0	0/6	0	0/7	0
Symptom Severity	3/8	38	0/2	0	1/1	100	2/2	100	2/3	67	1/2	50	1/3	33
Parental Anxiety	4/7	57	0/2	0			0/1	0	0/1	0	0/3	0	0/4	0

	Endpoint								Rate of Change					
	Diagnostic		Clinician		Parent		Child		Clinician		Parent		Child	
	Sig/NS	(%)	Sig/NS	(%)	Sig/NS	(%)	Sig/NS	(%)	Sig/NS	(%)	Sig/NS	(%)	Sig/NS	(%)
Maternal Anxiety	4/6	67			0/1	0	0/1	0	1/2	50	1/6	17	1/5	20
Maternal Depression	1/3	33			0/1	0	0/1	0	0/2	0	0/4	0	1/4	25
Paternal Anxiety	0/3	0			1/1	100	0/1	0	0/2	0	1/4	25	1/3	33
Paternal Depression	1/2	50			0/1	0	0/1	0	0/2	0	1/4	25	0/3	0
Parental Psychopathology	1/1	100					1/1	100	12	50	0/2	0	1/3	33

Note: sig/NS = study found a significant outcome / total number of studies; % = percentage of studies with a significant result out of the total number of studies.

Table 4:

Child demographic pre-treatment predictors of outcome.

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
Age								
	Alfano et al. (2009)	N/S				N/S		N/S
	Barmish (2009)	N/S						
	Beidel et al. (2000)[a]		N/S					
	Berman et al. (2000)	N/S						
	Bodden et al. (2008)	older -ve*					N/S	N/S
	Cooper et al. (2008)	N/S						N/S
	Festen et al. (2013)							N/S
	Hirshfeld-Becker et al. (2010)	N/S	N/S					
	Hedtke (2007)		N/S					
	Kendall et al. (1997)						N/S	N/S
	Kendall et al. (2004)	N/S					N/S	N/S
	Kerns et al.					N/S		

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	(2013)							
	Legerstee et al. (2009)	N/S						
	Legerstee et al. (2010)	N/S						
	Liber, van Widenfelt, Goedhart, et al. (2008)	N/S		N/S	N/S			
	Nauta et al. (2003)						N/S	N/S
	Ost et al. (2001)		N/S (CSR, PO/FU) N/S (BAT, PO/FU)		N/S (PO/FU)			
	Rapee (2000)[b]						N/S	
	Saavedra (2005)[c]				N/S			
	Shortt et al. (2001)					N/S	N/S	N/S
	Southam-Gerow et al. (2001)	Younger -ve* (PO) N/S (FU)						
	Storch, Merlo, Larson, Bloss, et		N/S					

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	al. (2008)							
Gender								
	Barmish (2009)	N/S						
	Berman et al. (2000)	N/S						
	Beidel et al. (2000)[a]		N/S					
	Cooper et al. (2008)	N/S						N/S
	Hirshfeld-Becker et al. (2010)	N/S	N/S					
	Hedtke (2007)		N/S					
	Kendall et al. (2004)	N/S					N/S	N/S
	Legerstee et al. (2009)	N/S						
	Legerstee et al. (2010)	Females -ve**						
	Liber, van Widenfelt, Goedhart, et al. (2008)	N/S		N/S	N/S			
	Manassis et al. (2002)					N/S	N/S	N/S
	Ost et al. (2001)		N/S (CSR,		N/S (PO/FU)			

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
			PO/FU) Females +ve* (BAT, PO/FU)					
	Rapee (2000)[b]						N/S	
	Saavedra (2005)[c]				N/S			
	Scully (2011)					N/S		
	Shortt et al. (2001)	N/S				N/S	N/S	N/S
	Southam-Gerow et al. (2001)	N/S						
	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S					
	Treadwell et al. (1995)	N/S					N/S	N/S
Ethnicity								
	Barmish (2009)	N/S						
	Beidel et al. (2000)[a]		N/S					
	Berman et al. (2000)	N/S						
	Hedtke (2007)		N/S					
	Pina et al. (2003)	N/S		N/S			N/S	N/S
	Saavedra (2005)[c]	N/S			N/S			N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Southam-Gerow et al. (2001)	N/S						
	Treadwell et al. (1995)	N/S					N/S	N/S
SES/ income								
	Barmish (2009)	N/S						
	Berman et al. (2000)	N/S						
	Hedtke (2007)		N/S					
	Kendall et al. (2004)	N/S					N/S	N/S
	Legerstee et al. (2009)	N/S						
	Legerstee et al. (2010)	N/S						
	Liber, van Widenfelt, Goedhart, et al. (2008)	N/S		N/S	N/S			
	Saavedra (2005)[c]				N/S			
	Southam-Gerow et al. (2001)	N/S						
	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S					

Note: *= p <.05; **= p <.01;+ve= better outcome;-ve= poorer outcome; N/S= non-significant; PO = Post; FU= Follow up. BAT = Behavioral Approach Task-Performance
[a]Outcome was measured using both a child and clinician reported endpoint cut-off and results are included under clinician-reported outcome
[b]Outcome was measured via a composite measure of parent and child reported anxiety and results are included under parent-reported outcome
[c]This paper assessed long term follow up. Youth self-report was the predominant measure of outcome so is summarized as child report.

Child demographic predictors.

Age. Age has been examined as a possible predictor of outcome for child anxiety treatment in a total of 22 studies. See Table 4.

Endpoint. Two studies indicated that younger children reported higher rates of diagnostic remission at post treatment compared to older children (Bodden et al., 2008; Southam-Gerow et al., 2001), however age did not predict remission at follow-up in either study. Additionally, eight studies (Alfano et al., 2009; Barmish, 2009; Berman et al., 2000; Cooper et al., 2008; Hirshfeld-Becker et al., 2010; Kendall et al., 2004; Legerstee et al., 2009; Liber, van Widenfelt, Goedhart, et al., 2008; Southam-Gerow et al., 2001) found that age did not predict diagnostic outcome at all timepoints. Five studies (Hirshfeld-Becker et al., 2010; Ost et al., 2001; Storch, Merlo, Larson, Bloss, et al., 2008) used endpoint clinician rating as the definition of outcome, with non-significant results, even when assessed via clinician observation and at follow-up (Ost et al., 2001). Similarly, three analyses (Liber, van Widenfelt, Goedhart, et al., 2008; Saavedra, 2005) found non-significant results using both parent and/or child reported endpoint.

Rate of change. Age was not found to predict symptom change after treatment; three analyses (Alfano et al., 2009; Beidel et al., 2000; Hedtke, 2007; Kerns et al., 2013) found non-significant results when using clinician reported symptom change, six analyses (Cooper et al., 2008; Kendall et al., 1997; Kendall et al., 2004; Nauta et al., 2003; Rapee, 2000) found non-significant results using parent reported symptom change and eight analyses (Alfano et al., 2009; Bodden et al., 2008; Cooper et al., 2008; Festen et al., 2013; Kendall et al., 1997; Kendall et al., 2004; Nauta et al., 2003) found non-significant results using child self report measures of symptom change.

Overall, results suggest that a child's age prior to treatment does not influence treatment outcome. Older children reported poorer outcome only when diagnostic endpoint was measured stringently (remission of all diagnoses) and only at post-treatment.

Gender. Overall, 19 studies examined the role of gender as a predictor of treatment outcome (see Table 4).

Endpoint. Only Legerstee et al. (2010) established a significant relationship between gender and diagnostic remission, with 98% of boys compared to 73% of girls being classified as a responder (remission of any anxiety disorder) following the second stage of a stepped care program. Interestingly, results from the first stage of the stepped care program (Legerstee et al., 2009) found no significant differences in remission between genders. The other ten studies (Barmish, 2009; Berman et al., 2000; Cooper et al., 2008; Hirshfeld-Becker et al., 2010; Kendall et al., 2004; Legerstee et al., 2009; Liber, van Widenfelt, Goedhart, et al., 2008; Shortt et al., 2001; Southam-Gerow et al., 2001; Treadwell et al., 1995) also found non-significant results. Similarly, the five studies that assessed outcome as endpoint clinician report found that gender was not a significant predictor (Beidel et al., 2000; Hirshfeld-Becker et al., 2010; Ost et al., 2001; Storch, Merlo, Larson, Bloss, et al., 2008). Yet, Ost et al. (2001)found that females improved more than males using a clinician observation measure of anxiety at post-treatment and at 12 month follow-up. Three studies (Liber, van Widenfelt, Goedhart, et al., 2008; Saavedra, 2005) examined endpoint according to parent and/or child report and similarly found that gender was not a significant predictor of treatment outcome.

Rate of change. When outcome has been assessed using rate of change, gender has not been found to predict treatment outcome. Specifically, three studies (Hedtke, 2007; Manassis et al., 2002; Scully, 2011; Shortt et al., 2001) found non – significant results using clinician rated symptom change, five studies (Cooper et al., 2008; Kendall et al., 2004; Manassis et al., 2002; Rapee, 2000; Shortt et al., 2001; Treadwell et al., 1995) found non significant results

using parent reported symptom change and five studies (Cooper et al., 2008; Kendall et al., 2004; Manassis et al., 2002; Shortt et al., 2001; Treadwell et al., 1995) found non-significant results using child-reported change.

Overall, results have mostly found that the child's gender does not predict response to treatment, regardless of how outcome is measured.

Ethnic background. Eight studies have examined ethnicity as a predictor of treatment outcome (see Table 4).

Endpoint. Ethnic background failed to predict outcome when measured using diagnostic status (Barmish, 2009; Berman et al., 2000; Pina et al., 2003; Saavedra, 2005; Southam-Gerow et al., 2001; Treadwell et al., 1995), clinician reported endpoint (Beidel et al., 2000), parent reported endpoint (Pina et al., 2003; Saavedra, 2005) or child-reported endpoint (Saavedra, 2005).

Rate of change. When outcome is measured using parent-rated (Pina et al., 2003; Treadwell et al., 1995) and child-rated (Pina et al., 2003; Treadwell et al., 1995) symptom change, ethnicity does not predict the child's response to treatment.

Overall, studies examining ethnic background have consistently found that it is not a significant predictor of treatment outcome, irrespective of definition of outcome and time-point.

SES/ Income. Ten studies have examined the role of socio – economic status (SES) and parental income (hereafter SES) on treatment outcome (see Table 4).

Endpoint. SES failed to predict outcome when measured using diagnostic status (Barmish, 2009; Hedtke, 2007; Kendall et al., 2004; Legerstee et al., 2010; Legerstee et al., 2009; Liber, van Widenfelt, Goedhart, et al., 2008; Saavedra, 2005; Southam-Gerow et al., 2001; Storch, Merlo, Larson, Bloss, et al., 2008), clinician reported endpoint (Storch, Merlo,

Larson, Bloss, et al., 2008), parent and/or child-reported endpoint (Liber, van Widenfelt, Goedhart, et al., 2008; Saavedra, 2005).

Rate of change. SES failed to predict outcome when measured using parent reported symptom change (Kendall et al., 2004) and child reported change (Kendall et al., 2004).

No studies found that SES predicted outcome, regardless of how and when outcome was measured. Thus, we can conclude that the family's income and status do not have any bearing on the child's response to treatment.

Child diagnostic predictors.

Primary diagnosis. Twelve studies have examined primary diagnosis as a predictor of treatment outcome with mixed results (see Table 5).

Endpoint. When outcome has been measured using diagnostic status, two[5] analyses (Barmish, 2009; Crawley et al., 2008) found that primary diagnosis significantly predicted outcome, while another three (Barrett et al., 1996; Berman et al., 2000; Shortt et al., 2001) found that the child's pre-treatment diagnosis (social phobia (SP), generalized anxiety disorder (GAD), or separation anxiety disorder (SAD)) did not predict the child's response to treatment. Barmish (2009) established that GAD was associated with significantly better outcomes; and both Barmish (2009) and Crawley et al. (2008) found that a primary diagnosis of SP was associated with significantly poorer outcomes. Only one study (Ost et al., 2001) examined endpoint clinician report (using two methods), and found that animal subtype improved more than any other subtype of specific phobia according to clinician observation of children's approach behavior towards the feared stimulus, but not according to clinician

[5] The Puleo and Kendall (2011) study was omitted from endpoint analyses due to the overlapping sample with Crawley et al. (2008).

Table 5:

Child diagnostic pre-treatment predictors of outcome.

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
Primary diagnosis								
	Barmish (2009)	GAD +ve* Social Phobia -ve*						
	Barrett et al. (1996)	N/S						
	Barrett et al. (2001)	N/S						
	Berman et al. (2000)	N/S						
	Crawley et al. (2008)	Social Phobia -ve*						
	Kendall et al. (1997)						OAD and SAD +ve*, AD –ve** (M STAI-C-P; M CBCL Int.) N/S (M CBCL-Anx. F STAI-C-P; F CBCL Int.	N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
							F CBCL Anx.)	
	Kerns et al. (2013)					SP –ve*		
	Legerstee et al. (2010)	N/S						
	Manassis et al. (2002)					N/S	GAD +ve*	N/S
	Ost et al. (2001)		N/S (CSR, PO/FU) Animal +ve* (BAT, PO) Animal +ve** (BAT, FU)		N/S (PO/FU)			
	Puleo and Kendall (2011)					-ve*		
	Shortt et al. (2001)	N/S						
Comorbid anxiety								
	Kendall et al. (2001)	N/S					N/S	N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Legerstee et al. (2010)	N/S						
	Ollendick et al. (2010)			N/S	N/S	N/S		
	Storch, Merlo, Larson, Geffken, et al. (2008)	N/S	N/S					
Comorbid depression								
	Alfano et al. (2009)	N/S				N/S		N/S
	Berman et al. (2000)	-ve*				N/S		
	Kendall et al (2004)	N/S					N/S	N/S
	Kerns et al. (2013)					N/S		
	Kley et al. (2012)						N/S	N/S
	Legerstee et al. (2010)	N/S						
	Saavedra (2005)[a]				-ve***			-ve**

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Southam-Gerow et al. (2001)	N/S (PO; FU)						
	Storch, Merlo, Larson, Geffken, et al. (2008)	-ve*	N/S					
Comorbid externalizing								
	Berman et al. (2000)	N/S				N/S		
	Ferguson (2002)	N/S					N/S	-ve*
	Kendall et al. (2001)	N/S					N/S	N/S
	Kendall et al. (2004)	N/S					-ve*	N/S
	Rapee (2000)[b]						N/S	
	Saavedra (2005)[a]				N/S			N/S
	Storch, Merlo, Larson,	DBD –ve** ADHD N/S	DBD -ve* ADHD -ve*					

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Geffken, et al. (2008)							
Presence of a comorbid disorder								
	Barrett et al. (2001)	N/S				N/S	N/S	N/S
	Beidel et al. (2000)[c]		N/S					
	Berman et al. (2000)	N/S						
	Cooper et al. (2008)	N/S						N/S
	Hedtke (2007)		N/S					
	Kendall et al. (1997)						N/S	N/S
	Kendall et al. (2001)	N/S					N/S	N/S
	Kendall et al. (2004)	N/S					N/S	N/S
	Legerstee et al. (2010)	N/S						
	Liber et al. (2010)	-ve*		N/S	N/S			

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Ollendick et al. (2010)			N/S	N/S	N/S		
	Ost et al. (2001)		N/S (CSR, PO/FU), N/S (BAT, PO/FU)		N/S (PO/FU)			
	Rapee et al. (2013)	-ve***				N/S	NS	N/S
	Saavedra (2005)[a]				-ve ***			
	Shortt et al. (2001)	N/S				N/S	N/S	N/S
	Southam-Gerow et al. (2001)	N/S (C; PO; FU) N/S (P; PO; FU)						
	Storch, Merlo, Larson, Bloss et al (2008)		N/S					
	Storch, Merlo, Larson, Geffken, et al. (2008)	-ve*	-ve*					

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
Anxiety Symptom Severity								
	Barmish (2009)	N/S						
	Berman et al. (2000)[d]	1-ve* (C) 2 N/S (C) 4 N/S (P)				1-ve* (C) 2 N/S (C) 4 N/S (P)		
	Hedtke (2007)		N/S					
	Kendall et al. (2004)	N/S					N/S	N/S
	Kerns et al. (2013)[e]					Social phobia symptoms -ve*		
	Kley et al. (2012)						1 -ve*** 1N/S	+ve *** 1 N/S
	Legerstee et al. (2009)	N/S						
	Legerstee et al. (2010)	N/S						
	Liber et al. (2010)	-ve***		+ve**	+ve*			
	Puleo and	N/S				N/S		

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Kendall (2011)							
	Saavedra (2005)[a]				-ve ***			N/S
	Southam-Gerow et al. (2001)[f]	1-ve* (PO) 3 N/S (PO) 2-ve* (FU) 2 N/S (FU)						
	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S					

Note: *= p <.05; **= p <.01; ***= p <.001+ve=better outcome;-ve= poorer outcome; N/S = non-significant; M= Mother reported; F= Father reported; C= Child reported; P=Parent reported; ADHD = Attention Deficit Hyperactive Disorder; BAT = Behavioral Approach Task-Performance; DBD = Destructive Behavioural Disorders; GAD= Generalized Anxiety Disorder; OAD = Overanxious Disorder; SAD = Separation Anxiety Disorder; AD = Avoidant Disorder

[a]Thispaper assessed long term follow up. Youth self-report was the predominant measure of outcome so is summarized as child report.

[b]Outcome was measured via a composite measure of parent and child reported anxiety and results are included under parent-reported outcome

[c]Outcome was measured using both a child and clinician reported endpoint cut-off and results are included under clinician-reported outcome

[d]possible predictors of outcome included the child and parent reported scores on the Revised Children's Manifest Anxiety Scale; Fear Survey Schedule for Children –Revised; State –Trait Anxiety Inventory for Children – Trait scale and parent reported Parental Rating of Severity; Child Behavior Checklist Internalizing subscale.

[e]possible predictors of outcome included the child reported Social Phobia and Anxiety Inventory for Children; parent reported scores on the Anxious subscale of the Child Behavior Checklist.

[f]possible predictors of outcome included the parent reported scores on the subscales of the Anxious/Depressed, parent reported scores on the State-Trait Anxiety Inventory for Children – Parent versions teacher reported scores on the subscales of the Teacher Report Form Anxious/Depressed;; child reported scores on the Revised Children's Manifest Anxiety Scale; State-Trait Anxiety Inventory for Children-Trait version

severity assessed post treatment or at follow-up. Ost and colleagues (2001) found that the type of primary specific phobia did not predict outcome according to child-reported endpoint.

Rate of change. Using symptom change scores, Puleo and Kendall (2011)[6] found that SP was associated with smaller clinician-rated changes on outcome compared to children with GAD or SAD and Kerns et al. (2013) found poorer improvement for individuals with SP compared to individuals without SP at long-term follow-up, while Manassis et al. (2002) found primary diagnosis did not predict clinician-reported symptom change. Manassis et al. (2002) found that mothers reported a greater reduction of anxiety symptoms following treatment for children with primary GAD compared to a primary diagnosis of a phobic disorder (including SAD, SP, specific phobia and panic disorder). Similarly, Kendall et al. (1997) found that primary anxiety diagnosis had a moderating effect on mother-reported symptoms: GAD (assessed as overanxious disorder) and separation anxiety disorder (SAD), but not SP (assessed as avoidant disorder), were associated with reduction of mother-reported symptoms following treatment. However, this result was only significant for two of the six mother reported rate of change measures and none of the father-reported rate of change measures analyzed in the study. One study (Kendall et al., 1997) examined whether primary diagnosis predicted outcome as defined as child reported change scores, and found non-significant results.

Although results are mixed, there is some evidence to suggest that children with a primary diagnosis of SP are significantly more likely to retain their diagnosis post treatment, while children with a primary diagnosis of GAD are more likely to be in remission post treatment. There is also some evidence that these diagnoses are related to improvement based on parent and clinician reported rate of change.

[6]The Hedtke (2007) dissertation was omitted from rate of change analyses due to the overlapping sample with Puleo and Kendall (2011).

Comorbid anxiety. Limited research (four studies, see Table 5) has specifically investigated the influence of comorbid anxiety disorders on treatment outcome.

Endpoint. Analyses examining outcome measured as diagnostic status (Kendall et al., 2001; Storch, Merlo, Larson, Geffken, et al., 2008), endpoint clinician report (Storch, Merlo, Larson, Geffken, et al., 2008), endpoint parent and child report (Ollendick et al., 2010) found that comorbid anxiety did not predict treatment outcome.

Rate of change. Analyses examining outcome measured as clinician-rated (Ollendick et al., 2010), parent and child rated (Kendall et al., 2001) symptom change found that comorbid anxiety did not predict treatment outcome.

Overall, the evidence consistently suggests that having comorbid anxiety diagnoses does not influence the child's response to treatment, irrespective of how and when outcome is measured.

Comorbid depression. Nine studies (see Table 5) examined the impact of comorbid depression on treatment outcome.

Endpoint. Two analyses (Berman et al., 2000; Storch, Merlo, Larson, Geffken, et al., 2008) found that comorbid depression predicted poorer diagnostic remission of primary anxiety, while four (Kendall et al., 2004; Southam-Gerow et al., 2001) found a non-significant result. Interestingly, the two studies that found comorbid depression was a predictor of treatment outcome used a diagnostic assessment of depression as the predictor variable, while the two non-significant results were found when the Children's Depression Inventory (CDI; Kovacs, 1992) was used to measure depression.

Only one study, (Storch, Merlo, Larson, Geffken, et al., 2008), examined endpoint via clinician report, finding a non – significant result for comorbid major depression while Saavedra (2005) found comorbid depressive symptoms predicted poorer long-term parent/child reported outcome.

Rate of change. Three studies (Alfano et al., 2009; Kerns et al., 2013) found non significant results using clinician-reported rate of change, using both diagnostic assessment of depression and self report measures of depressive symptoms. Two studies (Alfano et al., 2009; Kendall et al., 2004; Kley et al., 2012) have examined parent reported symptoms as the outcome variable, and four (Kendall et al., 2004; Kley et al., 2012; Saavedra, 2005) examined child reported symptoms as the outcome variable. Alfano et al. (2009), Kendall et al. (2004) and Kley et al. (2012) found that child reported depressive symptoms did not predict outcome, while Saavedra (2005) found that child reported depression predicted poorer long-term treatment response.

Overall, results suggest that comorbid depression is more strongly associated with worse treatment outcome, when assessed as endpoint and/or when depression is measured as a disorder rather than symptoms.

Comorbid externalizing. Six studies have investigated comorbid externalizing disorders as predictors of treatment outcome with mixed results (see Table 5).

Endpoint. Using diagnostic outcome, Storch, Merlo, Larson, Geffken, et al. (2008) found that comorbid Disruptive Behavioural Disorders (DBD) were associated with decreased outcome, however comorbid Attention Deficit Hyperactive Disorder (ADHD) was not. Berman et al. (2000), Kendall et al. 2001 and Kendall et al. (2004) found that comorbid externalizing disorders did not predict endpoint diagnostic measures of outcome. Ferguson (2002) found that externalizing symptoms did not predict diagnostic outcome. One analysis, (Storch, Merlo, Larson, Geffken, et al., 2008) examined whether comorbid ADHD and DBD predicted clinician-reported endpoint. In this study comorbid ADHD and DBD predicted poorer improvement post treatment. One analysis (Saavedra, 2005) found that comorbid externalizing symptoms did not predict parent/child reported endpoint.

Rate of change. When examining symptom change, one analysis (Rapee, 2000) found non-significant results using parent reported symptoms, while another (Kendall et al., 2004) found significant results suggesting that parent reported externalizing symptoms were associated with smaller change scores on a parent report outcome measure. Similarly, Ferguson (2002) found the same significant effects when using child reported rate of change scores, however Kendall et al. (2001), Kendall et al. (2004) and Saavedra (2005) found non-significant results. Berman et al. (2000) found non-significant results using clinician reported rate of change.

Overall, results are mixed regardless of the strategy used to assess outcome. When externalizing symptoms have predicted outcome, results indicate that comorbid externalizing symptoms are associated with poorer treatment response, yet this is not a consistent finding.

***Presence of a comorbid diagnosis*.** Eighteen studies[7] have examined whether the presence of, or number of, comorbid disorders is associated with treatment outcome (see Table 5).

Endpoint. Nine studies have been conducted using endpoint diagnostic status. Of these studies, three (Liber et al., 2010; Rapee et al., 2013; Storch, Merlo, Larson, Geffken, et al., 2008)) found that the presence of a comorbid disorder was associated with decreased remission. On the other hand eight studies (Barrett et al., 2001; Berman et al., 2000; Cooper et al., 2008; Kendall et al., 2004; Shortt et al., 2001; Southam-Gerow et al., 2001) found that the presence of a comorbid diagnosis was not associated with outcome.

Five studies examined the role of comorbidity on endpoint clinician report. One found that comorbidity (regardless of number) was related to poorer clinician reported treatment response (Storch, Merlo, Larson, Geffken, et al., 2008), while four studies found comorbidity did not predict outcome (Beidel et al., 2000; Ost et al., 2001).

[7]The Rapee (2003) was omitted from some analyses due to the overlapping sample with Rapee et al. (2013)

Using endpoint parent/child report as the outcome variable, Saavedra (2005) found that significantly more comorbid diagnoses were found in the treatment failure compared to treatment success group. Whereas Liber et al. (2010), Ollendick et al., (2010) and Ost et al., (2001) found that comorbidity did not predict parent and/or child reported endpoint.

Rate of change. Four studies (Barrett et al., 2001; Hedtke, 2007; Ollendick et al., 2010; Ost et al., 2001; Rapee et al., 2013; Shortt et al., 2001) found non-significant results for comorbidity as a predictor of treatment outcome using clinician reported rate of change scores. Similarly, only non-significant results were found in studies measuring outcome using child self report (Barrett et al., 2001; Cooper et al., 2008; Kendall et al., 2001; Kendall et al., 2004; Rapee, 2003; Shortt et al., 2001) and parent report (Barrett et al., 2001; Kendall et al., 2001; Kendall et al., 2004; Rapee et al., 2013; Shortt et al., 2001) change scores.

Overall, evidence does not consistently support the presence of a comorbid diagnosis as a predictor of decreased treatment outcome. There is some methodologically strong evidence for comorbid diagnoses predicting poorer treatment response when outcome is measured using endpoint diagnostic status (e.g., Rapee et al. (2013) which had both a sound methodology and a large sample size of n = 750).When measured as rate of change, there is no evidence to suggest that comorbidity affects treatment response. This suggests that although children with comorbid depression respond favourably to standard protocols, there is some evidence that they are more likely to retain their diagnosis at the end of treatment.

Anxious symptom severity. A large number of studies have examined the role of symptom severity as a predictor of outcome (see Table 5).

Endpoint. Looking at diagnostic remission, three analyses (Berman et al., 2000; Liber et al., 2010; Southam-Gerow et al., 2001) found significant effects, suggesting higher pre-treatment severity is associated with decreased remission of primary (Berman et al., 2000) and “any” anxiety diagnosis (Liber et al., 2010; Southam-Gerow et al., 2001), whereas

another seven found non-significant results (Berman et al., 2000; Kendall et al., 2004; Legerstee et al., 2010; Puleo & Kendall, 2011; Southam-Gerow et al., 2001). Two analysis measured outcome using clinician rated endpoint (Storch, Merlo, Larson, Bloss, et al., 2008) and found that diagnostic/symptom severity was not a significant predictor. When outcome has been investigated as parent and child reported endpoint, opposite effects were found. Liber et al. (2010) found that higher pre-treatment symptom severity predicted greater response on parent and child report measures of anxiety symptoms, while Saavedra (2005) found that pre-treatment severity of internalising symptoms predictor poorer outcome on endpoint parent/child report.

Rate of change. When examining symptom change, two analyses (Kerns et al., 2013; Storch, Merlo, Larson, Bloss, et al., 2008) found that greater pre-treatment severity predicted greater improvement according to clinician reported change, whereas two analyses (Barmish, 2009; Hedtke, 2007; Puleo & Kendall, 2011) found a non-significant relationship. One analysis (Kley et al., 2012) found that higher child-reported severity predicted more change in child reported symptoms following treatment, and three (Kendall et al., 2004; Kley et al., 2012) reported a non – significant effect. Lastly, three analyses (Berman et al., 2000; Kendall et al., 2004; Kley et al., 2012) that assessed outcome using parent reported change found that pre-treatment severity of symptoms was not a significant predictor, and one (Kley et al., 2012) found that parent reported severity predicted better parent-reported symptom change following treatment.

Overall, results across methods of measuring outcome are mixed. When symptom severity was found to predict outcome, results indicate that greater pre-treatment symptom severity predicts greater change on anxiety measures but poorer endpoints.

Parental predictors.

Parental anxiety.

Eight studies examined the role of parental anxiety as a predictor of outcome (see Table 6). Parental anxiety differed from maternal or paternal anxiety in that it did not specify which parent had the symptoms.

Endpoint. Using diagnostic status to measure outcome, six analyses (Bodden et al., 2008; Cobham et al., 1998; Hudson, Newall, et al., 2013) found that parental anxiety predicted decreased remission. However, Hudson, Newall, et al. (2013) found that parental anxiety was only a predictor of remission of any diagnosis and not a significant predictor of remission of primary diagnosis and Cobham et al. (1998) found non significant results at 12 month follow-up. Five additional analyses (Berman et al., 2000; Cobham et al., 2010; Hirshfeld-Becker et al., 2010; Kendall et al., 2008) found that parental anxiety was not a predictor of diagnostic status following treatment. Similarly, parent anxiety was not a predictor of outcome according to endpoint clinician report (Cobham et al., 2010) or parental/child report (Saavedra, 2005).

Rate of change. Bodden et al. (2008), Saavedra (2005), Cobham et al. (1998) and Cobham et al. (2010) conducted analyses measuring outcome using either parent and/or child reported symptoms and found that parental anxiety was not a predictor of change following treatment. Berman et al. (2000) found that parental anxiety did not predict clinician-reported rate of change.

Table 6:

Parental pre-treatment predictors of outcome.

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
Parental anxiety								
	Berman et al. (2000)	1 N/S 1 -ve*				N/S		
	Bodden et al. (2008)	-ve*					N/S	N/S
	Cobham et al. (1998)	-ve* (PO, 6mo FU) NS (12mo FU)					N/S	N/S
	Cobham et al. (2010)	N/S	N/S				N/S	N/S
	Hirshfeld-Becker et al. (2010)	N/S	N/S					
	Hudson, Newall, et al. (2013)	-ve* (PO/FU) (remission of any diagnosis) N/S (PO/FU) (remission of						

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
		primary diagnosis)						
	Kendall et al. (2008)	N/S						
	Saavedra (2005)[a]				N/S			N/S
Maternal anxiety								
	Barrington et al. (2005)	-ve**					N/S	N/S
	Cooper et al. (2008)[b]	N/S						2 N/S (maternal anxiety diagnosis) 2 N/S, 1-ve*§, 1-ve** (maternal anxiety symptoms)
	Crawford and Manassis (2001)					N/S	N/S (M) N/S (F)	N/S
	Kendall et al. (2008)	N/S (PO) -ve* (FU)					N/S (PO)-ve* (FU)	N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Legerstee et al. (2008)	+ve* (adolescents) N/S (children)				+ve* (adolescents) N/S (children)	N/S	
	Liber, van Widenfelt, Goedhart, et al. (2008)	N/S		N/S	N/S		N/S	N/S
	Rapee (2000)[c]						N/S	
	Southam-Gerow et al. (2001)	-ve* (PO, FU)						
Maternal depression								
	Cooper et al. (2008)							N/S
	Crawford and Manassis (2001)					N/S	N/S (M) N/S (F)	N/S
	Legerstee et al. (2008)	N/S				N/S	N/S	1 N/S (child) 1 –ve* (adol)
	Liber, van Widenfelt, Goedhart, et	N/S		N/S	N/S		N/S	N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	al. (2008)							
	Rapee (2000)[c]						N/S	
	Southam-Gerow et al. (2001)	-ve* (PO; FU)						
Paternal anxiety								
	Crawford and Manassis (2001)					N/S	N/S (M) N/S (F)	N/S
	Kendall et al. (2008)	N/S					N/S	-ve* (ICBT)
	Legerstee et al. (2008)	N/S				N/S	N/S	
	Liber, van Widenfelt, Goedhart, et al. (2008)	N/S		-ve*	N/S		N/S	N/S
	Rapee (2000)[c]						-ve** (PO) -ve* (FU)	
Paternal depression								
	Crawford and Manassis (2001)					N/S	N/S (M) N/S (F)	N/S

Predictor	Paper	Endpoint				Rate of change		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Legerstee et al. (2008)	N/S				N/S	N/S	N/S
	Liber, van Widenfelt, Goedhart, et al. (2008)	-ve*		N/S	N/S		-ve*	N/S
	Rapee (2000)[c]						N/S	
Presence of parental psychopathology								
	Berman et al. (2000)	-ve*				-ve*		
	Crawford and Manassis (2001)					N/S (Mo) N/S (Fa)	N/S (Mo/M) N/S (Fa/M) N/S (Mo/F) N/S (Fa/F)	N/S (Mo) N/S (Fa)
	Kley et al. (2012)						N/S	N/S
	Saavedra (2005)[a]				-ve **			-ve*

Note: *= p <.05; **= p <.01; ***= p <.001 +ve= better outcome;-ve= poorer outcome; N/S = non-significant; PO = post; FU= Follow up; M= Mother reported outcome variable; F=Father reported outcome variable; Mo= Mother reported predictor variable; Fa= Father reported predictor variable; Ch=Child reported predictor variable § no longer significant after controlling for maternal social anxiety symptoms.

[a]This paper assessed long term follow up. Youth self-report was the predominant measure of outcome so is summarized as child report.

[b] Diagnostic measures of maternal anxiety included maternal GAD and SP, symptom measures of maternal anxiety included the State Trait Anxiety Inventory and the Social Phobia Scale/Social Interaction Anxiety Scale.
[c]Outcome was measured via a composite measure of parent and child reported anxiety and results are included under parent-reported outcom

Overall, there is some evidence to suggest that the presence of parental anxiety predicts poorer remission of diagnoses, however, this is not a consistent finding. There is limited evidence upon which to make a conclusion about whether parental anxiety predicts outcome measured using rate of change scores. Studies have, however, assessed parental psychopathology separately for mothers and fathers, and these results will be reviewed in turn.

Maternal anxiety. A total of eight studies have examined the role of maternal anxiety as a predictor of outcome (see Table 6).

Endpoint. Four analyses (Barrington et al., 2005; Kendall et al., 2008; Legerstee et al., 2008; Southam-Gerow et al., 2001) found that maternal anxiety predicted endpoint diagnostic status while three (Cooper et al., 2008; Legerstee et al., 2008; Liber, van Widenfelt, Goedhart, et al., 2008) found a non- significant effect. Of the four analyses that found that maternal anxiety predicted outcome, three (Barrington et al., 2005; Kendall et al., 2008; Southam-Gerow et al., 2001) found that maternal anxiety was associated with decreased treatment outcome, whereas Legerstee et al. (2008) found that maternal anxiety predicted improved treatment outcome for adolescents aged 12 – 16 years old. One paper, Liber, van Widenfelt, Goedhart, et al. (2008) examined endpoint via parent and child report and found that maternal anxiety symptoms did not predict outcome.

Rate of change. Results are mixed using outcome measures that examine symptom change over time. For clinician report Legerstee et al. (2008) found a significant steeper change for adolescents whose mother had an anxiety disorder compared to those whose mothers did not report an anxiety disorder. However, these results were not found using the child sub-sample. Crawford and Manassis (2001) also found that maternal anxiety did not predict clinican-reported symptom change.

Barrington et al. (2005), Crawford and Manassis (2001), Kendall et al. (2008), Liber, van Widenfelt and Goedhart et al., (2008) all found maternal anxiety did not predict treatment

outcome using child reported rate of change scores. However, Cooper et al. (2008) found that maternal anxiety symptoms predicted poorer child-reported anxiety change on some but not all measures and not when maternal social anxiety symptoms were controlled for. When examining parent report change scores Barrington et al. (2005); Crawford and Manassis (2001); Legerstee et al. (2008); Podell and Kendall (2011); Rapee (2000), all found that maternal anxiety did not affect symptom change. Kendall et al. (2008) found that maternal anxiety predicted poorer parent-reported outcome at follow-up but not post-treatment.

Overall, results are inconclusive. There is emerging evidence to suggest that maternal anxiety may be more likely to affect treatment outcome when either outcome or predictor are endpoint/diagnostic; however this is not entirely consistent. Furthermore, when maternal anxiety predicts outcome, direction is mixed. Typically maternal anxiety predicts poorer outcome, however, two analyses from a single study found that maternal anxiety was associated with improved outcomes.

Maternal depression. Six studies examined the role of maternal depression as a pre-treatment predictor of outcome (see Table 6).

Endpoint. Maternal depressive symptoms or diagnosis were not associated with the child's posttreatment diagnostic status in two studies (Legerstee et al., 2008; Liber, van Widenfelt, Goedhart, et al., 2008; Southam-Gerow et al., 2001), however Southam-Gerow et al. (2004) found that higher maternal depression symptoms predicted poorer treatment response. Maternal depression did not predict parent and child-rated endpoint (Liber, van Widenfelt, Goedhart, et al., 2008).

Rate of change. Clinician reported (Crawford & Manassis, 2001; Legerstee et al., 2008), child reported (Crawford & Manassis, 2001; Legerstee et al., 2008) and parent reported (Cooper et al., 2008; Crawford & Manassis, 2001; Legerstee et al., 2008; Rapee, 2000) rate of change was not significantly predicted by maternal depressive symptoms or

diagnosis. However, for adolescents maternal depression was a predictor of poorer parent-reported rate of change (Legerstee et al., 2008).

Regardless of method of measuring outcome, most studies found that maternal depression failed to predict treatment outcome.

Paternal anxiety. Five studies examined the role of paternal anxiety as a predictor of outcome (see Table 6).

Endpoint. Paternal anxiety was not found to predict outcome when outcome was measured using endpoint diagnostic status (Kendall et al., 2008; Legerstee et al., 2008; Liber, van Widenfelt, Goedhart, et al., 2008) or parent and child report of endpoint (Liber, van Widenfelt, Goedhart, et al., 2008).

Rate of change. When outcome is measured by symptom change, then the impact of paternal anxiety on child outcomes is mixed. In studies using clinician and parent reported (Crawford & Manassis, 2001; Legerstee et al., 2008), parent reported (Kendall et al., 2008; Liber et al., 2008) and child reported rate of change (Crawford & Manassis, 2001), outcome was not associated with pre-treatment paternal anxiety. However, Rapee (2000) found that greater paternal self-reported pre-treatment anxiety symptoms was associated with less anxiety symptom change immediately and 12-months following treatment and Kendall et al. (2008) found that paternal anxiety was associated with poorer child-reported anxiety change.

Overall, results suggest that paternal anxiety is not associated with treatment outcome unless outcome is measured according to parent report (assessed as endpoint or rate of change) with opposite effects.

Paternal depression. Four studies have investigated paternal depression as a predictor of treatment outcome (see Table 6).

Endpoint. When examining the relationship between paternal depression and diagnostic status, one analysis (Liber, van Widenfelt, Goedhart, et al., 2008) found that paternal depression predicted poorer diagnostic remission, however another analysis (Legerstee et al., 2008) failed to find this effect. Liber, van Widenfelt, Goedhart, et al. (2008) found that paternal depression was not a predictor of child or parent reported endpoint measures of outcome.

Rate of change. Legerstee et al. (2008) and Crawford and Manassis (2001) found a non – significant relationship between paternal depression (measured diagnostically or via paternal report) and clinician reported symptoms. Similarly, parent-reported (Crawford & Manassis, 2001; Rapee, 2000) and child-reported symptom change (Crawford & Manassis, 2001)were not predicted by paternal diagnostic or depression symptoms. One study showed paternal depression significantly predicted less reduction in parent reported symptoms (Liber Widendelt, Goedhart, et al., 2008)

Overall, the weight of the evidence suggests that paternal depression is not associated with treatment outcome, however this conclusion is based on a small number of studies.

***Presence of parental psychopathology*.** Four studies examined the role of general parental psychopathology as a predictor of treatment outcome[8] (see Table 6).

Endpoint. Higher self-reported global parental psychopathology was associated with poorer treatment response, according to diagnostic (Berman et al., 2000) and parent/child reported endpoint (Saavedra, 2005).

Rate of change. Similarly, global parental psychopathology has been associated with poorer rate of change according to clinician report (Berman et al., 2000) and child

[8]When papers reported global measures of parental psychopathology specific subscales (other than parental anxiety which has been outlined in a separate section above) were not included.

report(Saavedra, 2005). However, studies have also found the parental psychopathology does not predict symptom change according to parent and child report (Crawford & Manassis, 2001; Kley et al., 2012) and clinician report (Crawford & Manassis, 2001).

Although based on limited studies, results suggest that increased parental psychopathology predicts worse outcome when measured as endpoint, but is mixed when assessed as rate of change. Note, parental psychopathology here has been assessed by parent self-report only.

Preliminary Investigation

Additional predictors were investigated in studies included in this review. However, when predictors were assessed in fewer than three studies, the investigation was considered to be preliminary, and conclusions were not drawn. Findings for predictors considered to be under preliminary investigation can be found in Table 6.

Table 7:

Preliminary Predictors:

Predictor	**Author**	**Endpoint Analysis**				**Rate of Change Analysis**		
		Diagnostic	**Clinician**	**Parent**	**Child**	**Clinician**	**Parent**	**Child**
Friendship Quality	Baker and Hudson (2013)	+ve *					N/S	N/S
Genetic Marker								
5HTTLPR	Eley et al. (2012)	N/S (PO) SS +ve* (FU)						
NGF rs6330	Lester et al. (2012)	N/S (PO) TT+ve*** (FU)						
BDNF rs6265	Lester et al. (2012)	N/S						
Expressed Emotion	Angelosante (2007)[a]	2 N/S (M PO; FU) 1 -ve* (M FU) 3 N/S (F PO; FU)	CGAS 3 N/S (M PO;FU) 3 N/S (F PO;FU) CSR 3 N/S (M PO; FU)					

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
			3 N/S (F PO; FU)					
Disclosure of Distress	Barmish (2009)	4 N/S				4 N/S	4 N/S	1+ve* 3 N/S
	Panichelli-Mindel et al. (2005)	N/S					High +ve***	High +ve***
IQ	Legerstee et al. (2009)	N/S						
	Legerstee et al. (2010)	N/S						
Selective Attention	Legerstee et al. (2009)	severe +ve* mild N/S						
Perfectionism	Mitchell et al. (2013)					Mo-SPP N/S (PO)	Mo-SPP N/S (PO)	Mo-SPP N/S (PO)
						Mo-SOP N/S (PO)	Mo-SOP N/S (PO)	Mo-SOP N/S (PO)
						Ch-SPP N/S (PO)	Ch-SPP N/S (PO)	Ch-SPP N/S (PO)
						Ch-SOP N/S (PO)	Ch-SOP –ve* (PO)	Ch-SOP N/S (PO)
						Mo-SPP N/S(FU)	Mo-SPP N/S (FU)	Mo-SPP N/S (FU)
						Mo-SOP	Mo-SOP N/S	Mo-SOP -ve**

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
						N/S (FU) Ch-SPP N/S(FU) Ch-SOP –ve* (FU)	(FU) Ch-SPP N/S (FU) Ch-SOP –ve* (FU)	(FU) Ch-SPP N/S (FU) Ch-SOP N/S (FU)
Autism Spectrum Symptoms	Puleo and Kendall (2011)	N/S				N/S		
Negative Life Events	Kendall et al. (2004)	N/S						-ve**
Temperament	Hirshfeld-Becker et al. (2010)	-ve**	-ve*					
	Festen et al. (2013)[b]							2 N/S 1 +ve*§
OCD Symptoms								
Aggressive/ Checking	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S			+ve*		
Symmetry/ Order	Storch,		N/S			N/S		

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
	Merlo, Larson, Bloss, et al. (2008)							
Hoarding	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S			N/S		
Contamination	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S			N/S		
Sexual Religious Symptoms	Storch, Merlo, Larson, Bloss, et al. (2008)		N/S			N/S		
Parental Temperament								
Maternal Temperament	Festen et al. (2013)[c]							+ve** (negative affect) -ve**§ (effortful

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
								control) 1 N/S
Paternal Temperament	Festen et al. (2013)[c]							3 N/S
Parenting Behaviours								
Maternal Overprotection	Liber, van Widenfelt, Goedhart, et al. (2008)[d]	2 N/S (Ch) 1 N/S (Mo)		2 N/S (Ch) 1 N/S (Mo)	2 N/S (Ch) 1 N/S (Mo)			
	Festen et al. (2013)[e]							1 N/S
Paternal Overprotection	Liber, van Widenfelt, Goedhart, et al. (2008)[d]	2 N/S (Ch) 1 N/S (Fa)		2 N/S (Ch) 1 N/S (Fa)	2 N/S (Ch) 1 N/S (Fa)			
	Festen et al. (2013)[e]							1 N/S
Maternal Rejection	Liber, van Widenfelt, Goedhart, et al. (2008)[f]	2 N/S (Ch) 2 N/S (Mo)		1 N/S (Ch) 1 +ve* (Ch) 2 N/S	2 N/S (Ch) 2 N/S (Mo)			

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
				(Mo)				
	Festen et al. (2013)[g]							1 N/S 1 –ve**
Paternal Rejection	Liber, van Widenfelt, Goedhart, et al. (2008)[f]	2 N/S (Ch) 2 N/S (Fa)		2 N/S (Ch) 2 N/S (Fa)	2 N/S (Ch) 2 N/S (Fa)			
	Festen et al. (2013)[g]							1 N/S 1 –ve**§
Marital Relationship	Berman et al. (2000)	N/S						
	Rapee (2000)[h]						N/S	
Marital Status	Kendall et al. (2004)	N/S					N/S	N/S
Family Functioning	Crawford and Manassis (2001)[i]					1-ve** 2 N/S	1 -ve* (M) 1 -ve**(M) 1 N/S (M) 3 N/S (F)	1 -ve* 2 N/S
Parental Stress/Dysfunctional	Crawford and					4 N/S (Mo)	1 -ve** (Mo/M)	4 N/S (Mo) 4 N/S (Fa)

Predictor	Author	Endpoint Analysis				Rate of Change Analysis		
		Diagnostic	Clinician	Parent	Child	Clinician	Parent	Child
Parenting	Manassis (2001)[j]					4 N/S (Fa)	3 N/S (Mo/M) 4 N/S (Fa/M) 4 NS (Mo/F) 4 NS (Fa/F)	
Parental Frustration	Crawford and Manassis (2001)[jk]					1 -ve**(Ch) 1 N/S (Mo) 1 N/S (Fa)	1 N/S (Ch/M) 1 N/S (Mo/M) 1 N/S (Fo/M) 1 N/S (Ch/F) 1 N/S (Mo/F) 1 N/S (Fa/F)	1 -ve** (Ch) 1 N/S (Mo) 1 N/S (Fa)
Parental Depression								
	Berman et al (2000)	-ve*				1 N/S 1 –ve*		
	Saavedra (2005)[l]				-ve*			N/S

Note: * = p <.05; ** = p <.01; *** = p <.001**'**+ve' = better outcome;'-ve' =poorer outcome; N/S = non-significant; § = was significant in bivariate analyses but no longer significant in multi-variate analyses; PO = Post; FU= Follow up; M= Mother reported outcome variable; F= Father reported outcome variable; C=Child reported outcome variable; Mo= Mother reported predictor variable; Fa= Father reported predictor variable; Ch=Child reported predictor variable

5HTTLPR = serotonin transporter gene promoter region (5HTTLPR); SS= Short short allele; NGF rs6330 = Nerve Growth Factor (rs6330); TT= TT allele; BDNF rs6265 =Brain Derived Neurotropic Factor (rs6265) SPP = Socially prescribed perfectionism; SOP = Self oriented perfectionism; CGAS= Children's Global Assessment Scale; CSR= Clinician Severity Rating

[a]possible predictors of outcome included the parent reported subscales on the Five-Minute Speech Sample: Criticism, Emotional Involvement, Overall Expressed Emotion

[b]possible predictors of outcome included child reported scores on the subscales of the Early Adolescent Temperament Questionnaire – Revised: Negative Affect, Effortful Control, Extraversion.

[c]possible predictors of outcome included mother and father report on the Adult Temperament Questionnaire (ATQ): Negative Affect, Effortful Control and Extraversion.

[d]possible predictors of outcome included mother, father and child reported scores on the EgnaMinnenBetraffandeUppfostran (My Memories of Upbringing): Protection, Anxious Rearing (child only)

[e]possible predictors of outcome included child report of the EgnaMinnenBetraffandeUppofstram: Overprotection reported separately for mothers and fathers
[f]possible predictors of outcome included mother, father and child reported scores on the EgnaMinnenBetraffandeUppfostran (My Memories of Upbringing): Rejection, Emotional Warmth
[g]possible predictors of outcome included child report of the EgnaMinnenBetraffandeUppofstram: Rejection, Emotional Warmth reported separately for mothers and fathers
[h]Outcome was measured via a composite measure of parent and child reported anxiety and results are included under parent-reported outcome.
[i]possible predictors of outcome included the mother, father and child scores on the Brief Family Assessment Measure III
[j]possible predictors of outcome included the mother and father reported scores on the subscales of the Parenting Stress Index: Child Domain, Parent Domain, Total Stress score, Stressful Life Events scale
[k]possible predictors of outcome included the mother, father and child reported scores on the Parental Frustration Questionnaire
[l]This paper assessed long term follow up. Youth self-report was the predominant measure of outcome so is summarized as child report.

Discussion

The current study aimed to review the literature examining all researched pre – treatment predictors of treatment outcome for childhood anxiety disorders, examining by type of outcome measurement. In doing so, it examined child demographic, (age, gender, ethnicity and parental income); diagnostic (primary diagnosis, severity and comorbidity), and parental psychopathology predictors, separating outcome into endpoint and rate of change. This study also provides a summary of variables that are currently being examined as possible pre – treatment predictors and require further examination.

Overview of Findings

Results of the current study echo the findings of Nilsen et al. (2013), with the current study determining that age, gender, ethnic background and SES/Income do not predict treatment outcome irrespective of outcome definition. These results can be accounted for by considering both the nature of the development of anxiety, and the treatment itself. While older children are more likely to have a diagnosed anxiety disorder, the content of the treatment manual varies depending on the age of a child and is designed to target the child at their level of cognitive development. The Cool Little Kids (Rapee, Lau, & Kennedy, 2010); Cool Kids (Rapee et al., 2006a) and Cool Kids 'Chilled' Adolescent Programs (Rapee et al., 2006b) are examples of this, with each comprising of similar components designed to work with children of a particular age. Similarly, treatment is designed to be gender neutral and as such, while females may be more predisposed to develop anxiety, there is no theoretical reason as to why it should be more effective for females. This concept also applies for both ethnic background and SES/Income. It should be noted though that most of the studies only conducted analyses using treatment completers, rather than an intent – to – treat sample, and as research (Kendall & Sugarman, 1997) has found higher rates of dropouts for lower

SES/Income and diverse ethnic backgrounds, the current results may be more due to the nature of the sample included rather than an accurate representation of the population.

Results were less definite for child diagnostic and parental psychopathology predictors. Some evidence was found to suggest that children with primary SP were more likely to retain their diagnosis post treatment, while children with primary GAD are more likely to in remission. However, the limited number of analyses and small sub samples mean that further examination is needed. Comorbid anxiety was found to be a non-significant predictor of treatment outcome irrespective of outcome definition, while comorbid depressive disorders were associated with significantly worse outcomes using endpoint outcomes only. Similarly there was some evidence that externalizing symptoms were associated with poorer outcomes, although this result was not found within all studies. Similarly, the current study determined that the presence of a comorbid diagnosis was associated with poorer treatment outcomes at endpoint, but not for rate of change, although this outcome was not consistent between studies. Although weak, there was some evidence that, in studies that had a significant finding, higher anxious symptom severity predicted greater change in scores anxiety measures (as measured by rate of change), but poorer endpoint outcomes suggesting that children with higher symptoms are associated with a greater change in symptoms over time, but are more likely to have a clinical diagnosis post treatment.

Our findings suggest that children with comorbid depression and externalizing disorders do not respond as well to treatment compared to children with no comorbid diagnoses when treatment outcome is defined as an endpoint and using a clinical measure to determine the diagnosis. This result was more consistent when examining comorbid depression rather than comorbid externalizing disorders. These findings suggest that it is perhaps more difficult to address multiple diagnoses concurrently in treatment. Similarly higher severity ratings at pre-treatment were associated with significantly decreased treatment

outcome when defined categorically, however non-significant results occurred when the outcome was continuous. These results suggest that it is possible that all children are responding to treatment in a similar rate, but that a child with a higher symptom severity prior to treatment has further to improve than a child with a less severe diagnosis.

When examining parental psychopathology predictors, both maternal and paternal depression did not significantly predict treatment outcome, while parental anxiety was an inconsistent predictor. Maternal anxiety appears to predict treatment outcome, although the direction of this effect is unclear, with two analyses finding that maternal anxiety predicted improved treatment outcome. Paternal anxiety did not significantly predict endpoint outcome, however results for the rate of change analyses are unclear. Lastly, the presence of parental psychopathology, albeit using a small sample, appears to predict worse outcome at endpoint, but remains mixed when examining rate of change. Overall, results regarding parental psychopathology are unclear, although there is some evidence that maternal anxiety is a predictor of treatment outcome (direction unclear) and parental psychopathology is a predictor of poorer treatment outcome. Overall, the evidence suggests that the more complex the child's health is, the more difficult it is to treat and the more likely it is that the child will still display symptoms following the cessation of treatment.

Results from the preliminary analyses also lend credence to the importance of using the aetiological model, with early suggestions that genetic markers, temperament, and parenting behaviours negatively affect treatment outcome. The early nature of the analyses prevents further interpretation, but do provide guidance for further research.

Methodological Implications

The study also highlights the importance of methodology in both the collection of data and the analyses undertaken. Many studies omitted the statistics associated with their non – significant results, or did not present descriptive information such as sample mean's and

standard deviations, preventing researchers from being able to compare their findings, or conducting meta analyses. As the current study was only a systematic review, not presenting the statistics associated with the non-significant effects did not cause any complications, however it did prevent meta- analyses from being conducted. Some attempts were made to gather this information, however the number of studies missing this information, made this task too difficult. Similarly, only a few studies conducted power analyses prior to the analyses, and of those that did, most were unpublished doctoral dissertations. Similarly, many of the studies conducted multiple analyses without controlling for the alpha level, risking a type 2 error occurring. To address these concerns, the current study only included articles that had a minimum sample size of 50; however, subsamples were significantly lower for some of the analyses.

The small samples used in the articles were most likely caused by the nature of the research being conducted. Most of the studies addressed secondary questions following on from a clinical trial and as such were limited to the sample size of the trial itself. Often, to address the issue of a smaller sample, researchers combined participants from multiple clinical trials and then published multiple papers, each examining a different predictor. Most studies attempted to specify where their sample came from, however it was not always clear, and in some cases the same sample, or parts of the same sample, was used multiple times examining the same predictor. In the current study attempts were made to exclude studies that used the same sample, however this was not always possible. While using a larger sample is of benefit when examining a single predictor, the reusing of a sample within multiple studies can hinder researchers in conducting more broad analyses such as systematic reviews and meta- analyses.

The results of the current study stress the importance of considering outcome definition when conducting these analyses. In the current study, a significant effect was more

likely to be found using an endpoint outcome, rather than a rate of change. This may be accounted for by both the type of measure used for the endpoint outcome, and also the nature of the outcome. Analyses conducted using an endpoint measure predominately relied on a clinical measure, in most cases the ADIS III or IV and the associated CSR rating. In these cases, the diagnosis and symptom severity has been made by a clinician who has been trained in conducting interviews and diagnoses these disorders in an objective manner. Similarly, within the studies examining rate of change, significant effects were more likely to be found using the clinician scores, with non significant results being found the least using parent and child self report measures. This finding emphases the importance of using diagnostic measures within research trials, especially as outcome measures, and their benefit as an objective measure of the child's diagnosis and symptom severity. The use of different measures of outcome was recently highlighted in a study by Compton et al. (2014), which included 488 children who received one of four conditions involving, placebo, medication, CBT, CBT and medication. Using an endpoint and continuous measure of outcome, their findings also highlighted the impact that the definition of treatment outcome can have on the results.

Alternatively, the differences found could be accounted for by the nature of the definition. The endpoint outcome compares all participants at the same time point and does not take into consideration the severity of their scores pre treatment. On the other hand, the rate of change scores looks at a child's improvement or response to treatment over time. The different results found when comparing these outcomes could suggest that while a particular predictor may determine whether a child still has a diagnosis at the end of treatment (i.e. an endpoint analysis), this predictor may not influence whether a child responds to the treatment (i.e. a rate of change analysis). As such, it may be of benefit for researchers in the future to

control for initial severity in their endpoint analyses to prevent this from confounding their results.

In line with the evidence that diagnostic measures are more effective at determining outcome, the use of a diagnostic measure as a predictor variable was associated with a significant effect. A significant negative effect was more likely to be found using a diagnostic measure for the comorbid depression, externalizing disorders, parental and maternal anxiety predictors, especially if they were used in conjunction with a clinical endpoint outcome. As such, these results highlight the importance of using diagnostic measures both as a measure of outcome and as a measure of the predictor.

Strengths, Weaknesses and Limitations

The current study only examined the impact of an individual predictor on treatment outcome, and did not include analyses of interactions between possible predictors. A few studies have conducted these types of analyses, predominately looking at the interaction between age and gender (e.g. Scully 2011), however these were beyond the scope of the current study. Similarly, the current study only examined pre treatment predictors of outcome, and excluded any examination of within treatment variables. It has been argued (La Greca, Silverman, & Lochman, 2009; March & Curry, 1998), however, that inclusion of both predictors and meditators (within treatment effects) is needed to fully understand which factors are important and should be examined concurrently.

In order to address concerns regarding the type 2 error rate, the current study only included studies with a sample size larger than 50. This arbitrary cutoff was consistent with (Nilsen et al., 2013), however Moncrieff et al. (2001) classified a subsample of 50 as ‘moderate’. It should be noted that a number of studies with samples between 45 and 50 were excluded, and their outcomes that may have affected the study’s outcomes. Additionally, the current study only examined pre treatment predictors and ratings. When examining the

influence of expressed emotion on treatment outcome, Angelosante (2007) determined that while pre-treatment ratings of expressed emotion did not significantly predict outcome, change in expressed emotion did; with results suggesting that parents who displayed a decease in expressed emotion predicted better treatment outcome. Similarly, Keeton et al. (2013) found that parental distress and parent rated family function improved only for treatment responders. It is therefore possible that changes in a parent's diagnosis or their parenting style may be a stronger predictor of outcome, rather than the actual variable itself, suggesting that a deeper understanding of how treatment works is needed.

It must also be noted that the current study did not control for treatment type, duration or other variations in treatment methodology, as there was not enough clarity within the articles themselves to effectively control for these variables. As such, differences between the treatment programs may have influenced the current finding. For example, Legerstee et al. (2010) found that females were more likely to retain diagnoses after stage two of a 'Stepped Care' program. These differences in treatment duration and structure may also account for some of the variation in findings, and further research is needed to examine the interaction between treatment characteristics and predictor variables.

Lastly, the current study grouped predictors based either on endpoint diagnosis, or rate of change. In either case, the outcome measure was related to a decrease in symptom severity, which, although important, may not be the only outcome of interest. Anxiety disorders are developed and maintained through genetic, behavioural and environmental factors, and it is possible that while a variable may not predict change in anxiety diagnosis, it may predict or influence another variable of interest. Future research would benefit from examining different definitions of outcome, not only in relation to predictors of treatment outcome, but also within clinical trials.

Future Research

With the exclusion of a few preliminary analyses, all studies examined predictors that have been previously determined to be a risk factor of a child developing an anxiety disorder. These predictors were then proposed to impact outcome as well. Similarly, research into predictors of treatment outcome for adults with an anxiety disorder have highlighted other variables that may be of interest (e.g. Steketee, 1993) and future research examining these predictors may be of benefit as well.

The current study also highlight the importance of developing a clear definition of a 'quality' analysis of predictors, similar to the CONSORT assessment for the quality of clinical treatment programs (Moher, 2001). Variables deemed to be important in differentiating the quality of the treatment program (e.g. sample size, power analyses, controlling the type II error rate, type of statistical analyses) were not always reported in the sample papers, preventing the researcher from being able to clearly assess the quality of the study. The development of a measure of quality assessment, and the use of this measure in designing, conducting and reporting these findings will allow for more conclusive findings in the future.

Similarly, researchers in the future would benefit from either including power analyses in their articles, or recruiting larger samples to ensure that their result has not been affected by a Type 1 or Type 2 error. By doing this, and also reporting all the relevant statistics, researchers can later conduct meta – analyses and meta – regressions in order to clearly examine what variables predict treatment outcome which can then be used to help design treatments for children who currently do not respond effectively.

Clinical and Theoretical Implications

The current study has implications both for the development and treatment of anxiety disorders. Although variables such as age, gender, are known risk factors for the development of anxiety (Rapee et al., 2009), our results strongly suggest that these factors do not affect treatment outcome. Alternatively, the early evidence of negative influence of parental psychopathology in treatment outcome reinforces the importance of this variable as both a risk and a maintain factor in childhood anxiety disorders (McLeod et al., 2007). Interestingly, the differences found between maternal and paternal psychopathology – namely that maternal anxiety and paternal depression is weakly associated with poorer outcome- suggests that mothers and fathers play a different role in the development and maintenance of childhood anxiety disorders. Recent research conducted by Bögels, Stevens, and Majdandžić (2011) have highlighted these differences, finding that a father's challenging behaviour is associated with lower childhood anxiety. It is therefore theoretically possible that a father who has depression may not be motivated to engage in these behaviours with their child, which may in turn increase the risk of the child developing an anxiety disorder, or maintaining their current disorder by preventing them from facing challenges (Bögels & Perotti, 2011). Research into the different roles of mothers and fathers is still recent and further research is required before any conclusions can be made.

In helping to design more effective treatments, results of the current study highlight the importance of focusing on understanding the child's symptoms and addressing them. As mentioned above, multiple, severe diagnoses are associated with poorer outcome, and there is a trend suggesting that the family environmental and parental psychopathology may influence the effects of treatment. As such, assessing children prior and then restructuring treatment dependent on these assessments may allow for the development of a more tailored treatment program.

Similarly, while treatment appears to be effective for treating children with GAD, current programs are not as effective for children with SP. Beidel, Turner, and Morris (1999) found that there was increased socially distressing situations and social isolation for children with social phobia. It may therefore be of benefit to adjust or enhance treatment for children with social phobia to allow for them to engage with their peers, perhaps within the school or social environment.

Lastly, as clinicians continue to monitor the progress of their clients throughout the duration of treatment, it may be of benefit for them to understand the impact that the psychometric measures themselves may have over time. The current study reiterates the importance of using multiples sources of information, and the different contributions that each source of information provides.

Conclusion

The current study reviewed the available literature examining all possible pre-treatment predictors of outcome. Results were grouped by outcome definition, and predictors that had had less than three analyses conducted were identified and highlighted as areas of future research. Results found that child demographic variables did not predict treatment outcome, while comorbid depression, higher symptoms severity and comorbid externalizing disorders were associated with poorer treatment outcomes. Results also found that maternal anxiety and parental psychopathology negatively affected treatment, although this finding was not consistent for maternal anxiety. The current study also showed that diagnostic measures, both as predictor variables and outcome variables, were the most effective at determining a significant effect. As such, the current study highlighted the importance of considering the form of outcome measurement used.

This study also showed the importance of developing a sound methodological framework for conducting analyses into predictors of outcome, as the studies reviewed often

lacked important information and highlighted some of the limitations of the current methodologies. It recommends the development of a quality assessment measure to allow researchers to gauge their research and ensure that the information needed to allow for meta-analyses and replication studies be provided. It also provided suggestions for researchers to apply these findings in a clinical setting and areas of future research.

Overall however, this study aimed to review the literature in order to provide guidance for researchers in optimising treatment and helping children who currently do not respond to treatment. This study has determined that some pre treatment predictors do impact outcome additional research into this area is required to allow for the development of more effective treatments.

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