Brief, Non-Pharmacological, Interventions for Pediatric Anxiety:

Meta-Analysis and Evidence Base Status

by

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ABSTRACT

There is a need to reinvent evidence-based interventions (EBIs) for pediatric anxiety problems to better address the demands of real-word service delivery settings and achieve public health impact. The time- and resource-intensive nature of most EBIs for youth anxiety has frequently been noted as a barrier to the utilization of EBIs in community settings, leading to increased attention towards exploring the viability of briefer, more accessible protocols. Principally, this research reports between-group effect sizes from brief-interventions targeting pediatric anxiety and classifies each as wellestablished, probably efficacious, possibly efficacious, experimental, or questionable. brief interventions yielded an overall mean effect size of 0.19 on pediatric anxiety outcomes from pre to post. Effect sizes varied significantly by level of intervention: Pre to post-intervention effects were strongest for brief-treatments (0.35), followed by brieftargeted prevention (0.22), and weakest for brief-universal prevention (0.09). No participant or other intervention characteristic emerged as significant moderators of effect sizes. In terms of standard of evidence, one brief intervention is well-established, and five are *probably efficacious*, with most drawing on cognitive and behavioral change procedures and/or family systems models. At this juncture, the minimal intervention needed for clinical change in pediatric anxiety points to in-vivo exposures for specific phobias (~3 hours), cognitive-behavioral therapy (CBT) with social skills training (~3 hours), and CBT based parent training (~6 hours, eight digital modules with clinician support). This research concludes with a discussion on limitations to available brief EBIs, practice guidelines, and future research needed to capitalize on the viability of briefer protocols in enhancing access to, and impact of, evidence-based care in the real-world.

TABLE OF CONTENTS

		Page
LIST OF	TABLES	iii
LIST OF	FIGURES	iv
CHAPT	ER	
1	INTRODUCTION	5
2	METHODS	12
3	RESULTS	22
4	DISCUSSION	29
REFERI	ENCES	

LIST OF TABLES

Table	Page
1.	Criteria for Study Selection and Evidence-Based Status
2.	RCTs Contributing to the Classification of Brief EBIs for Pediatric Anxiety 61
3.	Level of Support Designations for the Brief EBIs for Pediatric Anxiety
4.	Estimates of Pre to Post and Pre to Follow-Up Mean Effect Sizes
5.	Pre to Post Moderators of Effect Sizes
6.	Pre to Follow-Up Moderators of Effect Sizes

LIST OF FIGURES

Figure	Page
1.	Flowchart for Study Identification, Screening, and Inclusion
2.	Minimal EBI Effect Sizes for Pediatric Anxiety by Level of Intervention91

CHAPTER 1

INTRODUCTION

The past three decades of research show that non-pharmacological interventions are efficacious in the treatment and prevention of pediatric anxiety; however, few interventions are having public health impact (National Academies of Sciences, Engineering, and Medicine, 2017). There are several possible reasons for this need-toaccess gap, including complex organizational and systems-level factors (e.g., organizational culture, implementation climate; readiness for change; Aarons, Moullin, & Ehrhart, 2018; Aarons & Sawitzky, 2006; Powell et al., 2015). However, many could be related to the structural design features of most evidence-based interventions (e.g., number, length, and frequency of sessions, type of provider training and supervision. A number of studies have highlighted a misfit between the structure of evidence-based interventions for pediatric anxiety and the realities of youth, families, and community care providers. For instance, Ringle et al. (2015), Reid et al. (2017), and Salloum et al. (2016) indicated that: (a) the number and length of sessions is often incompatible with child and family schedules, (b) insurance inconsistently covers the cost of EBIs and outof-pocket expenses are high, (c) protocols are multi-component and complex, and (d) implementation often requires extensive provider and/or organizational resources (e.g., training, supervision, funding). And, because implementation of EBIs is often resourceintensive, these programs are increasingly limited to organizations in urban and higher resource areas, making them inconsistently accessible to youth and families, especially in rural or low-resource communities (Yancey, Glenn, Ford, & Bell-Lewis, 2018). As a result, when an evidence-based psychosocial intervention reaches the community,

provider-driven adaptations occur (e.g., as much as two-thirds of the intervention is adapted for real-word delivery; Rhoades, Bumbarger, & Moore, 2012), which probably compromise clinical outcomes (e.g., from deviations in protocol fidelity; Berkel et al., 2011).

To support evidence-based services in reaching the intended public health impact, a second wave of efficacy and effectiveness research has emerged and is focused on 'reinventing' evidence-based interventions (EBIs). More specifically, Brownson, Colditz, and Proctor (2018) defined reinvention as the modification of EBIs to maximize fit between interventions, the realities of delivery organizations, and the needs of end-users (e.g., service providers, youth, parents). One way in which EBIs could be reinvented is via a shift towards briefer, more streamlined protocols based on emerging empirical data indicating that longer treatments do not necessarily equate to superior clinical outcomes in pediatric populations, including those for anxiety. For instance, in a meta-analysis of 447 RCTs of psychological interventions targeting youth mental health problems (including 143 for anxiety), it was found that the magnitude of intervention effects was unrelated to the number of intervention sessions (Weisz et al., 2017). It was further reported that the number of treatment weeks was significantly and negatively related to the magnitude of ES at posttest, suggesting that as the total duration of intervention increases, intervention effects diminish.

With regards to pediatric anxiety specifically, Ost and Ollendick (2017) found initial support for brief, intensive, and concentrated protocols (referred to as brief hereafter) for the treatment of pediatric phobias. For pediatric anxiety, Ost and Ollendick only found four studies and thus cautioned against making strong conclusions regarding

2

efficacy or effectiveness of these brief protocols. Here, Ost and Ollendick operationalized interventions as *brief* if the intervention was at least 50% fewer total sessions versus standard treatments, *intensive* if the number of sessions and intervention duration was reduced versus standard treatment (e.g., one, 180 minute session treatment for specific phobia), and *concentrated* if the intervention had a standard number of sessions, but sessions were delivered in a shorter time period (e.g., 12 sessions of CBT delivered in 6 weeks). Using data corresponding to 23 RCTs, brief, intensive, and concentrated interventions produced a large overall within-group treatment effect sizes on anxiety outcomes at post-treatment (g = 1.50) and follow-up (M = 5.6 months; g = 1.53). Interestingly, overall effect sizes from the reinvented EBIs included in the Ost and Ollendick meta-analysis were more similar than different to those derived from typical length EBIs (e.g., 12 or more sessions). As such, reinvented EBIs that are briefer and more streamlined than typical length protocols might offer unique opportunities to positively affect population-level health outcomes.

This reinvention of typical EBIs into brief EBIs raises questions about the idea that there might be a minimal intervention needed for change (Glasgow et al., 2014). Such minimal interventions are intended to offer the smallest program dosage and intensity that can be delivered by an implementer with minimal expertise and resource set to achieve positive clinical outcomes. Ideally, these minimal interventions are theorized to work best by including only the strongest EBI elements, while considering design features that yield consumer friendly protocols (e.g., highly compatible with user needs, low application complexity). More specifically, and applied to EBIs for youth anxiety, a minimal intervention needed for change would include a minimal number of theoretical

3

and empirically based clinical elements (e.g., cognitive restructuring, exposure, relaxation) and incorporate the minimum intervention duration, or dosage, required to produce meaningful change. This minimal intervention would utilize the least trained and least expensive provider that can implement the intervention with quality and fidelity. In theory, this approach could provide a blueprint to guide EBI reinvention, potentially leading to briefer, more streamlined protocols for pediatric anxiety that are less costly and resource-intensive, more scalable and user-friendly, and better positioned for large-scale dissemination and implementation in real-world settings. The need for briefer, more streamlined interventions is consistent with recommendations established by frameworks in the dissemination and implementation science field. For instance, the Knowledge to Action framework, established by the Centers for Disease Control and Prevention (Wilson et al. 2011), emphasizes the need for translating evidence-based programs into more streamlined and consumer accessible versions to accelerate dissemination and implementation efforts. Likewise, the 'disruptive innovation' perspective outlined by Rotheram-Borus, Swendeman, and Chorpita (2012) recommends redesigning interventions to focus only on their most robust clinical features and meet the essential needs for the majority of consumers in order to enhance the public health impact of EBIs. Relatedly, the diffusion of innovations framework (Rogers, 2002; 2010) suggests innovations (like an EBI) are more likely to be adopted, and at a quicker rate, if they have high compatible with consumer needs and low application complexity. Similarly, the RE-AIM model suggests that EBIs are more likely to be delivered at broader scale and with higher quality under real-world conditions, if they are minimally intensive and include

only the elements necessary for implementation and clinical improvement (Harden et al., 2018; Kessler, Purcell, Glasgow, Kelsges, Benkeser, & Peak, 2012).

Since the initial work of Ost and Ollendick (2017), new randomized controlled trials (RCTs) targeting pediatric anxiety via brief, non-pharmacological, interventions have been published. In addition, about one third of these brief interventions deviate from cognitive and behavioral therapy theory, which is a positive step toward intervention innovation in pediatric anxiety. Armed with this knowledge, the time is ripe for shedding new light on progress made toward determining if there might be a viable minimal intervention for clinical change in the pediatric anxiety area. The focus on pediatric anxiety is important for several reasons. First, anxiety disorders are among the most prevalent psychiatric problems in youth, with rates ranging from 5% to 12% in children and as high as 31% in adolescents (Merikangas et al., 2010). Second, anxiety disorders are associated with impaired functioning across a number of areas, including academic achievement, social competence, and self-esteem (e.g., Ezpeleta, Keeler, Erkanli, Costello, & Angold, 2001). Third, without intervention, anxiety disorders often persist into adulthood and for some youth, are prospectively linked to later negative sequela, including clinical depression, suicidal ideation, conduct problems, and substance use disorders (Cummings, Caporino, & Kendall, 2013). Fourth, pediatric anxiety disorders carry a significant societal cost, exceeding \$17 billion per year, largely due to healthcare expenditures, school absenteeism, and lost productivity due to parents or primary caregivers missing work (e.g., therapy appointments, staying home with the child, medical visits) (Bodden, Dirksen, & Bogels, 2008; Chisholm et al., 2016). In this context, the relative financial costs of youth anxiety problems, combining absolute costs with

prevalence, are similar to conduct disorders and higher than those of autism spectrum disorders (Bui et al., 2017; Kilian, Losert, Park, McDaid, & Knapp, 2010).

In response to the public health impact of pediatric anxiety, substantial efforts have focused on developing, evaluating, and isolating several evidence-based interventions (EBIs) to treat these disorders. In 1998, Ollendick and King published the initial evidence-based status report of psychosocial interventions for phobic and anxiety disorders. This report largely focused on the classification of psychosocial interventions using criteria developed by Chambless and colleagues (Chambless et al., 1996, 1998; Chambless & Hollon, 1998) as part of a task force effort promoting the classification of treatments for disseminating interventions to practitioners, psychology training programs, consumers, and third-party payers (American Psychological Association, 1993). With treatment research focused on pediatric phobic and anxiety disorders using systematic diagnostic criteria just emerging, Ollendick and King identified just four randomized controlled trials (RCTs), relying mostly on single group treatment and case design studies with non-clinic-referred and undiagnosed youth. Based on their review, Ollendick and King concluded that imaginal and in vivo exposures and cognitive-behavioral procedures (with and without parents) were probably efficacious for childhood phobias and childhood anxiety disorders, respectively. A decade later, Silverman, Pina, and Viswesvaren (2008) published their seminal review and meta-analysis, updating the evidence-based status of psychosocial interventions for phobic and anxiety disorders in children and adolescents. Across 32 randomized trials, it was found that no treatment met the highest standard of evidence (i.e., well-established), but several treatment programs were identified as probably efficacious or possibly efficacious, with many drawing on

cognitive and behavioral therapy (CBT), exposure-based approaches, and family therapy models.

Recently, Higa-McMillan, Francis, Rith-Najarian, and Chorpita (2016) published an update to Silverman et al. (2008) that focused on examining progress made in securing psychological interventions for youth anxiety. In addition to expanding study selection criteria to include prevention and early intervention trials, Higa-McMillan et al. aggregated treatments by approach (e.g., exposure, CBT, parent training) rather than "brand name" (e.g., Coping Cat). More specifically, Higa-McMillan and colleagues used practice element profiling based on the work of Chorpita and Daleiden (2009) to identify the specific clinical elements (e.g., psychoeducation, relaxation, exposure) of the treatment protocols evaluated in each study. Then, to identify the 'treatment families' to be evaluated and classified using the Southam-Gerow and Prinstein (2014) guidelines, treatments were aggregated when they shared a majority of therapeutic elements with comparable theoretical foundations, while separating treatments with principally different theoretical underpinnings or unique clinical practices. Across 108 randomized trials published between 1967 and 2013, Higa-McMillan et al. (2016) found that six treatments met the *well-established* criteria, including CBT (with parent involvement and Sertraline) and behavior therapy (exposure, modeling), and produced an average within-group posttreatment effect size (ES) of 1.42. Eight treatments also were identified as probably *efficacious*, largely using CBT and/or family therapy approaches, with an average posttreatment ES of 1.13. Two treatments were classified as *possibly efficacious* and again used CBT-based approaches but did not report the necessary data for ES estimation (i.e., means and standard deviations for anxiety outcome variables). In addition, six treatments

were classified as *experimental* including psychodynamic therapy, rational emotive therapy, and biofeedback training, while eight treatments were classified as *questionable*, including teacher psychotherapy, psychoeducation, and Eye Movement Desensitization and Reprocessing (EMDR). Post-treatment effect sizes for *experimental* treatments averaged 0.93 and *questionable* treatments averaged 0.50. This expansion of empirically supported treatments for pediatric anxiety, including six *well-established and eight probably efficacious* treatments, highlights significant progress made since the prior evidence-based update report published ten years ago by Silverman et al. (2008).

Despite progress made in securing several efficacious treatments for pediatric anxiety, real-world access is strikingly low (each year, 60% to 80% of youth with anxiety and its related impairments never receive services; Beesdo, Knappe, & Pine, 2009; Kohn, Saxena, Levav, & Saraceno, 2003; National Academies of Sciences, Engineering, and Medicine, 2017). And, for youth who receive treatment services dropout rates are high and recovery rates, moderate. Specifically, youths attending an average of 25% of prescribed sessions (often 12 to 16 for typical pediatric anxiety protocols; Cummings et al., 2013; Weisz et al., 2017), while recovery rates of treatment completers tend to average 60% with remission falling by 10% over time (Barrett & Turner, 2004; Chavira et. al., 2004; Silverman et al., 2008). Given the substantial and significant problem of pediatric anxiety and identified limitations with treatment approaches, research efforts also have focused on advancing preventive interventions designed to interrupt the escalation of symptoms and the onset of anxiety disorders at the universal, selective, and indicated levels. Briefly, the Institute of Medicine classifies universal prevention as that which addresses the population at large; selective as that which targets those with an

elevated vulnerability; and indicated as that which focuses on those with disorder levels that are subsyndromal or high and projected to develop into diagnoses (O'Connell, Boat, & Warner, 2009). In the clinical child and adolescent area, the prevention of pediatric anxiety is as important as its treatment, particularly because prevention efforts appear to be promising in offering an additional avenue to maximize fit between the EBI, its possible delivery settings (e.g., schools, summer camps), and the needs of end-users (e.g., youth, caregivers, lay implementers) to reduce the burden posed by pediatric anxiety. In fact, several studies show that pediatric anxiety can be prevented via brief, nonpharmacological, interventions; and like most treatments, preventive interventions draw on cognitive and behavioral therapy theory. Also, as it should be anticipated, preventive effects on clinical outcomes take time to emerge and are expected to vary by level (e.g., universal vs. indicated) (Fisak, Richard, & Mann, 2011); while positive preventive effects also are relatively stable. For example, at the universal level, small effects are often found, followed by greater effects on clinical outcomes at the selective and then at the indicated prevention level. And, relative to treatment, prevention efforts offer strong promise in reducing anxiety symptom levels or risk for anxiety disorder development (e.g., effect sizes tend to range from 0.17 to 0.22 for indicated prevention and from 0.35 to 0.76 for treatment; Fisak et al., 2011; Reynolds et al., 2012; Weisz et al., 2017; Werner-Seidler et al., 2017). Moreover, given that preventive interventions are typically shorter in duration compared to treatments, delivered in non-clinical community settings (e.g., schools, after-school settings), and increasingly implemented by non-mental health professionals (teachers, behavior specialist), prevention offers another critical channel by

which EBIs could be disseminated and sustained at-scale for adequate public health impact.

As such, this research sought to achieve two principal objectives. First, this study aimed at quantifying (via meta-analysis) the magnitude of effects produced by brief, nonpharmacological interventions on pediatric anxiety outcomes. Within this aim, program effects were examined in the contexts of moderation, such that variations were expected as a function of two "families" of putative moderators relevant to intervention protocol (e.g., treatment versus prevention) and participant (e.g., youth age) characteristics. Second, this study aimed at evaluating the evidence-based status of brief, nonpharmacological interventions, in an effort to draw conclusions that would inform clinical practice.

In terms of hypotheses, relevant to the first aim, it was expected (and desired) that the interventions of focal interest herein would produce small to medium effect sizes on pediatric anxiety outcomes, which would be consistent with findings from meta-analyses of several brief non-pediatric anxiety interventions (e.g., Schleider & Weisz, 2017). Also expected (and desired) would be that larger effect sizes come from treatment, compared to prevention (indicated, selective, universal; Fisak et al. 2011; Reynolds et al., 2012). Clinically, one should desire large effects from treatment because this is the service often offered to youth that show the highest symptom levels and most severe impairment (empirically, higher risk levels tend to derive the earliest and greatest benefit from prevention; Spilt, Koot, & van Lier, 2013; Spoth, Trudeay, Redmond, & Shin, 2014; Verdurmen, Koning, Vollebergh, van den Eijnden, & Engels, 2014). In an exploratory way, other putative moderators (i.e., provider type, modality, theoretical approach, youth age, ethnicity representation, gender) were considered and tested in terms of its relation to effect size. In the past, few studies have shown moderation effects. Reynolds et al. (2012) reported larger effect sizes by delivery modality with individually-delivered cognitive and behavioral therapy (CBT) for adolescents showing greater effects than group-based CBT, non-CBT, or non-specific child-focused services (albeit formal subgroup analyses were not conducted to determine statistically significant variations). Fisak et al. (2011) reported larger effects for interventions delivered by mental health professionals compared to delivery by lay providers. This research did not uncover any other type of known moderator of response for interventions targeting pediatric anxiety. In terms of the second aim, it was anticipated that certain brief, non-pharmacological interventions could emerge as well-established or probably efficacious. For example, CBT (with and without parent involvement or medication), in vivo exposure, behavior therapy, and family therapy (e.g., CBT for child and parent) are already earmarked as well-established or probably efficacious in the treatment of pediatric anxiety. These levels of empirical support refer to typical protocols (Higa-McMillan et al., 2016); but since many reinvented EBIs (in both treatment and prevention) draw on the same efficacious components as typical protocols, some reinvented EBIs might already meet the well-established level (or at least probably efficacious), which would signal there is some level of readiness toward dissemination and implementation.

CHAPTER 2

METHODS

Study Search Procedure

The principal source to select each study for evaluation was a computer index search using a combination of PsychINFO and Web of Science. Parameter selections were: categories (psychology, psychiatry), topics (intervention, treatment, psychotherapy, training, modification), evaluation (clinical, randomized, comparison, effect, outcome), and population (child, youth, adolescent). In conjunction with selected parameter options, searches were carried out using combinations of the following keywords: anxiety, generalized anxiety, separation anxiety, social phobia, social anxiety, specific phobia, therapy, prevention, fears, phobic, worry, and panic. Auto-explode options were used in all computer searches to ensure that all relevant topics within the broader categories were included. The computer index search was supplemented with manual searches of studies cited in published meta-analyses, reviews, references in targeted studies citing other RCTs, and "in press" or "first online" studies recommended by the two search engines.

Study Selection

Studies were included for consideration based on the following criteria: (1) participants were selected and included in the original study on the basis of measures that had some degree of evidence of psychometric reliability (e.g., published Cronbach alpha reliability coefficients) due to: (1a) clinical anxiety as evidenced by diagnoses and/or symptoms, (1b) subsyndromal anxiety symptoms indicative of increased risk for disorder development, (1c) the presence of known risk factors associated with anxiety disorder development (e.g., parent with a diagnosed anxiety disorder), or (1d) universal prevention

efforts specifically targeting anxiety disorders; (2) the mean age of child participants was 18 years or younger; (3) participants did not have a co-morbid neurodevelopmental disorder (e.g., autism spectrum disorder) or significant externalizing disorder; (4) random assignment to conditions used; (5) at least one condition evaluated a nonpharmacological intervention with a maximum intervention "dosage" of 11 direct hours (including required booster sessions); and (6) condition protocols were clearly explained or specified. Studies of interventions involving medication only were excluded, as well as those focusing primarily on academic concerns, peer rejection or unpopularity, or "medical" problems (e.g., distress associated with a medical condition or procedure). Search efforts resulted 76 studies (including 26 published since Ost & Ollendick, 2017) meeting inclusion/exclusion criteria. Figure 1 shows the study search and study inclusion flowchart.

Study Coding Procedures

Studies were coded on variables relevant to quantitative characteristics used to calculate effect sizes. Anxiety outcome measures were included for effect size coding if the measure assessed anxiety symptoms, levels, or diagnoses using valid and reliable metrics (e.g., youth self-report, parent/caregiver report, behavioral observations, clinician assessments). For each eligible measure, the mean, standard deviation, and sample size was coded at each available assessment point.

Quantitative characteristics relevant to participant and intervention characteristics also were coded to evaluate effect size variations based on theory and published metaanalyses (Reynolds et al., 2012; Weisz et al., 2017; Schleider & Weisz, 2017). The variables were: sex (female), race/ethnicity (minority representation), age (in years), primary/targeted anxiety problem (specific phobia vs. mixed anxiety vs. another specific anxiety disorder), intervention level (treatment, targeted prevention, universal prevention) (the label name targeted is used to refer to selective and indicated prevention studies), focal intervention recipient or beneficiary (child, caregiver), primary delivery setting (research laboratory, community), provider (professional, non-professional or lay), training of providers (required vs. not required), supervision of providers (required vs. not required), supervision of providers (required vs. not required), intervention modality (individual, group, individual with digital supports, self-directed, digital only – e.g., attentional bias modification training), and comparator condition type (no-treatment or waitlist, placebo or attention control, another EBI). Two intervention design features relevant to dosage calculation also were coded: intervention length (the total time in weeks as defined by the intervention [e.g., 10 weeks]) and session duration, (average time per single session as defined by the intervention [e.g., 60 minutes]).

Quantitative data from measures assessing constructs of interest were entered into a Microsoft Excel (Microsoft Corp., Richmond, WA) database with algorithms programmed to calculate effect sizes. To ensure reliability, Lipsey and Wilson's (2001) recommendations were implemented. First, approximately three weeks following the original coding of the studies, 100% of the studies were re-coded in a separate database without access to the original coding file. Double-entered data were checked against the original database by an independent and trained rater. Data corresponding to sample and intervention variables were coded by two independent and trained raters and verified by the first author. Inter-rater reliability between the two coded databases was moderate to high (0.86 > ICCs > 0.94) for continuous effect size and moderator data and substantial to almost perfect ($0.79 > \kappa appas \ge 1.0$) for categorical variables.

Effect Size Calculation

To calculate between-group effect size estimates, the independent group pretestposttest (IGPP) procedure was used. The IGPP procedure allows for comparisons between two independent groups (e.g., intervention vs. control) on their corresponding mean change scores on some dependent measure using different standardized instruments. This increases confidence that observed differences are truly attributable to the intervention condition and not a result of nonspecific epiphenomenal factors (e.g., passage of time, attention; Hedges, 1982; Morris & DeShon, 2002). Between-group effect sizes were calculated for all anxiety outcomes from pre to post-intervention, as well as pre-intervention to follow-up using the following equation:

$$ES_{\text{IGPP}_g} = \left(\frac{\left(M_{Pre, Int} - M_{Post, Int}\right) - \left(M_{Pre, Cont} - M_{Post, Cont}\right)}{SD_{pooled, pre}}\right) * \left[1 - \frac{3}{4N - 9}\right]$$

In the first half of this equation, $(M_{Pre, Int} - M_{Post, Int})$ is the mean difference for the intervention group, $(M_{Pre, Cont} - M_{Post, Cont})$ is the mean difference for the control group, and $SD_{pooled, pre}$ is defined as:

$$SD_{pooled, pre} = \frac{(n_{G1} - 1)s_{G1}^2 + (n_{G2} - 1)s_{G2}^2}{(n_{G1} - 1) + (n_{G2} - 1)}$$

Here, n_{G1} is the number of subjects in the intervention group, n_{G2} is the number of subjects in the control group, s_{G1} is the pre-intervention standard deviation for the intervention group, and s_{G2} is the pre-intervention standard deviation for the control group. Pre-intervention standard deviations were used as they were measured before any intervention has happened and are thus more likely to be consistent across studies (Becker, 1988).

As noted by the second half of the IGPP equation, effect sizes were adjusted to yield Hedge's *g* estimates, which is recommended to account for the potential upward bias of Cohen's *d* effect sizes when based upon a collection of studies that include small sample sizes (e.g., N < 20; Lipsey & Wilson, 2001). Effect sizes also were weighted by the reciprocal of the standard error to account for differences among sample size and variances using the following equation (Lipsey & Wilson, 2001):

$$w_{sm} = \frac{1}{\left(\sqrt{\frac{n_{G1} + n_{G2}}{n_{G1}n_{G2}} + \frac{(ES_{\text{IGPP}_g})^2}{2(n_{G1} + n_{G2})}}\right)^2}$$

Finally, to calculate within-group effect sizes, the difference between pre and post-intervention (and follow-up) means was divided by the pre-intervention standard deviation, then adjusted for sample size bias using the Hedge's *g* correction. For both between- and within-group effect sizes reported herein, positive values reflect effects that are occurring in the expected direction (i.e., the intervention is related to the theorized clinical improvements) whereas a negative effect size value reflects continued clinical deterioration (i.e., the intervention is not related to the theorized clinical improvements). A 95% confidence interval was derived for each effect size aggregate and labeled using the standards suggested by Cohen (1988): small (0.20 or less), medium (about 0.50), and large (0.80 or over).

Meta-Analytic Procedure

Individual studies frequently reported multiple measures of anxiety outcomes. The inclusion of multiple measures per singular construct violates assumptions of independence that underlie meta-analysis (Rosenthal & DiMatteo, 2001). When this assumption is violated, sample sizes are inflated and standard error estimates are distorted, producing biased effect size estimates. To maintain assumptions of independence, and per the recommendation of Lipsey and Wilson (2001), multiple effect sizes for a single construct within studies were averaged prior to synthesis with effect sizes from other studies to ensure that each study only contributed a single effect size estimate.

In terms of statistical approach, random effects models were used to calculate overall weighted effect sizes. Unlike a fixed effect approach that assumes between-study differences are due to sampling error alone, random effects models assume that between-study differences are the result of both sampling error and other sources of variability (e.g., putative moderators, study design, random differences). In this way, random effects models enable broader generalizability of meta-analytic findings to populations beyond those under investigation (Cooper et al., 2009; Hedges, 1983; Rosenthal & DiMatteo, 2001). To confirm the a priori decision to use random effect model approach, and given the considerable methodological differences across studies, heterogeneity of effect sizes was determined using the *Q*-test.

$$Q = \sum_{i=1}^{k} W_i Y_i - \frac{\left(\sum_{i=1}^{k} W_i Y_i\right)^2}{\sum_{i=1}^{k} W_i}$$

The Q-test examines whether between-study variation could be completely explained by within-study sampling error or whether the variation among effect size values reflect real and important differences between studies. A significant Q-test value indicates there is significant variability in effect sizes beyond sampling error that should be examined via formal meta-moderation analyses. When the Q-test was significant, the I^2 statistic also was calculated:

$$I^2 = \frac{Q - (n-1)}{Q}$$

The I² statistic provides an estimate of the proportion of observed variance that reflects true differences among effect sizes, with guidelines suggesting that 25%, 50%, and 75% values represent low, moderate, and high levels of heterogeneity, respectively (Carpenter et al., 2018; Higgins, Thompson, Deeks, & Altman, 2003).

Validity Assessment

A byproduct of peer-reviewed publication standards and procedures is the "filedrawer problem," which suggests that published studies are more likely to report statistically significant results than unpublished studies (Rosenthal, 1991). As such, metaanalytic reviews may yield a systematic upward bias due to the omission of null findings such that effect sizes may not accurately represent the 'truth'. To address the file-drawer problem and potential publication bias, the fail-safe N (Rosenthal, 1991) was calculated using the following equation:

$$k_0 = \frac{K \left(K_Z^{-2} - 2.706 \right)}{2.706}$$

In this equation, K is the total number of studies assessing outcome or mediator variables in the meta-analysis and Z is mean effect size produced by the K studies. The

FSN represents the number of studies with a mean effect size of zero that would be needed to reduce an effect size to non-significance. Thus, the FSN approximates how resistant calculated effect sizes are to null effects.

Moderators of between-group effect sizes

To ascertain the potential moderators of between-group effect sizes, a series of analog to ANOVA tests were conducted using the International Business Machines' Statistical Package for the Social Sciences (IBM's SPSS) with the MetaF macros provided by Wilson (2003). This macro uses a mixed effect model to test for moderation, whereby studies within subgroups are pooled using random effects approaches, while tests of significance between subgroups are conducted using fixed effect approaches (Borenstein et al., 2009; Spinhoven et al., 2018). In addition, two approaches were taken to increase confidence in moderator findings. First, analyses were only conducted if the moderator category contained at least three studies per sub-group because meta-analytic moderation estimates have shown to be poor when the number of studies is very small (Weisz et al., 2017). Second, to maximize meta-analytic power in detecting significant moderation effects, separate analyses were conducted for each candidate moderator variable at each focal time point (i.e., pre- to post, pre to follow-up) (Raudenbush & Bryk, 2002; Van den Noortgate et al., 2013). To control for the increased possibility of Type I error due to the number of separate moderator analyses, a Holms-Bonferroni correction was applied. The Holms-Bonferroni adjusts *p*-values based on the total number of contrasts within a moderator family as to maintain experiment-wise error rates at a target alpha level while yielding more statistical power than a traditional Bonferroni correction. Briefly, and focusing on one moderator family at a time (intervention,

participant), analog-to-ANOVA tests were used (or meta-regression for continuous variables) to test for moderation. Then, the contrast with the smallest *p*-value was evaluated against an alpha of 0.05 divided by the total number of contrasts in the moderator family. If the contrast remained statistically significant, then the contrast with the next smallest *p*-value was tested using an alpha level of 0.05 divided by the number of remaining contrasts in the moderator family. This process continued until a nonsignificant difference within the family was observed; at which point, all remaining contrasts in the family were considered nonsignificant (for a more detailed discussion of the Holms-Bonferroni correction, see Jaccard and Guilamo-Ramos, 2002).

Dosage Calculation

For this research 'dosage' was calculated by multiplying intervention length by the product of session duration and session frequency. Session frequency was obtained by dividing total number of intervention sessions by intervention length. In addition, the average number of days in-between sessions was calculated by multiplying the inverse of intervention session frequency by seven (representing number of days in a week). From a dosage perspective, in-between session time could be considered an estimate of single dose duration or the time in which a single intervention dose (i.e., individual session) has the potential to have effects on targeted outcomes (via home practice assignments, for example). This approach to intervention dosage calculation is based on Berkel et al. (2011), Gierisch et al. (2010), and Glasgow et al. (2014).

Evidence Base Status Evaluation

Evaluation criteria adopted by the Society of Clinical Child and Adolescent Psychology (SCCAP; Southam-Gerow & Prinstein, 2014) from the guidelines originally articulated by Chambless and colleagues (Chambless et al., 1996, 1998; Chambless & Hollon, 1998) were used to determine the evidence-based status of brief, nonpharmacological, interventions for pediatric anxiety. Criteria are shown in Table 1. Using the M.1 to M.5 criteria listed in the table, two independent and trained evaluators rated the methodological robustness of each study, with coding verified by the first author. Initial agreement between evaluators was 98% with raters differing in their coding of the M.5 criterion for one study. The raters and first author discussed each discrepancy and then a 100% agreement between parties was reached.

Upon establishing the methodological robustness of each study, 'intervention families' were created to facilitate evidence-based status classification. Interventions were aggregated into distinct when they shared most therapeutic elements with comparable theoretical foundations and prescribed the same intervention dosage. When interventions had principally different theoretical underpinnings, they were separated into distinct intervention families. Distinct families also were created when interventions shared comparable theoretical foundations but prescribed different intervention dosage amounts. This approach was taken as to facilitate increased precision in identifying minimal intervention dosage thresholds necessary for clinically significant change in anxiety outcomes. Intervention families were then classified based on their empirical support into one of five "levels", including: well-established, probably efficacious, possibly efficacious, experimental, and questionable. Descriptions of each level, including evidentiary requirements, are presented in Table 1.

CHAPTER 3

RESULTS

Characteristics of Included Studies

The final sample of studies consisted of 76 RCTs, including 40 that were prevention focused (13 indicated, 4 selective, and 17 universal) and 36 treatment studies. The total sample size was 17,203 youth between the ages of 3 to 18 years (M = 10.61, SD = 2.75) with 55% being female. Twenty-five studies evaluated interventions with samples consisting mostly of Caucasian youth, while only three studies evaluated interventions with majority non-Caucasian samples (Ginsburg & Drake, 2002; Kato & Shumizu, 2017; Pina et al., in press). The remaining 48 studies did not report sample demographic information to ascertain ethnic minority representation. In terms of intervention characteristics, 114 independent intervention conditions were evaluated, with an overall average dosage of 6 direct hours (range = 1 to 10; SD = 2.64), delivered in about 8 sessions (range = 1 to 12; SD = 2.90), across 7.14 weeks (range = 1 to 16; SD = 3.47), with an average of 6 days between intervention sessions (range = 0 to 14; SD = 2.14). Approximately, 80% (n = 60) of interventions targeted pediatric anxiety broadly, without a focus on a particular disorder; of the remaining interventions, 13% (n = 10) targeted specific phobias and 8% (n = 6) focused on social anxiety. Most interventions were based on cognitive and/or behavioral therapy theory (63%), delivered by highly trained mental health professionals (67%), and largely focused on youth as the primary intervention recipient (91%). In terms of intervention modality, 48.5% (n = 32) of studies reported on protocols delivered in group format, 21.2% (n = 14) individual format, 21.2% (n = 14) used some form of technology in program delivery (e.g., internet, CD-ROMs, computer

games). About 14% (n = 9) of studies used attention-bias modification training (ABMt) protocols and 4.5% (n = 3) were bibliotherapy. With regard to comparator conditions, 53% (n = 41) of studies used a waitlist or no treatment control, 22% (n = 17) compared an intervention to an active control condition (e.g., attention control, education support), and 8% (n = 6) used another EBI as the comparator.

Included studies are summarized in Table 2. Column 1 names the investigatory team and publication date. Column 2 reports participants' characteristics (i.e., age, sex, clinical inclusion criteria) and randomization to condition and comparator. Column 3 describes significant program effects and condition comparison results. Finally, column 4 specifies Table 1 criteria relevant to the methodological robustness for each study. A total of 64 studies were considered methodological robust in that they met M.1 to M.5 criteria. Twelve studies did not meet M.5 criterion because the sample size per condition/comparator was too small to detect reliable effects (i.e., Attwood et al., 2012; Barrett et al., 2001; Dewis et al., 2001; Gallagher et al., 2004; Ginsburg & Drake, 2002; Hains, 1992; Muris et al., 1998; Muris et al., 2002; Sheslow et al., 1982; Stallard et al., 2011; Tillfors et al., 2011; Whiteside et al., 2015).

Effect Sizes Produced by Brief, Non-Pharmacological Interventions

Of the total sample of 76 studies, 64 reported data necessary to calculate betweengroup effect sizes (i.e., raw means and standard deviations for outcomes measured at a minimum of two time points for an intervention and comparator). Calculated effect sizes are reported in Table 4. In the table, column 1 corresponds to the level of intervention. Column 2 shows the number of studies that contributed to the effect size calculation, and column 3 indicates the meta-analytic sample size. Column's 4 through 7 report the weighted Hedge's *g* effect sizes of anxiety outcomes and corresponding effect size standard deviations, 95% confidence intervals, and fail-safe N. Finally, column 8 reports *Q*-test statistics relevant to heterogeneity among effect sizes.

Findings showed that brief treatments produced a pre to post effect size of 0.35 (SDg = 0.36) whereas brief prevention produced a pre to post effect size of 0.13 (SDg = 0.24). Within brief prevention, there was some variability. Indicated and selective prevention produced effect sizes of 0.22 (SDg = 0.24) and 0.31 (SDg = 0.25). Universal prevention produced an effect size of 0.09 (SDg = 0.19); from pre to post. Overall, fail-safe N calculations were generally robust; however, the fail-safe N for selective prevention was low (FSN = 2) suggesting that selective prevention effects would likely be altered by the presence of studies producing null effects. The fail-safe N for selective prevention could be related to the small number of included studies (n = 4).

Results for pre to follow-up effect sizes were based on the 30 studies that reported follow-up data with continuation assessments ranging from 1 month to 4 years postintervention. The meta-regression showed that follow-up duration did not account for significant differences in effect sizes (Q = 2.19 [1, 116], p = .14); accordingly, effects were averaged across assessments such that each study in the meta-analysis contributed a single estimate of pre to follow-up effects (M = 8.88 months; SD = 8.76). As shown in Table 4, the effect size for brief anxiety treatments decreased slightly (approximately 0.05 *g*-units) from pre-intervention to follow-up suggesting that these interventions are mostly maintaining effects overtime rather than showing growth in clinical outcome gains. In contrast, prevention produced an overall effect size from pre-intervention to follow-up and outcome gains. In contrast, prevention produced an overall effect size from pre-intervention to follow-up of 0.22 (SD = 0.23) representing an increase of 0.09 *g*-units from pre to postintervention effects. This suggests some growth in clinical outcome gains over time. It is important to highlight that the 0.09 g-unit increase appears to be largely driven indicated and universal prevention trials, not selective prevention efforts. Indicated prevention produced a pre to follow-up effect size of 0.30 (SD = 0.29), universal prevention produced a pre to follow-up effect size of 0.17 (SD = 0.17), and lastly, selective prevention produced a pre to follow-up effect size of 0.21 (SD = 0.10).

Moderators of Effect Sizes Derived by Brief, Non-Pharmacological Intervention

There was moderate to high degree of heterogeneity and thus moderation analyses were conducted as planned. That is, heterogeneity statistics indicated a significant degree of between-study variance in effect sizes from pre to post ($Q_w = 189.60$ [63], p < .001) and pre to follow-up ($Q_w = 91.94$ [29], p < .001). I² statistics indicated that 46.80% to 70.30% was true between-study variation; pre to follow-up effect sizes had I² rates of 68.40% to 73.77%. Tables 5 and 6 present findings for all analog-to-ANOVA moderation tests using pre to post intervention and pre to follow-up effects sizes.

From pre to post-intervention, only level of intervention emerged as a significant moderator of effect sizes ($Q_b = 28.11, p < .001$), such that treatment yielded larger effect sizes (g = 0.26; 95% CI 0.16 to 0.37) than targeted prevention (g = 0.21; 95% CI 0.12 to 0.30). Universal prevention (g = 0.09; 95% CI -0.05 to 0.23) effects were non-significant. In terms of the levels of intervention tested as targeted prevention, analyses revealed no significant differences in pre to post effect sizes between indicated and selective prevention ($Q_b = 0.32, p < .57$). However, there was a significant difference in pre to post effect sizes between targeted and universal prevention ($Q_b = 3.84, p < .05$). None of the participant and no other intervention level characteristic emerged as significant moderators of pre to post or pre to follow-up effect sizes.

Evidence-Based Status of Brief, Non-Pharmacological Interventions for Pediatric Anxiety

As shown in Table 3, in vivo exposure treatment (in one/single session with 3 intervention hours) for specific phobias met the well-established criteria. Five brief interventions met the probably efficacious criteria: CBT (9 to 10 hours), clinician supported digital parent training (6 hours), behavioral therapy (8 hours), and exposurebased CBT with social skills training (3 hours). Seventeen interventions were classified as possibly efficacious: CBT with or without parents (8 hours), clinician supported digital CBT (3 to 7 hours), and clinician supported bibliotherapy (3 to 5 hours). Also, possibly efficacious was ABMt: training away from threatening stimuli (1,920 trials across 4 sessions) and training toward positive stimuli (640 trials across 4 sessions). There is a handful of additional brief interventions meeting the possibly efficacious criteria (include stress inoculation training; growth mindset training, psychoeducation in-vivo exposures in 1.5 hours, and emotive imagery for specific phobias). Eleven interventions were classified as experimental. Neurofeedback with exposure and attention training (5 hours), as well as biofeedback interventions with relaxation training (6 hours) as delivered via immersive computer video games were classified as experimental because they produced significant within-group reductions in pediatric anxiety outcomes, but no between-group comparisons against active controls. Eye movement desensitization and reprocessing (EMDR) (2.5 hours) was found to be questionable because it was not better (or equivalent) when compared to in-vivo exposures.

26

Minimal Interventions Needed for Change in Pediatric Anxiety Outcome

Between-group, pre to post-intervention effect sizes were calculated for wellestablished and probably efficacious brief interventions along with the corresponding dosage scores (from number of sessions, length of session, frequency of sessions). Figure 2 illustrates the data. On the y-axis the between-group, pre to post-intervention effect sizes, are plotted. On the x-axis, level of intervention (treatment, targeted prevention, universal prevention) is plotted. The figure also references published meta-analysis effect size data from typical length interventions for pediatric anxiety.

In terms of treatment, as shown in the figure, approximately 3 hours of intervention for a specific phobia is able to produce a mean effect size of 0.36 (SD = 0.43; 95% CI = 0.08 to 0.80; k = 5) when in-vivo exposures are implemented. In addition, about 7 hours of CBT for pediatric anxiety disorders is able to produce a mean effect size of 0.50 (SD = 0.52; 95% CI = 0.02 to 0.97; k = 6). As shown in the figure, these effect sizes are below the average effect that can be obtained from a typical length EBI, <u>but</u> the upper confidence levels for these brief treatments fall within the confidence level range reported in the literature for typical length EBI (per Reynolds et al. 2012 meta-analysis). Focusing on this overlap, it might be the case that some youth could benefit from a brief EBI as much as youth who receive the typical length treatment for pediatric phobias and anxiety. With regard to pre to follow-up (about 5 months), the mean effect size for CBT was 0.33 (SD = 0.47; 95% CI = 0.04 to 0.71; k = 3). There was an insufficient amount of data (i.e., more than one study) to calculate pre to follow-up effect sizes for the other treatments (parent training, behavior therapy, in vivo exposures).

Turning to prevention, targeted (indicated and selective) efforts are able to produce to produce a mean effect size of 0.41 (SD = 0.30; 95% CI = 0.13 to 0.69; k = 6) from about 8 hours of cognitive and behavioral intervention. Interestingly, these 8 hours of EBI services are producing almost twice the effect size as typical length protocols (per Fisak et al., 2011 meta-analysis). In contrast, the effect size for universal prevention was small (0.09, SD = 0.15; 95% CI = 0.01 to 0.18; k = 12) and less than the corresponding universal prevention effect from typical length protocols. The pre to follow-up (about 9 month) effect size for targeted prevention was 0.45 (SD = 0.36; 95% CI = 0.08 to 0.82; k= 5) and for universal it was 0.20 (SD = 0.17; 95% CI = 0.04 to 0.35; k = 7; an increase of 0.08 g-units from pre to post-intervention).

CHAPTER 4

DISCUSSION

In the clinical child and adolescent psychology area, significant strides have been made to situate EBIs in community practice settings (Powell et al., 2015; Lyon & Koerner, 2016). Our research augments the knowledge-based by showing that a newwave of brief, non-pharmacological, interventions for pediatric anxiety might reach community practice settings. Some brief interventions for pediatric anxiety are now wellestablished or probably efficacious and demonstrate significant clinical changes, indexed by effect sizes resulting from our meta-analysis. Thus, the hypothesis that brief interventions produce small to medium effect sizes on pediatric anxiety outcomes was confirmed for one well-established intervention: in-vivo exposures for the treatment of specific phobias and four probably efficacious interventions: child focused exposurebased CBT with social skills training for indicated preventions and early intervention, CBT (no social skills training), behavior therapy, and parent focused training

Ollendick and King (1998) and Higa-McMillan et al. (2016) indicated that in-vivo exposures for specific phobias is an empirically supported treatment but data now show that significant positive change can be achieved in as little as one session (~3 hours; Ost et al., 2001; Ollendick et al., 2009). Silverman, Pina, and Viswesvaren (2008) and Higa-McMillan et al. reported that CBT for anxiety disorders is an empirically supported treatment but data now show that significant positive change can be achieved in as few as six sessions for indicated prevention and early intervention (~3 hours), and in as few as eight parent training sessions for anxiety treatment (~6 hours, delivered digitally with clinician support) (Donovan & March, 2014; Morgan et al., 2017). Similar to past research, we found larger effect sizes (ES) for treatment compared to prevention (indicated, selective, universal; Fisak et al. 2011; Reynolds et al., 2012) but results suggested that streamlining pediatric anxiety treatments into briefer formats might lead to declines in clinical effectiveness (e.g., ES: ~0.70 for typical length treatments; Reynolds et al., 2012; compared to 0.35 for brief-treatments). Such declines appear to be less apparent when it comes to prevention (ES: 0.17, Fisak et al., 2011; 0.13 for briefprevention); however, our findings from prevention require replication with additional studies because the fail-safe N estimates were low, and most protocols were universal or indicated.

At this juncture, the evidence suggests there are minimal interventions for clinical change: in-vivo exposures for specific phobias (~3 hours, one session), CBT with social skills training (~3 hours, six sessions for indicated prevention and early intervention, and parent training (~6 hours, eight digital modules with clinician support) (Donovan & March, 2014; Morgan et al., 2017; Pina et al., in press; Ost et al., 2001; Ollendick et al., 2009; Weersing et al., 2017). Importantly, brief interventions appear robust in the context of moderation. Only brief treatments produced larger effects than brief-prevention, which is desirable because diagnosed youth often show the largest breach between normal and clinical anxiety. No other candidate mediator variable was statistically significant. The null-moderation findings might be explained by low statistical power (only 44 studies or 58% reported data to afford moderation) and study design restrictions (little variability from data corresponding to controlled efficacy trials).

Moving forward, efforts should focus on facilitating the uptake of brief interventions in community practice settings. A major concern that needs to be addressed

is the gap between the architecture of brief-intervention and insurance system coverage. For instance, some brief interventions are designed to be intensive (e.g., one session lasting several hours) but providers only can submit service claims corresponding to onehour session per day. Another way to help facilitate uptake is to determine if briefinterventions are cost-effective and thus attractive to insurance companies or other organizations paying for the services. Clinically, facilitating the uptake of brief interventions in community practice settings might translate into informing providers about which intervention components could be augmented in dosage to achieve stronger clinical outcomes. Unfortunately, dosage augmentation is often linked to knowledge of mediators, which is scant. No study relevant to a well-established or probably efficacious brief-intervention has tested mediation using an analytic approach that involves temporal order (a requirement for inferring causality; Kraemer et al., 2002; Carper, Makover, Kendall, 2018). There are some data pointing to factors that might have potential mediation effects and thus could serve as targets for dosage augmentation. For instance, in Pella et al. (2016), changes in parental modeling of anxiety (at post and 6-month follow-up) emerged as a significant mediator between 8-sessions of family-based CBT and child anxiety severity at 1-year follow-up. In another study, Liu et al. (2018) found that changes in threat processing (indexed as ventrolateral prefrontal cortex (vlPFC) activation) mediated the relation between a single-session intervention (attention bias modification training in 320 digital trials - away from threat) and anxiety symptoms at immediate post-intervention. In Ollendick et al. (2017), changes in harm beliefs and coping efficacy emerged as significant mediators between one session (3 hours) of invivo exposures for specific phobia and changes in clinician severity ratings of anxiety

symptoms from pre to post and pre to 6-month follow-up. In addition, modifying anxious cognitions and improving coping self-efficacy could be targets for dosage augmentation as these two factors have emerged as likely mediators of typical length CBT interventions for pediatric anxiety based on findings from several RCTs (Treadwell & Kendall, 1996; Kendall & Treadwell, 2007; Lau et al., 2010; Pereira et al., 2018; Kendall et al., 2016; Maric et al., 2013; Essau et al., 2012; Spence et al., 2017); however, this would need to be established via additional research efforts.

Limitations and Future Directions

While the present dissertation advances knowledge regarding the viability and evidence-based status of brief, reinvented EBIs for pediatric anxiety, several limitations are important to consider when interpreting findings. First, as with all reviews, findings are limited to the studies included in the analyses. The inclusion criteria of the present research focused on identifying (and evaluating) published RCTs that met methodological indicators of robustness articulated by Southam-Gerow and Prinstein (2014) (e.g., utilization of treatment manuals, clearly delineated target population). This is particularly relevant when considering the current evidence-base of digital health interventions, an important avenue that will likely play an integral role in addressing need-to-access gaps in mental health services, have largely relied on non-randomized designs (e.g., open trials, time-series, match-control designs). Second, most studies included in the present research (86%) evaluated interventions under controlled, efficacyfocused conditions versus real-world, effectiveness conditions. This is important because intervention-related effect sizes often decrease when EBIs are transported into real-world conditions (Fixsen et al., 2018). Third, the evaluation and classification of identified

brief, non-pharmacological interventions focused on anxiety outcome measures without a systematic examination of the efficacy of the protocols in terms of performance in improving program targets (e.g., cognitions) or comorbid symptoms or disorders (e.g., depression). While this approach enabled a greater degree of generalizability, the tradeoff was less specificity of findings beyond anxiety outcome variables. Thus, a key avenue of future investigation is a deeper examination of these more specific and granular outcomes. Fourth, effect sizes were based on an aggregate of anxiety outcome measures that met a minimum criterion of psychometric strength (i.e., at least one documented psychometric property [e.g., reliability coefficients]). However, measurement error attenuates study-level effect sizes and can lead to incorrect conclusions regarding the presence of moderating variables and magnitude of summary effect sizes. Correcting for measurement error is crucial for obtaining a true picture of the stability of effect sizes across studies (Schmidt & Hunter, 2014). Thus, because of this lack of reporting, the impact of variability in measurement reliabilities on summary effect sizes is unknown. Fifth, this research did not examine, in depth, whether intervention effects varied by anxiety disorder type (e.g., GAD, SoP, SAD) because more than 60% of studies focused on youth with mixed or heterogenous anxiety problems. However, anxiety disorders are highly comorbid with themselves such that youth diagnosed with one disorder are more likely to experience other anxiety disorders (Higa-McMillan et al., 2016). More specifically, homotypic comorbidity rates between anxiety disorders in youth populations range from 33% to 67% (Bennett et al., 2013). Nevertheless, considering the role of comorbid anxiety disorders within intervention efforts is likely to be important as to facilitate the personalization of interventions to meet individual needs. Finally, while pre

to follow-up effect sizes were able to be calculated, these should be considered, with 30 studies including at least one follow-up assessment, most (75%) were 12 months or less. Thus, the long-term impact and durability of intervention effects (> 12 months) of these briefer protocols represents an important future research direction.

Research Recommendations

Moving forward, we must address important questions so that brief evidencebased psychosocial interventions can be adopted and sustained in real-word settings. Based on the findings from the present dissertation, below are several factors that the next generation of intervention research should consider when developing, evaluating, and disseminating brief, and reinvented treatment and prevention programs for pediatric anxiety.

1. Increased systematic evaluation of reinvented EBIs, including in the context of typical protocols. Effect sizes derived from brief EBIs for pediatric anxiety are smaller than those reported in published meta-analyses evaluating protocols of typical length (but are largely within effect size ranges). Although briefer protocols are producing smaller effect sizes, they may be more efficient in producing significant improvements in anxiety symptoms and diagnostic recovery based on dosage. For example, the dosage for typical length treatments often ranges from 12 to 16 hours compared to versus brief treatments that range from 3 to 10 hours. By reducing the amount of time to implement, for instance, the greater the service capacity available in community settings, as providers would be able to serve more youth and families in shorter amount of times. In addition to exploring this possibility, future research also should evaluate reinvented against

34

typical length efforts to gain a more precise understanding as to the difference or equivalence of program effects (e.g., using non-inferiority randomized designs). This also includes longer-term follow-up assessments (> 12 months) in order to gain a better sense as to the durability of effects obtained by brief, reinvented efforts.

2. Move beyond efficacy testing with increased attention towards external validity, real-world implementation, and use of novel designs. An important step in supporting the adoption and sustainment of evidence-based psychosocial interventions in real-world settings is to start moving beyond efficacy studies. Efficacy trials adhere to generally stringent inclusion/exclusion criteria and tend to rely on interventionists (e.g., graduate students, clinical psychologists) serving under high levels of fidelity to manuals and emphasize internal validity. In this way, efficacy evaluations embody significant barriers in the provision and access of evidence-based care in community settings and observed effects may be representative of what might be obtained under real-world conditions. At this point, this research advocates for focused efforts to move intervention research serving anxious youth and their families into effectiveness settings and to implement novel treatment designs (e.g., hybrid trials; Curran et al., 2012) to ascertain real-world effects of reinvented EBIs. Effectiveness trials and hybrid designs emphasize external validity (few inclusion and exclusion criteria), lessen geographical and transportation restrictions in the provision of care (Yancey, Glenn, Bell-Lewis, & Ford, 2012), and capitalize on the typical infrastructures of the community, such as primary care, schools, neighborhood clinics, emergency

rooms, and child welfare (Chavira et al., 2014; Asarnow et al., 2005). In effectiveness settings, providers generally have diverse experiences and expertise (guidance counselors, behavior specialists, social workers, parent liaisons, promotores/as) and have experience working with different types of youth and families (Durlak, 2015; Grumbach & Mendoza, 2008). Moreover, real-world evaluations of reinvented EBIs with minimal inclusion/exclusion criteria might enable more diverse and heterogeneous samples of youths and families to inform potential adaptations. For example, in the present research, only three trials reported included a significant degree of ethnic minority representation and none tested moderation by ethnicity/race. Moreover, similar to concerns outlined by Polo et al. (2018) and Pina et al. (2019), Asian American and Native American youth are still largely absent from the treatment outcome literature in general, and almost non-existent in brief EBIs for anxiety. For these reasons, effectiveness research offers rich opportunities for advancing intervention science for anxious and phobic youth, while providing opportunities for increased understanding as to how these protocols could be made more robust and amenable to the unique needs of youth and families.

3. *Examine dose-response relations*. The present research represents an important step towards identifying minimal dosage thresholds for well-established and probably efficacious interventions. However, dosage recommendations reported herein represent mean estimates that do not take into account individual dosage needs. As such, future research should systematically evaluate relations between intervention dosage and youth responsiveness as demonstrated by gains in clinical

outcomes. Knowledge derived from these efforts could provide more specific insight relevant to dosage levels that are optimal for change. Understanding dosage thresholds, for example, could enable the use of dynamical and adaptive interventions that are responsive to individual needs, strengths, and contexts. In this way, deeper examinations of intervention dose-response effects could identify ways in which protocols could be adapted to align with personalized medicine initiatives (Riley et al., 2014).

4. Increase understanding of how reinvented, brief EBIs for pediatric anxiety are achieving effects (i.e., mediators). Knowledge of intervention-related mechanisms of change is important as it may help improve the precision of established interventions, guide new intervention models, enhance measurement tools, refine psychosocial change theories, and inform practice efforts in the absence of validated intervention packages (Holly, Stoll, Rapp, Pina, & Chavira, 2018). Illustratively, interventions could become more precise in affecting planned outcomes by amplifying the critical components or mediators, while minimizing or removing unsuccessful components. In this way, mediator-driven interventions could help augment short- and long-term program effectiveness, while yielding streamlined interventions that are more amenable for large-scale, sustainable dissemination in real-world practice settings. Out of the 76 studies included in the present research, just six conducted formal mediation analyses. Of those, only one reported significant finding from a test of mediation that used a robust analytic approach that accounted for temporal order (Pella et al., 2016), a critical requirement for classifying a mediator as a causal mechanism between

interventions and planned outcomes (Kraemer et al., 2002; Carper, Makover, Kendall, 2018). The remaining studies that evaluated mediation utilized concurrent or cross-sectional mediation design, where the mediator variable was assessed concurrently with the outcome variable and precludes the possibility of elucidating the precise sequence of changes that might establish temporal precedence. The limited scope of mediation, however, is not due to a lack of available data. Specifically, most interventions in the present research included clinical content linked to two significant mediators of typical length protocols for pediatric anxiety (changing anxious cognitions and improving coping selfefficacy). And, 50% (n = 38) of studies assessed change in at least one candidate mediator variable. Collectively, this represents a considerable number of missed opportunities to evaluate mediation effects of brief interventions for pediatric anxiety. Thus, future RCTs should more frequently measure theory-driven mediators, be responsive to timing of change in mediators in relation to assessment (as guided by theoretical model), and test mediation using robust analytic strategies such as the product of coefficients method using bias-corrected bootstrapped and asymmetric confidence interval (see MacKinnon, 2008; Toglifi & MacKinnon, 2011). Better measurement and analysis of the supposed mediators underlying changes in intervention outcomes would assist in identifying successful and unsuccessful portions of treatment and prevention efforts (MacKinnon et al., 2013). This information could help determine which intervention components and targets are crucial for changes in planned outcomes, allowing reinvented EBIs to be made more precise and streamlined.

5. Improve readiness of interventions for real-world implementation. Despite the EBIs evaluated in the present research being briefer in terms of planned dosage (as compared to typical length protocols), other structural characteristics need to be systematically examined to gain insight into other design features of brief EBIs that need to be reinvented to be more usable, scalable, and sustainable. For example, briefer EBIs likely have greater potential for reaching individuals not currently served or those not being well served (with evidence-based practices) and scalability by function of their brevity. However, because most of the identified EBIs have evaluated under highly controlled efficacy conditions, other design features need to be examined and likely reinvented to be positioned to reach public health impact. For example, 66% of the interventions evaluated in the present research were delivered by highly trained service providers (e.g., doctoral students, psychologists, counselors) with extensive pre-service training (10 or more hours) and on-going supervision or clinical consultation (usually on a weekly basis for 60 to 90 minutes). While training and supervision or consultation efforts are important in ensuring, as best as possible, high quality and fidelity of implementation, most current variants also are resource-intensive (e.g., financial, infrastructure) and limit the capacity of potential providers, along with potential reach and scalability (Aarons et al., 2012; Rogers, 2002; Lyon & Koerner, 2016). Thus, to leverage the potential benefits of briefer interventions, additional attention to other structural and design features is necessary to ensure the protocols being reinvented are responsive to issues relevant to readiness for largescale dissemination and implementation.

Concluding Comments

Looking ahead, the future is bright for briefer, more streamlined EBIs for pediatric anxiety. There is reason for optimism, given the number of well-established and probably efficacious brief, non-pharmacological interventions for anxious that are available to clinicians and other service providers, expanding intervention options noted by Higa-McMillan et al. (2016). It is important to keep in mind, however, that earmarking a psychosocial intervention as well-established does not necessarily translate into policy or readiness for adoption, sustainability, or scaling-up. As articulated by Elliott and Mihalic (2004), Fagan and Mihalic (2003), Flay, Biglan, Boruch, Gonzales Castro, Gottfredson, et al. (2005), Greenberg et al. (2005), and Fixen et al. (2018), there need to be resources for adequate dissemination and implementation. Initial resources may include high quality training, monitoring and technical assistance, and disclosures about costs (e.g., staff training, on-site time, space, equipment, reproduction of materials; Chatterji et al., 2001; Foster et al., 2003; Lyon, Stanick, & Pullman, 2017). Over time, there needs to be ongoing communication between researchers and providers about the conditions under which the intervention is working, maintenance of program effects, booster sessions, availability of innovative efficiencies (e.g., digital health tools), and regular cost-benefit evaluation reports. Therefore, scaling-up evidence-based psychosocial interventions that are brief and thus more sustainable means having essential armamentaria in place so that anxious children and their families can live productive and happy lives.

At the same time, it also is important to consider the pipeline in which these protocols are currently being developed, evaluated, and disseminated. It takes, on average, 17 years for evidence-based programs to become "certified" as efficacious and considered for broad diffusion (Balas & Boren, 2000; Rotheram-Borus, Swendeman, & Chorpita, 2012); largely due to the research process. That is, becoming certified as efficacious often includes two years for development and pilot testing, five-years for an efficacy trial (evaluation under ideal or controlled conditions), an additional five-year efficacy trial (for intervention refinement and replication), and then five more years for effectiveness testing (under real-world conditions) (Rotheram-Borus et al., 2012; Schoenwald & Hoagwood, 2001). And, beyond the point of being deemed efficacious, there exists little to no infrastructure to disseminate EBIs at a large enough scale to achieve intended public health impact in ways that are responsive to the needs and realities of youth, families, providers, and delivery organizations. Thus, if we aim to realize the public health promise of evidence-based practices and reduce the incidence and prevalence of anxiety disorders in sustainable manner, prioritizing these research directions would likely greatly expand the availability and accessibility of EBIs, thus benefiting more youth, families, and communities.

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Design and Methods (M)

M.1. Group design: Study involved a randomized controlled design

M.2. Independent variable: Intervention was manualized or logical equivalent

M.3. **Population:** Specified problems based on clearly delineated inclusion/exclusion criteria

M.4. **Dependent variable:** Reliable and valid measures used to ascertain outcomes M.5. **Analyses:** Appropriate analytic approach with sufficient sample size to detect effects

Evidence-Based Status Criteria (Level 1 to 5)

Well-Established (Level 1)

Effects demonstrated on most primary outcomes by showing:

1.1a. Statistically significant superiority to pill, psychological placebo, or another active intervention

OR

1.1b. Equivalent (or not significantly different) to an already well-established intervention,

AND

1.1c. In at least two independent research settings and by two independent investigatory teams, AND

1.2. M.1 to M.5

Probably Efficacious (Level 2)

Effects demonstrated for the intervention by showing:

2.1. Statistically significant superiority to a waitlist or no intervention control, in at least two good experiments,

OR 2.2. Well-Established criteria except for 1.1c AND 2.3. M.1 to M.5

Possibly Efficacious (Level 3)

Effects demonstrated for the intervention by showing:

3.1 Statistically significant superiority to a waitlist or no intervention control, in at least one experiment,AND

3.2. M.1 to M.5

OR

3.3 Statistically significant effects, in at least two clinical studies, with two or more studies meeting the M.2 to M.5.

Experimental (Level 4)

Effects demonstrated for the intervention by showing:

4.1. Statistically significant effects, but not tested in an experiment OR

4.2. Statistically significant effects, in at least one experiment, but no sufficient to meet Level 3 criteria

Questionable (Level 5)

Effects demonstrated for the intervention by showing:

5.1. Inferiority to another intervention, waitlist, and/or control, OR

5.2. No beneficial effects.

Notes: Relevant to R.1, Kazdin and Bass found that a sample size of 12 per condition, with treatment vs. no-treatment main effect comparisons, yielded large effects while intervention versus placebo main effect comparisons yielded small to medium effects. Criteria from Silverman and Hinshaw (2008), Division 12 Task Force on Psychological Interventions' reports (Chambless et al., 1996, 1998), Chambless and Hollon (1998), and Chambless and Ollendick (2001). Editorial revisions were made to the M criteria and Levels 1 to 5 to expand scope of interventions from treatments to also include preventive interventions; however, classification of interventions remained unaffected by our editorial revisions.

Study	Sample Characteristics	Significant Program Effects	Criteria
	Anxiet	y (<i>n</i> = 66)	
Hains (1992) [1]	N = 25. Ages 15 to 16 years. Mean age not reported. 0% girls. Randomized to individual plus group cognitive- behavioral stress management intervention (CBSMI), individual plus group anxiety management training (AMS), or waitlist control.	Intervention conditions did not differ from one another. At posttest and 3-week FU, CBSMI and AMS led to lower STAIC state anxiety, STAIC trait anxiety, STAXI trait anger, STAXI state anger, and RCADS depression than control.	M.1 to M.4
Kiselica et al. (1994) [2]	$N = 48.9^{\text{th}}$ grade students. Mean age not reported. 46% girls. Top 12 highest scores in classroom on STAIC-T anxiety. Randomized to stress inoculation training or usual care.	At posttest and 1-month FU, stress inoculation training led to lower STAIC trait anxiety than usual care. No other between- group differences emerged on SOSI stress or GPA. Stress inoculation training led to lower SOSI stress at posttest and FU.	M.1 to M.5
Barrett et al. (2000) [3]	N = 20. Ages 14 to 19 years ($M = 16.30$). Former Yugoslavian Refugee in Australia. Randomized to group cognitive behavior therapy (GCBT) or waitlist control.	At posttest, GCBT led to reduced YSR anxiety/ depression than control. GCBT led to lower YSR internalizing, SCAS anxiety, and ASP cognitive styles but between-group differences were not evident.	M.1 to M.4
Barrett et al. (2001) [4]	N = 204. Ages 7 to 19 years ($M = 12.50$). Non- English-speaking Background in Australia. Randomized to GCBT or waitlist control.	At posttest, GCBT led to more improvements in BHS future outlook/hopelessness, SEI school self-esteem, RSES self-esteem, and reductions in RCMAS anxiety than control.	M.1 to M.5
Ginsburg & Drake (2002) [5]	N=12. Ages 14 to 17 years ($M=15.60$). 83% girls. DSM GAD, Specific Phobia, SoP, SAD. Randomized to GCBT or attention control.	At posttest, GCBT led to lower ADIS-C/P CSR severity and SCARED-C anxiety than control. GCBT led to lower SAS-A social anxiety, but between-group differences were not evident.	M.1 to M.4

Table 2. RCTs Contributing to the Classification of Brief EBIs for Pediatric Anxiety

Heyne et al. (2002) [6]	N = 61. Ages 7 to 14 years (M = 11.50). 46% girls. Met school refusal criteria as defined by Berg et al. (1969) and DSM GAD, Specific Phobia, SAD, SoP. Randomized to ICBT, ICBT plus parent teacher training (ICBT+PTT), or parent/teacher training (PTT) only.	At posttest, ICBT+PTT and PTT led to higher school attendance than ICBT. ICBT+PTT also led to lower CBCL internalizing symptoms than ICBT at posttest. In addition, PTT led to significantly less FSSC-R fear of the unknown, RCMAS worry and oversensitivity, and RCMAS physiological anxiety than ICBT. Additional between-group differences were not evident for any outcome at posttest or 4.5- month FU. All conditions led to lower FSSC-R fears, RCMAS anxiety, CDI depression, and higher SEQSS self-efficacy at posttest. At FU, ICBT+PTT led to lower FSSC-R fear and RCMAS worry/ oversensitivity, ICBT led to lower FSSC-R fear, RCMAS physiological complaints and higher SEQSS self-efficacy. At FU, 69% (across all conditions) no longer met ADIS-C/P CSR diagnostic criteria for any anxiety disorder.	M.1 to M.5
Muris et al. (2002) [7]	N = 30. Ages 9 to 12 years ($M = 10.00$). 43% girls. RCADS \geq 9 (boys) or 11 (girls) anxiety symptoms and DSM GAD, Specific Phobia, SAD, SoP. Randomized to ICBT, emotional disclosure treatment (ED), or no treatment control.	At posttest, ICBT led to lower RCADS anxiety, RCADS depression, STAIC trait anxiety than control and ED. At posttest, RCADS anxiety recovery rates were 80% in ICBT vs 40% in ED and 30% in control.	M.1 to M.4
Gallagher et al. (2004) [8]	N = 23. Ages 8 to 11 years. Mean age not reported. 52% girls. DSM SoP. Randomized to GCBT or waitlist control.	At posttest, GCBT led to lower CBCL anxiety/ depression than control. At 3-week FU, GCBT led to lower SPAI-C social anxiety, RCMAS anxiety, CDI	M.1 to M.4

		depression, and CBCL anxiety/depression than control. No other between-group differences emerged on SASC-R social anxiety, CBCL social competence, CBCL activities, or CBCL school problems. Both GCBT and control led to lower SASC-R social anxiety at posttest and FU.	
Bernstein et al. (2005) [9]	N = 61. Ages 7 to 11 years (M = 9.00). 66% girls. 53% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to GCBT, GCBT plus parent training, or no-treatment control.	At posttest, 3-, and 6-month FU, GCBT and GCBT plus parent training led to lower MASC-P anxiety, SCARED-P anxiety, ADIS-C/P CSR severity, and CGI functioning than control. Stronger pre to posttest changes were found for GCBT plus parent training than GCBT for CGI functioning. At 12-month FU, GCBT and GCBT plus parent training led to lower CSR severity ratings than control, with improvements in CGI functioning being maintained at 3-year FU (Lee et al. 2016).	M.1 to M.5
Mifsud & Rapee (2005) [10]	N = 425. Ages 8 to 11 years ($M = 9.5$). 59% girls. RCMAS ≥ 18 RCMAS anxiety symptoms. Randomized to GCBT or waitlist control.	At posttest and 4-month FU, GCBT led to lower RCMAS anxiety, SCAS anxiety, SCAS-P anxiety, CATS automatic thoughts, and TRF emotional and behavioral problems than control.	M.1 to M.5
Rapee et al. (2005) [11]	N = 146. Ages 3 to 5 years ($M = 3.9$). 55% girls. STSC-approach subscale > 30. Randomized to parent- education intervention or assessment only control.	Between-group differences were not evident for any outcome at posttest. Both parent-education intervention and control led to improvements in STSC-C/P temperament, TABC-R-P temperament, and behavioral inhibition measured via approach tasks. At 12-month FU, parent- education intervention had fewer	M.1 to M.5

		ADIS-C/P CSR anxiety disorder diagnoses than control. At 11- year FU, girls in the parent- education intervention had lower SCAS-P anxiety, CALIS-C life interference, and fewer ADIS- C/P anxiety disorder diagnoses than control and boys in the intervention condition (Rapee et al., 2013).	
Dadds & Roth (2008) [12]	N = 734. Ages 3 to 7 years. Mean age not reported. 47% girls. Randomized to parent-focused ICBT or no intervention control.	At posttest, parent-focused CBT led to lower teacher reported SCBE child anxious-withdrawn and angry-aggressive behaviors and higher SCBE social competence than control. Between group differences were not evident at 7-month FU.	M.1 to M.5
Aune & Stiles (2009) [13]	N = 1,439. Ages 12 to 14 years. Mean age not reported. 52% girls. Randomized to GCBT or no intervention control.	At posttest, GCBT led to lower SPAI-C social anxiety and SCARED anxiety than control.	M.1 to M.5
	N = 190. Ages 12 to 14 years. Mean age not reported. 52% girls. Post- hoc subsample. SPAI-C \geq 18 social anxiety symptoms. Randomized to GCBT or no intervention control.	At posttest, GCBT led to lower SPAI-C social anxiety and SCARED anxiety than control.	M.1 to M.5
Calear et al. (2009) [14]	N = 1,477. Ages 12 to 17 years. Mean age not reported. 56% girls. Randomized to internet- delivered CBT (iCBT) or waitlist control.	At posttest and 6-month FU, iCBT led to lower RCMAS anxiety than control.	M.1 to M.5
Ginsburg (2009) [15]	N = 40. Ages 7 to 12 years ($M = 8.94$). 45% girls. Parents met DSM criteria for GAD, Specific Phobia,	At posttest, 6-month, and 12- month FU, family-focused ICBT led to lower ADIS-CSR severity and SCARED-P anxiety than	M.1 to M.5

	SAD, SoP. Randomized to parent-focused ICBT or waitlist control.	control. At 12-month FU, family- focused ICBT led to fewer ADIS anxiety disorder diagnoses than control (0% vs. 30%).	
Hunt et al. (2009) [16]	$N = 260.9^{\text{th}}$ grade students. Mean age and sex not reported. 43% girls. RCMAS >1 SD above mean of a normative sample. Randomized to GCBT or assessment only control.	Between-group differences were not evident for any outcome at posttest, 2-year FU, and 4-year FU. Both GCBT and control led to lower RCMAS anxiety at 4- year FU.	M.1 to M.5
Balle & Tortella- Feliu (2010) [17]	N = 92. Ages 11 to 17 years. Mean age and sex not reported. CASI > 80 th percentile for anxiety sensitivity. Randomized to GCBT, waitlist, or no intervention control.	Between-group differences were not evident at posttest. Both GCBT and control conditions led to lower CASI anxiety sensitivity and SCAS-P anxiety symptoms. At 6-month FU, GCBT led to lower CASI anxiety sensitivity than controls.	M.1 to M.5
Khanna & Kendall (2010) [18]	N = 49. Ages 7 to 13 years ($M = 10.10$). 33% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to computer- assisted CBT (CCAL), ICBT, or computer-assisted attention control.	Intervention conditions did not differ from one another. At posttest and 3-month FU, CCAL and ICBT led to lower ADIS-C/P CSR severity than control. No other between-group differences emerged on CGAS functioning, MASC-C anxiety, or CDI-C depression. CCAL and ICBT demonstrated reductions in MASC-C anxiety and CDI-C depression at posttest.	M.1 to M.5
Miller et al. (2010) [19]	N = 118. Ages 7 to 12 years ($M = 9.75$). 50% girls. Randomized to GCBT or waitlist control.	Between-group differences were not evident for any outcome at posttest. Both GCBT and control led to lower MASC anxiety and BASC-IC internalizing symptoms.	M.1 to M.5
	N = 33. Ages 7 to 12 years ($M = 9.75$). 50% girls.	At posttest, GCBT led to lower MASC anxiety than control.	M.1 to M.5

	Post-hoc subsample. MASC anxiety > 56. Randomized to GCBT or waitlist control.		
Pahl & Barrett (2010) [20]	N = 263. Ages 4 to 6 years ($M = 4.56$). 48% girls. Randomized to GCBT or waitlist control.	At posttest, GCBT led to lower BIQ-T behavioral inhibition and higher BERS-T social-emotional competence than control. No other between-group differences emerged on PAS anxiety, BIQ-P behavioral inhibition, or BERS-P social-emotional competence. GCBT led to lower PAS anxiety, BIQ-P behavioral inhibition, and higher BERS-P social-emotional competence at posttest and 12- month FU.	M.1 to M.5
Bar Haim et al. (2011) [21]	N = 34. Age 10 years ($M = 10.10$). 71% girls. SCARED > 50 th percentile for anxiety symptoms. Randomized to attention bias modification training (ABMt) or attention control training (ACT).	At posttest, ABMt led to higher rates of disengagement from threat and were less vulnerable to the stress induced by the stressor task than ACT. No additional between-group differences were evident, with both ABMt and ACT having led to lower STAIC trait anxiety at posttest.	M.1 to M.5
Miller et al. (2011a1) [22]	N = 191. Ages 9 to 12 years ($M = 10.1$). 48% girls. MASC anxiety <i>T</i> - score of \geq 56. Randomized to GCBT or attention control.	Between-group differences were not evident for any outcome at posttest, 5-month, or 17-month FU.	M.1 to M.5
	N = 42. Ages 9 to 12 years ($M = 10.1$). Post-hoc subsample. MASC anxiety <i>T</i> -score of > 65. Randomized to GCBT or attention control.	Between-group differences were not evident for any outcome at posttest, 5-month, or 17-month FU.	M.1 to M.5

Miller et al. (2011a2) [23]	N = 253. Ages 9 to 12 years ($M = 9.80$). 54% girls. Randomized to GCBT or attention control.	Between-group differences were not evident for any outcome at posttest or 17-month FU.	M.1 to M.5
	N = 64. Ages 9 to 12 years ($M = 9.80$). Post-hoc subsample. MASC anxiety <i>T</i> -score of > 65. Randomized to GCBT or attention control.	Between-group differences were not evident for any outcome at posttest or 17-month FU.	M.1 to M.5
Miller et al. (2011b) [24]	N = 553. Ages 9 to 12 years ($M = 9.77$). 50% girls. Randomized to GCBT or waitlist control.	Between-group differences were not evident. At posttest and 3- month FU, GCBT led to lower MASC anxiety.	M.1 to M.5
Stallard et al. (2011) [25]	N = 20. Ages 11 to 16 years. Mean age and sex not reported. Seeking services at Tier 3 Child and Adolescent Mental Health Services with DSM GAD, Specific Phobia, SAD, SoP or mild-moderate depression. Randomized to computer-assisted CBT or waitlist control.	No between-group analyses were conducted. Both computer- assisted CBT and control led to improved RSEI self-esteem and SQC cognitive schemas. Computer-assisted CBT also led to lower SCAS social phobia SDQ-P emotional difficulties, SDQ-P hyperactivity, AWS depression, and improved SDQ-P total strengths and difficulties. Control also led to lower SCAS physical injury fears.	M.1 to M.4
Tillfors et al. (2011) [26]	N = 19. Ages 15 to 21 years ($M = 16.50$). 89% girls. DSM SoP. Randomized to iCBT or waitlist control.	At posttest and 1-year FU, iCBT led to lower SPSQ-C social anxiety, LSAS-RS social anxiety, BAI anxiety, and MADRS-S depression than control.	M.1 to M.4
Attwood et al. (2012) [27]	N = 13. Ages 10 to 12 years ($M = 10.6$). 0% girls. Randomized to computer- assisted CBT or computer gaming control.	At posttest, computer-assisted CBT led to lower SCAS anxiety, SCAS social anxiety, and SCAS generalized anxiety than control.	M.1 to M.4
Eldar et al. (2012)	N = 40. Ages 8 to 14 years ($M = 9.84$). 45% girls.	At posttest, ABMt led to greater reductions in dot-probe task	M.1 to M.5

[28]	DSM GAD, Specific Phobia, SAD, SoP. Randomized to ABMt away from threat, placebo attention control training using ABMt stimuli, or placebo attention training using neutral stimuli.	attentional bias and ADIS-IV- C/P anxiety symptom counts compared to controls. At posttest, 33% of children in the ABMt condition no longer met ADIS- C/P CSR diagnostic criteria for any anxiety disorder, compared to 13.3% in the placebo condition, and 0% in the neutral placebo condition. No other between-group differences emerged on SCARED-anxiety or CDI-C depression, with all three conditions having led to lower SCARED-C anxiety and CDI-C depression.	
Ginsburg et al. (2012) [29]	N = 32. Ages 8 to 12 years ($M = 10.28$). 63% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to modular ICBT or usual care.	Between-group differences were not evident for any outcome at posttest and 1-month FU. Both modular ICBT and usual care led to lower ADIS-C/P CSR anxiety severity, SCARED-C anxiety, and SDQ emotional difficulties, and higher CGAS functioning.	M.1 to M.5
McLoone & Rapee (2012) [30]	N = 152. Ages 7 to 12 years ($M = 9.8$). 62% girls. 10% of SCAS scores for their age group. Randomized to school- based GCBT, bibliotherapy w/o clinician support, or waitlist control.	At posttest and 12-month FU, school-based GCBT and bibliotherapy w/o clinician support led to lower SCAS-P anxiety and CALIS life interference than control.	M.1 to M.5
Thirlwall et al. (2013) [31]	N = 194. Ages 7 to 12 years. Mean age not reported. 52% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to full guidance parent- delivered CBT, brief guidance parent-delivered CBT, or waitlist control.	At posttest, full guidance parent- delivered CBT led to lower CAIS-P interference and SMFQ low mood than brief guidance parent-delivered CBT and control. At posttest, participants in both treatment conditions were 85% more likely to have recovered from their ADIS-C/P CSR principal anxiety disorder	M.1 to M.5

		than control, with full guided parent-delivered CBT leading to higher diagnostic recovery (50%) than brief guided parent- delivered CBT (39%). ADIS-C/P CSR diagnostic recovery rates at 6-month FU were comparable for both treatment conditions (76% for full guided, 71% for brief guided). Improvements for the intervention conditions were maintained at 3- to 5-year FU (Brown et al., 2017).	
Waters et al. (2013) [32]	N = 37. Ages 7 to 13 years ($M = 9.60$). 65% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to ABMt- attention-towards-positive (ABMt-ATP) or ABMt attention training control (ABMt-ATC).	At posttest, ABMt-ATP led to lower ADIS-C/P CSR severity and higher attentional bias towards positive stimuli (via dot- probe task) than control. At posttest, 50% in ABMt-ATP no longer met ADIS-C/P CSR diagnostic criteria for principal anxiety disorder compared to 8% in control. No other between- group differences emerged on SCAS anxiety, SCAS-P anxiety, or CES-DC depression, with both conditions having led to lower SCAS anxiety, SCAS-P anxiety, and CES-DC depression at posttest.	M.1 to M.5
Collins et al. (2014) [33]	N = 317. Ages 9 to 10 years. Mean age not reported. 45% girls. Randomized to psychologist-led GCBT, teacher-led GCBT, or usual care.	Intervention conditions did not differ from one another at posttest. At posttest and 6-month FU, psychologist-led GCBT and teacher-led GCBT led to lower SCAS anxiety, CSI avoidance coping, and higher CSI problem solving coping and CSI social coping than control. At 6-month FU, psychologist-led GCBT led to lower SCAS anxiety than teacher-led GCBT and control, whereas teacher-led GCBT led to	M.1 to M.5

		lower CSI avoidant coping was lower in teacher-led GCBT than psychologist-led GCBT and control.	
Donovan & March (2014) [34]	N = 52. Ages 3 to 6 years ($M = 4.08$). 54% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to parent focused iCBT or waitlist control.	At posttest, 6-month, and 12- month FU, iCBT led to lower PAS anxiety, CBCL internalizing, and higher CGAS functioning than control. At posttest, ADIS-C/P CSR diagnostic recovery rates were 39.1% in iCBT and 25.9% in control; with iCBT ADIS-C/P CSR recovery rates improving to 70.6% in iCBT at 12-month FU.	M.1 to M.5
Stallard et al. (2014) [35]	N = 1,362. Ages 9 to 10 years. Mean age not reported. 51% girls. Randomized to school staff-led GCBT, health facilitator-led GCBT, or personal social and health education (PSHE) control.	At 12-month FU, health facilitator-led and school staff- led GCBT led to lower RCADS- C anxiety than control, with more pronounced changes occurring for those in school staff GCBT. At FU, health facilitator GCBT led to lower RCADS separation anxiety than school staff GCBT and control (Skryabina et al., 2016).	M.1 to M.5
	N = 99. Ages 9 to 10 years. Mean age not reported. Post-hoc subsample. ≥ 49 on RCADS anxiety. Randomized to school staff-led GCBT, health facilitator-led GCBT, or personal social and health education (PSHE) control.	Between-group differences were not evident for any outcome at posttest or 3-month FU. Both school staff-led and health facilitator-led GCBT led to lower RCADS-C anxiety.	M.1 to M.5
Wong et al. (2014) [36]	N = 976. Ages 14 to 16 years. Mean age not reported. 70% girls. Randomized to iCBT for anxiety, iCBT for depression, or usual care.	Intervention conditions were not different from one another. At posttest, iCBT for anxiety led to reductions in GAD-7 generalized anxiety than control. iCBT for depression led to reductions in	M.1 to M.5

PHQ-5 depression than usual care.

Rodgers & Dunsmuir (2015) [37]	N = 62. Ages 12 to 13 years. Mean age not reported. 69% girls. Randomized to GCBT or waitlist control.	At posttest and 4-month FU, GCBT led to lower SCAS and SCAS-P anxiety than control. No other between-group differences emerged on CRS school adjustment, with both GCBT and control having led to improvements in CRS school adjustment.	M.1 to M.5
Ginsburg et al. (2015) [38]	N = 136. Ages 6 to 13 years ($M = 8.70$). Parents met DSM criteria for GAD, Specific Phobia, SAD, SoP. Randomized to family-focused ICBT or information-monitoring control.	At posttest, 6-month, and 12- month FU, family-focused ICBT led to reduced ADIS-CSR severity than control. At 12- month FU, family-focused ICBT led to fewer ADIS anxiety disorder diagnoses than control (5.26% vs. 30.65%). Over time, family-focused ICBT also led to lower SCARED-P anxiety, CBCL anxiety/depressive symptoms, and CBCL behavior problems than control (Pella et al., 2016).	M.1 to M.5
Waters et al. (2015) [39]	N = 59. Ages 6 to 17 years ($M = 8.75$). 53% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to ABMt or waitlist control.	At posttest and 6-month FU, ABMt led to lower ADIS-C/P CSR severity, SCAS-P anxiety, SMFQ-P strengths and difficulties, CBCL internalizing, and higher CGAS functioning than control. At posttest, 35% no longer met ADIS-C/P CSR diagnostic criteria for principal anxiety disorder for ABMt vs. 7% in control.	M.1 to M.5

Whiteside et al. (2015) [40]	N = 14. Ages 7 to 14 ($M = 10.20$). 71% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to ICBT or parent-coached exposure therapy (PC- Exp).	At posttest and 3-month FU, PC- Exp led to lower CBCL internalizing, CBCL externalizing, PARS anxiety, ADIS CSR severity, CGI severity, SCAS anxiety, SCAS-P anxiety, CSDS-P disability, and CATS negative cognitions than ICBT.	M.1 to M.4
Calear et al. (2016a) [41]	N = 1,767. Ages 12 to 18 years ($M = 14.86$). 75% girls. Randomized to school supported iCBT, health service supported iCBT (e-GAD HS), or waitlist control.	Intervention conditions did not differ from one another. At posttest and 6-month FU, school supported iCBT led to lower SAS-A social anxiety, GAD-Y generalized anxiety, and higher WEMWBS psychological wellbeing than control. At 6- month FU, e-GAD HS led to higher WEMWBS wellbeing than control. No between-group differences were evident for any outcome at 12-month FU.	M.1 to M.5
Calear et al. (2016b) [42]	N = 225. Ages 12 to 18 years ($M = 14.86$). 75% girls. Randomized to school-based iCBT or waitlist control.	No between-group differences were evident for any outcome at posttest or 3-month FU.	M.1 to M.5
Fitzgerald et al. (2016) [43]	N = 130. Ages 15 to 18 years ($M = 15.94$). 57% girls. SPAI-C ≥ 24 . Randomized to ABMt or attention control training (ACT).	Between-group differences were not evident at post-test or 4- month FU. Both ABMt and ACT led to lower SPAI-C social anxiety and SCARED anxiety.	M.1 to M.5
Infantino et al. (2016) [44]	N = 24. Ages 5 to 11 years ($M = 7.46$). 54% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to audio- based CBT or waitlist control.	At posttest and 3-month FU, audio-based CBT led to lower ADIS-C/P CSR severity, SCAS anxiety, and SCAS-P anxiety than control. At posttest, ADIS- C/P CSR diagnostic recovery rates for principal disorder were 58.3% for audio-based CBT and 16.7% for control. At FU,	M.1 to M.5

		diagnostic recovery rates for audio-based CBT was 66.67%.	
Morgan et al. (2016) [45]	N = 51. Ages 3 to 6 years ($M = 4.75$). 49% girls. STSC > 30. Randomized to parent-focused iCBT with clinician support or parent- focused iCBT without clinician support.	Intervention conditions did not differ from one another. At posttest, both conditions led to lower PAS-R anxiety, SDQ emotional difficulties, OAPA number of child anxiety diagnoses, and CALIS-PV life interference of anxiety.	M.1 to M.5
Pergamin- Hight et al. (2016) [46]	N = 67. Ages 6 to 18 years ($M = 12.67$). 57% girls. DSM SAD. Randomized to ABMt or ACT.	Between-group differences were not evident. Both ABMt and ACT led to lower ADIS-C/P social anxiety severity, SPAI-C social anxiety, dot probe response latencies, and higher dot probe accuracy at post and 3- month FU.	M.1 to M.5
Pophilat et al. (2016) [47]	N = 206. Ages 6 to 9 years. Mean age not reported. 51% girls. Randomized to GCBT or usual care control (health education classes).	At posttest, GCBT led to lower SCAS-P anxiety. No other between-group differences emerged. Both conditions led to lower ACES emotional skills.	M.1 to M.5
Ruttledge et al. (2016) [48]	N = 709. Ages 9 to 13 years ($M = 10.83$). 51% girls. Randomized to GCBT or usual care control (health education classes).	A posttest, GCBT led to improved BSC-Y self-concept, CES coping efficacy, and SCS school connectedness. GCBT also led to lower SCAS anxiety, however change was not significantly different than control.	M.1 to M.5
Scholten et al. (2016) [49]	N = 139. Ages 11 to 15 years ($M = 13.27$). 65% girls. SCAS ≥ 1 SD above the mean total anxiety symptoms or ≥ 1 SD above the mean on two SCAS subscales. Randomized to Biofeedback w/ relaxation	Linear change of the top scoring SCAS subscale from pretest to posttest to FU was greater for Dojo than control. No between- group differences emerged for SCAS total anxiety.	M.1 to M.5

	training video game (Dojo) or control video game.		
Schoneveld et al. (2016) [50]	N = 136. Ages 7 to 13 years ($M = 9.95$). 55% girls. SCAS ≥ 1 SD above the mean total anxiety symptoms or ≥ 1 SD above the mean on two SCAS subscales. Randomized to neurofeedback w/ exposures and ABMt video game (Mindlight) or control video game.	Between-group differences were not evident at posttest and 3- month FU. Both Mindlight and control led to lower SCAS and SCAS-P anxiety.	M.1 to M.5
Vigerland et al. (2016) [51]	N = 93. Ages 8 to 12 years ($M = 10.10$). 51% girls. DSM GAD, PD, SAD, SoP, or Specific Phobia. Randomized to internet- delivered CBT with therapist-support or waitlist control.	At posttest, internet-delivered CBT led to lower ADIS-C/P CSR severity, SCAS-P anxiety, and improved CGAS functioning than control, with ADIS-C/P CSR severity and CGAS functioning improvements maintained at 3-month FU. No other between-group differences emerged on SCAS anxiety or QOLI-C quality of life. Internet- delivered CBT led to reduced SCAS anxiety and QOLI-C quality of life at posttest.	M.1 to M.5
Ahlen et al. (2017) [52]	N = 695. Ages 8 to 11 years ($M = 9.60$). 48% girls. Randomized to GCBT or waitlist control.	Between-group differences were not evident for any outcome at posttest and 12-month FU.	M.1 to M.5
Cobham et al. (2017) [53]	N = 61. Ages 7 to 14 years ($M = 9.30$). 49% girls. DSM Specific phobia, SAD, GAD, or SoP. Randomized to parent- focused GCBT or waitlist control.	At posttest, 3-, 6-, and 12-month FU, parent-focused intervention led to lower ADIS-C/P CSR severity, SCAS-P anxiety, SCAS anxiety, and CBCL internalizing than control. At posttest, 64.5% in parent-focused intervention were free from principal anxiety disorder vs. 16.2% in control based on ADIS-C/P CSR	M.1 to M.5

		criterion. Parent-focused intervention ADIS-C/P CSR recovery rates improved to 84% at 12-month FU.	
Creswell et al. (2017) [54]	N = 136. Ages 5 to 12 ($M = 9.21$). 53% girls. Referred to mental health clinic for anxiety-related impairments. Randomized to brief guidance parent- delivered CBT or solution focused brief therapy (SFBT)	Between-group differences were not evident for any outcome at posttest or 3-month FU. Both brief guidance parent-delivered CBT and SFBT led to higher CGI improvement scores and lower KFQ-C fears, SCAS anxiety, SCAS-P anxiety, and ADIS-C/P CSR.	M.1 to M.5
De Voogd et al. (2017) [55]	N = 108. Ages 11 to 19 years ($M = 14.45$). 67% girls. SCARED > 16 anxiety symptoms and/or CDI > 7 depressive symptoms. Randomized to internet-based visual search ABMt (VS-ABMt), VS placebo-training, or no intervention control.	At posttest, VS-ABMt led to lower EVST attentional bias and Recognition Task interpretation bias than controls. No other between-group differences emerged on SCARED anxiety, CDI-C depression, RSES self- esteem, PTQ perseverative thinking, and SDQ-P strengths and difficulties, with all conditions having led to improvements in these outcomes at posttest and 6-month FU.	M.1 to M.5
Kato & Shumizu (2017) [56]	N = 74. Ages 8 to 9 years. Mean age not reported. 43% girls. Randomized to GCBT or no intervention control.	At posttest, GCBT led to lower SCAS-P anxiety. No other between-group differences emerged on SCAS anxiety, DSRS-depression, Hope, or SDQ-P total difficulties.	M.1 to M.5
Morgan et al. (2017) [57]	N = 433. Ages 3 to 6 years ($M = 4.80$). 53% girls. STSC > 30. Randomized to parent-focused iCBT (with clinician support as needed) or waitlist control.	At posttest, parent-focused internet-based CBT led to lower PAS-R anxiety, CALIS life interference, and fewer PAS-R anxiety disorder diagnoses than control (40% vs. 54%, respectively).	M.1 to M.5

Schoneveld et al. (2017) [58]	N = 174. Ages 7 to 12 years ($M = 9.97$). 59% girls. Randomized to cognitive-behavioral neurofeedback training video game video game (Mindlight) or GCBT. SCAS ≥ 1 SD above the mean total anxiety symptoms or ≥ 1 SD above the mean on two SCAS subscales.	Between-group differences were not evident. Both Mindlight and GCBT led to lower SCAS and SCAS-P anxiety at posttest, 3- month and 6-month FU.	M.1 to M.5
Weersing et al. (2017) [59]	N = 185. Ages 8 to 17 ($M = 11.30$). 58% girls. DSM GAD, Specific Phobia, SAD, SoP. Randomized to brief behavioral therapy (BBT) or assisted referral to care (ARC).	At posttest, BBT led to lower CGI severity, PARS anxiety, improvements in CGAS functioning, and higher rates of CGI clinical improvement (56.8% vs. 28.2%) than ARC.	M.1 to M.5
Bayer et al. (2018) [60]	N = 545. Ages 3 to 6 years ($M = 4.55$). 48% girls. Australian Temperament Project approach/inhibition > 30. Randomized to parent-focused GCBT or treatment as usual control (access to community mental health services).	Between-group differences were not evident. Both parent-focused GCBT and control led to lower SDQ internalizing symptoms and fewer DSM anxiety disorder diagnoses based on ADIS-C/P criteria.	M.1 to M.5
Chavira et al. (2018) [61]	N=31. Ages 8 to 13 years ($M=11.25$). Mean age and sex not reported. DSM Specific phobia, SAD, GAD, or SoP. Randomized to parent-mediated CBT bibliotherapy telephone- delivered, therapist-assisted bibliotherapy or parent- mediated CBT bibliotherapy minimal contact, self-directed.	At posttest, ADIS-C/P CSR recovery rates were 50% in telephone-therapist and 36% for self-directed.	M.1 to M.5

Liu et al. (2018) [62]	N = 84. Ages 9 to 12 years. Mean age and sex not reported. Met criteria for behavioral inhibition per BIQ. Randomized to ABMt or attention control training.	At posttest, ABMt led to lower C-DISC-IV separation anxiety than control. ABMt also led to reduced amygdala and insulate activation and enhanced activation in ventrolateral prefrontal cortex relative to control via fMRI data.	M.1 to M.5
Ollendick et al. (2018) [63]	N = 58. Ages 12 to 16 years ($M = 14.29$). 71% girls. DSM SAD. Randomized to ABMt or ACT.	At posttest, ABMt led to lower SCARED-C social anxiety than control. No other between-group differences emerged on SCARED-P social anxiety, EATQ-R-P attention control, or dot probe threat bias. Both ABMt and control led to lower SCARED-P social anxiety at posttest.	M.1 to M.5
Schleider & Weisz (2018) [64]	N = 96. Ages 12 to 15 years ($M = 13.33$) 55% girls. RCADS-P $\ge 84^{\text{th}}$ percentile, has school- based accommodations for internalizing symptoms, or sought treatment for anxiety in past 3 years. Randomized to computer- based growth mindset intervention (GMI) or computer-based supportive-therapy.	GMI led to lower CDI-P depression, SCARED-P anxiety, PCSC behavioral control, and CDI-C depression than computer-based supportive- therapy.	M.1 to M.5
Suveg et al. (2018) [65]	N = 92. Ages 7 to 12 years. Mean age not reported. 42% girls. DSM GAD, SAD, SoP. Randomized to emotion-focused CBT (ECBT) or ICBT.	Intervention conditions did not differ from one another. At posttest, both ECBT and ICBT led to lower ERC emotion regulation, ERC emotion dysregulation, ERC anger regulation, CEMS anger, sadness, and worry dysregulation, ADIS-C/P CSR severity, and CGI severity. At posttest, 72% in ECBT and 74%	M.1 to M.5

		in ICBT no longer met ADIS-IV diagnostic criteria for their principal anxiety disorder.	
Pina et al. (In Press) [66]	$N = 109$. Ages 8 to 12 ($M = 9.64$). 78% girls. SCAS \geq 42 anxiety symptoms. Randomized to GCBT with social skills training or active control.	At 12-month FU, GCBT led to higher SEQSS self-efficacy for managing anxiety, SSIS-RS social competence, and lower CNCEQ cognitive interpretation biases than control.	M.1 to M.5
	Phobia	s(n = 10)	
Sheslow (1982) [67]	N = 32. Ages 4 to 5 years. Mean age not reported. 50% girls. Darkness phobia as measure by < 8 seconds of darkness tolerance. Randomized to graduated exposure, verbal coping skills, graduated exposure plus verbal coping skills, or contact-only control.	At posttest, graduated exposure condition and graduated exposure plus verbal coping skills conditions led to improved BAT dark tolerance than verbal coping skills and control, with more pronounced improvements occurring for graduated exposure condition.	M.1 to M.4
Menzies & Clark (1993) [68]	N = 48. Ages 3 and 8 years ($M = 5.50$). BRS < 15 and water phobic (no DSM diagnosis). Randomized to in vivo exposure plus vicarious exposure (IVVE), vicarious exposure (VE), in vivo exposure (IVE), or assessment only control.	At posttest, IVVE and IVE led to lower CWP water phobia, PCWP water phobia, OR phobic reactions, and greater improvements in BRS approach behavior and ability than control. At FU, IVVE, VE, and IVE led to further reductions in OR phobic reactions, and improvements in BRS approach behavior and ability than control. At FU, IVE demonstrated poorer effect maintenance and improvement on all outcomes than IVVE.	M.1 to M.5
Cornwall et al. (1996) [69]	N = 24. Ages 7 to 10 years ($M = 8.25$). DSM Specific Phobia (darkness). Randomized to individual emotive imagery therapy (EAT) or waitlist control.	At posttest and FU, EAT led to improvements in behavioral responses to darkness tolerance and lower FSSC-R fears, RCMAS anxiety, DFBQ	M.1 to M.5

		darkness fear behavior than control.	
Muris et al. (1998) [70]	N = 28. Ages 8 to 17 years ($M = 12.58$). 100% girls. DSM Specific Phobia (spiders). Randomized to EMDR, in vivo exposure (IVE), or computerized exposure control.	At posttest and FU, IVE led to lower SPQ-C spider fear, SAM fear/arousal, BAT state anxiety, and BAT spider avoidance than EMDR and control. At posttest, EMDR led to lower SPQ-C spider fear than control.	M.1 to M.4
Dewis et al. (2001) [71]	N = 28. Ages 10 to 17 years. Mean age not reported. 64% girls. DSM Specific Phobia (spiders). Randomized to live graded exposure (LGE), computer- aided vicarious exposure (CAVE), or waitlist control.	At posttest and 1-month FU, LGE and CAVE had lower SPQ- C spider phobia, PT phobic severity, BAT approach avoidance, and SUDS than control. At posttest, LGE improved more than CAVE on SPQ-C and SUDS and led to greater reductions in SPQ-C spider phobia and PT phobic severity than CAVE at FU.	M.1 to M.4
Ost et al. (2001) [72]	N = 60. Ages 7 to 17 years ($M = 11.70$). 61% girls. DSM Specific Phobia. Randomized to child only one session treatment for specific phobia (OST), parent present OST, or waitlist control.	At posttest and 1-year FU, child only OST and parent present OST led to lower BAT avoidance than waitlist control, with greater improvements emerging for child only OST. No other between- group differences emerged on FSSC-R fears, RCMAS anxiety, CASI anxiety sensitivity, STAIC trait anxiety, STAIC state anxiety, and CDI depression. Both child-only OST and parent present OST led to lower FSSC- R fear, RCMAS anxiety, STAIC anxiety, and CASI anxiety sensitivity at post and FU.	M.1 to M.5
Ollendick et al. (2009) [73]	N = 196. Ages 7 to 16 years ($M = 11.00$). 55% girls. DSM Specific Phobia. Randomized to OST, education support	At posttest, OST led to lower BAT SUDS and ADIS-C/P CSR than EST and control. At 6- month FU, OST led to lower ADIS-C/P CSR severity than	M.1 to M.5

	treatment (EST), or waitlist control.	EST and control. Both OST and EST led to lower CBCL anxiety/depression, MASC anxiety, and FSSC-R fears than control at posttest and FU; OST and EST did not differ from one another for these outcomes. At posttest, ADIS-C/P CSR diagnostic recovery rates were 55% in OST, 23% in EST, and 2% in control; recovery rates were similar at FU (52% in OST and 21% in EST).	
Flatt & King (2010) [74]	N = 43. Ages 7 to 17 years ($M = 11.23$). 60% girls. DSM Specific Phobia. Randomized to OST, psychoeducation, or waitlist control.	Intervention conditions did not differ from one another. At posttest and 1-year FU, OST and Psychoeducation led to lower BAT approach avoidance and FSSC-II fearfulness, as well as higher SEQ-SP self-efficacy than control.	M.1 to M.5
Waters et al. (2014) [75]	N = 37. Ages 6 to 17 years ($M = 10.56$) 57% girls. DSM Specific Phobia. Randomized to Attention training towards positive stimuli plus OST (ATP+OST) or attention control training plus OST (ACT+OST).	At posttest and 3-month FU, ATP+OST led to lower danger expectancies to fear stimuli and greater bias towards positive stimuli at posttest than ACT+OST. No other between- group differences emerged on SCAS anxiety, SCAS-P anxiety, and SMFQ-C/P depression, with both ATP+OST and ACT+OST having led to lower SCAS anxiety, SCAS-P anxiety, and SMFQ-C/P depression at posttest and FU.	M.1 to M.5
Ollendick et al. (2015) [76]	N = 97. Ages 6 to 15 years. Mean age not reported. 52% girls. DSM Specific Phobia. Randomized to OST or parent-augmented OST (A-OST).	Intervention conditions did not differ from one another at posttest or 1-month FU. Both OST and A-OST led to lower ADIS-C/P CSR severity and higher PIR and CIR anxiety improvement. At 6-month FU,	M.1 to M.5

OST resulted in marginally superior ADIS-C/P diagnostic recovery ratings than A-OST (67.39 % for OST and 49.02% for A-OST).

Notes: ACES = Assessment of Children's Emotional Skill; ADIS-C/P = Anxiety Disorders Interview Schedule for DSM-IV; ADIS-C/P CSR =Clinician Severity Rating; ASP = Ambiguous Situations Protocol; AWS = Adolescent Wellbeing Scale; BAI = Beck Anxiety Inventory; BASC-PRS = Behavior Assessment System for Children-Parent Rating Scales; BAT = Behavioral Assessment Test; BERS-P = Behavioral and Emotional Rating Scale (Parent); BERS-T = Behavioral and Emotional Rating Scale (Teacher); BHS = Beck Hopelessness Scale; BIQ = Behavioral Inhibition Questionnaire; BRS = Behavior Response Scale; BSC-Y = Beck Self-Concept Inventory for Youth; C-DISC-IV= Diagnostic Interview Schedule for Children; CALIS = Child Anxiety Life Interference Scale: CALIS-P = Child Anxiety Life Interference Scale (Parent); CALIS-PV-P = Child Anxiety Life Interference Scale – Preschool Version (Parent); CASI = Child Anxiety Sensitivity Index; CATS = Children's Automatic Thoughts Scale; CBCL = Child Behavior Checklist; CDI = Children's Depression Inventory; CEMS = Children's Emotion Management Scales; CES = Coping Efficacy Scale; CES-DC = Center for Epidemiological Studies Depression Scale; CGAS = Children's Global Assessment Scale; CGI = Clinical Global Impressions; CIR = Child Improvement Rating; CNCEQ = Children's Negative Cognitive Error Questionnaire; CRS = Child Rating Scale; CSDS-P = Child Sheehan Disability Scale (Parent); CSI = Coping Strategy Indicator; CWP = Water Phobia Survey Schedule; DFBQ = Darkness Fear Behavior Questionnaire; DSRS-C = Depression Self Rating Scale for Children; EATQ-R-P = Early Adolescent Temperament Questionnaire-Revised Short Form; ERC = Emotion Regulation Checklist; FSSC-R = Fear Survey Schedule for Children-Revised; GAD = Generalized Anxiety Disorder; GAD-7 = Generalized Anxiety Disorder - Seven item scale; Hope = Children's Hope Scale; KFQ-C = Koala Fear Questionnaire - child report; LSAS - RS= Liebowitz Social Anxiety Scale-Revised; MADRS-S = Montgomery-Asberg Depression Rating Scale-Self Report; MASC = Multidimensional Anxiety Scale for Children; MASC-P = Multidimensional Anxiety Scale for Children (Parent); MES = Motivation and Engagement Scale; OAPA = Online Assessment of Preschool Anxiety; OR = Overall Reaction; PAS = Preschool Anxiety Scale; PAS-R = Preschool Anxiety Scale-Revised; PCSC = Perceived Control Scale for Children; PCWP = Water Phobia Survey Schedule (Parent); PHQ = Patient Health Questionnaire; PIR = Parent Improvement Rating; PT = Phobic Target; PTQ = Perseverative Thinking Questionnaire; QOLI-C Quality of Life Inventory-Child Version; RCADS = Revised Children's Anxiety and Depression Scale; RCMAS = Revised Children's Manifest Anxiety Scale; RSEI = Rosenberg Self-Esteem Inventory; RSES = Rosenberg Self-Esteem Scale; RTAS = Revised Test Anxiety Scale; SAD = Separation Anxiety Disorder; SAM = Self-Assessment Manikin; SAS-A = Social Anxiety Scale for Adolescents-Avoidance; SASC-R = Social Anxiety Scale for Children-Revised;

SCARED = Screen for Child Anxiety Related Emotional Disorders; SCARED-P = Screen for Child Anxiety Related Emotional Disorders (Parent); SCAS = Spence Children's Anxiety Scale; SCAS-P = Spence Children's Anxiety Scale SCAS-P (Parent); SCBE = Social Competence and Behavior Evaluation; SCS = School Connectedness Scale; SDQ-P = Strengths and Difficulties Questionnaire (Parent); SEI = Self Esteem Inventory; SEQ-SP Self-Efficacy Questionnaire for Specific Phobias; SEQSS = Self Efficacy Questionnaire for School Situations; SMFQ = Short Mood and Feelings Questionnaire; SMFQ-P = Short Mood and Feelings Questionnaire (Parent); SoP = Social Phobia; SOSI = Symptoms of Stress Inventory; SPAI-C = Social Phobia and Anxiety Inventory; SPQ-C = Spider Phobia Questionnaire for Children; SPSQ-C = Social Phobia Screening Questionnaire for Children; SQC = Schema Questionnaire for Children; SSIS-RS = Social Skills Improvement System-Rating Scales; STAIC = State-Trait Anxiety Inventory for Children; STAXI = State Trait Anger Expression Inventory; STSC = Short Temperament Scale for Children; STSC-P = Short Temperament Scale for Children (Parent); SUDS = Subjective Units of Distress; TABC-R-P = Temperament Assessment Battery for Children-Revised (Parent); TASC = Test Anxiety Scale for Children; TRF = Teacher's Report Form; WEMWBS = Warwick-Edinburgh Mental Wellbeing Scale; YSR = Youth Self Report

Within-group Esg None Vertications Possibly Efficacions Experimental Questionabli (under Post) Pre to Follow-Up - 0.34 - </th <th></th> <th>Level 1</th> <th>Level 2</th> <th>Level 3</th> <th>Level 4</th> <th>Level 5</th>		Level 1	Level 2	Level 3	Level 4	Level 5
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Within-group ESg 069	Clinician- bibliother 3 hours 5 hours 5 hours 1920 tri 1280 tri 1280 tri	Clinician-guided bibliotherapy ^{a,b} 3 hours [31] 5 hours [31]	[UC] JUDDAN SUPPORT	
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	Emot 4 h	Emotive imagery 4 hours [69]		
<i>Notes:</i> Level of Support Designations are described in Table 1. Number in [] refers to the studies cited in Table 2. ESg = Hedge's g effect size: ns = not statistically significant; ABMt = Attention Bias Modification Training; CB = Cognitive-behavioral; CBT = Cognitive behavior therapy; dash indicates not enough data to meta-analyze (i.e., at least two studies) ^d = Clinician here broadly refers to any person in a position in which they are delivering these interventions (e.g., clinical psychologists, counselors, social workers, nurses, prevention or behavior specialists, school psychologists) ^b = <i>Overcoming your Child's Fears and Worries: A Self-help Guide using Cognitive-Behavioural Techniques</i> (Creswell & Willetts, 2007) ^b = <i>Dovercoming your Child's Fears and Worries: A Self-help Guide using Cognitive-Behavioural Techniques</i> (Creswell & Willetts, 2007) ^c = <i>Helping Your Anxious</i> cognitive-behavioral skills relevant to managing parent anxiety and/or supporting child in managing their anxiety ^e = Teocused on traching cognitive-behavioral skills relevant to managing parent anxiety and/or supporting child in managing their anxiety ^e = Teocused on traching cognitive-behavioral skills relevant to managing parent anxiety	ribed in Table 1. Number in ntion Bias Modification Train e (i.e., at least two studies) in a position in which they a or behavior specialists, scho- ies: A Self-help Guide using (p Guide (Rapec, Spence, Col p Guide (Rapec, Spence, Col askills relevant to managing pa and telephone consultation	[] refers to the studies ing: CB = Cognitive-l re delivering these inte psychologists) "Cognitive-Behavioural anam & Wignall, 2000 and anxiety and/or su with a clinician	cited in Table 2. ESg = Hed, behavioral; CBT = Cognitive rentions (e.g., clinical psyc rechniques (Creswell & Wi 0)	ige's g effect size; e behavior therapy; chologists, illetts, 2007) their anxiety

K N g SDg 95% CI FSN Qw Pre to Post 0.10 0.27 0.11 0.27 287 189.60 (63)**** Pre to Post 37 9.084 0.19 0.27 0.11 0.27 287 189.60 (63)*** Teatment 25 1,300 0.35 0.36 0.20 0.56 80 24.65 (24) \$**** Treatment 37 9.084 0.19 0.27 0.010 0.36 24.65 (24) \$***** Targeted Prevention 17 2,315 0.22 0.24 0.10 0.36 24 30.10 (16)*** Selective Prevention 17 6,769 0.09 0.19 0.01 0.20 58.42 (16)*** Vet to Follow-up 7.22 0.23 0.24 0.10 0.34 27.45 (23) Universal Prevention 17 6,769 0.09 0.19 0.01 0.20 58.42 (16)**** Pre to Follow-up <th></th> <th></th> <th></th> <th></th> <th></th> <th></th>						
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Selective Prevention4400Universal Prevention17 $6,769$ Pre to Follow-up30 $6,440$ Overall30 $6,440$ Overall 6 235 Prevention 23 $5,855$ Treatment 6 235 Prevention 15 $2,057$ Indicated Prevention 15 $2,057$ Indicated Prevention 15 $2,057$ Indicated Prevention 23 $5,855$ Vargeted Prevention 23 $5,855$ Mote: $K = number of studies; N = sample size; g = weighte95\% confidence interval; FSN = Fail Safe N, which is the interval$		0.24	0.10	0.30	14	22.56 (12)*
Universal Prevention17 $6,769$ Pre to Follow-up 30 $6,440$ Overall 30 $6,440$ Overall 30 $6,440$ Treatment 6 235 Prevention 23 $5,855$ Targeted Prevention 23 $5,855$ Indicated Prevention 15 $2,057$ Indicated Prevention 15 $2,057$ Indicated Prevention 23 $5,855$ Mote: K = number of studies; N = sample size; g = weighte 95% confidence interval; FSN = Fail Safe N, which is the		0.27	0.06	0.46	2	7.22 (3)
Pre to Follow-upOverall 30 $6,440$ Overall 30 $6,440$ Treatment 6 235 Prevention 23 $5,855$ Targeted Prevention 15 $2,057$ Indicated Prevention 15 $2,057$ Indicated Prevention 13 $1,881$ Selective Prevention 2 176 Universal Prevention 2 176 Solective Prevention 8 $3,798$ Solective Prevention 8 $3,798$ Solective Interval, FSN = Fail Safe N, which is the set of studies; $N = \text{sample size}; g = \text{weighte}$		0.19	-0.01	0.20		58.42 (16)***
Overall30 $6,440$ Treatment6235Prevention23 $5,855$ Targeted Prevention15 $2,057$ Indicated Prevention13 $1,881$ Selective Prevention2 176 Universal Prevention2 176 Wate: K = number of studies; N = sample size; g = weighte95% confidence interval; FSN = Fail Safe N, which is the						
Treatment6235Prevention235,855Prevention152,057Indicated Prevention152,057Indicated Prevention131,881Selective Prevention2176Universal Prevention2176Vote: $K =$ number of studies; $N =$ sample size; $g =$ weighte95% confidence interval; FSN = Fail Safe N, which is the		0.24	0.13	0.34	80	$91.94(29)^{***}$
Prevention235,855Targeted Prevention152,057Indicated Prevention131,881Selective Prevention2176Universal Prevention23,798 $Note: K =$ number of studies; $N =$ sample size; $g =$ weighte95% confidence interval; FSN = Fail Safe N, which is the	-	0.50	0.04	0.56	4	13.74(5)*
Targeted Prevention15 $2,057$ Indicated Prevention13 $1,881$ Selective Prevention2 176 Universal Prevention8 $3,798$ 95% confidence interval; FSN = Fail Safe N, which is the	5,855 0.22	0.23	0.12	0.33	43	77.45 (23)***
Indicated Prevention131,881Selective Prevention2176Universal Prevention8 $3,798$ Note: K = number of studies; N = sample size; g = weighte95% confidence interval; FSN = Fail Safe N, which is the i		0.28	0.14	0.44	24	17.94 (14)
Selective Prevention2176Universal Prevention8 $3,798$ Note: $K =$ number of studies; $N =$ sample size; $g =$ weighte95% confidence interval; FSN = Fail Safe N, which is the i		0.29	0.13	0.47	18	17.01 (12)
Universal Prevention8 $3,798$ Note: $K =$ number of studies; $N =$ sample size; $g =$ weighte95% confidence interval; FSN = Fail Safe N, which is the 1	176 0.21	0.10	-0.09	0.51	ı	0.004(1)
<i>Note:</i> $K =$ number of studies; $N =$ sample size; $g =$ weighte 95% confidence interval; FSN = Fail Safe N, which is the	3,798 0.17	0.17	0.03	0.32	4	26.70 (7)***
95% confidence interval; FSN = Fail Safe N, which is the	= weighted mean effec	tt size; SDg	= weighted	l standare	d deviatio	on of g; 95 % CI =
	ch is the number of sai	mples with	an effect si	ze of zer	o that sho	ould have been left
out in order to reduce estimated effect size to non-significance; $Q_w = Variability$ among effect sizes; Pre to follow-up is average of	-significance; $Q_w = V_a$	ariability an	nong effect	sizes; Pr	te to follo	w-up is average of
8.88 months; ¹ = not including studies reporting post-hoc intervention effects for indicated or selective sub-groups; $*p<.05$, **	ost-hoc intervention ef	ffects for inc	dicated or s	selective	sub-grouj	ps; * <i>p<</i> .05, **
p<.01, ***p<.001;						

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Table 5. Pre to Post Moderators of Effect Sizes Using Analog to ANOVA Test							
		Subg	roup Ar	nalysis	Moder	Ioderator Test	
	Studies,	ES	95%	6 CI	Qb	<i>p</i> -	
	k	(g)	207	0.01	νυ	value	
Study Moderators ^a							
Sample type							
Recruited	51	0.20	0.10	0.29	0.02	0.88	
Referred	13	0.21	0.01	0.42			
Study Location							
North America	19	0.19	0.02	0.35	0.08	0.96	
Outside North America	44	0.21	0.11	0.31			
Type of evaluation							
Efficacy	43	0.15	0.09	0.21	4.05	0.04	
Effectiveness	21	0.07	0.02	0.12			
Comparator condition							
No treatment or waitlist	39	0.26	0.16	0.37	3.72	0.05	
Psychotherapy placebo or active	25	0.09	-0.05	0.23			
Participant Moderators ^b							
Youth ethnicity							
Majority Caucasian sample	25	0.19	0.01	0.37	0.37	0.54	
Majority non-Caucasian	3	0.01	-0.54	0.56			
sample							
Youth gender							
Majority male ($\leq 50\%$ boys)	21	0.24	0.10	0.39	0.65	0.42	
Majority female (>50% girls)	42	0.17	0.07	0.27			
Developmental period							
Childhood ($M_{age} \leq 12$ years)	32	0.16	0.07	0.25	0.78	0.38	
Adolescence ($M_{age} > 12$ years)	14	0.24	0.09	0.39			
Focal Anxiety Concern							
Specific Phobia	7	0.41	0.08	0.73	1.72	0.42	
Mixed Anxiety	52	0.18	0.09	0.27			
Social Anxiety	5	0.22	0.03	0.41			
Intervention Moderators ^c	-	-		-			
Level of Intervention							
Treatment	25	0.33	0.22	0.44	28.11	0.001*	
Targeted Prevention	17	0.21	0.12	0.30	20.11	0.001	
Universal Prevention	17	0.04	-0.01	0.09			
Primary Format	1 /	0.04	0.01	0.07			
Individual	9	0.30	0.05	0.55	3.70	0.59	
Group	30	0.17	0.05	0.28	5.70	0.59	
Digital	11	0.17	0.00	0.20			
ABMt	8	0.23	-0.15	0.37			
Recipient of Intervention	0	0.11	0.15	0.57			
Child	57	0.20	0.11	0.29	0.02	0.88	
China	51	0.20	0.11	0.27	0.02	0.00	

Table 5. Pre to Post Moderators of Effect Sizes Using Analog to ANOVA Tests

Parent	6	0.18	-0.08	0.43		
Delivery Setting						
Research	18	0.34	0.15	0.53	2.85	0.24
Community	35	0.15	0.05	0.26		
Home/Digital	11	0.22	0.02	0.40		
Provider Type						
Professional	42	0.26	0.15	0.37	3.36	0.07
Non-Professional	22	0.08	0.02	0.13		
Training for Providers						
Required	29	0.16	0.04	0.25	0.02	0.90
Not required	7	0.14	0.06	0.38		
Supervision for Providers						
Required	27	0.12	0.01	0.23	0.84	0.34
Not required	15	0.20	0.06	0.35		

Note: Q_b = Between group Q-test value (analog-to-ANOVA); Significant Q_b value indicates moderation effect; * = statistically significant at the family-level p-value ascertained from the Holm's modified Bonferroni correction

^a = Significance threshold for study-level moderator family was ≤ 0.0125

^b = Significance threshold for participant-level moderator family was ≤ 0.0125

^c = Significance threshold for participant-level moderator family was ≤ 0.008

		Subg	oup Analysis		Moderator	
		U	1	5		est
	Studies, <i>k</i>	ES (g)	95%	6 CI	Qb	<i>p-</i> value
Study Moderators						
Sample type						
Recruited	27	0.25	0.14	0.37	0.46	0.50
Referred	3	0.08	-0.40	0.56		
Study Location						
North America	19	0.19	0.02	0.35	0.04	0.85
Outside North America	44	0.21	0.11	0.31		
Type of evaluation						
Efficacy	20	0.30	0.15	0.44	1.41	0.24
Effectiveness	10	0.16	0.02	0.34		
Comparator condition						
No treatment or waitlist	18	0.32	0.18	0.47	3.50	0.06
Psychotherapy placebo or active	12	0.11	-0.07	0.29		
Participant Moderators						
Youth ethnicity						
Majority Caucasian sample	8	0.24	0.02	0.47	-	-
Majority non-Caucasian sample	1	-	-	-		
Youth gender						
Majority male (> 50% boys)	9	0.28	0.09	0.48	0.46	0.50
Majority female (>50% girls)	20	0.20	0.07	0.34		
Developmental period						
Childhood ($M_{age} < 12$ years)	17	0.16	0.06	0.25	4.49	0.03
Adolescence ($M_{age} > 12$ years)	5	0.07	0.12	0.26		
Focal Anxiety Concern						
Specific Phobia	2	0.75	0.13	1.37	3.12	0.21
Mixed Anxiety	25	0.24	0.12	0.35		
Social Anxiety	3	0.09	-0.31	0.50		
Intervention Moderators						
Level of Intervention						
Treatment	6	0.32	0.01	0.65	0.84	0.66
Targeted Prevention	15	0.29	0.14	0.44		
Universal Prevention	8	0.19	0.01	0.37		
Primary Format		-	-			
Individual	4	0.24	-0.14	0.62	3.21	0.52
Group	16	0.33	0.17	0.48		
Digital	5	0.11	-0.13	0.35		
ABMt	4	0.08	-0.26	0.42		
Recipient of Intervention						
Child	26	0.23	0.11	0.35	0.06	0.81

Table 6. Pre to Post Moderators of Effect Sizes Using Analog to ANOVA Tests

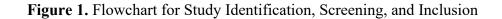
Parent	3	0.27	-0.07	0.62		
Delivery Setting						
Research	6	0.32	0.00	0.63	0.29	0.86
Community	20	0.23	0.09	0.36		
Home/Digital	4	0.26	-0.02	0.55		
Provider Type						
Professional	21	0.29	0.15	0.43	1.01	0.31
Non-Professional	9	0.17	-0.02	0.35		
Training for Providers						
Required	13	0.19	0.07	0.32	0.14	0.71
Not required	3	0.14	-0.11	0.39		
Supervision for Providers						
Required	8	0.11	0.08	0.30	0.69	0.41
Not required	9	0.18	0.05	0.31		

Note: Average time of follow-up assessments used in effect size calculations was 8.88 months (SD = 8.76 months); - denotes less than 2 studies available to meta-analyze; Q_b = Between group Q-test value (analog-to-ANOVA); Significant Q_b value indicates moderation effect; * = statistically significant at the family-level *p*-value ascertained from the Holm's modified Bonferroni correction

^a = Significance threshold for study-level moderator family was ≤ 0.0125

^b = Significance threshold for participant-level moderator family was ≤ 0.0125

^c = Significance threshold for participant-level moderator family was ≤ 0.008



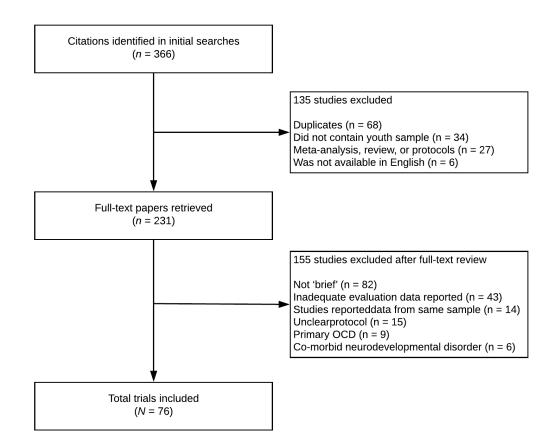
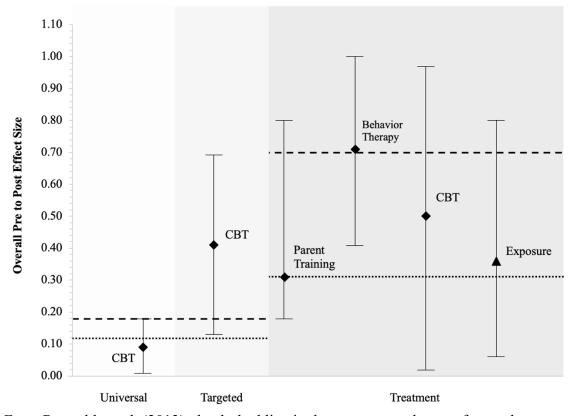


Figure 2. Minimal EBI Effect Sizes for Pediatric Anxiety by Level of Intervention



From Reynolds et al. (2012), the dashed line in the treatment column refers to the overall pre to post effect size of 0.70 (UCL: 1.17; LCL: 0.32) resulting from typical length treatments for pediatric anxiety whereas the dotted line refers to the lower confidence level of the overall effect size (the UCL is not illustrated). The dashed and dotted lines in the targeted and universal prevention columns refers to the overall pre to post effect size of 0.18 (UCL: 0.23; LCL: 0.13) and lower confidence limit, respectively, as reported by Fisak et al. (2011).



Note: Effect sizes reported are between-group