Identifying Mediators of Youth Anxiety and Depression Intervention Outcomes: A Meta-Analytic Path Analysis

by

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A Thesis Presented in Partial Fulfillment
of the Requirements for the Degree
Master of Arts

Approved October 2015 by the
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ARIZONA STATE UNIVERSITY

December 2015

ABSTRACT

Anxiety and depression are among the most prevalent disorders in youth, with prevalence rates ranging from 15% to 25% for anxiety and 5% to 14% for depression. Anxiety and depressive disorders cause significant impairment, fail to spontaneously remit, and have been prospectively linked to problematic substance use and legal problems in adulthood. These disorders often share a high-degree of comorbidity in both clinical and community samples, with anxiety disorders typically preceding the onset of depression. Given the nature and consequences of anxiety and depressive disorders, a plethora of treatment and preventative interventions have been developed and tested with data showing significant pre to post to follow-up reductions in anxiety and depressive symptoms. However, little is known about the mediators by which these interventions achieve their effects. To address this gap in the literature, the present thesis study combined meta-analytic methods and path analysis to evaluate the effects of youth anxiety and depression interventions on outcomes and four theory-driven mediators using data from 55 randomized controlled trials (N = 11,413). The mediators included: (1) information-processing biases, (2) coping strategies, (3) social competence, and (4) physiological hyperarousal. Meta-analytic results showed that treatment and preventative interventions reliably produced moderate effect sizes on outcomes and three of the four mediators (information-processing biases, coping strategies, social competence). Most importantly, findings from the path analysis showed that changes in information-processing biases and coping strategies consistently mediated changes in outcomes for anxiety and depression at both levels of intervention, whereas gains in social competence and reductions in physiological hyperarousal did not emerge as significant mediators.

Knowledge of the mediators underlying intervention effects is important because they can refine testable models of treatment and prevention efforts and identify which anxiety and depression components need to be packaged or strengthened to maximize intervention effects. Allocating additional resources to significant mediators has the potential to reduce costs associated with adopting and implementing evidence-based interventions and improve dissemination and sustainability in real-world settings, thus setting the stage to be more readily integrated into clinical and non-clinical settings on a large scale.

ACKNOWLEDGMENTS

I would like to first thank my mentor and committee chair, Dr. Armando Pina for his ongoing support and commitment to my graduate training. His feedback, advice, and encouragement throughout this thesis project made me a better and more confident researcher. I also would like to thank my thesis committee, including Dr. David MacKinnon and Dr. George Knight for their time, advice, and encouragement. Their thoughtful feedback and suggestions helped expand my thinking and shape the development of this thesis. Finally, I would like to thank my research lab mates from the Child and Family Intervention Program who offered helpful advice and constant support throughout this project.

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INTRODUCTION

Anxiety and depression are among the most prevalent disorders in children and adolescents (Albano, Chorpita, & Barlow, 2003; Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Kesler et al., 2007; Lewinsohn, Hops, Roberts, Seeley, & Andrews, 1993) with lifetime prevalence rates ranging from 15% to 25% for anxiety (Beesdo, Knappe, & Pine, 2010; Costello, Egger, & Angold, 2005) followed by 5% to 14% for depression (Angold & Costello, 2001; Merikangas et al., 2010; Lewinsohn et al., 1993). Anxiety and depressive disorders often persist throughout the lifespan and are associated with negative outcomes, including increased somatic reactions, low self-esteem, and severe disruptions in social functioning and achievement (Costello et al., 2003; Gotlib; Lewinsohn, & Seeley, 1995). These disorders also have been found to precede problematic substance use and legal problems in adulthood (Albano, Chorpita, & Barlow, 2003; Collins & Dozois, 2008; Kendall, Safford, Flannery-Schroeder, & Webb, 2005; Merikangas et al., 1998) while showing a high-degree of comorbidity in both clinical and community samples. That is, about 10% to 15% of youth with primary anxiety meet criteria for depression and approximately 15% to 75% of depressed youth are diagnosed with anxiety (Angold, Costello, & Erkanli, 1999; Brady & Kendall, 1992; Costello et al., 2003; Cummings, Caporino, & Kendall, 2013). Moreover, copious evidence suggests a relatively temporal relation between anxiety and depression in that anxiety disorders tend to precede the onset of depressive disorders (Beesdo, Bittner, Pine, et al., 2007; Brady & Kendall, 1992; Chorpita & Daleiden, 2002; Kovacs, Gatsonis, Paulauskas, & Richards, 1989; Pine, Cohen, Gurley, Brook, & Ma, 1998; Watson & Kendall, 1989). For instance,

in a clinical sample of depressed youth with comorbid anxiety, two thirds of participants had the anxiety disorder prior to the depression (Kovacs et al., 1989) suggesting that an anxiety disorder at a young age may increase the risk for the development of a concurrent depressive disorder at older ages. For these reasons, it is important to consider both anxiety and depression, when possible, in research relevant to disorder development and its reversal (Garber & Weersing, 2010).

Theoretically, and also based on empirical data, anxiety and depression may co-occur due to common diatheses, temperamental factors, neural-circuitry dysfunctions, and genetic influences (Barlow, 2000; Costello, Egger, & Angold, 2005; Garber & Weersing, 2010; McLeod, Weisz, & Wood, 2007; Phillips, Drevets, Rauch, & Lane, 2003). In fact, intervention research supports this possibility in several ways. First, intervention effects are often nonspecific in that anxiety interventions produce changes in depressive symptoms and depression interventions produce changes in anxiety symptoms (e.g., Flannery-Schroeder & Kendall, 2000; Pattison & Lynd-Stevenson, 2001; Stopa, Barrett, & Golingi, 2010). Second, anxiety and depression interventions rely on similar strategies, such as improving emotional understanding (psychoeducation), modifying antecedent cognitive reappraisals (cognitive restructuring), preventing emotional or behavioral avoidance (exposures), and facilitating action tendencies contrary to emotional symptoms (skills training) (Chorpita, Daleiden, & Weisz, 2005). Third, several randomized controlled trials (RCTs) have demonstrated that when targeted using a variety of intervention formats (e.g., individual, family, peer group), reductions in anxiety and depression reduce the risk for common negative sequela, including academic difficulties, strained interpersonal relationships, substance use disorders, and behavioral

problems (Fine, Forth, Gilbert, & Haley, 1991; Kendall et al., 1997; Lock & Barrett, 2012; Puleo, Conner, Benjamin, & Kendall, 2011; Stice, Rohde, Seeley, & Gau, 2008). Lastly, the strategies used across psychosocial interventions for anxiety and depression tend to target similar etiological factors such as information-processing biases, coping strategies, social competence, and physiological hyperarousal (Kendall, 2006; Weersing, Rozenman, Maher-Bridge, & Campo, 2012). Together, these data suggest that a joint examination of anxiety and depression might be worthwhile as it can provide new insights into the common factors between both disorders and identify ways to enhance treatment and prevention programs.

Moving forward, one important next step for the next generation of research focused on anxiety and depression is to identify potential mediators driving intervention outcomes. Identifying potential mediators may help to: (a) isolate causal mechanisms, (b) refine testable models of intervention efforts, and (c) identify which anxiety or depression intervention components need to be packaged or strengthened. The need to identify potential mediators has been emphasized previously (Kazdin & Kendall, 1998; Kazdin & Nock, 2003; Weersing & Weisz, 2002), yet a significant gap still exists in the research literature. The paucity of meditational analyses is not due to a lack of available information regarding assessment of mediator measures. In their seminal review, Weersing and Weisz (2002) found that approximately 50% of youth internalizing disorder trials included an assessment of at least one potential mediator of intervention response but only eight examined mediating effects, leaving the authors to conclude, 'considerable evidence exists but has not been fully exploited,' (*p.*22). Findings from the little work that has been done to identify mediators of anxiety and depression

interventions have typically treated mediator variables as simple outcome measures, on par with changes in symptoms and diagnoses. Thus, most of the existing RCTs have not examined the mechanisms by which programs achieve their effects using methodologically robust techniques (i.e., utilizing a temporal design with more than two assessment points; Kraemer et al., 2002). Namely, in the anxiety literature only three studies (Hoogendorn et al., 2014; Maric, Heyne, MacKinnon, van Widenfelt, & Westenberg, 2013; Alfano et al., 2009) have examined mediation using a methodologically robust approach, while three treatment studies (Kendall & Treadwell, 2007; Lau, Chan, Li, & Au, 2010; Treadwell & Kendall, 1996) and one prevention study (Essau, Conradt, Sasagawa, & Ollendick, 2012) have tested the mediator concurrently with the outcomes. Similarly in the depression literature, three treatment trials (Ackerson, Scogin, McKendree, & Lyman, 1998; Kaufman, Rohde, Seeley, Clarke, & Stice, 2005; Shirk, Crisostomo, Jungbluth, & Gudmundsen, 2013) and five prevention trials (Compas et al., 2009; Gilham & Reivich, 1999; Jaycox & Seligman, 1995; Sarin, Abela, & Auerbach, 2005; Yu & Seligman, 2001) have tested the mediator concurrently with the outcomes while none have examined mediation using a robust framework. This limitation precludes the possibility of elucidating the precise sequence of changes that might establish temporal precedence, a critical requirement for classifying a mediator as a causal mechanism that could be used to strengthen the potency of interventions efforts (Kraemer, Wilson, Fairburn, & Agras, 2002).

Turning to knowledge from three meta-analyses, data show preliminary support for cognitive, behavioral, physiological, and coping-related variables that may operate as mediators of anxiety and depression interventions (Chu & Harrison, 2007; Prins &

Ollendick, 2003; Stice, Shaw, Bohon, Marti, & Rodhe, 2009). However, these meta-analyses focused on particular modalities (e.g., cognitive-behavioral therapy), intervention types (e.g., treatment or prevention), or certain categories of potential mediators (e.g., behavioral or cognitive) rather than on the constructs, processes, or putative mechanisms by which interventions are assumed to achieve their effects. Regardless, these meta-analyses advanced knowledge relevant to the pursuit of identifying mediators and mechanisms of intervention response.

Thus, I believe an important next step is to begin organizing knowledge secured to date into an integrative framework that can be used to elucidate and test potential mediators from across published studies. In this thesis, I therefore propose to synthesize the empirical literature relevant to interventions that target youth anxiety and depression via meta-analytic methods to identify potential mediators of intervention response. I then plan to test these variables using meta-analytic path analyses to ascertain the relation between the potential mediators and change in anxiety and depressive symptoms. To set the stage for achieving these goals, I organized the thesis document into three sections. First, I offer an overview of the research literature investigating the efficacy (and effectiveness when available) of treatment and prevention efforts for anxiety and depression. Second, I critically review putative mediator variables suggested in the RCT literature and theory. Third, I articulate the aims of the proposed study with a description of the methods I plan to use to evaluate data from youth 6 to 18 years old who participated in intervention studies that met "robustness" criteria articulated by Chambless and Hollon (1998).

Chapter 2

INTERVENTIONS FOR YOUTH ANXIETY AND DEPRESSION

The high prevalence and severe consequences associated with anxiety and depression has prompted the development and evaluation of psychosocial interventions with cognitive-behavioral therapy (CBT) consistently identified as the modality of choice. Broadly, CBT is a collaborative, problem-focused approach that aims to address factors underlying the maintenance of youth distress (Barlow, Allen, & Choate, 2004; Chu & Harrison, 2007; Kendall, 1990; Kendall, 2006). Notably, about 97% of treatment efforts for youth internalizing problems have been developed on the basis of CBT theory (see Chorpita & Daleiden, 2007; Hollon et al., 2002 for a review). Of note, CBT interventions for anxiety and depression utilize similar implementation strategies to improve emotional understanding, modify information-processing biases, counter patterns of avoidant behaviors, and advance skill sets relevant to improving global functioning (Albano & Kendall, 2002; Chorpita, Daleiden, & Weisz, 2005; Chorpita & Daleiden, 2009; Kendall et al., 2006; James, Soler, & Weatherall, 2009). Combined, CBT provides youth with strategies and tools that allow effective management of anxiety and depression.

Treatment Interventions

Focusing on the treatment of anxiety disorders, eight meta-analyses have reported moderate to large pre to post-treatment effect sizes, ranging from 0.44 to 1.27 (Brendel & Maynard, 2014; Chu & Harrison, 2007; In-Albon & Schneider, 2007; Ishikawa, Okajima, Matsuoka, & Sakano, 2007; Prins & Ollendick, 2003; Reynolds, Wilson, Austin, & Hooper, 2012; Silverman, Pina, & Viswesvaran, 2008; Spielmans, Pasek, & McFall,

2007). Several methodologically robust trials show that CBT is efficacious. For example, Kendall (1994) conducted the first RCT examining the efficacy of individual CBT (ICBT; the *Coping Cat Program*) and found significantly greater pre to post-treatment reductions in youth and parent reports of anxiety symptoms for ICBT than the waitlist control condition. In addition, 64% of participants in ICBT no longer met criteria for an anxiety disorder diagnosis as compared to 5% in the waitlist at post-treatment. These findings were replicated in a later RCT (Kendall et al., 1997) with treatment gains maintained across several indices of anxiety for up to seven years (Kendall et al., 2004). Subsequently, Barrett, Dadds, and Rapee (1996) developed an Australian adaptation of Kendall's Coping Cat program and reported similar beneficial gains while also finding support for a CBT plus a family anxiety management component (ICBT+FAM). At post-treatment, 57 % of youth in the ICBT condition and 84% in the ICBT+FAM condition no longer met diagnostic criteria for an anxiety disorder compared to 26% in the waitlist control. At the one-year follow-up, treatment gains were maintained on all youth and parent report measures for both conditions, with ICBT+FAM remaining statistically greater to ICBT on diagnostic recovery rates. Support for ICBT+FAM was also reported across several additional trials (Cobham, Dadds, & Spence, 1999; King et al., 1998; Wood, Piacentini, Southam-Gerow, Chu, & Sigman, 2006).

Computer-assisted cognitive behavioral therapy also has emerged as an efficacious approach to the treatment of youth anxiety. These approaches often involve using computers or the internet to deliver interactive media games teaching youth the intervention strategies common to traditional ICBT (e.g., cognitive restructuring, emotion identification, relaxation training). For instance, Khanna and Kendall (2010) compared

the effects of a computer-delivered CBT protocol based upon Kendall's Coping Cat program (*Camp Cope-A-Lot;* Kendall & Khanna, 2008) to ICBT and an attention control condition. Post-treatment results indicated that the percentage of youth no longer meeting criteria for an anxiety disorder diagnosis in the computer-assisted condition (81%) was comparable to ICBT (70%) and both were significantly larger than the attention control (19%). In another trial, Spence et al. (2011) compared the efficacy of online versus clinic delivery of CBT for anxious youth. Statistically significant reductions for anxiety diagnoses and symptoms were comparable for both the online and clinic-based CBT conditions at post-treatment. At one-year follow-up, 78% of youth in the online group no longer met diagnostic criteria for an anxiety disorder compared with 81% in the clinic-based condition. These findings are supported by several additional trials reporting similar findings (e.g., March, Spence, & Donovan, 2009; Spence, Holmes, March, & Lipp, 2006; Stallard, Richardson, Velleman, & Attwood, 2011; Wuthrich et al., 2012).

Group cognitive-behavioral therapy (GCBT) also has been used with efficacious findings. Namely, Silverman et al. (1999a, 1999b) evaluated an exposure-based GCBT program and found that post-treatment results indicated that the percentage of youth free of anxiety disorder diagnosis was significantly larger in that condition (64%) then the waitlist control condition (13%). Statistically significant improvements were observed pre to post-treatment for GCBT on clinicians' ratings of diagnostic severity, youth reports, and parent reports of anxiety for GCBT but not the waitlist. Treatment gains were maintained at the 1-year follow-up and diagnostic recovery rates for GCBT increased to over 75%. Several trials have reported similar results supporting GCBT (e.g., Flannery-Schroeder, Choudhury, & Kendall, 2005; Manassis, Avery, Butalia, & Mendlowitz;

Mendlowitz et al., 1999; Rapee, Abbott, & Lyneham, 2006; Shortt, Barrett, & Fox, 2001; Spence, Donovan, & Brechman-Toussaint, 2000).

Finally, in the Child-Adolescent Anxiety Multimodal Study (CAMS), the largest anxiety treatment trial ever conducted, the efficacy of CBT, medication, and their combination were compared (Walkup et al., 2008). While the best outcomes were produced by the CBT plus medication condition, results indicated that clinicians rated 60% of youth in the CBT condition as "very much improved" or "much improved," which was slightly greater than the medication condition (54.9%). In addition, anxiety disorder remission rates (i.e., becoming nearly symptom-free) in the CBT condition (46.2%) were comparable to the medication condition (45.9%;), with less somatic side effects (e.g., fatigue, insomnia) being associated with CBT than for medication (Ginsburg et al., 2011). Given the efficacious results of CBT in treating youth anxiety using a variety of formats, trials using this approach continue to be refined and tested. In fact, a search in PsycInfo of CBT treatment outcome studies with anxious youth published after Kendall (1994) produces 173 results, with about 80% of these trials having been published within the last decade.

Turning to the treatment of depression, seven meta-analyses have reported moderate to large effect sizes, ranging from 0.55 to 1.39 (Arnberg & Ost, 2014; Chu & Harrison, 2007; Klein, Jacobs, & Reinecke, 2007; Lewisohn & Clarke, 1999; Michael & Crowley, 2002; Reinecke, Ryan, & Dubois, 1998). Various formats of CBT have been used with depressed youth, including ICBT, computer-assisted CBT, and GCBT. Brent et al. (1997), for example, found support for ICBT, with significant pre to post-treatment changes on several youth report indices of depression (no parent report measures of

depression symptoms were assessed). At post-treatment, 64.7% of youth in the ICBT condition no longer met diagnostic criteria for a depressive disorder. In another trial, Merry et al., (2012) compared a computer-delivered CBT condition to treatment as usual (i.e., ICBT). Results indicated significantly higher remission rates in depression diagnoses for the computer-assisted CBT group (43.7%) compared to the treatment as usual condition (26.4%), with improvements maintained at the three-month follow-up. These findings were supported across several other trials (Clarke, Rohde, Lewinshohn, Hops, & Seeley, 1999; Rossello & Bernal, 1999; Vostanis, Feehan, Grattan, & Bickerson, 1996; Wood, Harrington, & Moore, 1996).

Subsequently, Lewinsohn, Clarke, Hops, Andrews, and Clarke (1990) evaluated GCBT with post-treatment results showing that the percentage of youth free of a depressive disorder was significantly larger in the treatment condition (43%) then the waitlist control condition (5.3%). Significant pre to post-treatment improvements were also found on clinicians' diagnostic severity ratings and youth and parent reports of depressive symptoms. Additional evidence for the efficacy of GCBT has been reported (Clarke et al., 2001; Kahn, Kehle, Jenson, & Clark, 1990; Liddle & Spence, 1990; Reynolds & Coats, 1986; Weisz, Thurber, Sweeney, Proffitt, & LeGagnoux, 1997) with symptom and diagnostic improvements being maintained for up to 1-year post-treatment (De Cuyper, Timbremont, Braet, De Backer, & Wullaert, 2004).

Finally, in the Treatment for Adolescent Depression Study (TADS), the largest RCT comparing the efficacy of treatment modalities for depressed youth, those receiving CBT following the cessation of a psychopharmacological intervention were found to have increasing rates of positive response or recovery over time (The TADS Team,

2007). While initial response rates of CBT were lower at the 12-week assessment, at the 36-week assessment, CBT produced response rates comparable to medication and CBT plus medication groups. Moreover, the addition of CBT also appeared to improve the safety of medication, as those in the CBT plus medication condition experienced significantly fewer suicidal events than youth in the medication only condition (Kennard et al., 2010). Since the initial wave of RCT's in the late 1980's, results from PsycInfo indicate that 182 evaluations of CBT treatment for youth depression have been conducted, with 106 being published within the last 10 years. This suggests that this approach continues to be used across a spectrum of populations and formats, with consistent efficacious findings.

Preventative Interventions

In contrast to treatment protocols that aim to mitigate pre-existing emotional and/or behavioral problems, the goal of prevention is to reduce the likelihood of the development of these difficulties. Preventative interventions can be classified as either universal or targeted (Mrazek & Haggerty, 1994). Universal prevention programs are delivered to entire populations of youth without the identification of risk factors. Targeted prevention programs can further be classified as selective in which an intervention is delivered to youth on the basis of various group level risk factors (e.g., gender, ethnicity, parent psychopathology, exposure to stress) or indicated in which participants are selected on the basis of elevated subsyndromal symptoms, suggestive of developing an anxiety or depressive disorder in the future. Although the prevention literature is less extensive than the treatment literature, efforts have demonstrated significant promise in preventing anxiety and depressive disorders in youth, with

cognitive-behavioral approaches included in nearly 100% of prevention protocols (O'Connell, Boat, & Warner, 2009).

In general, preventative interventions for anxiety have demonstrated efficacious findings using GCBT (no study to date has evaluated computer-assisted CBT within anxiety prevention). More specifically, four meta-analyses have evaluated the efficacy of anxiety prevention efforts (Fisak, Richard, & Mann, 2011; Mychailysyn, Brodman, Read, & Kendall, 2012; Teubert & Pinquart, 2011; Zalta, 2011) and have reported effect sizes ranging from 0.18 to 0.32, depending on the type of prevention program. That is, universal prevention programs generally produce smaller effect sizes than targeted programs as they are delivered to a large number of youth with a small need for emotional and behavioral services (Horowitz & Garber, 2006). Starting with universal prevention, for example, Lock and Barrett (2003) examined the efficacy of a school-based GCBT intervention based upon Kendall's Coping Cat program (FRIENDS; Barrett, Lowry-Webster, & Turner, 2000) with 733 youth and found significant pre to post-intervention reductions in youth report measures of anxiety for GCBT but not the monitoring control condition. Significant gains were maintained for up to 3-years post-treatment (Barrett, Farrell, Ollendick, & Dadds, 2006). Similar findings were reported across other universal RCTs (Barrett, Lock, & Farrell, 2005; Barrett & Turner, 2001; Lowry-Webster, Barrett, & Dadds, 2003). Universal prevention programs also have proven to be efficacious across socioeconomically disadvantaged communities (Stopa et al., 2010) and a myriad of cultural settings (Barrett, Sonderegger, & Xenos, 2003; Essau et al., 2012; Gallegos, Rodriguez, Gomez, Rabelo, & Gutierrez, 2012).

Turning to targeted prevention, for example, Dadds, Spence, Holland, Barrett and Laurens (1997) focused on youth with subclinical levels of youth and teacher reported anxiety and reported superior pre to post-intervention reductions of youth reports anxiety symptoms in the GCBT condition but not in the control condition. At the 6-month follow-up, results further indicated that the percentage of youth who met diagnostic criteria for an anxiety disorder was significantly less in GCBT condition (16%) than the control condition (54%). The 2-year follow-up revealed maintenance of intervention effects across all youth rating scales, suggesting a significant preventative effect (Dadds et al., 1999). Barrett, Moore, and Sondergger (2000), Liddle and Macmillan (2010), Pina, Zerr, Villalta, and Gonzales (2012), Roberts et al. (2010), and Siu (2007) reported similar findings, providing further support for targeted prevention programs for anxiety. In the last decade, PsycInfo indicates that 44 RCTs evaluating anxiety prevention programs have been published, indicating that researchers continue to refine the CBT approach.

Focusing on the prevention of depression, support for depression prevention programs has increasingly emerged in the literature with seven meta-analytic reviews (Brunwasser, Gillham, & Kim, 2009; Calear & Christensen, 2010; Gillham, Shatte, & Freres, 2000; Horowitz & Garber, 2006; Jane-Llopis, Hosman, Jenkins, & Anderson, 2003; Merry, McDowell, Hetrick, Bir, & Muller, 2004; Mychailysyn et al., 2012; Stice et al., 2009) reporting pre to post-intervention effect sizes ranging from 0.21 to 0.40. Findings from universal programs for depression seem variable with respect to efficacy. Results from Shochet et al. (2001) indicated that at post-intervention, the percentage of youth that transitioned from the subclinical depression category to the healthy category was significantly larger in the GCBT condition (71.4%) than the control condition

(31.6%). Significant improvements continued to be found at the 10-month follow-up, with the percentage of youth transitioning from the subclinical category to the healthy category increasing to 75%. In another universal trial, Spence, Sheffield, and Donovan (2003) reported significant pre to post-intervention reductions in youth reports of depressive symptoms, however at the 2-, 3-, and 4-year follow-up, these results were not maintained and the intervention and control conditions were no longer significantly different. Other universal trials have reported no significant reductions in depressive symptoms at post or follow-up assessments (Gillham et al., 2007; Harnett & Dadds, 2004; Pattison & Lynd-Stevenson, 2001; Roberts, Kane, Thomson, Bishop, & Hart, 2003; Sheffield et al., 2006).

These relatively "weak" universal intervention effects have led to a greater focus and evaluation of targeted prevention approaches. In general, targeted prevention programs have demonstrated more consistent and efficacious results across GCBT and computer-assisted CBT formats. Clarke et al. (2001), for example, aimed at treating subclinical levels of depression in youth who had parents with a depressive disorder. Statistically and clinically relevant preventative results were found post-intervention in GCBT condition on youth and parent-reports of depressive symptoms but not in the control condition. In addition, youth in GCBT had an average of 33 fewer depressed days in the year following the start of the intervention than did the control condition. Symptom improvements and reductions in depression incidence rates were maintained at the 1-year follow-up (Clarke et al., 2001). Three recent trials have demonstrated further support for targeted prevention programs for depression (Beardslee et al., 2013; Punamaki, Paavonen, Toikka, & Solantaus, 2013; Stice et al., 2008). Relevant to the use of

computer-assisted CBT in the targeted prevention of youth depression, O'Kearney, Kang, Christensen, and Griffiths (2009) reported significant reductions in depressive symptoms in computer-assisted CBT group compared to passive control condition at 6-month follow-up, with those having higher initial depression levels showing the greatest improvement. Four additional trials evaluating computer-assisted or internet delivered CBT protocols demonstrated similar changes in depressive symptoms at post and follow-up (Calear et al., 2009; O'Kearney et al., 2006; Stallard et al., 2011; Van Voorhees et al., 2009.) As with the other efforts noted earlier, refinements of cognitive-behavioral prevention programs for depression continue to be examined in the field. In fact, a search of such evaluations on PsycInfo yielded 89 RCT's, with over 83% being published since 2004.

Transdiagnostic Interventions

Interventions targeting one disorder may have advantageous effects on symptoms of the other disorder (Garber & Weersing, 2010). As such, the inclusion of both anxiety and depression treatment and prevention trials in the current thesis study has the potential to make a valuable contribution to the understanding of how to address, treat, and prevent the negative sequela associated with both disorders. In a recent meta-analysis, Chu and Harrison (2007) compared effect sizes across 28 CBT trials targeting anxiety or depression found that anxiety treatments significantly reduced depressive symptoms (ES = 0.55) and depression treatments produced a small, but significant, effect in reducing anxiety symptoms (ES = 0.28). In a more specific example, Saavedra, Silverman, Morgan-Lopez, and Kurtines (2010) beyond the reductions of anxiety disorders and symptoms directly targeted by the treatment, clinically significant reductions of youth

reported symptoms of depression were also found 8 to 13 years post-treatment. Numerous RCTs in the prevention literature have found similar results (Dobson, Hopkins, Fata, Scherrer, & Allan, 2010; Liddle & Macmillan, 2010; Lock & Barrett, 2003; Lowry-Webster, Barrett, & Lock, 2003; Roberts, Kane, Thomson, Bishop, & Hart, 2003; Sheffield et al., 2006; Spence et al., 2003). Lowry-Webster et al. (2003), for example, evaluated a CBT-based universal prevention program for anxiety and in addition to significantly reducing anxiety symptoms, significant reductions in youth reported depressive symptoms were also found at the 1-year follow-up.

These findings have prompted the development of "unified" or "transdiagnostic" protocols that build on CBT theory to target the underlying commonalities between the two disorders. For example, the content from the Unified Protocol for the Treatment of Emotional Disorders in Youth (UP-Y) aims to provide education regarding emotions and behaviors to increase affective awareness, modify incorrect situational appraisals, increase experience with uncomfortable emotions, and provide tools to prevent future relapse (Trosper, Buzzella, Bennett, & Ehrenreich, 2009). Preliminary data from the three trials evaluating transdiagnostic protocols with youth (Bilek & Ehrenreich-May, 2012; Ehrenreich-May & Bilek, 2011; Ehrenreich et al., 2008) suggests that they may be efficacious in treating anxiety and depression concurrently. Bilek and Ehrenreich-May (2012), for example, evaluated a transdiagnostic protocol with clinically anxious and/or depressed youth. Significant pre to post-treatment reductions were found across clinicians' diagnostic severity ratings and youth reports of anxiety and depressive symptoms. Post-treatment results also indicated that about 83% of youth receiving the intervention no longer met diagnostic criteria for an anxiety or depressive disorder post-

treatment. Diagnostic recovery and symptom reductions were maintained at the 6-month follow-up (Bilek & Ehrenreich-May, 2012). Together, these findings provide evidence suggesting that interventions may not only ameliorate the immediate adverse consequences of the primary targeted disorder (e.g., anxiety in anxiety interventions), but also influence the trajectory of the other disorder in the process (e.g., depression in anxiety interventions).

Evaluative Summary

A large body of research has accumulated that supports the assertion that CBT is one of the most efficacious approaches in the treatment and prevention of youth anxiety and depression. Most studies report significant pre to post-intervention improvements across a variety of indices, including youth report, parent report, diagnostic interviews, and clinician ratings. Furthermore, most studies that included a follow-up assessment report maintenance of intervention gains across time. In addition, evidence across many of these trials shows that CBT interventions often produce non-specific effects. That is, anxiety interventions reduce depressive symptoms and depression interventions reduce anxiety symptoms. Finally, youth in CBT conditions demonstrate statistically significant improvements across a variety of intervention formats, including individual, group, computer-assisted, and parent interventions. These conclusions are supported by the 23 meta-analyses examining the efficacy of CBT in the treatment and prevention of anxiety and depression (Arnberg & Ost, 2014; Brendel & Maynard, 2014; Brunwasser et al., 2009; Calear & Christensen, 2010; Chu & Harrison, 2007; Fisak et al., 2011; Gillham et al., 2000; Horowitz & Garber, 2006; In-Albon & Schneider, 2007; Ishikawa et al., 2007; Jane-Ellopis et al., 2003; Klein et al., 2007; Lewisohn & Clarke, 1999; Manassis et al.,

2014; Merry et al., 2004; Michael & Crowley, 2002; Mychailysyn et al., 2012; Prins & Ollendick, 2003; Reinecke et al., 1998; Reynolds et al., 2012; Silverman et al., 2008; Spielmans et al., 2007; Stice et al., 2009; Teubert & Pinquart, 2011; Zalta, 2011). Taken together, several studies support the efficacy of CBT interventions for youth anxiety and depression by way of pre to post improvement, improvement over waitlist or monitoring conditions, and maintenance at short-term and long-term follow-ups.

Chapter 3

PUTATIVE MEDIATORS OF INTERVENTION RESPONSE

An evaluation of the underlying mediators that putatively account for intervention effects in RCTs is an understudied topic, but a critical next step of intervention research (Kazdin & Nock, 2003; Kazdin & Weisz, 1998; Weersing & Weisz, 2002). A mediator specifies how (or the mechanism through which) a given effect occurs (Baron & Kenny, 1986; Holmbeck, 1997; MacKinnon, Lockwood, Hoffman, West, & Sheets, 2002). Using Baron and Kenny's (1986) causal steps approach, four conditions must be met to demonstrate mediation: (a) the predictor must be significantly associated with the mediator, (b) the predictor must be significantly associated with the dependent measure, (c) the mediator must be significant associated with the dependent variable, and (d) the impact of the predictor on the dependent variable must be less after controlling for the mediator (Baron & Kenny, 1986). Here, step (a) tests the action theory, which assesses the strength of the link between the intervention strategies (e.g., cognitive restructuring) and the mediating variable the intervention was designed to change (e.g., information-processing biases). Whereas step (c) tests the conceptual theory, which assesses the mediator's theoretical basis for being included by assessing whether there is a causal influence of the mediator on the outcome (MacKinnon et al., 2002). In addition to the four conditions of the causal steps approach, in order to classify a mediator as a causal mechanism, change in the mediator must also follow the onset of the independent variable, or intervention in this case, and precede change in the dependent variable temporally (Kazdin & Nock, 2003). Establishing this temporal relationship requires that measurement of mediators occur prior to the assessment of intervention target outcomes

(e.g., anxiety, depressive symptoms) and need to be measured during the intervention and not only at pre and post (Weersing & Weisz, 2002).

Identifying mediators of intervention response is important for four reasons. First, mediation analysis permit the testing of theoretical mechanisms suggested to drive intervention effects. If intervention effects were noted in the lack of changes in the putative mediator, then the theoretical conceptualization underlying the intervention would appear to be incorrectly specified, thus providing an opportunity to refine testable models of intervention effects (Kraemer et al., 2002; MacKinnon, 2011). Second, mediation analysis may improve measurement of general and specific aspects of a mediating variable (MacKinnon, Lockhart, Baraldi, & Gelfand, 2013). If an intervention produces null effects on a known mediating variable, this might suggest that the measures used to assess the mediator were not reliable enough to capture change, thus prompting the refinement of measurement instruments to be more appropriate, reliable, and valid for specific variables. Third, mediation analysis can assist in identifying which intervention components are successful and unsuccessful at producing expected change in planned program effects. Interventions would cost less, have more robust effects, and be better positioned for large-scale dissemination and implementation into clinical and non-clinical settings by identifying and amplifying the critical components while minimizing or removing unsuccessful components (MacKinnon et al., 2013; Spoth & Greenberg, 2005). Finally, mediation analyses can help isolate causal mechanisms. Just as all causal factors are risk factors, but not all risk factors are causal factors, all mechanisms are mediators, but not all mediators are mechanisms (Kraemer et al., 1997). Yet, establishing a mediator as a mechanism is a much more stringent process than establishing a variable as a

mediator, suggesting that the investment of time and effort to narrow the search for causal factors by first identifying and testing mediators is both necessary and worthwhile (Kraemer et al., 2002).

Although the benefits and methods of mediation analysis have been described extensively (Kazdin, 2007; Kraemer et al., 2002; MacKinnon, 2008, 2011), little research has formally evaluated the mechanisms driving planned program effects. In fact, only twenty RCTs have conducted mediation analyses across the youth anxiety and depression treatment and prevention literature. However, with the exception of three studies (Hogendoorn et al., 2014; Maric et al., 2013; Silverman et al., 2009), these trials did not use methodologically robust mediation techniques (i.e., tested the mediator concurrently with the outcome), thus severely limiting conclusions about causality. Regardless, fifteen of these trials have provided some support for four theory-driven mediators, including reductions in negative cognitions (Ackerson et al., 1998; Gilham et al., 1995; Jaycox & Seligman, 1995; Kaufman et al., 2005; Kendall & Treadwell, 2007; Lau et al., 2010; Shirk et al., 2013; Treadwell & Kendall, 1996; Yu & Seligman, 2001), decreases in feelings of social isolation (Alfano et al., 2009), increases in self-efficacy (Maric et al., 2013), and more frequent use of adaptive coping strategies (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001; Essau et al., 2012; Hogendoorn et al., 2014; Lau et al., 2010; Sarin et al., 2005). These findings are in line with two meta-analyses that explored candidate mediators across published treatment outcome studies. First, Prins and Ollendick (2003) evaluated 25 RCTs and focused on evidence for cognitive and coping variables as candidate mediators of CBT for anxious youth. Across the trials that included a coping measure, CBT was found to produce a moderate mean effect size (ES =

0.65) on youth reports and a large effect size (ES = 1.26) on parent reports of youth coping. A small effect size (ES = 0.36) was found on youth report measures of cognitions, with no studies including a parent-report measure of cognitions. In a more recent and comprehensive review, 28 RCTs for anxious and depressed youth were meta-analyzed to examine the magnitudes of effect CBT has on candidate mediators related to techniques derived from cognitive-behavioral theories of change (Chu & Harrison, 2007). Chu and Harrison separated measurement data into outcome measures (e.g., anxiety, depression symptoms, global functioning) and process measures, or measures targeted by implementation strategies, into four broad mediator categories: cognitive, behavioral, physiological, and coping. In the 14 anxiety studies, CBT was found to produce a large effect size for behavioral measures (ES = 1.02), and moderate effect sizes for physiological (ES = 0.49), coping (ES = 0.73), and cognitive measures (ES = 0.50). Across the 14 depression studies, CBT was found to produce a small effect size for cognitive measures (ES = 0.35) with nonsignificant effects being found for behavioral or coping measures (no depression trials reported data relevant to physiological measures).

Findings from these meta-analyses provided useful knowledge relevant to identifying candidate mediators of CBT for youth anxiety and depression, however two important questions remain unexplored. First, the focus in both meta-analyses was placed on identifying candidate mediators in treatment programs only, with early intervention and prevention trials being excluded. As research emerges providing support that anxiety and depressive disorders are preventable psychiatric conditions, identifying potential mediators across prevention efforts is increasingly important. By understanding the mechanisms underlying prevention effects, programs could place a stronger emphasis on

these targets, potentially streamlining protocols in ways that can integrate with emerging electronic health technologies (e.g., smartphone applications, internet-based tools) and improve dissemination, sustainability, and cost-effectiveness in "real-world" conditions (i.e., schools, community centers, hospitals). Second, candidate mediators in these meta-analyses were organized around broad implementation strategies as opposed to constructs, processes, or theoretical mechanisms. For example, Chu and Harrison (2007) aggregated measures of self-esteem and negative cognitive errors into a "cognitive" candidate mediator. It is true that these measures are tapping on cognitive techniques employed by these interventions, but conceptually and psychometrically self-esteem and negative cognitive errors are distinct and can be targeted in a variety of ways using methods that could result in non-overlapping outcomes. Thus, for the proposed thesis, I plan to include anxiety and depression treatment and prevention programs. In addition, instead of organizing process measures around categories of implementation strategies, data will be separated into four constructs identified by anxiety and depression etiological and conceptual theories: (1) information-processing biases, (2) coping strategies, (3) social competence, and (4) physiological hyperarousal. The remainder of this section will review the empirical evidence (and theoretical rationale) for exploring these putative mediators in the context of youth anxiety and depression intervention research.

Information-Processing Biases

According to prominent cognitive theories, information-processing biases play a central role in the pathogenesis of behavioral and emotional difficulties (Beck, 1976; Beck & Clark, 1997; Crick & Dodge, 1994; Kendall, 1985; Kendall & Ingram, 1989). Broadly, these theories propose that information-processing is directed by cognitive

frameworks, or schemas, that determine how information is attended to, interpreted, and recalled. More specifically, and focusing on child anxiety and depression, Kendall (1985) adapted these models to psychotherapy and suggested that chronic activity of danger-related schemas results in information-processing resources that are chronically focused on threat-related information, which then produces dysfunctional and maladaptive thoughts that maintain feelings of anxiety. Beck and Clark (1988) proposed a similar model for depressive disorders in that maladaptive schemas results in dysfunctional processing of information which in return, frequently override more functional beliefs thereby confirming the faulty schemas and maintaining depressogenic symptoms and cognitions.

Building on these models, information-processing biases manifest in anxious and depressed youth in several ways including negatively interpreting neutral or ambiguous information, selectively attending to negative stimuli, overestimating the likelihood of negative events occurring, recalling negatively valence information, making internal, stable, and global attributions for interpersonal failures, and having more negative view of themselves and their future (Crick & Dodge, 1994; Daleiden & Vasey, 2001; Dalgleish et al., 2003 Garber & Weersing, 2010; Kendall & Chansky, 1991; Kendall & Ronan, 1990). These biases have been associated with greater levels of anxiety (Kendall & Chansky, 1991; Leitenberg, Yost, & Carrol-Wilson, 1986; Schniering & Rapee, 2002; Villabo et al., 2013) and depression (Horowitz et al., 2007; Kolko, Brent, Baugher, Bridge, & Birmaher, 2000; O'Kearney et al., 2009; Seligman et al., 1984) across the treatment and prevention literature. More specifically, Villabo et al. (2013) found that when compared to non-symptomatic peers, clinically anxious youth were found to make

significantly more negative statements about themselves. A greater number of these statements were also associated with more severe self-reported anxiety and greater functional impairment. Similarly, Seligman et al., (1984) observed a greater frequency of causal internal attributions for negative events (i.e. blaming an outcome on some aspect of themselves) was predictive of depressive symptoms in youth six months later. In another example, Leitenberg et al. (1986) found evidence suggesting that anxious and depressed youth tend to expect a disproportionate amount of negative outcomes and assign a low probability to effectively cope with such outcomes. Thus, attention toward and interpretation of negative information may exacerbate symptoms of anxiety and depression and in turn, likely intensifying attention to and interpretation of negative information thereby creating a feedback loop. It has been suggested that changes in this information-processing feedback loop are essential to reduce internalizing symptoms, thus disrupting anxiety and/or depressive disorder development (Garber & Weersing, 2010; Muris, Mayer, den Adel, Roos, & van Wamelen, 2009).

Anxiety and depression treatment and prevention programs consistently target the deficits above-mentioned using cognitive restructuring techniques. Cognitive restructuring focuses on making automatic and biased thoughts more controlled thus allowing anxious and depressed youth to more readily identify and challenge information-processing biases and replace them with more functional cognitions (Dozois, Seeds, & Collins, 2009; Kendall et al., 2003). Information-processing biases in anxious and depressed youth have been found to decrease following completion of treatment and prevention protocols (Bar-Haim, Morag, & Glickman, 2011; Horowitz et al., 2007; Jaycox, Reivich, Gillham, & Seligman, 1994; Silverman et al., 1999). In particular, Bar-

Haim et al., (2011) reported that after attention-bias training, highly anxious youth were better able to disengage their attention away from threat on an emotional attention spatial cueing task. In a depression prevention trial, post-intervention results indicated that depressed youth in the intervention condition were less likely to attribute negative events to stable and enduring causes (i.e., themselves) than youth in the no-treatment control condition (Jaycox et al., 1994). These findings are further complemented by nine mediation trials across the anxiety and depression intervention literature (Ackerson et al., 1998; Gilham et al., 1995; Kaufman et al., 2005; Jaycox & Seligman, 1995; Kendall & Treadwell, 2007; Lau et al., 2010; Shirk et al., 2013; Treadwell & Kendall, 1996; Yu & Seligman, 2001). For instance, three treatment trials (Kendall & Treadwell, 2007; Lau et al., 2010; Treadwell & Kendall, 1996) found that a decrease in negative self-referent cognitions preceded reductions in anxiety symptoms. Likewise, in a depression prevention trial, Jaycox and Seligman (1995) found that changes in attributional style mediated change in youth reported depressive symptoms. However, as noted earlier, these trials tested the mediator concurrently with the outcomes, thus preventing strong conclusions about temporal precedence. Nonetheless, these findings are in line with cognitive theories of anxiety and depression briefly discussed above, and thus support the mediational role of information-processing biases as proposed by this thesis study. To further knowledge gained by these RCTs and similar meta-analyses (Chu & Harrison, 2007; Prins & Ollendick, 2003), the present thesis plans to examine the impact of information-processing biases as a potential mediator at general and specific levels (i.e., internally focused biases [self-esteem, self-efficacy], externally focused biases [cognitive

errors relevant to external stimuli]). In this way, I hope to determine if specific biases are stronger potential mediators of intervention outcomes than others.

Coping Strategies

Coping can be defined as the strategies in which individuals are able to mitigate or handle the harmful effects of stress and everyday problems (Compas et al., 2001). Some coping strategies may serve as a buffing agent by regulating the negative emotions and outcomes associated with stressful events, thereby reducing the adverse psychological consequences of stress, whereas others may exacerbate the effects of stress and contribute to the development and maintenance of emotional and behavioral problems (Seiffge-Krenke & Klessinger, 2000). In this regard, researchers have made conceptual distinctions between adaptive and maladaptive coping strategies (Billings & Moos, 1981; Cronkite & Moos, 1995; Folkman & Lazarus, 1980; Seiffge-Krenke, 1995). Briefly, adaptive coping strategies refers to efforts to manage a problem or stressful event by actively seeking support from others, reflecting on possible solutions, and taking concrete actions towards solving the problem (Garnefski, Kraaij, & Spinhoven, 2001; Seiffge-Krenke & Klessinger, 2000). Adaptive coping strategies are considered to be a protective factor against the adverse effects of stress and are associated with better psychological adjustment, greater peer support, and lower rates of emotional and behavioral problems in youth (Garnefski, Kraaij, & Spinhoven, 2001). Conversely, maladaptive coping strategies refer to efforts to withdrawal from or deny the existence of the stressor, avoid seeking support from peers, and attempt to regulate stress-related emotions using cognitive coping strategies (e.g., rumination, self-blame, catastrophizing) (Garnefski et al., 2001). A robust relation between greater use of maladaptive coping

strategies and youth anxiety and depression is well documented in the empirical literature (Barrett, 1998; Burwell & Shirk, 2007; Hofman et al., 1992; Kendall, 1997; Lewinsohn et al., 1990) and meta-analyses (Chu & Harrison, 2007; Prins & Ollendick, 2003). Beyond this strong association, these strategies are related to reduced self-efficacy, poor social adjustment, and lead to increased stress and higher rates of anxiety and depression in youth, which in turn lead to a greater use of these types of coping strategies, creating a vicious feedback loop (Chan, 1995; Compas et al., 2001; Seiffge-Krenke, 2000; Tolor & Fehon, 1987). This feedback loop is consistent with theoretical models of coping and psychopathology (e.g., Asarnow, Carlson, & Guthrie, 1987; Cicchetti & Schnieder-Rosen, 1986; Nolen-Hoeksema, 1991) and empirical studies. To be more specific, Seiffge-Krenke (2000) found cross-sectional and longitudinal of effects on internalizing symptoms supporting this reciprocal process: maladaptive coping strategies increased concurrent anxiety or depressive symptoms, which then increased withdrawal or avoidant coping a year later. Therefore, disrupting this feedback loop by targeting maladaptive coping strategies may reduce the effects of stress thereby reducing anxiety and depression symptoms and diagnoses in youth.

Youth anxiety and depression treatment and prevention programs that include a coping component have consistently reported that following completion of treatment, maladaptive coping decreased and adaptive coping strategies were more highly endorsed via self-, parent-, and teacher-report measures (Barrett et al., 1996; Blalock et al., 2007; Essau et al., 2012; Kendall, 1994; Michl, McLaughlin, Shepherd, & Nolen-Hoeksema, 2013; Rivet-Duval, Heriot, & Hunt, 2011). For example, Rivet-Duval et al. (2011) evaluated a universal prevention program for depressed youth aimed at teaching

resources for managing stress, including adaptive coping strategies. Post-intervention results indicated significant changes in self-reported depressive symptoms and coping strategies. This is further complemented by four anxiety (Essau et al., 2012; Hogendoorn et al., 2014; Lau et al., 2010; Roberts et al., 2010) and six depression (Blalock et al., 2007; Michl et al., 2013; Moberly & Watkins, 2008; Roberts et al., 2010; Sarin, Abela, & Auerbach, 2005; Spence et al., 2003) studies that examined coping strategies within mediation models. Michl et al. (2013), for example, found that maladaptive coping strategies (e.g., rumination) mediated the longitudinal relations between self-reported stressors and symptoms of depression. Likewise, Hoogendorn et al. (2014) and Lau et al. (2010) reported that an increase in youth and parent reports of adaptive coping strategies (e.g., seeking support from peers) preceded change in youth anxiety symptoms. Together, these data suggest that examining the role of coping strategies in general, and adaptive and maladaptive coping strategies in particular as mediators within the present thesis study would be advantageous to better understand the role coping plays in the reversal and prevention of youth anxiety and depression.

Social Competence

Social competence can broadly be operationalized as the ability to interact with others in a way that is successful (e.g., does not violate social norms, values) and maximizes the rate of positive social reinforcement (Libet & Lewinsohn, 1973; Segrin, 1992). Social competence covers a wide array of abilities including eye-contact, clarity of speech, identifying appropriate moments to initiate a conversation, starting a conversation, selecting appropriate topics for conversation, leaving a conversation, being assertive, and requesting help or information (Spence, 2003). Theoretical views have

suggested that deficits in social competence can reduce peer-support, lead to lower quality of interpersonal relationships with peers, parents, and teachers, and may act as a risk factor for the development of anxiety and depression (La Greca & Harrison, 2005; Prinstein, Boergers, & Vernberg, 2001). That is, deficits in social competence may serve as a diathesis in the development of internalizing symptoms and disorders when combined with stressful events. In this way, social competence is related to coping behaviors in that individuals with strong social competence can garner the high quality and positive social support networks that are effective in buffering the negative consequences of stressful situations. Conversely, those with deficits in social competence are expected to (a) experience more stressors, and (b) have a reduced ability to secure assistance and social support for handling stressors when they do occur (Segrin, 2000). There is a substantial body of evidence indicating that deficits in social competence are highly common among anxious and depressed youth across self-, parent-, teacher-, and peer-report indices (Chan, 1997; Hamilton, Asarnow, & Tompson, 1997; Hops, Lewinsohn, Andrews, & Roberts, 1990; Flannery-Schroeder & Kendall, 2000; Roberts et al., 2010; Spence et al., 2000). Further, deficits in social competence has been linked to negative peer interactions, lower friendship satisfaction, lower self-esteem, increased feelings of social dissatisfaction, and greater feelings of loneliness or social isolation (Alfano et al., 2009; Altmann & Gotlib, 1988; Baker & Hudson, 2013; Biggs, Nelson, & Sampilo, 2010). Thus, deficits in social competence in anxious and depressed youth may lead to these related consequences, which can decrease the youth's rate of positive social reinforcement and minimize their opportunities to form and maintain healthy social networks, thereby exacerbating internalizing symptoms and avoidance behaviors relevant

to social situations (La Greca, 2001). Therefore, by targeting social competence within a larger intervention context, more adaptive tools can be taught and used during stressful events to disrupt the consequences associated with these difficulties, which may assist in reducing anxiety and depressive symptoms (Lewinsohn, 1974; Pahl & Barrett, 2007).

Interventions targeting youth anxiety and depression that include a social skills training component have consistently reported improvements in social competence and reductions in internalizing symptoms via self- and parent-report measures (Essau et al., 2012; Kaufman et al., 2005; Kendall, 1994; Kraag, Van Breukelen, Kok, & Hosman, 2009; Liddle & Spence, 1990; Spence et al., 2000). For example, Spence et al. (2000) utilized a CBT program that included social skills training and found that youth with social phobia reported significantly greater reductions in social anxiety symptoms and increases in social competence as compared to a waitlist control condition. Likewise, Mufson et al. (1994) examined the efficacy of IPT-A, a treatment protocol with a large focus on improving interpersonal skills and behaviors. Post-intervention results indicated statistically significant changes in self-reported depressive symptoms and overall social functioning, and across several social domains including school, friends, and family. These findings are further complemented by several studies that examined social competence within mediation models (e.g., Alfano et al., 2009; Baker & Hudson, 2013; Biggs, Nelson, & Sampilo, 2010; Essau et al., 2012; Horowitz, Garber, Ciesla, Young, & Mufson, 2007). Alfano and colleagues (2009), in particular, showed that reductions in subjective feelings of social isolation mediated reductions in in social anxiety symptoms in socially phobic youth. Moreover, an improvement in observer-rated social effectiveness during a role-play task with peers predicted a reduction in social anxiety

symptoms in socially phobic youth. Additionally, Biggs et al. (2010) reported that among a sample of adolescents, the association between anxiety and depressive symptoms was significantly mediated by peer acceptance and peer victimization. Together, these data provide support for the hypothesized role that social competence plays as a potential mediator of intervention response in the present thesis study.

Physiological Hyperarousal

Physiological hyperarousal is broadly defined as the overarousal of the sympathetic branch of the nervous system that manifests as somatic complaints (Joiner et al., 1999; Lang, Bradley, & Cuthbert, 1998; Watson et al., 1995). These complaints cover a wide array of physical symptoms including, but not limited to, headaches, stomachaches, muscle tension or pain, racing heart, cold sweats, hot flashes, chills, and unexplained fatigue (Crawley, 2011). Although physiological hyperarousal was originally hypothesized as a factor unique to anxiety (Clark & Watson, 1991), a significant correlation between physiological hyperarousal and youth depression also has been reported (Chorpita & Daleiden, 2002). A more recent investigation reported that heart rate variability and resting response, both physiological markers of hyperarousal, were unable to differentiate between anxiety and depression in adolescents (Greaves-Lord et al., 2007), suggesting that physiological hyperarousal is a common factor across both disorders. While no study has explicitly examined the role of physiological hyperarousal as a mediator of intervention response, the hypothesized mediational role of physiological hyperarousal and related somatic complaints in the current thesis study is consistent with the three-response system described by Lang et al. (1998) and Barlow's model of emotional disorders (2000). More specifically, these theoretical views broadly suggest

that heightened noncued arousal may prompt youth with anxiety or depressive symptoms to exhibit a greater physiological response during situations perceived as threatening or negative, thus resulting in more frequent and severe somatic complaints (Barlow, 2000; Lang et al., 1998).

A strong relation between somatic complaints and youth anxiety and depressive disorders has been well documented (Beidel, Christ, & Long, 1991; Bernstein, Massie, Thuras, & Perwein, 1997; Campo, Jansen-McWilliams, Comer, & Kelleher, 1999; Egger, Costello, Erkanli, & Angold, 1999; Kendall & Pimentel, 2003; Masia-Warner, Reigada, Fisher, Saborsky, & Benkov, 2009). In particular, Beidel, Christ, and Long (1991) found that anxious youth endorsed significantly more somatic complaints on self-report measures compared to non-anxious controls. Similarly, McCauley, Carlson, and Calderon (1991) reported that approximately 70% of clinically depressed youth had significant somatic symptoms as reported by self-report assessments and the frequency of somatic complaints increased with the severity of depression symptoms. These findings are complemented by the functional pain literature, wherein unexplained somatic complaints are consistently found to be associated with anxiety and depressive symptoms and diagnoses (Campo et al., 2004; Campo & Fritz, 2007; Egger et al., 1999). Campo and colleagues (2004) for example, found that within youths in primary care presenting with recurrent and unexplained abdominal pain, 80% met diagnostic criteria for an anxiety disorder and over 40% met criteria for depression. Beyond this robust association, reductions in somatic complaints, gastrointestinal symptom severity, diastolic and systolic blood pressure levels, and galvanic skin response has been linked to anxiety or depression symptom reductions in several treatment trials (e.g., Holley et al., 2013;

Logan & Simons, 2010; Masia-Warner et al., 2011; Ost, Svensson, Hellstrom, Lindwall, 2001; Weiss et al., 2013). Moreover, CBT interventions focused on reducing somatic complaints through psychoeducation and relaxation techniques also have been found to significantly reduce anxiety and depression symptoms (Masia-Warner et al., 2011; Sanders, Shepherd, Cleghorn, & Woolford, 1994). Although formal mediation tests were not conducted, indirect evidence from these trials supports the hypothesized meditational role of physiological hyperarousal and related somatic complaints in the current thesis study.

Meta-Analytic Path Analysis Approach

The present thesis seeks to address three main aims via meta-analyses of RCTs targeting anxiety and depression in youth across treatment and prevention efforts. The first aim will be to estimate effect size (ES) values of intervention efforts on primary targeted outcomes (i.e., anxiety, depressive symptoms and diagnoses) and for potential mediator constructs identified in the literature that are likely linked to changes in these primary outcomes. These include: (1) information-processing biases, (2) coping strategies, (3) social competence, and (4) physiological hyperarousal. It is hypothesized that anxiety interventions will produce larger ES values on primary outcomes and potential mediators as compared to interventions targeting depression. Additionally, it is expected that interventions will produce larger ES values for targeted outcomes (e.g., anxiety symptoms in interventions targeting anxiety) than for non-targeted outcomes (e.g., depressive symptoms in interventions targeting anxiety). The second aim will be to compare the magnitude of ES values for potential mediators across types of interventions to determine if they vary significantly between treatment and prevention efforts. The

hypothesis for this aim is that ES values for potential mediators will be greater for treatment than those in prevention. Finally, to advance knowledge gained from meta-analyses, the present thesis study will be the first in the youth anxiety and depression intervention literature to combine meta-analytic findings with path analyses. Meta-analytic path analysis is a highly informative strategy for testing theoretical models (Hunter & Schmidt, 2004; Shadish, 1996; Viswesvaran & Ones, 1995). This novel contribution will use published data to examine the temporal relation between change in each mediator and change in anxiety and/or depression outcomes. It is expected that changes in physiological hyperarousal will be a significant mediator in anxiety interventions but not depression interventions and changes in information-processing biases, coping strategies, and social competence will be significant mediators for both anxiety and depression interventions.

Chapter 4

METHODS

Study Search Procedures

Fifty-five studies were identified through two search strategies. First, several comprehensive psychotherapy and prevention reviews and meta-analyses (e.g., Cartwright-Hatton et al., 2004; Chu & Harrison, 2007; Compton et al., 2002; Fisak et al., 2011; Horowitz & Garber, 2006; In-Albon & Schnieder, 2006; Klein et al., 2007; Lewisohn & Clarke, 1999; Michael & Crowley, 2002; Merry et al., 2009; Mychailysyn et al., 2012; Prins & Ollendick, 2003; Reinecke et al., 1998; Reynolds et al., 2012; Silverman et al., 2008; Spielmans et al., 2007; Teubert & Pinquart, 2011; Sutton, 2007; Watanabe et al., 2007; Weersing et al., 2002; Weisz et al., 2007; Zalta, 2011) were evaluated and relevant studies were included in the pool for this research. Second, computer index searches were conducted using PsychINFO, MEDLINE, PubMed, and Web of Science using the following keywords: *depression, dysthymia, depressive, anxiety, generalized anxiety, separation anxiety, social phobia, social anxiety, specific phobia, CBT, cognitive-behavioral therapy, cognitive therapy, behavior therapy, exposure therapy, behavioral activation, attention bias, interpersonal therapy, social skills, RCT, randomized controlled trial, prevention, treatment, and early intervention*. The auto-explode option was used in computer searches as to ensure that all relevant topics within the broader categories were also included. Reference sections of identified studies also were reviewed for additional articles.

Study Selection

Studies were included based on the following inclusion criteria: (1) participants were between the ages of 6 and 18 years old; (2) participants were selected and included in the study on the basis of reliable and validated measures due to: (2a) clinical anxiety and/or depression, as evidenced by diagnoses and/or symptoms or (2b) increased risk for developing clinically significant anxiety and/or depressive symptoms or diagnoses; (3) random assignment to conditions was used; (4) reported means and standard deviations at three assessment points (i.e., pre-, post-, and follow-up) along theoretically relevant variables using empirically supported measures; and (5) condition protocols were clearly explained with intervention practice elements adequately specified. Studies were excluded if participants had a comorbid conduct disorder or other significant behavioral difficulties as intervention outcomes for anxiety and depression are likely to vary drastically in the presence of significant externalizing psychopathology. Further, to preserve independence, studies were excluded if the sample being assessed overlapped partially or completely with the sample of another included study. In such instances, the study that was conducted first or had more complete data relevant to the present study was included.

Validity Assessment

A significant concern of current peer review practices is that of the "file-drawer problem," which suggests that published studies are more likely to included statistically significant findings than those that are unpublished (Rosenthal, 1991). As such, in meta-analytic reviews, there may be a systematic upward bias due to the omission of studies reporting null findings and the resultant summary effect sizes may not accurately

represent true findings. To address the file-drawer problem and publication bias, the fail-safe N (FSN; Rosenthal, 1991) was calculated using the following formula:

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

In this formula, *K* is the total number of studies assessing outcome or mediator variables in the meta-analysis and *Z* is mean effect size attained from 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. This value offers an approximation of how resistant calculated effect sizes are to null effects.

Study Coding Procedures

The studies were coded on variables relevant to quantitative characteristics used to calculate effect sizes for anxiety and depression outcome measures and mediators as well as sample, intervention, and methodological characteristics selected on the basis on developmental theory, results from RCTs, and findings from previously published meta-analyses. The purpose of these coding and examining these characteristics is to elucidate moderators of intervention response to establish potential considerations for future randomized controlled trials. In terms of sample characteristics, percentage of female participants, percentage of White/Caucasian participants, and age descriptors were coded. Information related to intervention characteristics that also were coded including: (1) target disorder (i.e., anxiety, depression), (2) intervention type (i.e., treatment, prevention), (3) intervention duration (i.e., number and duration of sessions), (4) implementer characteristics (e.g., MS/PhD, teacher, school psychologist), (5) intervention setting (e.g., school, community, clinic, lab), (9) intervention condition format (i.e.,

individual, group, combined, internet-based), and (10) control group format (e.g., wait-list, no-treatment, education support, alternative intervention). These were coded either as continuous variables or as categorical dummy variables to determine whether effect sizes vary as a function of any of these variables (i.e., moderators of intervention response). Anxiety and depression outcome measures were included for effect size coding if they assessed anxiety or depressive symptoms. Measures assessing information-processing biases, coping strategies, social competence, or physiological hyperarousal were coded as mediators. Moreover, who reported on included outcome and mediator of change measures also was coded (i.e., youth report, parent report, clinician report).

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 and following training recommendations adapted from Lipsey and Wilson (2001) in cases of meta-analyses performed by a single analyst were used. First, approximately three weeks following the original coding of the studies, 100% of the studies were coded again in a separate database without access to the original coding file. Double-entered data was then checked against the original coding database. Inter-rater reliabilities calculated using Intra-class correlation (ICC) for variables capturing continuous measures of outcome and mediator data and Cohen's kappa (κ) for categorical variables met moderate to high quality standards (per criteria for Landis & Koch, 1977) for all coded variables (see Results section).

Effect Size Calculation

Individual studies frequently reported multiple measures of a construct; the inclusion of multiple measures per singular construct would violate assumptions of independence that underlie meta-analysis (Rosenthal, 1991), resulting in inflated sample sizes, and distorted standard error estimates. Per the recommendation of Lipsey and Wilson (2001) multiple effect sizes for a single construct within single studies were averaged. This was done prior to synthesis with effect sizes from other studies to ensure that each study only contributed a single effect size per construct.

To compute effect size estimates, the independent group pretest-posttest design (IGPP) procedure was used. The IGPP procedure was chosen over the more commonly used standardized mean gain (SMG; Becker, 1988) effect size because the SMG would have required the correlations amongst study variables for pre to post-intervention, post-intervention to follow-up, and pre-intervention to follow-up and none of the trials in the present study provided these values or the raw data needed to calculate them. In fact, only one study provided correlations amongst study variables (Gillham et al., 2012), but only at pre-intervention. As such the IGPP procedure was chosen. Much like the SMG, 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 such as passage of time, simply receiving therapeutic attention, or differences in experimental design (Hedges, 1982; Morris & DeShon, 2002). Cohen's *d* was calculated for all outcome and mediators at pre to post-intervention, post-

intervention to follow-up, and pre-intervention to follow-up assessment points using the following equation:

$$d_{\text{IGPP}} = \frac{\left(M_{Pre,\ Int} - M_{Post,\ Int}\right) - \left(M_{Pre,\ Cont} - M_{Post,\ Cont}\right)}{SD_{pre}}$$

In this formula, $(M_{Pre,\ Int} - M_{Post,\ Int})$ is the mean difference for the intervention group, $(M_{Pre,\ Cont} - M_{Post,\ Cont})$ is the mean difference for the control group for Group 2, and SD_{pre} is defined as:

$$s_p = \frac{(n_{G1} - 1)s_{G1}^2 + (n_{G2} - 1)s_{G2}^2}{(n_{G1} - 1) + (n_{G2} - 1)}$$

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). However, because the distribution of Cohen's *d* may be upwardly biased if it is based upon a collection of studies that include small sample sizes (e.g., $N <$ 20; Lipsey & Wilson, 2001), effect sizes were adjusted to yield Hedge's *g* (Hedges, 1981) using the following formula:

$$g_{\text{IGPP}} = \left[1 - \frac{3}{4N - 9}\right] d_{\text{IGPP}}$$

Additionally, to account for differences amongst sample size and variances, mean effect sizes were weighted by the reciprocal of the standard error to produce a more accurate estimate. To calculate this weight, the standard error for each effect size for each study must first be calculated using the following formula (Lipsey & Wilson, 2001):

$$SE_{sm} = \sqrt{\frac{n_{G1} + n_{G2}}{n_{G1} n_{G2}} + \frac{(g_{\mathrm{IGPP}})^2}{2(n_{G1} + n_{G2})}}$$

Then, the weights of each effect size were calculated using inverse variance weights, which is the reciprocal of the standard error and results in greater weight to be given to studies with less random variation as well as those with larger sample sizes:

$$w_{sm} = \frac{1}{SE_{sm}^2}$$

Positive effect size values reflect effects occurring in the expected direction (e.g., improved over the course of the intervention), whereas a negative effect size value reflects effects worsening overtime. Additionally, all effect sizes were calculated with a 95% confidence interval and interpreted using the standards established by Cohen (1988), in which effect sizes are considered small (0.2), medium (0.5), and large (0.8).

Homogeneity of Effect Sizes

An important step in examining and comparing effect sizes is testing the assumption that all effect size values are estimated from the same population or are homogeneous (Lipsey & Wilson, 2001). In a homogeneous distribution, it is expected that each individual effect size would diverge from the population mean effect size *only* as a result of sampling error. However, if the homogeneity assumption is violated, it is presumed that the variation in effect sizes is a result of a source beyond sampling error, including moderators and random differences that cannot be identified among the overall distribution of included studies. Homogeneity was tested using the *Q*-statistic:

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

Two statistical approaches can be applied to the synthesis and summarization of data as dictated by the primary goal of meta-analysis and results of the *Q* statistic; each differs in their approach to describe the inferences that can be made from the collection of studies being reviewed. A fixed effects approach is described as a model that assumes that between-study differences are due to sampling error alone (Cooper, Hedges, & Valentine, 2009). In contrast, a random effects model assumes that between-study differences are due to both sampling error and other sources of between-study variability, which provides broader generalizability of inferences and results (Cooper et al., 2009; Hedges, 1983; Rosenthal & DiMatteo, 2001). Borenstein, Hedges, Higgins, and Rothstein (2009) suggest that a fixed effects model should only be employed if it is thought that all studies included in the meta-analysis are functionally equal and the goal is to not generalize to other populations. Given that there are considerable methodological differences across the included studies and generalizing findings to populations beyond those under investigation, the present study adopted a random effects model approach for analyses.

Calculation of Mediation Effects

Because the causal steps approach outlined by Baron and Kenny (1986) tends to have low power for detecting mediation effects when the mediated effect and/or sample size is small, the product of the coefficients method was used (Fritz & MacKinnon, 2007; MacKinnon et al., 2002). The product of the coefficients method evaluates mediation by multiplying the path "a" and "b" coefficients and dividing by a standard error, with significance tests typically using a bias-corrected bootstrapped and asymmetric confidence interval to adjust for non-normal distributions resulting from multiplying path

coefficients (Fritz & MacKinnon, 2007; MacKinnon, Lockwood, & Williams, 2004; Toglifi & MacKinnon, 2011). In this case, mediation or indirect effects were tested using RMediation (Tofighi and MacKinnon, 2011), which requires regression coefficients and their standard errors. Several steps were taken to obtain the necessary regression coefficients and standard errors from the effect sizes derived from the meta-analytic procedures. First, the Hedge's *g* summary effect size for the pre to post path for each mediator and pre to follow-up path for targeted and non-targeted outcomes were transformed into correlation coefficients (*r*) using guidelines from Lipsey and Wilson (2001). Second, to obtain the estimated correlation between mediators and outcomes, a bivariate correlation was run between post to follow-up effect sizes for mediators and pre to follow-up effect sizes for outcomes using aggregated study effect sizes. This process provided each mediator with a three-variable correlation matrix. Using these matrices, single mediator path models were tested in MPlus software version 7.1 (Muthén & Muthén, 2012) to obtain the two coefficients and standard errors needed to test for mediation. That is, the standardized regression coefficient and standard error for the intervention to mediator path (pre to post; path a) and the standardized regression coefficient and standard error for the mediator to the target outcome (post to follow-up; path b), controlling for the intervention to target outcome relation (path c; see Figure 1). Full mediation occurred when the intervention effect (c') was reduced to non-significance after considering the mediator whereas partial mediation occurred when the intervention effect was smaller but still significant after controlling for the mediator. Because the sample size in each path varied, the harmonic mean of the sample size across each path was used per the recommendation of Viswesvaran and Ones (1995) was used to the

standard errors. The harmonic mean is equal to or less than an arithmetic mean and as a result, provides reasonably conservative estimates that are not as stringent as using the lowest *n* in a pairwise deletion matrix (Albarracin et al., 2005). Finally, regression estimates and standard errors were entered into RMediation to estimate the mediation effects and calculate 95% confidence intervals using the distribution of the product method. The indirect effect value can be considered a standardized effect size of the mediating variable (Preacher & Kelley, 2011) and can be interpreted using the standards established by Cohen (1988), in which effect sizes are considered small (0.01), medium (0.09), and large (0.25). Further and with regards to "real-world" or clinical significance, the indirect effect value represents changes in standard deviation units of the intervention effects on targeted or non-targeted outcome variables via the mediator variable(s).

Chapter 5

RESULTS

Descriptive Characteristics of Included Studies

The final sample of studies consisted of 28 anxiety studies (8 prevention, 20 treatment) and 30 depression studies (21 prevention, 9 treatment) with a total of 11,413 children and adolescents. Youth ages ranged from 6 to 17 years (M = 12.93, SD = 2.23), of which 54% were female. The number of sessions varied between 4 and 24 (M = 10.79, SD = 4.5) and the length of protocol sessions ranged from 40 to 120 minutes (M = 71.47, SD = 22.03) (additionally, three studies reported on the one-session, 180-minute, exposure protocol for specific phobias outlined by Ost and colleagues). The time between the end of the intervention and follow-up assessments ranged from 1 to 48 months (M = 9.98, SD = 8.18). In terms of program characteristics, 67.2% (n = 37) of studies reported on protocols delivered in group format, 41.8% (n = 23) individual format, and 3.6% (n = 2) combined group plus individual formats. Forty-one studies used a waitlist control, 13 studies compared an intervention to an active control condition (e.g., attention control, education support, nonspecific treatment), and 5 used an alternative evidence-based intervention as a comparison control (e.g., modified version of primary intervention condition). Approximately 71% (n = 39) of the studies used professionally trained interventionists as program leaders, with 45% of studies employing doctoral students, 27% using PhD level clinicians, and 8% utilizing school counselors, psychologists, and/or social workers. Parents were involved at the level of facilitator (e.g., assisting youth with skills practice) or co-participant (e.g., family therapy, parent management training) in 31% (n = 17) of the included studies. In terms of relevant statistical

methodology, 45% (n = 26) reported Cronbach alpha reliability coefficients for at least one measure in the study, with only 11 of those providing study-level reliability data for all measures used. Finally, only six studies conducted formal mediation tests (Ackerson et al., 1998; Cardemil, Reivich, & Seligma, 2002; Essau et al., 2012; Horowitz et al., 2007; Kraag et al., 2009; Yu & Seligman, 2002). Descriptive information about included studies is reported in Appendix A.

Focusing on the measures used to assess each of the mediator variables (see Appendix A), a majority of studies, 75% (n = 21) of anxiety studies and 83% (n = 25) depression studies, assessed information-processing biases. Thirty-five different measures were used, of which 33 were youth report and 2 were parent report, with 25% of the studies using the Children's Attributional Style Questionnaire (CASQ; 48-items; Seligman et al. 1984) or Children's Attributional Style Questionnaire-Revised (CASQ-R; 24-items; Kaslow & Nolen-Hoeksema, 1991). Coping strategies were measured in 39% (n = 11) of anxiety studies, and 27% (n = 8) of depression studies using 19 different measures; thirteen of these were youth-report, 7 were parent report, and 2 measures were clinician report. Twenty-one percent of studies (n =4) used the Coping Questionnaire (CQ-C/P; 3-items; Kendall, 1994), making it the most commonly used measure to assess cognitive strategies in the present study. About 36% (n = 10) of anxiety studies and 46% (n = 14) of depression studies assessed social competence. Twenty-five different measures were used, with 18 being youth report, followed by 5 parent report measures, and 2 clinician report measures. Thirty-eight percent of studies (n = 9) used the Matson Evaluation of Social Skills with Youngsters (MESSY; 62-items; Matson et al., 2010), Child and Adolescent Social and Adaptive Functioning Scale (CASAFS; 24-items; Price

et al., 2002), or the Social Adjustment Scale-Youth version (SAS-SRY; 17-items; Weissman, Orvaschel, & Padian, 1980). Physiological hyperarousal was assessed infrequently, with only 21% (n = 6) anxiety studies and 7% (n = 2) depression studies including measures of physiological hyperarousal. Ten different measures were used, 5 of which were youth self-report, 2 were objective (i.e., heart rate, blood pressure), 2 were parent report, and 1 was clinician report.

Appendix A also provides the outcome measures used across the included studies. In terms of anxiety outcome measures, 36 different measures were used, 21 of which were youth self-report, 9 were parent report, and 6 were clinician report. Twenty-nine percent (n = 16) of studies used the Revised Children's Manifest Anxiety Scale (RCMAS; 37-items; Reynolds & Richmond, 1985), 22% (n = 12) used the State-Trait Anxiety Inventory for Children (STAIC; 40-items; Spielberger, Edwards, Lushene, Montuori, & Platzek, 1973); and 16% (n = 9) used the Spence Child Anxiety Scale (SCAS; 44-items; Spence, 1998). With regards to assessing depression, 15 measures were used; of which 13 were youth self-report and 2 were parent report. Fifty-three percent (n = 29) of studies used the Children's Depression Inventory (CDI; 27-items; Kovacs, 1983).

Based on criteria for Landis and Koch (1977), 100% of the included studies were coded twice and entered into databases. Inter-rater reliability between the two coded databases was moderate to high ($0.86 > ICC > 0.94$) for continuous effect size outcomes and mediator data was substantial to almost perfect ($0.79 > \kappa \geq 1.0$) for categorical variables.

Homogeneity Analysis

Primary outcome effect sizes were evaluated using *Q*-tests and the I^2 statistic for each assessment point (i.e., pre to post, post to follow-up, pre to follow-up) to determine whether variation could be completely explained by sampling error within studies or whether the variation among effect size values reflects real and important differences between studies. The *Q*-test examines the null hypothesis that each outcome and mediator variable assessed across all studies share a common effect size, while the I^2 statistic provides an estimate of the proportion of observed variance that reflects true differences among effect sizes. In terms of the I^2 statistic, 25%, 50%, and 75% are generally used standards, signifying "low," "moderate," and "high" amounts, respectively, of the amount of variance accounted for by true differences.

Across the studies examining anxiety interventions, the null hypothesis that all studies share a common effect size was rejected for pre to post ($Q = 214.04$, $p < .0001$), post to follow-up ($Q = 54.47$, $p < .001$), and pre to follow-up ($Q = 279.32$, $p < .0001$) effect sizes indicating that the true effects vary. Additionally, the I^2 statistic indicated that approximately 50% to 90% of the observed variance is accounted for by true differences. Similarly, for depression intervention studies, the null hypothesis was rejected for pre to post ($Q = 154.06$, $p < .0001$), post to follow-up ($Q = 97.53$, $p < .0001$), and pre to follow-up ($Q = 117.07$, $p < .0001$), providing evidence indicating the true effects vary. Within these studies, the I^2 statistic indicated that roughly 70% to 80% of the observed variance is accounted for by true differences.

These results support the a priori decision to utilize a random effects model to calculate the mean effect sizes. In addition, these findings show that studies likely do not

share one true effect size and that factors that could impact effect sizes are not identical across all the studies included in meta-analyses. As a result, a series of analog to ANOVA tests were conducted for each of the outcome and mediators using disaggregated data to study characteristics that are accounting for sources of variance and to ascertain the levels of analysis moving forward. A significant Q_b value indicates the factor accounts for a significant amount of variance and likely moderates the effect size value. To identify the most robust moderators, the following criteria were used as decision points to move forward with analyses based on grouping sample characteristics: (1) moderators need to be significantly associated with more than 50% of the outcome and mediator variables; and (2) such association needs to occur across a majority of assessment points (i.e., two of the three considered in this research). Applying criteria (1) and (2), the following factors emerged as robust sources of variance: principal target problem (Q_b = 0.69 to 94.82, p < .05 to .01; anxiety vs. depression), intervention type (Q_b = 6.98 to 179.70, p < .01; treatment vs. depression), intervention format (Q_b = 4.98 to 91.12, p < .05 to .01; individual vs. group), intervention setting (Q_b = 7.34 to 126.10, p < .01; clinical vs. non-clinical), use of professional interventionist (Q_b = 5.37 to 83.84, p < .05 to .01; yes vs. no), and two mediator subtypes (Q_b = 4.48 to 9.87, p < .05 to .01; Information-processing biases: internally focused vs. externally focused information-processing biases; Q_b = 3.13 to 4.60, p < .05; Coping strategies: adaptive vs. maladaptive coping strategies). These factors were assumed to be confounded with intervention type because nearly all protocols were delivered using individual formats in clinical settings with professional interventionists and most preventative interventions were delivered with group formats in nonclinical settings. As a result, mean effect sizes were not

calculated and examined separately for intervention format, intervention setting, and use of professional interventionist. As a result, the focus was on intervention type, target disorder, and mediator subtypes. In addition, six factors did not meet the criteria: control group format, parent involvement, reporter, age, reported measurement reliability information, and time at follow-up. With regard to the latter, because follow-up duration consistently did not account for significant variance in effect sizes, post to follow-up and pre to follow-up effect sizes were calculated using aggregated follow-up duration data (the average post to follow-up was 8.53 months; the average pre to follow-up was 9.17 months). Results from the analog to ANOVA analyses are shown in Tables 1 to 3.

Intervention Effects on Targeted Outcomes

Consistent with previously published meta-analyses, pre to post-intervention effect sizes for targeted outcomes compared to control conditions (passive and active controls combined) were calculated (see Appendix B). Overall, treatment and prevention programs produced significant small to large effect sizes across anxiety and depression outcomes. Mean effect sizes for targeted outcomes in anxiety prevention studies was 0.22 and the mean effect size in anxiety treatment studies was 0.88 (*SDg* = 0.67). Pre to post intervention changes for anxiety treatment studies had an especially large 95% confidence interval (0.55 to 1.20). As such, the mean effect size value for anxiety treatment studies may be inflated, in part, because behavioral observations were included in the calculation of the mean effect size and this measurement type typically produces very large effect sizes. Further, although both mean effect sizes were significant, the variability was substantial across both anxiety prevention and treatment studies. More specifically, of the observed variance, only 18% of anxiety prevention studies and 12% of

anxiety treatment studies could be attributed to sampling error indicating there are likely several moderating influences on these outcomes. For depression trials, preventative interventions produced almost a one-quarter SD improvement over control conditions in depressive outcomes (g = 0.23; SDg = 0.26; 95% CI = 0.11 to 0.35) and half a SD improvement in depression treatment programs (g = 0.52; SDg = 0.52; 95% CI = 0.12 to 0.91). Much like the anxiety studies, only a small percentage of the observed variance across the prevention and treatment studies targeting depression could be attributed to sampling error (*%VarSE* = 17% and 28%, respectively).

Pre to follow-up effect sizes produced consistently significant effect sizes for targeted outcomes in interventions targeting anxiety and depression (see Appendix D). More precisely, pre to follow-up mean effect size for anxiety outcomes in prevention studies was 0.17 and 1.15 (SDg = 0.73; 95% CI = 0.79 to 1.51) for anxiety treatment studies. For anxiety prevention studies, the effect size decreased slightly (approximately 0.05 g-units) from pre-intervention to follow-up. However, there was a considerable increase of 0.27 g-units for anxiety treatment from pre-intervention to follow-up. Nonetheless, there are likely factors that are moderating these findings given the large residual standard deviations (*ResSD* = 0.19 and 0.71, respectively) and small percentages of variance attributed to sampling error. With regards to changes in primary outcomes across depression studies, preventative interventions targeting depression, the pre to follow-up mean effect size was 0.16, with depression treatment producing a mean effect size of 0.24. Consistent with previously discussed targeted outcome effect sizes, significant variability was observed across depression treatment and prevention studies, indicating the existence of moderating variables. Post-intervention to follow-up effect

sizes also were calculated for targeted outcomes; however, these were mostly non-significant as shown in Appendix C.

Intervention Effects on Mediators

Pre to post-intervention effect sizes were calculated for the mediator variables (see Appendix B). Overall, anxiety prevention studies had significant pre to post-intervention effects on four of the eight mediators: overall information-processing biases ($g = 0.38$), externally focused information processing biases ($g = 0.18$), overall coping strategies ($g = 0.13$), and maladaptive coping strategies ($g = 0.22$). Focusing on anxiety treatment, interventions showed statistically significant pre to post-intervention effects on each of the mediators ($g = 0.24$ to 0.92). Moving to depression, prevention programs produced significant pre to post-intervention effects for overall information-processing biases ($g = 0.18$), internally focused information-processing biases ($g = 0.23$), externally focused information-processing biases ($g = 0.10$), overall coping strategies ($g = 0.16$), and adaptive coping strategies ($g = 0.12$). Pre to post-intervention mean effect size for physiological hyperarousal could not be computed for depression prevention because only one study assessed this variable (at least two effect sizes from different studies are required). Moderate to large effects were found for pre to post-intervention changes for depression treatment programs on mediators, with overall information-processing biases ($g = 0.76$), internally focused information-processing biases ($g = 0.77$), and social competence ($g = 0.41$) producing significant effects. Only one study assessed for externally focused information-processing biases and physiological hyperarousal and no studies incorporated a measure of coping strategies in the depression treatment studies, thus, mean effect sizes for these variables could not be calculated. Fail-safe *N*

calculations for effect sizes for these variables across anxiety and depression interventions indicated that many were not very robust and could be altered by the presence of unidentified studies reporting null effects. However, this may be the result of only a small number of studies examining each of the mediator variables. In addition, the percentage of variance attributed to sampling error varies drastically across the mediators variables in both anxiety (*%VarSE* = 4.93% to 100%) and depression interventions (*%VarSE* = 28.8% to 100%) suggesting the need for future studies to examine potential moderators of these variables.

Appendix C shows post-intervention to follow-up mean effect sizes. In general, anxiety and depression interventions were associated with significant post-intervention to follow-up mean effect sizes across most of the mediators. Specifically, preventative interventions targeting anxiety produced significant changes in all mediators (g = 0.09 to 0.25), except adaptive coping strategies (g = 0.07; *SDg* = 0.02; 95% CI = -0.01 to 0.19). Anxiety treatment programs produced significant post to follow-up mean effects for overall information-processing biases (g = 0.18), internally focused information-processing biases (g = 0.43), overall coping strategies (g = 0.23), adaptive coping strategies (g = 0.25), and social competence (g = 0.35). Focusing on depression prevention, interventions had small to moderate effects (g = 0.11 to 0.32) on the mediators, of which internally focused information-processing biases (g = 0.10; 95% CI = -0.01 to 0.21) and social competence (g = 0.08; 95% CI = -0.05 to 0.21) were not statistically significant. Only overall information processing biases (g = 0.23) and internally focused information-processing biases (g = 0.28) had significant post to follow-up effect sizes within depression treatment studies. However, as previously

mentioned, for depression treatment, only one study reported externally focused information-processing biases and physiological hyperarousal and none reported changes in coping strategies; therefore, these indicators could not be meta-analyzed. The fail-safe *N* calculations suggest that two of the summary effect sizes for anxiety and depression prevention studies are not very robust: overall coping strategies (*FSN* = 1) and maladaptive coping strategies (*FSN* = 1). In contrast and unlike the pre to post intervention effect sizes, much of the variance in post to follow-up effects could be attributed to sampling error for anxiety interventions (*%VarSE* = 58.34% to 100%) but less so for interventions targeting depression (*%VarSE* = 4.61 to 100%).

Pre-intervention to follow-up effect size values for intervention effects on the mediators also were calculated and, in general, produced consistently significant findings (see Appendix D). More specifically, all of the mediators within the anxiety prevention studies (g = 0.09 to 0.41) and anxiety treatment studies (g = 0.59 to 1.42) had statistically significant pre to follow-up changes. Of note, anxiety treatment studies produced very large pre to follow-up changes in overall coping strategies and adaptive coping strategies. Again, these effect sizes may be inflated due to the inclusion of behavioral observations (observations tend to produce large effect size values). Small to moderate effects on the mediators were generated in depression prevention studies (g = 0.15 to 0.21), of which externally focused information-processing biases (g = 0.04; 95% CI = -0.03 to 0.11), overall coping strategies (g = 0.10; 95% CI = -0.01 to 0.20), and maladaptive coping strategies (g = 0.05; 95% CI = -0.08 to 0.17) were not statistically significant. Finally, for depression treatment studies, pre to follow-up effect sizes were significant for three mediators: overall information-processing biases (g = 0.67), internally focused

information-processing biases (g = 0.72), and social competence (g = 0.34) were significant. In terms of the fail-safe N calculations, pre to follow-up effect sizes appear to be consistently more robust than pre to post and post to follow-up. In addition, the percentage of variance accounted for by sampling error across all studies ranged from 10% to 100%, again indicating the likelihood that additional factors are influencing these effects.

Evidence of Mediation from Interventions to Outcomes

Using the methods described by MacKinnon et al. (2007), Tofighi and MacKinnon (2011), and Viswesvaran and Ones (1995), tests of the indirect effects were performed for all of the mediators using RMediation to ascertain whether they mediated the relation between interventions and primary outcomes. Table 4 presents these findings. Focusing on anxiety prevention studies, results indicate that changes in four of the mediators significantly mediated the relation between anxiety prevention programs and anxiety outcomes: overall information-processing biases (indirect effect = .006; 95% CI = .003, .010), internally focused information processing biases (indirect effect = .012; 95% CI = .006, .019), externally focused information processing biases (indirect effect = .007; 95% CI = .001, .014), and maladaptive coping strategies (indirect effect = .011; 95% CI = .004, .019). All of the significant indirect effects for anxiety prevention mediators were small in magnitude. Within anxiety treatment studies, significant small to moderate indirect effects for overall information-processing biases (indirect effect = .054; 95% CI = .026, .086), internally focused information-processing biases (indirect effect = .085; 95% CI = .018, .166), overall coping strategies (indirect effect = .071; 95% CI = .043, .104), and adaptive coping strategies (indirect effect = .064; 95% CI = .010, .122)

were statistically significant mediators between treatment programs and anxiety outcomes. It should be noted that all of the significant mediators within anxiety prevention and treatment partially mediated intervention effects on primary outcomes. That is, the direct path from the intervention to anxiety outcomes did not reduce to non-significance after taking into consideration the impact of the mediator on the outcomes.

Turning to depression prevention, changes in five mediators emerged as significant mediators between interventions and depressive symptoms: overall information-processing biases (indirect effect = .011; 95% CI = .001, .023), externally focused information-processing biases (indirect effect = .008; 95% CI = .003, .012), overall coping strategies (indirect effect = .003; 95% CI = .003, .014), adaptive coping strategies (indirect effect = .028; 95% CI = .016, .043), and maladaptive coping strategies (indirect effect = .005; 95% CI = .001, .011). In general, these mediators are small in overall magnitude, with intervention effects on depression outcomes improving by 0.01 to 0.03 standard deviation units via the mediating variables. Two mediators emerged as significant within depression treatment trials and both produced moderate indirect effects: overall information-processing biases (indirect effect = .17; 95% CI = .054, .308) and internally focused information processing biases (indirect effect = .13; 95% CI = .009, .262). This translates to depression treatments improving depression outcome effects by 0.13 to 0.17 standard deviation units via overall and internally focused information-processing biases. Coping strategies, physiological hyperarousal, and externally focused information-processing biases could not be examined as mediators between depression treatments and depression outcomes as no depression treatment studies reported coping strategies or physiological hyperarousal and only one study assessed externally focused

information-processing biases. Furthermore, and similar to anxiety interventions, all of the significant mediators within depression trials only partially mediated the relation between depression interventions and depression outcomes.

Tests of indirect effects also were conducted to examine whether these variables mediated the relation between interventions and non-targeted outcomes (see Table 5). That is, mediation from anxiety interventions on depression outcomes and from depression interventions on anxiety outcomes was examined. For anxiety prevention, changes in overall information-processing biases (indirect effect = .003; 95% CI = .002, .005), internally focused information-processing biases (indirect effect = .005; 95% CI = .003, .007), externally focused information-processing biases (indirect effect = .003; 95% CI = .001, .008), overall coping strategies (indirect effect = .004; 95% CI = .001, .008), and maladaptive coping strategies (indirect effect = .004; 95% CI = .001, .006) significantly mediated the relation between anxiety prevention programs and reductions in depressive symptoms. In contrast, only internally focused information-processing biases (indirect effect = .043; 95% CI = .006, .096) was a significant mediator for depression outcomes as a result of anxiety treatments. While these mediators emerged as significant, all with the exception of internally focused information-processing biases within anxiety treatment, do not meet the threshold for a small effect (i.e., 0.01; Cohen, 1988).

Focusing on depression prevention, changes in five of the mediators significantly mediated the relation between depression prevention programs and anxiety symptoms: internally focused information-processing biases (indirect effect = .003; 95% CI = .001, .005), externally focused information-processing biases (indirect effect = .004; 95% CI =

.001, .007), overall coping strategies (indirect effect = .006; 95% CI = .002, .010), adaptive coping strategies (indirect effect = .008; 95% CI = .003, .014), and maladaptive coping strategies (indirect effect = .004; 95% CI = .001, .008). For depression treatment, only internally focused information processing biases (indirect effect = .092; 95% CI = .004, .188) was a significant mediator between depression treatments and anxiety outcomes.

Chapter 6

DISCUSSION

The present thesis study combined meta-analytic and mediational path analysis to evaluate the effects of youth anxiety and depression intervention efforts on outcomes and on four theory-driven mediators using data from 55 randomized controlled trials (RCTs). More specifically, this research evaluated targeted outcomes (e.g., anxiety symptoms in anxiety interventions), non-targeted outcomes (e.g., depression symptoms in anxiety interventions), moderators of effects (e.g., intervention format, control group type) and four mediator variables. The mediators included: (1) information-processing biases (e.g., inaccurate and/or overly negative interpretations of events, internal attributions to external negative events), (2) coping strategies (e.g., behavioral and/or cognitive avoidance, active problem solving), (3) social competence (e.g., appropriate conversation skills, assertiveness, asking for help), and (4) physiological hyperarousal (e.g., increased somatic complaints; headaches, stomachaches, unexplained fatigue). In general, meta-analytic findings showed that treatment and prevention programs reliably produced moderate effect sizes on targeted outcomes and three of the four mediator variables (i.e., information-processing biases, coping strategies, social competence) and small effect sizes on non-targeted outcomes. Most important, mediational path analysis results showed that information-processing biases and coping strategies consistently mediated pre to follow-up changes in outcomes for anxiety and depression at both levels of intervention (e.g., treatment, prevention), whereas social competence and physiological hyperarousal did not emerge as statistically significant mediators.

These findings are robust and advance the research literature (including results from the other 23 published meta-analyses that reported on youth anxiety and depression interventions) for several reasons. First, and particularly noteworthy, the present study is the first in the youth anxiety and depression intervention literature to combine meta-analytic findings with mediational path analysis to estimate the indirect relations between each mediator variable and changes in outcomes. Separate assessment points for mediators (i.e., pre to post, post to follow-up) and outcomes (i.e., pre to follow-up) were used as opposed to using concurrent assessment points that reflects the extant literature (e.g., Ackerson et al., 1998; Essau et al., 2012; Kendall & Treadwell, 2007; Yu & Seligman, 2001). This approach provided a more robust framework to estimate indirect effects so that findings could be more confidently be used to guide possible revisions of intervention efforts or development of future streamlined interventions by focusing on the "significant" mediators in ways that can increase potency of effects and transportability for real-world or effectiveness settings. Second, conservative meta-analytic procedures (e.g., random effects modeling, inverse-variance weighting, sampling error corrections) were used to calculate intervention effects on outcome and mediator variables. This is in contrast to past published meta-analyses that relied exclusively on fixed effects modeling or weighted effect sizes by inverse of sample size only, which tend to overestimate program effects (e.g., Fisak et al., 2011; Spielmans et al., 2007; Stice et al., 2009). Third, in contrast to 18 of the 23 published meta-analyses, study-level effect sizes herein were calculated using the independent group pre–test post-test procedure (IGPP; Morris & DeShon, 2002). The IGPP procedure takes into account pre-existing differences between conditions, control group effects overtime, and experimental design differences (e.g.,

repeated measures, independent group designs). This approach increases confidence that changes in summary effect sizes for outcome and mediator variables can be attributed to intervention conditions and not a result of epiphenomenal factors (e.g., passage of time, therapeutic attention) or significant differences in experimental designs. Forth, separate pre to post, post to follow-up, and pre to follow-up effect sizes were calculated for outcomes and mediators across principal target problem and level of intervention. No meta-analysis to date has calculated post to follow-up effects, two have estimated effects for mediators separately from outcomes (Chu & Harrison, 2007; Prins & Ollendick, 2003), and only one focused on intervention effects for a both anxiety and depression (Chu & Harrison, 2007). Calculating effect sizes separately for each mediator and outcome variable across time, disorder, and intervention type allowed for a comprehensive examination of the "time" intervention effects seem to occur (e.g., pre to post and/or post to follow-up), for what variables, and for what disorder. Finally, separate effect sizes were calculated for specific facets of information-processing biases (internally vs. externally focused biases) and coping strategies (adaptive vs. maladaptive strategies). Although conceptual distinctions exist between these facets of information-processing biases and coping strategies, the two meta-analyses that have examined mediators separately from outcomes did not examine these mediator subtypes (Chu & Harrison, 2007; Prins & Ollendick, 2003). Doing so in the present thesis study allowed for the unique opportunity to shed light on the importance of intervention content specificity for targeting anxiety versus depression, if any.

Specific findings relevant to changes in outcomes and mediator variables, as well as limitations of the present meta-analytic path analysis approach, considerations for

future research, and implications for the treatment and prevention of youth anxiety and depression are discussed in the subsequent sections.

Information-Processing Biases

Interventions targeting youth anxiety and depression consistently produced moderate to large effects across information-processing biases, with findings being consistent with cognitive-behavioral theories and prior meta-analyses (Barlow, Allen, & Choate, 2004; Beck & Clark, 1997; Chu & Harrison, 2007; Prins & Ollendick, 2003). In addition, based on fail-safe *n* calculations, effect sizes observed in this research were robust and the presence of unidentified studies producing null effects would likely not alter these findings. Information-processing biases in general and internally focused biases (e.g., negative self-statements, negative self-views) in particular consistently mediated pre to follow-up changes to outcomes across principal target problem and level of intervention, with small to moderate indirect effect estimates. Moreover, internally focused biases significantly mediated pre to follow-up improvements to depression outcomes in interventions targeting anxiety and anxiety outcomes in interventions targeting depression, suggesting that this factor is a robust mediator for both disorders in terms of disorder reversal and prevention. Collectively, these findings are supported by both theoretical views emphasizing the importance of modifying distorted cognitions, especially those relevant to negative self-views (Beck & Clark, 1997; Enrenreich-May & Bilek, 2012; Kendall, 1985) and empirical evidence supporting the mediational role these constructs play in youth anxiety and depression interventions (e.g., Gillham et al., 1995; Kendall & Treadwell, 2007; Lau et al., 2010; Yu & Seligman, 2001).

Externally focused information-processing biases (e.g., negatively biased interpretations of neutral or ambiguous situations) also significantly mediated pre to follow-up changes to targeted and non-targeted outcomes for anxiety and depression preventative interventions. This was not the case for anxiety and depression treatment programs. This finding suggests that specific aspects of information-processing biases may mediate outcomes differently for prevention and treatment efforts such that type of bias is influenced by the developmental trajectory of anxiety and depressive disorders. For instance, at-risk youth might benefit more from improvements to externally focused biases because information-processing resources might not yet be as chronically focused on threat-related or negatively valenced information as with clinically anxious or depressed youth (Beck & Clark, 1988; Garber & Weersing, 2010; Kendall, 1985). Although this interpretation fits within conceptual models emphasizing the importance of externally focused information-processing biases in the development and maintenance of anxiety and depression (e.g., Beck, 1976; Daleiden & Vasey, 2001; Kendall & Ingram, 1989), prior mediational examinations of this construct have reported mixed results. For example, Stice et al. (2010) reported that across three depression prevention programs, externally focused information-processing biases did not mediated changes in depression outcomes. Kolko et al. (2000) reported similar findings for depression treatment programs. Conversely, both Kaufman et al. (2005) and Lau et al. (2010) found that within anxiety treatment efforts, modifying externally focused information-processing biases was a significant mediator of outcomes. However, with the exception of Stice et al. (2010), mediational analyses in these trials tested the mediator concurrently with the outcomes, which limits conclusions derived from these prior findings. Given the

relatively little research examining externally focused information-processing biases, more frequent measurement of this construct might be necessary to better understand its role in the treatment and prevention of youth anxiety and depression. For example, more RCTs evaluating change in externally focused information-processing biases would allow future meta-analytic path analyses to examine the mediational role of this construct in greater depth. Furthermore, because specific aspects of information-processing biases appear to mediate outcomes differently across levels of intervention, more treatment and prevention RCTs should measure change in both externally and internally focused biases utilizing distinct measures (e.g., Children's Automatic Thought Scale [CATS; Hogendoorn et al., 2010]; Children's Negative Cognitive Error Questionnaire [CNCEQ; Leitenberg et al., 1986]; Negative Affect Self-Statement Questionnaire [NASSQ; Ronan et al., 1994]). This multi-measure approach would enable future efforts combining meta-analytic results with mediational path analysis to examine these factors at greater depth to help further understand these differential findings and determine the unique and common contributions of these specific biases.

Coping Strategies

Youth anxiety treatment programs produced moderate to large effect sizes on coping strategies whereas anxiety and depression prevention efforts produced small to moderate effect sizes on coping strategies. These effect sizes are in accordance with previously published meta-analytic investigations (Chu & Harrison, 2007; Prins & Ollendick, 2003) and conceptual theories of anxiety and depression (Compas et al., 2001; Nolen-Hoeksema, 1991; Seiffge-Krenke, 2000). Turning to results from the mediational path analyses, coping strategies in general was a significant mediator for anxiety at both

levels of intervention and for depression prevention efforts. However, some differences emerged between specific types of coping strategies. That is, modifying maladaptive coping strategies (e.g., behavioral avoidance) was a significant mediator for anxiety and depression preventative interventions, but not anxiety treatment programs. Changing adaptive coping strategies on the other hand was a significant mediator for anxiety treatment on targeted outcomes and depression prevention on targeted and non-targeted outcomes but not for anxiety prevention. A possible explanation of these differences is that change in maladaptive coping strategies within anxiety treatment efforts and change in adaptive coping strategies in prevention efforts might occur at more long-term assessments given that increased usage of adaptive coping would likely decrease engagement in maladaptive coping and vice versa. This might be especially true given that the pre to post summary effect size for maladaptive coping in anxiety treatment was small whereas the pre to follow-up summary effect size was large in magnitude according to the standards established by Cohen (1988). A similar pattern was found for adaptive coping in anxiety prevention. Based on this interpretation, it is recommended that future RCTs assess change in general and specific aspects of coping strategies using longer-term follow-up assessments to identify how these specific strategies interact with each other and the impact this interaction may have on outcomes long after completion of the intervention.

Regarding depression interventions, the role of coping at general and specific levels in depression treatment is largely unknown. In the present meta-analysis, although all of the depression treatment studies utilized intervention strategies targeting coping (e.g., problem-solving, behavioral activation), none measured changes in the construct.

This was surprising given that coping strategies have long been identified as an important facet in conceptualizations of depression (Lewinsohn et al., 1990; Nolen-Hoeksema, 1991). As such, more frequent measurement and reporting of findings is necessary to fully understand the role of coping in the treatment of youth depression.

Social Competence

Anxiety and depression treatment programs produced moderate to large effect sizes for social competence across pre to post, post to follow-up, and pre to follow-up assessments. For preventative interventions, pre to post and post to follow-up changes in social competence were non-significant, with pre to follow-up effect sizes being small and likely altered by the presence of unidentified studies producing null effects (fail-safe n = 1 to 4). Although improving social competence has been identified as a central factor in reducing anxiety and depression, promoting mental health, and increasing resilience to stress (Alfano et al., 2009; Beidel, Turner, & Morris, 2000; La Greca & Harrison, 2005), changes in social competence was not as a significant mediator for anxiety or depression at either level of intervention. One possibility for these null findings may relate to failure of measurement issues. More specifically, social competence was frequently assessed using youth self-report measures that may prove problematic in measuring change over time given that social competence spans verbal and non-verbal skills. In fact, anxious and depressed youth may have a greater difficulty in identifying what social behaviors are acceptable and how well they can perceive themselves as having being competent in prior social situations (Yates et al., 2008). Furthermore, developmental research has found there to be a high degree of difficulty in accurately capturing change in non-verbal social competence using only self-reported data (Henderson & Meisels, 1994). Given that

changes in social competence in anxious and depressed youth may be difficult to capture using only self-report questionnaires, non-significant mediation findings in the present thesis study might reflect the use of measurement strategies that are not optimal or appropriate in capturing change. As a result, future RCTs targeting youth anxiety or depression that utilize intervention strategies targeting social competence should, at minimum, assess change in this construct using both questionnaire and behavioral observation (e.g., clinician, teacher, parent) methods to more accurately evaluate change.

Null findings also may suggest that social competence is not a direct mediator of outcomes. This is likely true in considering that after aggregating all studies that examined social competence to increase statistical power, social competence still failed to mediate outcomes. Instead, social competence might better be conceptualized as a long-term outcome given effect sizes for pre to follow-up changes were consistently significant across principal target problem and level of intervention, but not pre to post and post to follow-up changes. Alternatively, social competence maybe working in conjunction with another mediating variable such as coping strategies to impact planned outcomes. For instance, deficits in social competencies in anxious and/or depressed youth can lead to negative interactions with peers, whereby rates of positive social reinforcement are decreased thus minimizing youth opportunities to form and maintain healthy social networks, resulting in increased usage of maladaptive coping strategies (e.g., behavioral avoidance) relevant to social situations (La Greca, 2001). Together, this suggests that social competence, in isolation, may not mediate intervention effects. Instead, a bidirectional relation between coping strategies and social competence may exist in that increased social abilities facilitates reductions in avoidant behaviors and

these reductions in return improve social competence. This relation could then positively impact intervention outcomes. Thus, the role of social competence may be difficult to detect if assessed together with coping strategies. This emphasizes the need for continuous assessment of social competence during interventions and/or across multiple follow-up points (using youth self-report and/or observational methods) to better understand both the unique contribution, the potential interplay between this construct and other mediators, and how this may impact short and long-term anxiety and depression outcomes.

Physiological Hyperarousal

Physiological hyperarousal was not a significant mediator with summary effect sizes tending to be small and mostly non-significant. These null findings are likely due to four possibilities. First, though conceptualized as an important facet of youth anxiety and depressive disorders, only a small percentage of the included studies, 15% (n = 8), assessed changes in physiological hyperarousal. This greatly limited the ability to calculate reliable estimations of summary effect sizes and subsequent mediational effects. Second, the studies assessing physiological hyperarousal mostly relied on crude subjective estimates (e.g., youth self-report) that typically correlate poorly with changes in arousal states and somatic symptoms (Hoehn-Saric & McLeod, 2000), as opposed to objective measurement strategies (e.g., heart rate variability, galvanic skin response, blood pressure) that demonstrate greater accuracy in capturing physiological changes (Greaves-Lord et al., 2007; Laurent & Ettelson, 2001). Thus, more frequent assessment of physiological hyperarousal utilizing more robust and appropriate objective measures of arousal would likely provide a clearer and more accurate evaluation of the role this

construct plays between anxiety and depressive disorders, interventions, and planned outcomes. Third, physiological hyperarousal might not be a mediator, but rather an outcome variable. This possibility is consistent with recent emotion-focused theory and empirical data suggesting that, for some youth, changes in physiological hyperarousal during intervention efforts may not be necessary to produce immediate changes in planned outcomes, but rather reductions in physiological hyperarousal occurs over time (Chu et al., 2013; Greenberg, 2002; Tiwari et al., 2012). Finally, current intervention strategies targeting physiological hyperarousal (e.g., relaxation, guided imagery) may not yet be adequately developed to produce significant impact. As evidence emerges that anxiety and depression interventions produce inconsistent findings regarding significant change in physiological factors (Crawley et al., 2014; McKee et al., 2014), future research should reexamine the current strategies used to target this construct in youth-focused interventions to ensure they are appropriately modifying arousal states and somatic symptoms.

Limitations and Future Directions

While the present thesis advances knowledge regarding the mediators underlying treatment and prevention effects on youth anxiety and depression symptoms, three limitations are important to consider when interpreting the findings. First, as with all meta-analyses, results are limited to the studies included in the analyses. The inclusion criteria of the present review focused on identifying RCTs that met "robustness" criteria articulated by Chambless and Hollon (1998) and reported basic descriptive data (e.g., unadjusted means, standard deviations, and sample sizes) for both outcomes and mediator variables at pre, post, and follow-up assessment points. Unfortunately, 106 RCTs,

including prominent and methodologically rigorous anxiety (Child-Adolescent Anxiety Multimodal Study (CAMS); Walkup et al., 2008) and depression (Treatment for Adolescent Depression Study (TADS); The TADS Team, 2007) trials, as well as trials evaluating non-CBT interventions (e.g., acceptance and commitment therapy, interpersonal therapy) were excluded because they did not meet one or more of the criteria set by the present thesis study. For the CAMS and TADS trials, although findings advanced knowledge regarding the treatment of youth anxiety and depression, neither reported assessments of change for information-processing biases, coping, social, of physiological hyperarousal related variables. Instead studies evaluating the impact of these trials reported change across indices of diagnostic or broadband symptoms of affective disorders (e.g., Pediatric Anxiety Rating Scale, Child Behavior Checklist, Children's Depression Rating Scale-Revised). Turning to non-CBT interventions, only one trial using alternative theoretical frameworks met inclusion criteria for the present study (Horowitz et al., 2007). Thus, the number of studies was too low to make meaningful use of meta-analytic procedures to identify mediators likely unique to these approaches (e.g., cognitive and emotional acceptance in ACT; Arch & Craske, 2008). It is important that more research evaluates alternative intervention modalities as the information gained may provide useful insights into the phenomenological similarities between anxiety and depression as well as how to best serve youth that do not respond to traditional CBT protocols. Focusing on the other excluded trials, 18 studies did not measure or report data relevant to at least one mediator variable, 33 studies did not report the basic data necessary to compute effect sizes (e.g., unadjusted means, standard deviations, sample sizes), and 55 did not include a follow-up assessment. Future studies

need to report basic descriptive statistics for each experimental and control group, Furthermore, all of the studies excluded because they did not measure or report data for at least one mediator variable utilized common intervention strategies (e.g., cognitive restricting, exposures, relaxation, problem-solving skills practice, social skills building) that target each of the four mediators tested in the present review. Had these trials assessed and reported data for at least one mediator variable, findings from the present thesis study would be more representative of the body of literature testing interventions targeting anxiety and depression in youth. Prospective RCTs should provide more frequent measurement of putative mediator variables to improve understanding of how interventions are working and enhance future meta-analytic path analysis investigations.

Second, for included studies, there was a general lack of completeness of reporting of participant, intervention, and study-level information. More specifically, there was inconsistent reporting of participant characteristics (e.g., ethnicity, medication use, concurrent external treatment/prevention services, comorbidity), intervention details (e.g., dosage of intervention strategies), and study-level information (e.g., measurement reliability statistics, correlations between study variables, how attrition data was handled or considered). These missing data precluded more extensive moderator analyses on summary effect sizes as well as evaluations of factors that moderate the significant mediators (i.e., moderated mediation). Relevant to this, additional factors likely moderating the mediation effects as outlined by previously published RCTs and meta-analyses were planned (e.g., intervention format, use of professional interventionist, age group, gender, parental involvement), however there were not enough degrees of freedom within each factor to meaningfully analyze these on the pre to post and post to follow-up

paths, thus precluding the planned analyses. Therefore, additional evaluations of potential moderators are necessary before future meta-analytic path analyses in the anxiety and depression intervention literature can analyze the potential influence of these factors on significant mediators and determine under what conditions findings are true. Turning to study-level measurement reliability, only 20% (n = 11) of studies reported complete reliability information and only one study (Gillham et al., 2012) provided correlations amongst study variables, thus limiting the ability to correct for measurement error. 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 and correlational relations on summary effect sizes is unknown. The omission of such critical participant, intervention, and study-level details could limit conclusions regarding generalizability and robustness of effect sizes in meta-analyses in general and reduces overall confidence regarding inferences made about single studies in particular. Moving forward, studies should assess and report these details to enhance prospective meta-analytic investigations and allow for greater interpretation and generalizability of findings.

Finally, as with all indirect effect estimates derived from the meta-analytic path analyses, findings are based on correlational data. In addition, summary effect sizes were calculated and analyzed separately for disorder type and level of intervention given that they accounted for significant proportions of variance across variables and assessment points, however nearly all of the pre to post and pre to follow-up effect sizes indicated a

significant degree of heterogeneity. The use of heterogeneous samples is less than optimal (Shadish, 1996) and indicates potential study-level moderators. As a result, strong conclusions regarding causality cannot be made. Instead, significant mediators should be considered as potential directions for more in-depth examination in future randomized controlled trials (e.g., dismantling studies, additive studies, temporal or longitudinal studies; Kraemer et al., 2002; MacKinnon et al., 2013).

Recommendations and Implications

Based on the findings from the present thesis study, below are four factors to that the next generation of intervention research should consider when developing and testing treatment and prevention programs for anxious and/or depressed youth.

1. *Increase measurement and evaluation of supposed mediators of treatment and prevention outcomes.* The continued examination of youth anxiety and depression interventions has resulted in a considerable body of evidence demonstrating efficacy in disorder reversal and prevention. In fact, as exhibited in the present thesis study and consistent with prior meta-analyses (e.g., Fisak et al., 2011; Horowitz & Garber, 2007; Reynolds et al., 2012; Silverman, Pina, & Viswesvaran, 2008), treatment and prevention programs consistently produced moderate to large effect sizes on targeted (e.g., anxiety symptoms in anxiety interventions) outcomes and small to moderate effect sizes on non-targeted outcomes (e.g., depression symptoms in anxiety interventions). However, as noted previously, 18 RCTs were excluded from the present thesis study because they did not report data relevant to at least one mediator variable. Although these 18 interventions appeared to utilize strategies targeting these mediators, without measuring change in them, one cannot conclude that any of these

mediators accounted for change in outcomes. In addition, 89% (n = 49) of studies included in the present meta-analysis, did not conduct formal tests of mediation, thus missing a considerable number of opportunities to evaluate mediation effects. Thus, future RCTs should more frequently measure potential mediators *and* test them using robust mediation analysis 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.

2. *Specifically target and evaluate the mediational effects of information-processing biases and coping strategies on intervention outcomes.* Findings relevant to robustness of effect sizes and consistency of significant mediation effects across disorder and level of intervention suggest that increasing the dosage of strategies targeting information-processing biases and coping strategies at general and specific levels might represent the investment with the greatest potential to increase potency of intervention effects. Future treatment and prevention RCTs could be conducted comparing an intervention enhanced with strategies targeting information-processing biases and/or coping to the original intervention. Alternatively, current interventions could be augmented with promising electronic health technologies (e.g., smartphone applications, internet-based tools) to increase dosage of strategies targeting information-processing biases and/or coping strategies over and above what is

generally prescribed in the intervention and then compared to the original protocol without the technology component. In addition to assessing information-processing biases and coping strategies at pre and post-intervention, change should also be measured during the intervention, separately from the outcome to allow for establishing the precise sequence of change to establish temporal precedence. Following these recommendations, any demonstration of improved intervention effects favoring the intervention with greater dosage of strategies targeting information-processing biases and/or coping strategies would provide formal evidence that this construct mediates intervention outcomes and is most likely a causal mechanism (Kraemer et al., 2002; MacKinnon et al., 2002).

3. *Measure and evaluate mediational effects of specific types of coping strategies on anxiety intervention outcomes.* Differences emerged between specific types of coping strategies for anxiety treatment and prevention. Given that type of coping strategy mediated pre to follow-up changes differentially across level of intervention for anxiety, more in-depth examinations are needed. More specifically, prospective treatment and prevention RCTs should delineate intervention strategies targeting coping into distinct components of adaptive and maladaptive strategies and assess these constructs using measures that include distinct adaptive and maladaptive strategy subscales (e.g., Children's Coping Strategy Checklist [CCSC-R1; Ayers & Sandler, 1999]; Coping Response Inventory-Youth [CRI-Y; Moos, 1993]; Coping Scale for Children and Youth [CSCY; Brodzinsky et al., 1992]). Immediate post intervention and follow-up changes for each type of coping strategy could then be

compared to gain some sense about potential differences in effects and how to best leverage these differences to produce more potent intervention outcomes in the future.

4. *Integrate and measure change in strategies that target constructs identified as important to the development, maintenance, and reversal of youth affective problems.* Additional constructs identified as potential mediators could not be included in this meta-analysis because measurement of these factors across RCTs is scarce. In particular, a number of parenting factors, parental over-control, inter-parental conflict, decreased levels of autonomy granting, over-involvement, and reduced parental monitoring (Beardslee et al., 2003; Mcleod, 2007). Likewise, non-specific intervention factors such as therapeutic alliance, therapist adherence (i.e., the extent to which the techniques set by the intervention are followed) and competence (i.e., the skill in which the techniques are applied) have been suggested as important factors for anxiety and depressive symptom improvement in youth (Chu et al., 2004; Marker, Comer, Abramova, & Kendall, 2013; Shirk, Karver, & Brown, 2011; Webb, Auerbach, & DeRubeis, 2012). Most existing intervention efforts for anxiety and depression in youth have yet to specifically target or measure many of these factors, leaving the effects of improved parenting processes or implementation quality (e.g., alliance, adherence, competence) on youth distress largely unknown (Chu et al., 2004; Sandler, Schoenfelder, Wolchik, & MacKinnon, 2011). Therefore, more in-depth and frequent examination and measurement of these factors should be conducted. The information gained from these additional examinations would provide a clearer understanding into additional processes that might be underlying changes in

intervention outcomes. These data could then be used to inform the revision or development of interventions targeting youth anxiety and/or depression.

5. *Consider and evaluate mediators within the context of multiple mediator and/or cascading effects models.* Because the goal of the present thesis study was to evaluate overall mediational evidence, indirect effects were evaluated using single mediator models to preserve statistical power as to detect small mediational effects. However, multiple mediator models tend to be the rule rather than the exception (Kazdin & Nock, 2003) as intervention strategies are often non-orthogonal. That is, intervention strategies may impact more than one theoretical mediator. For example, reducing the use of maladaptive coping strategies will likely reduce avoidant coping patterns but may also provide the opportunity to disconfirm information-processing biases related to whatever stimuli was being avoided or provide more opportunities to engage with peers, thus improving social competencies. Relatedly, mediators may work with other mediators to improve intervention outcomes via a cascading model framework. For example, in the present thesis study, modifying adaptive coping strategies and internally focused information processing biases were significant mediators for anxiety treatment programs. Although not tested here, it might be the case that improving internally-focused biases provides youth with increased self-efficacy for handling stressful situations thus increasing the use of adaptive coping because youth feel more competent doing so, thereby leading to improvements in targeted outcomes. Therefore, future RCTs should evaluate mediators within a multiple mediator or cascading model framework. One possibility is for future treatment and prevention trials to conduct mediation analysis using cross-lagged panel models. Though this

method is typically employed with longitudinal data, recent applications have supported the utilization of cross-sectional designs that had at least three time points (Bollen & Brand, 2008), including pre, post, mid-intervention, and/or follow-up. This would allow for an examination of how specific mediators interact with other mediators and how this impacts targeted outcomes. Further, more frequent examination of mediators using this framework would then allow for future meta-analytic path analyses to synthesize findings and provide a more comprehensive overview regarding the interplay between mediators on outcomes across time.

Concluding Comments

The present thesis study marks a distinct departure from current meta-analytic investigations that commonly focus on the efficacy or effectiveness of interventions. While these meta-analyses have provided useful information demonstrating that CBT treatment and prevention efforts for anxiety and depression are efficacious, little attention has been paid to how these interventions work. Given the emphasis and need for more Type 2 translational research that examines ways evidence-based interventions can be more sustainable and readily integrated into clinical and non-clinical settings on a large scale (Spoth & Greenberg, 2005; Spoth et al., 2013), delineating the underlying factors producing change in anxiety and depression outcomes is critical to develop interventions that achieve these goals. The combination of meta-analytic findings and mediational path analyses allowed for the present thesis study to identify support for several mediators driving intervention outcomes. Findings from these meta-analytic path analyses conducted here have the potential to not only help in developing or selecting intervention targets for new protocols that have demonstrated a significant mediational effect on

outcomes, but also can assist in evaluating and improving existing interventions deemed effective or efficacious.

Although careful evaluation of changes or revisions to current interventions would be essential, by redirecting resources from factors that did not emerge as significant mediators (i.e., social competence, physiological hyperarousal) to targets that consistently mediated outcomes for both disorders and levels of intervention, such as modifying information-processing biases and coping strategies, effective and efficacious programs are likely to become more potent with longer lasting effects. Such revisions also may minimize costs relevant to adopting and implementing evidence-based interventions in real-world or effectiveness settings by shortening intervention length, reducing interventionist burden in training, and setting the stage for improved sustainability and large-scale diffusion capabilities.

Table 1. Analog to ANOVA Results for Pre to Post Effect Sizes

	Information-Processing Biases		Coping Strategies		Social Competence		Physiological Hyperarousal		Primary Outcomes		Secondary Outcomes	
Moderator	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b
Target Disorder												
Anxiety	0.29	0.79*	0.42	10.81**	0.74	5.18*	0.34	0.69	0.52	6.35*	0.15	0.21
Depression	0.23		0.15		0.23		0.19		0.28		0.19	
Intervention Type												
Treatment	0.49	13.24**	0.61	41.39**	0.81	9.92**	0.42	14.58**	0.59	23.26**	0.35	18.77**
Prevention	0.19		0.14		0.15		0.08		0.17		0.00	
Intervention Format												
Individual	0.46	7.70*	0.61	21.85**	0.80	3.85	0.42	14.58**	0.54	4.98*	0.34	10.75**
Group	0.21		0.19		0.35		0.08		0.35		0.05	
Control Condition												
Passive	0.36	1.23	0.39	1.07	0.45	0.10	-	-	0.41	3.07	0.04	0.50
Active	0.23		0.27		0.37		-		0.27		-0.02	
Intervention Setting												
Clinical	0.43	8.29*	0.68	36.33**	0.67	2.03	0.42	14.58**	0.53	7.62**	0.32	10.15**
Nonclinical	0.19		0.17		0.34		0.08		0.28		0.05	
Reported Reliabilities												
Yes	0.15		-		0.32	0.11	-	-	0.33	0.72	0.28	1.85
No	0.37	2.59	-	-	0.39		-		0.50		0.12	
Parent Involvement												
Yes	0.19	0.61	0.16	13.20**	0.83	2.75	0.35	1.79	0.47	0.22	0.24	4.42*
No	0.26		0.45		0.40		0.15		0.43		0.04	
Professional Interventionist												
Yes	0.32	5.45*	0.37	5.37*	0.58	2.24	0.25	1.43	0.50	7.68**	0.25	6.74**
No	0.17		0.18		0.12		0.06		0.19		0.01	
Reporter												
Child	0.25		0.20		0.37		0.18		0.31		0.18	
Parent	0.19	0.07	0.71	21.66**	0.38	9.02*	0.65	3.02	0.59	106.2**	0.07	0.43
Clinician	-		0.32		1.28		-		2.47		-	
Age Group												
Child	0.20	0.39	0.18	0.00	0.21	4.36*	-	-	0.44	0.04	0.10	3.03
Adolescent	0.26		0.18		0.49		-		0.46		0.30	
Mediator Subtype												
Subtype 1	0.33	4.48*	0.36	3.13*	-	-	-	-	-	-	-	-
Subtype 2	0.19		0.21		-		-		-		-	

Note: ES = Inverse-variance weighted mean effect size; Q_b = Between group Q statistic for homogeneity calculated using a maximum likelihood model; Subtype 1 for Information-Processing Biases = Internally focused information-processing biases; Subtype 2 for Information-Processing Biases = Externally focused information-processing biases; Subtype 1 for Coping Strategies = Adaptive coping strategies; Subtype 2 for Coping Strategies = Maladaptive coping strategies; - = Not enough degrees of freedom to analyze; Analyses conducted using disaggregated data;* $p < .05$, ** $p < .01$

Table 2. Analog to ANOVA Results for Post to Follow-Up Effect Sizes

	Information-Processing Biases		Coping Strategies		Social Competence		Physiological Hyperarousal		Primary Outcomes		Secondary Outcomes	
Moderator	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b
Target Disorder												
Anxiety	0.16	0.01	0.16	2.89	0.16	9.44**	0.13	0.02	0.14	20.49**	-0.02	2.39
Depression	0.17		0.09		0.00		0.12		-0.04		0.04	
Intervention Type												
Treatment	0.25	0.83	0.30	10.86**	0.20	6.98**	0.19	0.48	0.13	8.54**	0.11	7.88**
Prevention	0.15		0.10		0.04		0.12		0.01		-0.01	
Intervention Format												
Individual	0.27	0.67	0.28	6.03*	0.16	1.69	0.19	0.48	0.10	1.56	0.11	6.13**
Group	0.16		0.11		0.07		0.12		0.05		-0.01	
Control Condition												
Passive	0.22	0.11	0.24	2.65	0.20	5.43*	-	-	0.10	17.20**	0.01	2.31
Active	0.16		0.11		0.04		-		-0.10		-0.09	
Intervention Setting												
Clinical	0.22	0.20	0.29	7.34**	0.15	2.39	0.19	0.49	0.11	2.84	0.12	8.00**
Nonclinical	0.16		0.11		0.06		0.12		0.04		-0.01	
Reported Reliabilities												
Yes	0.13	0.35	-	-	0.18	1.66	-	-	0.08	0.52	0.03	0.16
No	0.28		-		0.07		-		0.02		0.01	
Parent Involvement												
Yes	0.22	0.22	0.17	3.01	0.08	0.02	0.19	1.70	0.12	4.53**	-0.02	0.88
No	0.16		0.10		0.09		0.19		0.03		0.02	
Professional Interventionist												
Yes	0.16	0.02	0.16	3.22	0.11	3.79	0.14	0.10	0.12	21.93**	0.04	1.13
No	0.18		0.09		-0.01		0.12		-0.10		0.00	
Reporter												
Child	0.17		0.11		0.08		0.13		0.06		0.03	
Parent	0.42	0.57	0.32	8.04*	0.07	0.74	0.46	2.26	0.05	0.32	-0.18	8.69**
Clinician	-		0.37		0.17		-		0.13		-	
Age Group												
Child	0.17	0.03	0.10	0.68	0.13	3.59	0.12	0.01	0.11	3.39	0.02	0.00
Adolescent	0.19		0.07		0.02		0.14		0.02		0.02	
Putative Mediator Subtype												
Subtype 1	0.22	0.97	0.14	1.86	-	-	-	-	-	-	-	-
Subtype 2	0.13		0.09		-		-		-		-	
Follow-up Duration												
0 to 12 months	0.19	1.60	0.11	3.34	0.09	1.03	-	-	0.08	4.26*	0.01	0.01
> 12 months	-0.03		0.22		-0.06		-		-0.09		0.02	

Note: ES = Inverse-variance weighted mean effect size; Q_b = Between group Q statistic for homogeneity calculated using a maximum likelihood model; Subtype 1 for Information-Processing Biases = Internally focused information-processing biases; Subtype 2 for Information-Processing Biases = Externally focused information-processing biases; Subtype 1 for Coping Strategies = Adaptive coping strategies; Subtype 2 for Coping Strategies = Maladaptive coping strategies; - = Not enough degrees of freedom to analyze; Analyses conducted using disaggregated data;* $p < .05$, ** $p < .01$

Table 3. Analog to ANOVA Results for Pre to Follow-Up Effect Sizes

	Information-Processing Biases		Coping Strategies		Social Competence		Physiological Hyperarousal		Primary Outcomes		Secondary Outcomes	
Moderator	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b	*ES*	Q_b
Target Disorder												
Anxiety	0.36	18.94**	0.60	29.58**	0.64	14.74**	0.26	0.01	0.74	94.82**	0.26	5.60**
Depression	0.13		0.11		0.21		0.27		0.16		0.10	
Intervention Type												
Treatment	0.60	43.37**	0.90	105.6**	0.85	63.08**	0.57	18.82**	0.81	130.6**	0.47	179.7**
Prevention	0.14		0.11		0.15		0.19		0.16		-0.02	
Intervention Format												
Individual	0.46	12.82**	0.87	34.03**	0.80	16.72**	0.57	18.82**	0.76	52.07**	0.45	91.12**
Group	0.17		0.21		0.28		0.19		0.33		0.01	
Control Condition												
Passive	0.50	8.89**	0.50	1.34	0.52	4.36**	-	-	0.57	26.24**	0.13	4.93*
Active	0.18		0.32		0.29		-		0.23		-0.04	
Intervention Setting												
Clinical	0.44	22.83**	0.98	64.21**	0.76	24.37**	0.57	18.82**	0.75	71.29**	0.44	126.1**
Nonclinical	0.13		0.17		0.22		0.19		0.23		-0.01	
Reported Reliabilities												
Yes	0.19	0.73	-	-	0.34	0.05	-	-	0.35	0.68	0.27	0.42
No	0.29		-		0.31		-		0.53		0.18	
Parent Involvement												
Yes	0.42	11.03**	0.64	35.57**	0.55	1.78	0.37	3.47	0.71	30.43**	0.20	0.40
No	0.17		0.12		..3623		0.21		0.37		0.16	
Professional Interventionist												
Yes	0.32	24.41**	0.56	30.79**	0.48	7.72**	0.28	2.78	0.67	83.89**	0.31	38.93**
No	0.09		0.09		0.11		0.18		0.04		-0.02	
Reporter												
Child	0.20	1.92	0.20	48.76**	0.31	28.09**	0.23	6.83**	0.37	158.5**	0.18	0.74
Parent	0.52		1.05		0.35		0.83		0.77		0.09	
Clinician	-		0.68		1.36		-		2.53		-	
Age Group												
Child	0.18	0.07	0.15	0.89	0.27	0.78	-	-	0.55	14.80**	0.09	1.56
Adolescent	0.20		0.21		0.34		-		0.28		0.18	
Putative Mediator Subtype												
Subtype 1	0.31	9.87**	0.46	4.60*	-	-	-	-	-	-	-	-
Subtype 2	0.15		0.25		-		-		-		-	
Follow-up Duration												
0 to 12 months	0.24	6.67**	0.37	3.21	0.43	1.28	-	-	0.57	13.92**	0.19	4.16*
> 12 months	0.06		0.01		0.20		-		0.12		-0.01	

Note: ES = Inverse-variance weighted mean effect size; Q_b = Between group Q statistic for homogeneity calculated using a maximum likelihood model; Subtype 1 for Information-Processing Biases = Internally focused information-processing biases; Subtype 2 for Information-Processing Biases = Externally focused information-processing biases; Subtype 1 for Coping Strategies = Adaptive coping strategies; Subtype 2 for Coping Strategies = Maladaptive coping strategies; - = Not enough degrees of freedom to analyze; Analyses conducted using disaggregated data; * $p < .05$, ** $p < .01$

Table 4. Indirect Effect Estimates for Targeted Outcomes

Mediating Variable	Harmonic Mean	Intervention to Mediator Coefficient (*SE*)	Mediator to Outcome Coefficient (*SE*)	Indirect Effect	95% Confidence Interval
Anxiety Prevention					
Information-Processing Biases-Overall	2242	.04 (.004)	.16 (.044)	.006[sig]	[.003, .010]
Information-Processing Biases-Internal	1527	.05 (.004)	.26 (.062)	.012[sig]	[.006, .019]
Information-Processing Biases-External	1865	.05 (.012)	.15 (.060)	.007[sig]	[.001, .014]
Coping Strategies-Overall	3123	.06 (.065)	.23 (.074)	.014	[-.016, .050]
Coping Strategies-Adaptive	2817	.02 (.009)	.33 (.195)	.006	[-.001, .018]
Coping Strategies-Maladaptive	3123	.03 (.006)	.32 (.093)	.011[sig]	[.004, .019]
Social Competence	1597	.05 (.033)	.17 (.065)	.008	[-.002, .024]
Physiological Hyperarousal	2624	.12 (.078)	.22 (.101)	.026	[-.007, .079]
Anxiety Treatment					
Information-Processing Biases-Overall	975	.11 (.028)	.49 (.061)	.054[sig]	[.026, .086]
Information-Processing Biases-Internal	307	.18 (.070)	.48 (.094)	.085[sig]	[.018, .166]
Information-Processing Biases-External	855	.03 (.220)	-.87 (.361)	-.026	[-.466, .396]
Coping Strategies-Overall	842	.14 (.015)	.51 (.096)	.071[sig]	[.043, .104]
Coping Strategies-Adaptive	476	.19 (.019)	.33 (.142)	.064[sig]	[.010, .122]
Coping Strategies-Maladaptive	450	.14 (.052)	-.08 (.164)	-.011	[-.066, .037]
Social Competence	520	.26 (.026)	-.05 (.065)	-.013	[-.047, .020]
Physiological Hyperarousal	183	.17 (.060)	.09 (.142)	.015	[-.034, .073]
Depression Prevention					
Information-Processing Biases-Overall	4919	.19 (.090)	.06 (.006)	.011[sig]	[.001, .023]
Information-Processing Biases-Internal	1970	.004 (.008)	.10 (.052)	.0004	[-.001, .002]
Information-Processing Biases-External	4648	.13 (.038)	.06 (.006)	.008[sig]	[.003, .012]
Coping Strategies-Overall	5289	.08 (.014)	.10 (.029)	.008[sig]	[.003, .014]
Coping Strategies-Adaptive	5008	.09 (.018)	.32 (.033)	.028[sig]	[.016, .042]
Coping Strategies-Maladaptive	3049	.04 (.007)	.14 (.061)	.005[sig]	[.001, .011]
Social Competence	3477	.06 (.015)	.04 (.021)	.002	[.000, .006]
Physiological Hyperarousal	-	-	-	-	-
Depression Treatment					
Information-Processing Biases-Overall	406	.60 (.079)	.29 (.100)	.170[sig]	[.054, .308]
Information-Processing Biases-Internal	406	.54 (.070)	.24 (.114)	.130[sig]	[.009, .262]
Information-Processing Biases-External	-	-	-	-	-
Coping Strategies-Overall	-	-	-	-	-
Coping Strategies-Adaptive	-	-	-	-	-
Coping Strategies-Maladaptive	-	-	-	-	-
Social Competence	269	.45 (.134)	-.01 (.183)	-.005	[-.180, .169]
Physiological Hyperarousal	-	-	-	-	-

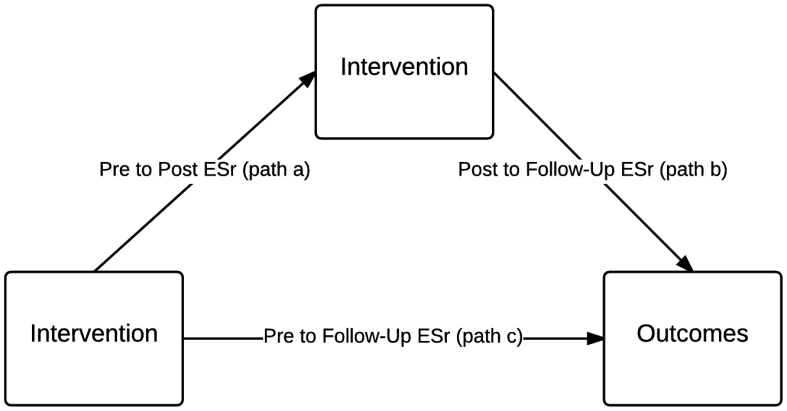
Note: [sig] = Significant indirect effect; - = not enough data to analyze

Table 5. Indirect Effect Estimates for Non-Targeted Outcomes

Mediating Variable	Harmonic Mean	Intervention to Mediator Coefficient (*SE*)	Mediator to Outcome Coefficient (*SE*)	Indirect Effect	95% Confidence Interval
Anxiety Prevention					
Information-Processing Biases-Overall	2077	.04 (.004)	.08 (.016)	.003[sig]	[.002, .005]
Information-Processing Biases-Internal	1448	.05 (.004)	.10 (.022)	.005[sig]	[.003, .007]
Information-Processing Biases-External	1749	.05 (.012)	.06 (.021)	.003[sig]	[.001, .006]
Coping Strategies-Overall	2811	.05 (.016)	.08 (.026)	.004[sig]	[.001, .008]
Coping Strategies-Adaptive	2561	.02 (.010)	.12 (.069)	.002	[.000, .007]
Coping Strategies-Maladaptive	2811	.03 (.006)	.12 (.033)	.004[sig]	[.001, .006]
Social Competence	1512	.05 (.034)	.06 (.022)	.003	[-.001, .009]
Physiological Hyperarousal	2400	.12 (.082)	.08 (.036)	.010	[-.003, .029]
Anxiety Treatment					
Information-Processing Biases-Overall	784	.11 (.032)	.09 (.057)	.010	[-.002, .026]
Information-Processing Biases-Internal	286	.18 (.073)	.24 (.08)	.043[sig]	[.006, .096]
Information-Processing Biases-External	704	.03 (.005)	-.27 (.333)	-.008	[-.029, .012]
Coping Strategies-Overall	696	.14 (.016)	.05 (.088)	.007	[-.017, .032]
Coping Strategies-Adaptive	426	.19 (.021)	-.02 (.126)	-.004	[-.051, .044]
Coping Strategies-Maladaptive	404	.14 (.055)	.02 (.144)	.003	[-.041, .049]
Social Competence	460	.26 (.027)	.07 (.057)	.018	[-.011, .048]
Physiological Hyperarousal	175	.17 (.061)	.13 (.121)	.022	[-.018, .074]
Depression Prevention					
Information-Processing Biases-Overall	4106	.19 (.033)	.003 (.003)	.001	[-.001, .003]
Information-Processing Biases-Internal	1825	.04 (.008)	.07 (.026)	.003[sig]	[.001, .005]
Information-Processing Biases-External	3915	.13 (.041)	.03 (.003)	.004[sig]	[.001, .007]
Coping Strategies-Overall	4421	.08 (.015)	.07 (.021)	.006[sig]	[.002, .010]
Coping Strategies-Adaptive	4223	.08 (.019)	.10 (.023)	.008[sig]	[.003, .014]
Coping Strategies-Maladaptive	2739	.04 (.007)	.10 (.041)	.004[sig]	[.001, .008]
Social Competence	3049	.06 (.026)	.04 (.011)	.002	[.000, .005]
Physiological Hyperarousal	-	-	-	-	-
Depression Treatment					
Information-Processing Biases-Overall	344	.60 (.086)	.11 (.073)	.066	[-.020, .159]
Information-Processing Biases-Internal	365	.61 (.086)	.15 (.073)	.092[sig]	[.004, .188]
Information-Processing Biases-External	-	-	-	-	-
Coping Strategies-Overall	-	-	-	-	-
Coping Strategies-Adaptive	-	-	-	-	-
Coping Strategies-Maladaptive	-	-	-	-	-
Social Competence	241	.45 (.142)	-.02 (.129)	-.009	[-.135, .113]
Physiological Hyperarousal	-	-	-		

Note: [sig] = Significant indirect effect; - = not enough data to analyze

Figure 1. Meta-Analytic Path Model Structure



Note: ESr= Effect Size Correlation; Putative Mediators = Information-Processing Biases, Coping Strategies, Social Competence, Physiological Hyperarousal; Outcomes = Anxiety and/or depression symptoms

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APPENDIX A

DESCRIPTIVE CHARACTERISTICS OF INCLUDED STUDIES

Table 1 Descriptive Characteristics of Included Studies ($N = 55$)

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Targeted Outcome Measures	Non-Targeted Outcome Measures
Ackerson et al., 1998	Depression	Treatment	22	15.91	63.6	DAS ATQ				CBCL-Dep CDI HRSD	
Barrett et al., 2003	Anxiety	Prevention	320	12.3	47.8	BHS KHS RSES SEI					RCMAS
Beidel et al., 2000	Anxiety	Treatment	67	10.5	60	EPQ-N LS	EPQ-E RPE RAE RPA RAA			ADIS-C CBCL-Int SPAI-C STAIC-S STAIC-T	CDI
Cardemil et al., 2002	Depression	Prevention	152	NR	50.5	ATQ CASQ-R WIAL HS				CDI	
De Cuyper et al., 2004	Depression	Treatment	20	10	75	SPPC-GSW				CBCL-Int CDI	STATIC-T
Dobson et al., 2010	Anxiety/ Depression	Prevention	46	15.28	69.5	RSES				CBCL-Anx/Dep BAI STAIC-T MASC-Anx MASC-Dep CES-D CDI BAI	
Essau et al., 2012	Anxiety	Prevention	638	10.91	45.7	CAPS (self)	CASAFS CAPS (social)	SSQ (P) CSCY	SCAS (physio)	SCAS	RCADS
Flannery-Schroeder & Kendall, 2000	Anxiety	Treatment	37	NR	49	LS	FM-C SPPC	CQ-C/P		CBCL-Int RCMAS SASC-R STAIC (P) STAIC-S STAIC-T TRF	CDI
Garcia-Lopez et al., 2002	Anxiety	Treatment	59	15.92	77.97	SAS-A-FNE		SAS-A-NEW SAS-A-GEN		CSR SPAI-B	

Table 1. continued

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Targeted Outcome Measures	Non-Targeted Outcome Measures
Gillham et al., 2007	Depression	Prevention	271	NR	NR	CASQ				CDI	
Gillham et al., 2012	Depression	Prevention				CASQ HSC		CCSC		CDI RADS	RCMAS
Hains, & Szyjakowski, 1990	Anxiety	Treatment	21	NR	0	CSE ASSQ Thought Listing				STAIC-S STAIC-T	BDI
Hains, 1992	Anxiety	Treatment	25	NR	0	CSE ASSQ				STAIC-S STAIC-T	RADS
Harnett & Dadds, 2004	Depression	Prevention	212	13.58	100	HSPPA	SCS	ACS		RADS	RCMAS
Herbert et al., 2009	Anxiety	Treatment	73	14.7	57.6		BAT (clinician) BAT (child)			BAT (suds rating) SAS-C/P SPAC-C	
Horowitz et al., 2007	Depression	Prevention	380	14.43	54	CASQ-R	CBQ-P	COPE		CDI CES-D	
Ingul et al., 2014	Anxiety	Treatment	128	14.50	56.14	STABS				SCARED SPAI-C	CDI
Kendall, 1994	Anxiety	Treatment	47	NR	40	NASSQ	TBO CBCL-Social	CQ-C/P	CBCL-Health	CBCL-Int FSSC-R RCMAS STAIC-S STAIC-T TCBCL-Int	CDI
Kendall et al., 1997	Anxiety	Treatment	94	NR	38	NASSQ	TBO	CQ-C/P		CBCL-Anx/Dep CBCL-Int FSSC-R RCMAS STAIC (P) STAIC-T TRF-Int TRF-Anx/Dep	CDI
Kendall et al., 2008	Anxiety	Treatment	161	NR	44			CQ-C/P		CBCL-Anx CBCL-Int MASC TRF-Anx TRF-Int	

Table 1. continued

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Primary Outcome Measures	Secondary Outcome Measures
King et al., 1998	Anxiety	Treatment	34	11	47	SEQSS				CBCL-Int FSSC-II FT RCMAS TRF-Int	CDI
Kowalenko et al., 2005	Depression	Prevention	126	14.5	63	CATS		ACS		CDI	
Kraag et al., 2009	Anxiety/ Depression	Prevention	1467	10.3	49.9		SPSI-Social Support	SPSI	MUSIC	STAIC-T	SDIC
Lock & Barrett, 2003	Anxiety	Prevention	733	NR	NR	RSES		CSCY		RCMAS SCAS	CDI
Masia Warner et al., 2005	Anxiety	Treatment	35	14.8	74.3	LS SAS-A (FNE)		SAS-A (General) SAS-A (New) LSAS-CA-Social Avoidance LSAS-CA-Performance Avoidance		SPAI-C SPDSCF LSAS-CA-Social Anxiety	CDI
Masia Warner et al., 2007	Anxiety	Treatment	36	15.1	83	SAS-A (FNE) (P) SAS-A (FNE) (C)		SAS-A (General)-C/P SAS-A (New)-C/P		SPAI-C	BDI-II
Masia Warner et al., 2011	Anxiety	Treatment	40	12.4	65				GI-CSI PAIN	SCAS PSR	
McLoone & Rapee, 2012	Anxiety	Treatment	152	9.7	61.8	CATS				AIQ (P) SAS (T) SCAS SDQ (T)	
Merry et al., 2012	Depression	Treatment	187	15.56	65.7	HPLS			SCAS (physio)	CDRS-R MFQ RADS-2	SCAS
Mifsud & Rapee, 2005	Anxiety	Prevention	91	9.5	59	CATS				SCAS-C/P TRF-Int	
O'Kearney et al., 2009	Depression	Treatment	157	NR	100	CASQ-R ATD DLC				CESD	

Table 1. continued

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Targeted Outcome Measures	Non-Targeted Outcome Measures
Ollendick et al., 2009	Anxiety	Treatment	196	NR	45.3			BAT-CR		BAT (suds) CBCL-Anx/Dep CBCL-Int CSR FSSC-R MASC	CDI
Ost et al., 2001	Anxiety	Treatment	40	NR	33				Blood Pressure Heart Rate	CASI FSSC-R RCMAS STAIC-T STAIC-S	CDI
Pattison & Lynd-Stevenson, 2001	Depression	Prevention	66	10.44	52	CTI-C	MESSY			CDI	STAIC-T
Possel et al., 2005	Depression	Prevention	324	14	51.2	ATQ	FESU			CES-D	
Puskar et al., 2003	Depression	Treatment	89	16	73			CRI-Y		RADS	
Reynolds & Coats, 1986	Depression	Treatment	30	15.65	61.2	RSES ASCS				BDI BID RADS	STAIC-T
Rivet-Duval et al., 2011	Depression	Prevention	160	13.95	50	BHS RSES		YCI		RADS-2	
Roberts et al., 2003	Depression	Prevention	189	11.89	49.7	CASQ-R	MESSY			CDI	RCMAS
Roberts et al., 2010	Anxiety/ Depression	Prevention	496	11.99	54.4	CASQ-R	MESSY			CBCL-Int RCMAS CDI	
Rohde et al., 2004	Depression	Treatment	93	15.1	44.8		SAS-SRY			BDI-II HDRS	
Rohde et al., 2014	Depression	Prevention	378	15.2	68		SAS-SRY			BDI	
Rooney et al., 2006	Depression	Prevention	120	9.1	43	CASQ-R				CDI	RCMAS
Rooney et al., 2013	Depression	Prevention	910	8.75	48.6	CASQ-R SDQ-P	SDQ-Social-P			CDI SDQ-Total	SCAS
Rossello & Bamal, 1999	Depression	Treatment	48	14.7	54	PHCSCS	SASCA CBCL-Social			CDI	
Salusberry et al., 2013	Depression	Prevention	83	17.3	57	LS				CESD-10 PHQ-A	

Table 1. continued

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Targeted Outcome Measures	Non-Targeted Outcome Measures
Sheffield et al., 2006	Depression	Prevention	521	14.34	54	CATS BHS	CASAFS	NPS		CDI CES-D	SCAS
Spence et al., 2000	Anxiety	Treatment	50	10.62	38		SSQ-P SCQ-P BAT-C SWQ-PU			ADIS-P RCMAS SCAS	
Spence et al., 2003	Depression	Prevention	751	NR	50.6	CASQ-R	CASAFS	SPSI-R		BDI YSR-Int	
Sportel et al., 2013	Anxiety	Treatment	240	14.1	73.7	ATRA				RCADS TAI	
Stice et al., 2008	Depression	Prevention	341	15.6	56		SAS-SR			BDI	
Tomba et al., 2010	Anxiety	Prevention	162	11.41	58	PWB RCMAS-Concentration RCMAS-Worry	SQ-Friendliness, Contentment, Hostility		SQ-Physio SQ-Relax RCMAS-Physio SQ-well-being	SQ-anx RCMAS-total	SQ-Dep
Vostanis et al., 1996	Depression	Treatment	56	12.7	56	SEI	SAICA			MFQ (P)	RCMAS-CP
Waters et al., 2014	Anxiety	Treatment	37	10.6	57.1	Attention Bias (Positive Affect)				CGAS CSR SCAS-C/P SMFQ-C/P STAIC-S	
Yu & Seligman, 2002	Depression	Prevention	220	11.8	45	CASQ				CDI	

Note: NR indicates that the data was not reported; ACS = adolescent coping scale; ADIS = Anxiety Disorder Interview Schedule; AIQ-C = adolescent interpersonal competence questionnaire; ASCS = academic self-concept; ASSQ = anxious self-statements questionnaire; ATQ = automatic thoughts questionnaire; ATRA = automatic threat related associations; BAI = beck anxiety inventory; BAT-CR = revised behavior assertiveness test for children; BDI = Beck depression inventory; BHS = beck hopelessness scale; BID = Bellevue index of depression; CAPS = child and adolescent perfectionism scale; CAS =coping actions scale; CASAFS = child and adolescent social and adaptive functioning scale; CASI = child anxiety sensitivity index; CASQ-R = children's attributional style questionnaire revised; CATS = children's automatic thoughts scale; CBCL = children's behavior checklist; CCSY = children's coping strategies checklist; CDI = children's depression inventory; CESD = center for epidemiologic studies depression scale; CF-SEQ = culture free self-esteem questionnaire; CGAS = clinician's global assessment of severity; CLQ = Asher and Wheeler loneliness and social dissatisfaction scale; CNCEQ = children's negative cognitive error questionnaire; COPE = coping orientation to problems experienced inventory; CQ-C/P = coping questionnaire, child, parent versions; CRI-Y = coping response inventory for youth; CSCY= coping scale for children and youth; CSE = Coopersmith self-esteem inventory; CSR = clinician's severity rating; CTI-C = cognitive triad inventory for children; DAS = dysfunctional attitudes scale; EPQ-E = Eysenck Personality Questionnaire-extroversion, neuroticism subscales; FESU = questionnaire of social support; FSSC-R = fear survey schedule for children revised; FT = fear thermometer; HPLS = Kazdin hopelessness scale; HRSD = Hamilton rating scale for depression; HS = hopelessness scale; LSAS = Liebowitz social anxiety scale; LS = loneliness scale; LSSP = list of social situation problems; MASC = multidimensional anxiety scale for children; MESSY = Matson evaluation of social skills with adolescents-social skills, inappropriate assertiveness subscales; MSPS = multidimensional scale of perceived support; MUSIC = Maastricht University stress instrument for children; NASSQ = negative affect self-statement questionnaire; NPS = negative problem solving skills; NRS = numerical rating scale for somatic symptoms; PHCSCS = Piers-Harris children's self-concept scale; PHQ-A = patient health questionnaire adolescent-cognitive subscale; PWB = psychological well-being scales; PSR = psychiatric status rating; PSS = Perceived Social Support; RCADS = revised children's anxiety and depression scale; RCMAS = revised children's anxiety manifest

Table 1. continued

Study	Target Disorder	Level of Intervention	Sample Size	Mean Age	% Female	Information-Processing Measures	Social Measures	Coping Measures	Physio Measures	Targeted Outcome Measures	Non-Targeted Outcome Measures

Note continued: RCMAS = revised children's anxiety manifest scale; RPE (observer) = role play effectiveness, observer version; RSES = Rosenberg self-esteem scale; SAICA = social adjustment inventory for children and adolescents; SAS-A: social anxiety scale for adolescents-avoidance, fear of negative evaluation subscales; SAS-SR = social adjustment scale, self-report version; SCARED = screen for child anxiety related disorders; SCAS = Spence children's anxiety scale; SCQ-P = social competence questionnaire, parent version; SCS =social competence scale; SDIC = short depression inventory for children; SDQ = strengths and difficulties questionnaire, child and parent versions; SEQ-DA: self-efficacy questionnaire for depressed adolescents; SEQSS = self-efficacy questionnaire for school situations; SMFQ = short mood and feelings questionnaire; SPAI-C = social phobia and anxiety inventory; SPDSCF = social phobic disorders severity and change form; SPPC = self-perception profile for children; SPSI = social problem solving inventory; SPSI-R = social problem solving inventory-revised-social support, avoidance style, negative problem solving orientation subscales; SQ: symptom questionnaire-depression, friendliness, physiological, relaxation subscales; SSQ-P = social skills questionnaire, parent version; SSRS-C/P = social skills rating system, child, parent versions; STABS = social thoughts and beliefs scale; STAIC-T= state-anxiety inventory for children; SWQ-PU = social worries questionnaire, pupil version; TBO = therapist behavioral observations; TRF = teacher rating form; WIAL = what I am like; YCI = youth coping index; YSR = youth severity rating

APPENDIX B

ESTIMATES OF PRE TO POST MEAN EFFECT SIZES

Estimates of Pre to Post Mean Effect Sizes

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Anxiety Prevention										
Program Outcomes										
Targeted	8	2574	0.22	0.25	0.05	17.78	[0.02	0.43]	10	39.37***
Non-Targeted	5	2810	0.03	0.10	0.01	66.45	[-0.07	0.14]	-	6.02
Putative Mediators										
Information-Processing Biases-Overall	7	1837	0.38	0.54	0.54	4.93	[0.28	0.47]	20	121.75***
Information-Processing Biases-Internal	4	1166	0.57	0.57	0.57	3.42	[-0.17	1.31]	-	87.71***
Information-Processing Biases-External	5	1471	0.18	0.16	0.13	44.20	[0.01	0.36]	4	9.05
Coping Strategies-Overall	3	2812	0.13	0.06	0.04	75.47	[0.04	0.22]	1	2.65
Coping Strategies-Adaptive	3	2812	0.07	0.04	0.01	100.00	[-0.02	0.13]	-	0.91
Coping Strategies-Maladaptive	3	2812	0.22	0.13	0.12	16.60	[0.14	0.29]	4	12.05**
Social Competence	3	1228	0.07	0.06	0.00	100.00	[-0.04	0.18]	-	0.98
Physiological Hyperarousal	3	2237	0.06	0.00	0.00	77.52	[-0.02	0.16]	-	0.003
Anxiety Treatment										
Program Outcomes										
Targeted	20	1696	0.88	0.67	0.47	12.26	[0.55	1.20]	156	154.97***
Non-Targeted	13	901	0.39	0.38	0.13	40.32	[0.16	0.62]	38	29.76**
Putative Mediators										
Information-Processing Biases-Overall	11	848	0.24	0.25	0.24	100.00	[0.09	0.40]	15	9.65
Information-Processing Biases-Internal	7	240	0.29	0.21	0.16	100.00	[0.03	0.55]	13	2.61
Information-Processing Biases-External	7	703	0.45	0.32	0.28	47.06	[0.15	0.75]	25	12.75
Coping Strategies-Overall	9	690	0.64	0.40	0.37	32.47	[0.34	0.94]	49	24.64**
Coping Strategies-Adaptive	5	373	0.92	0.42	0.35	28.84	[0.45	1.37]	41	13.87**
Coping Strategies-Maladaptive	4	317	0.26	0.11	0.00	100.00	[0.04	0.49]	6	0.98
Social Competence	7	411	0.88	0.62	0.31	19.44	[0.39	1.38]	55	30.86***
Physiological Hyperarousal	3	127	0.43	0.29	0.04	77.52	[0.02	0.48]	10	2.58
Depression Prevention										
Program Outcomes										
Targeted	23	8076	0.23	0.26	0.06	16.80	[0.11	0.35]	30	130.93***
Non-Targeted	9	2650	0.06	0.11	0.01	96.62	[-0.08	0.08]	-	8.28
Putative Mediators										
Information-Processing Biases-Overall	17	3903	0.18	0.24	0.23	28.80	[0.05	0.31]	14	55.56***
Information-Processing Biases-Internal	9	1512	0.23	0.26	0.24	34.19	[0.04	0.41]	12	23.40**
Information-Processing Biases-External	14	3834	0.10	0.21	0.21	31.55	[0.03	0.16]	1	41.20***
Coping Strategies-Overall	9	4583	0.16	0.11	0.10	58.78	[0.07	0.25]	5	13.61
Coping Strategies-Adaptive	7	4148	0.12	0.08	0.07	86.46	[0.05	0.19]	1	6.94
Coping Strategies-Maladaptive	5	2341	0.20	0.18	0.14	21.18	[-0.02	0.41]	-	18.89***
Social Competence	8	2706	0.13	0.20	0.03	27.83	[-0.03	0.28]	-	25.15***
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Estimates of Pre to Post Mean Effect Sizes (continued)

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Depression Treatment										
Program Outcomes										
Targeted	7	480	0.52	0.44	0.15	27.68	[0.12	0.91]	29	21.68**
Non-Targeted	4	294	0.27	0.29	0.01	29.21	[-0.26	0.80]	-	10.27*
Putative Mediators										
Information-Processing Biases-Overall	6	387	0.76	0.19	0.16	100.00	[0.55	0.97]	40	3.01
Information-Processing Biases-Internal	5	365	0.77	0.19	0.15	100.00	[0.55	0.99]	34	2.85
Information-Processing Biases-External	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Overall	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Adaptive	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Maladaptive	-	-	-	-	-	-	-	-	-	-
Social Competence	3	221	0.41	0.08	0.01	100.00	[0.14	0.69]	9	0.30
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Note: *K* = number of studies; *N* = sample size; *g* = weighted mean effect size; *SDg* = weighted standard deviation of *g*; *ResSD* = standard deviation of the residual; %VarSE = percentage of variance attributed to sampling error; CI = 95% confidence interval; Fail Safe N = number of samples with an effect size of zero that should have been left out in order to reduce estimated effect size to non-significance; Q_w = Variability among effect sizes; - = not enough data to analyze; *Targeted Outcomes* = Outcome targeted by intervention***;*** *Non-Targeted Outcomes* = Secondary disorder outcomes; *p<.05, ** p<.01, p<.001

APPENDIX C

ESTIMATES OF POST TO FOLLOW-UP MEAN EFFECT SIZE

Estimates of Post to Follow-Up Mean Effect Sizes

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Anxiety Prevention										
Program Outcomes										
Targeted	9	4011	0.02	0.17	0.16	28.13	[-0.11	0.16]	-	28.44***
Non-Targeted	5	2810	-0.04	0.05	0.03	197.04	[-0.11	0.04]	-	2.03
Putative Mediators										
Information-Processing Biases-Overall	7	1837	0.16	0.10	0.09	100.00	[0.07	0.25]	4	4.32
Information-Processing Biases-Internal	4	1166	0.25	0.09	0.07	100.00	[0.13	0.36]	6	2.14
Information-Processing Biases-External	5	1471	0.13	0.08	0.06	100.00	[0.03	0.24]	2	2.49
Coping Strategies-Overall	3	2812	0.12	0.05	0.03	100.00	[0.05	0.20]	1	1.92
Coping Strategies-Adaptive	2	2174	0.07	0.02	0.00	100.00	[-0.01	0.15]	-	0.16
Coping Strategies-Maladaptive	3	2812	0.14	0.04	0.01	100.00	[0.07	0.22]	1	1.20
Social Competence	3	1228	0.14	0.08	0.06	100.00	[0.02	0.25]	1	1.94
Physiological Hyperarousal	3	2237	0.09	0.04	0.00	100.00	[0.01	0.18]	1	1.05
Anxiety Treatment										
Program Outcomes										
Targeted	19	1502	0.18	0.27	0.26	80.79	[0.05	0.31]	15	22.28
Non-Targeted	12	707	0.06	0.14	0.12	100.00	[-0.09	0.21]	-	3.22
Putative Mediators										
Information-Processing Biases-Overall	10	811	0.18	0.22	0.20	100.00	[0.02	0.34]	8	7.14
Information-Processing Biases-Internal	6	203	0.43	0.27	0.23	100.00	[0.15	0.71]	20	3.57
Information-Processing Biases-External	7	703	0.08	0.04	0.00	100.00	[-0.09	0.26]	-	0.19
Coping Strategies-Overall	8	690	0.23	0.16	0.13	100.00	[0.05	0.42]	10	2.90
Coping Strategies-Adaptive	5	339	0.25	0.15	0.10	100.00	[0.04	0.47]	8	1.89
Coping Strategies-Maladaptive	4	351	0.09	0.12	0.04	100.00	[-0.13	0.31]	-	1.19
Social Competence	6	374	0.35	0.32	0.29	58.34	[0.06	0.63]	15	8.57
Physiological Hyperarousal	3	127	0.29	0.23	0.14	100.00	[-0.06	0.64]	-	1.61
Depression Prevention										
Program Outcomes										
Targeted	23	8076	-0.04	0.18	0.17	34.04	[-0.12	0.05]	-	64.63***
Non-Targeted	9	2650	0.04	0.10	0.09	100.00	[-0.04	0.12]	-	6.16
Putative Mediators										
Information-Processing Biases-Overall	19	4351	0.29	0.52	0.50	6.37	[0.03	0.54]	36	282.71***
Information-Processing Biases-Internal	8	1355	0.10	0.09	0.07	100.00	[-0.01	0.21]	-	2.30
Information-Processing Biases-External	14	3834	0.32	0.56	0.54	4.61	[0.01	0.63]	31	282.27***
Coping Strategies-Overall	8	4308	0.11	0.11	0.10	60.14	[0.02	0.20]	1	11.64
Coping Strategies-Adaptive	7	4148	0.13	0.10	0.09	60.12	[0.04	0.22]	2	9.98
Coping Strategies-Maladaptive	5	2274	0.10	0.07	0.06	100.00	[0.02	0.18]	1	2.65
Social Competence	8	2706	0.08	0.17	0.16	37.67	[-0.05	0.21]	-	18.58**
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Estimates of Post to Follow-Up Mean Effect Sizes (continued)

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Depression Treatment										
Program Outcomes										
Targeted	7	480	0.16	0.50	0.44	21.13	[-0.29	0.61]	-	28.40***
Non-Targeted	4	294	0.15	0.17	0.12	100.00	[-0.09	0.38]	2	2.14
Putative Mediators										
Information-Processing Biases-Overall	5	367	0.23	0.27	0.25	61.92	[0.02	0.44]	7	6.46
Information-Processing Biases-Internal	5	365	0.28	0.24	0.20	78.59	[0.02	0.54]	9	5.09
Information-Processing Biases-External	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Overall	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Adaptive	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Maladaptive	-	-	-	-	-	-	-	-	-	-
Social Competence	3	221	-0.04	0.17	0.14	100.00	[-0.31	0.23]	-	1.50
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Note: *K* = number of studies; *N* = sample size; *g* = weighted mean effect size; *SDg* = weighted standard deviation of *g*; *ResSD* = standard deviation of the residual; %VarSE = percentage of variance attributed to sampling error; CI = 95% confidence interval; Fail Safe N = number of samples with an effect size of zero that should have been left out in order to reduce estimated effect size to non-significance; Q_w = Variability among effect sizes; - = not enough data to analyze; *Targeted Outcomes* = Outcome targeted by intervention***;*** *Non-Targeted Outcomes* = Secondary disorder outcomes; *p<.05, ** p<.01, p<.001

APPENDIX D

ESTIMATES OF PRE TO FOLLOW-UP MEAN EFFECT SIZE

Estimates of Pre to Follow-Up Mean Effect Sizes

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Anxiety Prevention										
Program Outcomes										
Targeted	9	4011	0.17	0.21	0.19	18.04	[0.01	0.34]	6	44.35***
Non-Targeted	5	2810	-0.04	0.07	0.06	100.00	[-0.12	0.03]	-	3.36
Putative Mediators										
Information-Processing Biases-Overall	7	1837	0.28	0.27	0.24	19.24	[0.04	0.52]	13	31.18***
Information-Processing Biases-Internal	4	1166	0.41	0.21	0.15	23.66	[0.13	0.68]	12	12.68**
Information-Processing Biases-External	5	1471	0.26	0.24	0.23	19.82	[0.15	0.36]	8	20.18***
Coping Strategies-Overall	3	2812	0.09	0.08	0.07	43.76	[0.02	0.17]	1	4.57
Coping Strategies-Adaptive	3	2812	0.12	0.01	0.00	100.00	[0.05	0.20]	1	0.11
Coping Strategies-Maladaptive	3	2812	0.12	0.17	0.11	9.50	[-0.13	0.38]	-	21.06***
Social Competence	3	1228	0.14	0.09	0.06	82.30	[0.01	0.27]	1	2.43
Physiological Hyperarousal	3	2237	0.19	0.05	0.03	100.00	[0.11	0.27]	3	1.41
Anxiety Treatment										
Program Outcomes										
Targeted	19	1502	1.15	0.73	0.71	12.06	[0.79	1.51]	200	149.31***
Non-Targeted	12	707	0.65	0.42	0.40	38.73	[0.38	0.91]	66	28.40**
Putative Mediators										
Information-Processing Biases-Overall	10	811	0.65	0.34	0.32	54.74	[0.41	0.90]	55	16.44
Information-Processing Biases-Internal	6	203	0.81	0.14	0.00	100.00	[0.52	1.10]	43	0.82
Information-Processing Biases-External	7	703	0.59	0.32	0.28	47.51	[0.29	0.89]	34	12.63*
Coping Strategies-Overall	9	690	1.14	0.58	0.54	16.66	[0.71	1.57]	94	48.03***
Coping Strategies-Adaptive	5	339	1.42	0.72	0.60	10.85	[0.65	1.76]	66	36.88***
Coping Strategies-Maladaptive	4	351	0.82	0.32	0.24	41.55	[0.39	1.24]	29	7.22
Social Competence	6	374	0.95	0.16	0.11	100.00	[0.73	1.17]	51	2.10
Physiological Hyperarousal	3	127	0.65	0.20	0.08	100.00	[0.29	1.01]	17	1.21
Depression Prevention										
Program Outcomes										
Targeted	23	8076	0.16	0.21	0.20	25.93	[0.06	0.26]	14	84.84***
Non-Targeted	10	4087	0.01	0.10	0.09	100.00	[-0.05	0.07]	-	8.99
Putative Mediators										
Information-Processing Biases-Overall	20	5988	0.08	0.16	0.15	52.97	[0.01	0.16]	1	35.87*
Information-Processing Biases-Internal	9	1512	0.21	0.26	0.24	33.94	[0.02	0.40]	10	23.57**
Information-Processing Biases-External	15	5334	0.04	0.12	0.11	71.28	[-0.03	0.11]	-	19.64
Coping Strategies-Overall	9	4583	0.10	0.13	0.12	43.15	[-.01	-0.20]	-	18.54*
Coping Strategies-Adaptive	6	2648	0.15	0.06	0.05	100.00	[0.08	0.23]	3	2.25
Coping Strategies-Maladaptive	7	4049	0.05	0.13	0.11	33.73	[-0.08	0.17]	-	17.79**
Social Competence	10	4547	0.14	0.16	0.15	32.75	[0.03	0.26]	4	27.48**
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Estimates of Pre to Follow-Up Mean Effect Sizes (continued)

Category	*K*	*N*	*g*	*SDg*	*ResSd*	*%VarSE*	*95% CI*		*Fail Safe N*	Q_w
Depression Treatment										
Program Outcomes										
Targeted	7	480	0.24	0.51	0.50	20.20	[0.06	0.43]	10	29.70***
Non-Targeted	4	294	0.21	0.34	0.24	36.99	[-0.26	0.68]	4	8.11*
Putative Mediators										
Information-Processing Biases-Overall	5	316	0.67	0.25	0.21	89.29	[0.41	0.94]	29	4.48
Information-Processing Biases-Internal	4	294	0.72	0.24	0.18	79.58	[0.42	1.02]	25	3.77
Information-Processing Biases-External	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Overall	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Adaptive	-	-	-	-	-	-	-	-	-	-
Coping Strategies-Maladaptive	-	-	-	-	-	-	-	-	-	-
Social Competence	3	221	0.34	0.05	0.00	100.00	[0.07	0.61]	7	0.11
Physiological Hyperarousal	-	-	-	-	-	-	-	-	-	-

Note: *K* = number of studies; *N* = sample size; *g* = weighted mean effect size; *SDg* = weighted standard deviation of *g*; *ResSD* = standard deviation of the residual; %VarSE = percentage of variance attributed to sampling error; CI = 95% confidence interval; Fail Safe N = number of samples with an effect size of zero that should have been left out in order to reduce estimated effect size to non-significance; Q_w = Variability among effect sizes; - = not enough data to analyze; *Targeted Outcomes* = Outcome targeted by intervention***;*** *Non-Targeted Outcomes* = Secondary disorder outcomes; **p*<.05, ** *p*<.01, *p*<.001